MORTALITY OF QUEBEC CHRYSOTILE WORKERS

IN RELATION TO

RADIOLOGICAL FINDINGS WHILE STILL EMPLOYED

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A B S T R A C T

Two cohorts of Quebec chrysotile miners and millers were selected in order to study how radiological changes in asbestos workers predict mortality. *Cohort "A"* comprised 988 men, with dust exposure, smoking history, respiratory symptoms and lung function recorded in 1967-68, and six independent readings of an earlier chest radiograph; 130 men had died before 1976. *Cohort "B"* comprised all 4,559 men not in Cohort "A" who were born 1891 through 1920, with at least a month's employment and an x-ray; dust exposure, smoking history and a single x-ray reading were obtained. By the end of 1975, there had been 1,453 deaths in Cohort "B".

A high Relative Risk (RR) of *total mortality* was related to each radiographic feature, cigarette smoking, excessive dust exposure, and, in Cohort "A", dyspnoea and poorer function.

Death from *pneumoconiosis* was associated with small irregular parenchymal opacities, of profusion usually greater than 1/0, and with heavy dust exposure, but not with smoking.

Almost all who died from *chest cancer* had smoked cigarettes (very high RR), had been heavily exposed to asbestos (high RR), or both. About half their x-rays showed pneumoconiotic change. There was insufficient evidence to determine whether chest cancer was a complication of asbestosis or an independent reaction to asbestos exposure. Five of the six *mesothelioma* cases had positive but varied radiographic appearances.

The contribution of asbestos dust to some deaths attributed to *heart disease* was substantiated by high RRs for severe exposure, and for small parenchymal opacities; smokers, and the breathless, also had high risks.

Other neoplasms showed no consistent dust or x-ray patterns. RRs of deaths from some other causes were high for certain radiographic features. Not all these results can yet be explained.

The main findings validated the UICC/Cincinnati classification convincingly. Studies to permit generalization are recommended.

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I N T R O D U C T I O N

The uses of asbestos, the "magic mineral" are stated to be innumerable, and certainly more than three thousand have been identified by Rosato (1959). The President of the Quebec Asbestos Mining Association called asbestos "a unique product, a natural material of great value providing characteristics which are found in no other product and which are of inestimable service" (Messel, 1978). Although these views are not disinterested, they are largely endorsed by unbiased experts. Thus, Gilson (1965) commented on the surprisingly diverse and expanding uses of asbestos, and that it had much to its credit, for example such products as incombustible buildings, fire-resistant bulkheads, fire-proof clothing, brake shoes and clutch plates, all affecting safety and health. Further, it allowed great savings in energy through its use for insulation.

World production of asbestos has grown steadily from a few tons a year just a century ago (50 tons were produced in Quebec in 1878) to nearly six million short tons in 1977 (Quebec Asbestos Mining Association, 1978), at a value of almost 2,000 million dollars. Although the bulk comes from Russia (44.8 per cent in 1977, all chrysotile), Canada (27.4 per cent, chrysotile) and South Africa (13.0 per cent, chrysotile, crocidolite and amosite), all countries make use of it.

Undoubtedly, asbestos is a most important mineral; but equally certainly it can have adverse effects on health. It has been known since the turn of the century to lead to pulmonary fibrosis, which in those days of extremely severe exposure was often rapidly fatal. Before this and other health hazards were officially recognized (in the 1930s in Britain), environmental hygiene for both production and manufacture was poor in the extreme. After implementation of the Asbestos Regulations 1931, concentrations of airborne dust were much reduced, but remained very high, by modern standards, both here and in the mines and mills which produce asbestos. However, even in such conditions, the deleterious effects on the health of those exposed to asbestos dust are not usually manifest for many years. Thus, studies over the last quarter of a century, which have shown excess mortality and morbidity in asbestos workers, and which have aroused great concern about hygiene standards, have been examining responses to past doses which, in most parts of the world, would be unthinkably high today.

Nevertheless, it remains essential to make the most thorough possible epidemiological investigations of health in relation to asbestos exposure. These are, indeed, aided by knowledge of severe historical exposures, although interpolation of fitted dose-response relationships within the region of present day exposures is of great difficulty. Further, although concerns about the health of asbestos workers have led to attempts to develop substitutes, there is little likelihood of complete replacement of asbestos for many years. Nor are substitutes necessarily the answer; it may be that the properties needed to mimic those of asbestos are themselves fibrogenic or carci-

nogenic.

There are known to be interrelationships between all the responses to exposure, and many of the responses exhibit dose-response relationships, although these differ markedly from one fibre type to another. However, little is known as to the sequence in which the various signs and symptoms manifest themselves; nor has there been any reliable evidence on how any particular sign, symptom or combination can predict mortality. Radiological changes can, since 1967, be categorized into a classification of the pneumoconioses which was devised with particular reference to asbestos, the appearances of which could not be adequately recorded in earlier classifications. The irregular small opacities in the lung parenchyma and the pleural changes that are often observed in the x-ray films of asbestos workers, but comparatively seldom in others, can therefore be taken as reasonably specific to asbestos exposure, and thus might be expected to be better than other signs in indicating state of asbestos-related disease and so of predicting mortality in populations of asbestos workers.

The broad objective of this research was to determine the extent to which radiographic changes could predict mortality in persons who had been exposed to asbestos. Should prediction turn out to be good, it would serve as the best possible validation of the radiological classification. Further, light might be cast on the value of radiology in the surveillance of a working population. When the research reported here was already well advanced, a meeting of x-ray readers from six countries, at an important symposium on asbestos in Johannesburg (Glen, 1978), agreed that research on morbidity and mortality

associated with x-ray abnormality was "crucial", because "the importance of radiology depends on its ability to predict illness and death, rather than on its sensitivity to asbestos exposure" (Irwig, 1978).

This thesis is organized in a further thirteen chapters, of which the first considers the history, forms, uses and consumption of asbestos, and its health effects. Chapter 3 discusses radiological classification, tracing development from 1930 to the latest international classification, i.e. the ILO U/C (1971) international classification of radiographs of the pneumoconioses. Full details are given of the U/C classification agreed to in 1967; this, the basis of the ILO U/C (1971) classification, was used for the film reading essential to this research. The next two chapters are concerned with the health of Quebec chrysotile miners and millers, by far the largest group exposed to a single fibre to have been investigated: Chapter 4 describes the populations and measures of exposure defined and studied by Professor J. Corbett McDonald and his colleagues (including the author) at McGill University; the findings from these studies are reviewed in Chapter 5. The following chapter examines findings from many diverse investigations in the context of those from McGill; particular attention is paid to (a) the relationships between dose (exposure to asbestos dust) and response (lung cancer mortality) and (b) the interaction between asbestos and smoking in carcinogenesis.

Chapter 7 details the thesis objectives, and the materials used in the research, and Chapter 8 discusses methodology, in both general and specific terms. The study populations are described fully in

Chapter 9, and the findings are presented in the following chapters.

Discussion, synthesis and recommendations are in Chapter 14, followed by statements on originality and acknowledgements. The leading data are given in a 350-page Annex which is bound as Volume 2 of the thesis.

Chapter 2

A S B E S T O S : F O R M S , U S E S A N D H E A L T H E F F E C T S

2.1 Introduction

According to Friedrichsen (1963), the word *asbestos* is from the Latin *absestus* itself from the Greek *a-* (not) with *sbestos* (quenchable). It was certainly in English use by the early 18th Century, and has commonly been employed as a collective term for a wide variety of minerals composed of silicates of magnesium and iron.

The fire-resistant properties of asbestos were known to the ancient Romans, as is clear from the account by Herodotus, in 450 B.C. (quoted by Hunter, 1975, p. 972), of its use in the enshrouding of corpses before cremation to facilitate collection of ashes for burial. This may be the reason for another Latin word for the mineral, *amiant(h)us (cf.* the French *amiante),* literally without miasma, or undefiled, incorruptible. Several classical writers appeared to have been referring to one of the asbestiform minerals, particularly in its use for lamp wicks; the flames of ancient temple lamps were kept replenished with oil so that they would never be extinguished, and this is thought to be the origin of the term unquenchable. These early uses must have involved spinning and weaving, and there is some evidence that cloth made with a mixture of asbestos and vegetable fibre was

used to deaden the sounds of tree-felling in Roman times. Thereafter, there were occasional references to asbestos, for example by Marco Polo (around 1250 A.D.), by Dr. Robert Plot (in 1686) and by Benjamin Franklin (in 1725), this last probably being to asbestos from Quebec (*Asbestos*, 1970; Zussman, 1972). However, much the earliest reported use was in Finland, described by Dr. L. Noro and by Dr. R. Kiviluoto (quoted by Gilson, 1965); Seaton (1975) states that fibres of the indigenous Finnish type of asbestos have been found in the clay of pots made in Finland four thousand years ago, having been added presumably to strengthen the final article.

2.2 Forms and supplies of asbestos

Asbestos has been defined variously. Statements by Hendry and Gaze, at the 1964 Conference on Biological Effects of Asbestos held by the New York Academy of Sciences, that the word asbestos was "a broad term embracing a number of fibrous mineral silicates that differ in chemical composition" and that it was "used to describe any mineral that breaks down into fibres when it is crushed or processed" were echoed by Zussman (1972) who wrote that asbestos was the name for "a mineral which is made up of fine fibres and which is resistant to high temperatures". However, in a 1970 publication, *Asbestos* magazine stated that the term "is not the name of a distinct mineral species but is a commercial term applied to fibrous varieties of several minerals differing widely in chemical composition, the fibres being diverse in length, strength, flexibility and consequent usefulness". More recently, in a report (Ward, 1977) of the International Agency for Research on Cancer (IARC) Working Group on the Evaluation of the Carcinogenic Risks of Chemicals to Man: Asbestos, the use of asbestos

as a generic term is confirmed, but it is also stated that current usage of the term is restricted to six specific naturally-occurring mineral silicate fibres. In fact, commercial use is almost entirely of only four of these fibres: chrysotile, crocidolite, amosite and anthophyllite.

Asbestos is so invaluable because it is made up of fine fibres which are flexible, of great tensile strength, and resistant to high temperatures; they provide good accustic, electrical and thermal insulation and, to varying degrees, resist corrosion (Zussman, 1972). The three or four commercially important types of asbestos differ in physical properties and in chemical composition and in their health effects.

Chrysotile, or white asbestos, makes up the bulk of world production and use, about nine-tenths in 1977. The fibres are fine, highly flexible and have good heat resistance, while the long fibres can be spun easily into textiles (*Asbestos*, 1970); Hendry (1965) adds to these properties tensile strength, absorption and filtration, stating that in combination they place chrysotile in a class by itself as a mineral of "unparalleled properties". This form of asbestos is a mineral of the so-called serpentine group, and contains mainly magnesium oxide in addition to silica. Most of it is produced in Russia and Canada, but Ward (1977) mentions half-a-dozen other countries where comparatively small deposits are exploited.

Crocidolite, or blue asbestos, and *amosite* are both of the amphibole group of minerals, which are more brittle and of harsher texture

than serpentine fibres; their main chemical constituents in addition to silica are ferrous and ferric oxides. These forms of asbestos now come only from South Africa, although small amounts of crocidolite used to be mined in Australia and Bolivia, while India is about to produce a fibre very similar to amosite (which is a name based on the initial letters of the Asbestos Mine of South Africa where it was first mined) to be called "mysorite" (Ward, 1977). *Anthophyllite* is another amphibole, which used to be produced and used almost exclusively in Finland; according to Ward (1977), production in Finland ceased in 1974. In this form of asbestos there is more silica than in chrysotile, less magnesium oxide and more ferrous oxide.

The modern asbestos industry dates from the discovery in the 1870s of large deposits of fibre in Quebec and Russia (Bogovski *et al.*, 1973, p. xiii); the Canadian and Russian form of asbestos is chrysotile. Slightly later came the commercial exploitation of the three amphibole fibres, crocidolite, amosite and anthophyllite. The Quebec Asbestos Mining Association (QAMA, 1978a) has kindly provided annual figures of asbestos shipments from 1878 (50 short tons) to 1977 (1,289 thousand short tons). Production in Quebec for each decade of the hundred years' existence of this industry, in millions of short tons, was:-

1878-87	0.015	1928-37	2.345
1888-97	0.101	1938-47	4.492
1898-1907	0.490	1948-57	8.771
1908-17	1.267	1958-67	11.449
1918-27	2.075	1968-77	13.399

Quebec has always produced the vast bulk of Canadian asbestos (82.1 per cent of the total production in Canada of 16.311 million short tons in the decade 1968-77). The other provinces that produce asbestos, all chrysotile, are the Yukon territories (109.2 thousand short tons in 1977), British Columbia (103.5 thousand tons), Newfoundland (71.2 thousand tons) and Ontario (16.4 thousand tons). Until recently, Quebec also produced the bulk of the world's total supplies; according to the IARC Working Party on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Asbestos (Ward, 1977), who had to assemble data from several sources, Canada's share of world production was 45 per cent in 1960. However, this share had fallen to 29 per cent in 1976 (Ward, 1977) and was 27.4 per cent in 1977; Quebec's share in 1977 was 22.3 per cent (QAMA, 1978b). The USSR is now the main producer of asbestos, having increased its share of world production from 29 per cent in 1960 to 44 per cent in 1976 (Ward, 1977); in 1977, Russia produced 2,596 thousand short tons, all chrysotile, 44.8 per cent of the total, 5,790 thousand short tons (QAMA, 1978b).

It is difficult to obtain a direct statement of how much of each fibre is produced, but it seems that crocidolite and amosite are nowadays mined only in South Africa, which in 1977 produced 750 thousand short tons of all forms of asbestos, while anthophyllite production in Finland ceased in 1974 (Ward, 1977; QAMA, 1978b). As about nine-tenths of the current South African labour force produce amphibole (Gibbs and DuToit, 1978), the proportion of chrysotile in the grand total must be close to ninety per cent. The countries that mined chrysotile in 1977 were, in addition to Russia and Canada, China (220 thousand short tons), Cyprus and Italy (a total of 330 thousand tons),

with the USA, South America, Australia and "other countries" accounting for 105, 80, 75 and 45 thousand short tons, respectively (QAMA, 1978b). Small amounts of fibre have been produced in the past in Corsica (chrysotile) and in Australia and Bolivia (crocidolite); some limited amounts of another asbestiform mineral, tremolite, have been mined in the USA and the Far East (Ward, 1977).

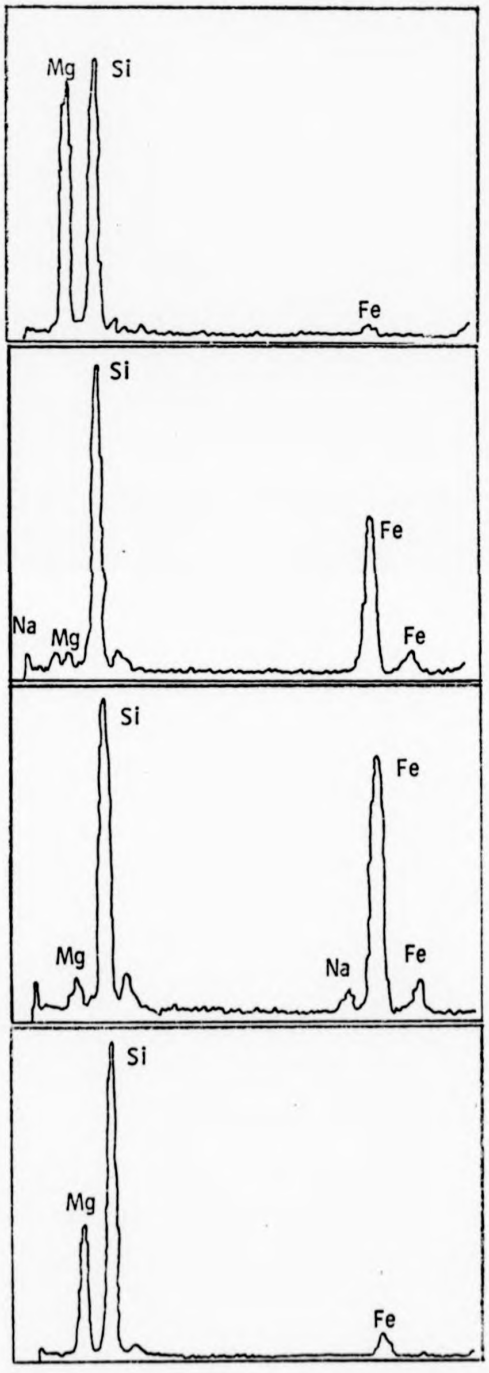
The four main types of asbestos fibre differ greatly in their chemical composition, as shown in the x-ray spectra (after Dr. Fred Pooley) in Figure 2.1, which are in close conformity with the analyses given by Gaze (1965). All types of fibre contain silica (Si), but there is less in chrysotile (40 per cent) than in the amphiboles (around 50 per cent in crocidolite and amosite; 57 per cent in anthophyllite). The main constituent of chrysotile is 42 per cent of magnesium oxide (MgO), but that forms only a small part of crocidolite (1 per cent) and amosite (6 per cent), although up to 29 per cent in anthophyllite. On the other hand, iron is scarce in chrysotile, ferrous oxide (FeO) and ferric oxide (Fe_2O_3) totalling less than 3 per cent, but common in the South African amphiboles (around 40 per cent) and intermediate in anthophyllite. Water of crystallization is 14 per cent of chrysotile but only 2 per cent of the amphiboles.

2.3 Uses of asbestos

At least three thousand uses of asbestos have been identified (Rosato, 1959); a short list of over 150 is given by *Asbestos* (1970). Raw asbestos is used for the "rovings" from which all spun and woven textiles are made; in felts, paper, millboard and wall board; for reinforcement of cement products; for thermal, electrical and acoustic

Figure 2.1

X-RAY SPECTRA



insulation; for friction materials and filtration media; for fire-proofing and other protective clothing. Asbestos yarn is used for cloth, electrical tape, brake linings, clutch facings, gaskets, rope, twine, sewing thread, wicks, etc. The cloth woven from asbestos has many uses, not least because of its strength - clothing, draperies, mailbags, ironing boards, for example. Asbestos millboard, being fireproof, has a large number of applications in industry and building; here, too, asbestos-cement sheets, flat and corrugated, and asbestos-cement wall boards are popular. Asbestos-cement pipe is used for flues and chimneys in the home and elsewhere, and for high and low pressure systems.

Since the introduction of asbestos into the Paris Mētro (1903) to prevent the repetition of a disastrous fire, and particularly in the interior of RMS Queen Mary (1932) to prevent the spread of any possible fire, its pre-eminence as a fire-resistant material has been recognized. It is used for firefighters' suits, helmets, ropes and sheets. With the introduction (1912) of a method of constructing seamless asbestos-cement pipes, such pipes have been very widely used for a multitude of purposes, and asbestos itself is used for jointing and lagging steam pipes, etc, and for many other forms of heat insulation. It can be exploited in the form of building materials such as tiles - for roof or floor - and even linoleum, for cements and cement sheets, and for fireproof panels, including theatre safety curtains. Because it is acid-resistant, asbestos is used for filter cloths, linings for certain chemical vessels, valve packings and gaskets. Re-inforced with wire threads, asbestos is the major ingredient of brake linings and clutch facings. On top of these largely industrial uses, asbestos

has many domestic applications, for example in ironing boards, oven cloths, cooking mats, and other kitchen materials. Finally, it has been a favourite material in the "Do-It-Yourself" market.

Although about ninety per cent of asbestos usage is of chrysotile, the other forms have often been specified for particular purposes. One set of examples is given in an important section of the doctoral thesis of Surgeon Commander P. G. Harries (1971). Until 1950, the Admiralty specified crocidolite for certain materials used in the insulation of machinery in steam-propelled warships. With higher steam temperatures after 1950, large pipe sections, almost wholly of amosite fibre, were tried but found less efficient than had been expected. More recently, pipe lagging has been with calcium silicate bound by amosite, the binder amounting to 12 per cent of the material. Further, until the late 1950s, insulation of steel decks and hull was mainly with crocidolite. One other specified use of crocidolite was for the filter pads in service gas masks in World War II, while chrysotile was used for civilian masks (Jones *et al.*, 1976; McDonald and McDonald, 1978; Dr. J. C. Gilson, personal communication).

Perhaps the most important present-day use of asbestos is in cement products, and particularly for asbestos-cement pipes. Here, fast drying is essential in manufacture, especially for the pipes of largest diameter, and this process is greatly accelerated by a mixture of about one-sixth of crocidolite; see below. Certain other processes have been devised for specific amphibole fibres and cannot easily be adapted to the exclusive use of chrysotile.

The IARC Working Group (Ward, 1977, quoting Clifton, 1974) gives the following distribution of asbestos use, by type, in the USA for 1974 *(sic)*:-

Chrysotile	720.8 million kg	94.5 per cent
Crocidolite	33.8 "	4.4 "
Amosite	8.4 "	1.1 "
Anthophyllite	1.0 "	0.1 "
	763	100

Most of the crocidolite (33 million kg) was used for asbestos-cement pipes, but accounted for only one sixth of the asbestos used for this product. The amosite was used mainly for asbestos-cement sheet (3.9 million kg), thermal insulation (1.6 million kg) and roofing products (1.5 million kg), but chrysotile was still the most commonly used fibre for these products, accounting for 95.3, 82.5 and 98.5 per cent, respectively, of the asbestos in each. QAMA (1978b) provide approximate fibre content in various products, as follows:-

Asbestos cloth and yarn	85-90	per cent by weight
Asbestos papers (felts)	80-90	" "
Asbestos water-proofing and anti-leak joints	50-70	" "
Woven friction material	70	" "
Moulded friction material	50-60	" "
Asbestos pipes	16	" "
Asbestos finished sheets	10-15	" "
Asbestos-vinyl tiles	8-14	" "

2.4 Geology and exploitation

Zussman (1972) concluded his article on the nature and history of asbestos with the statement that: "Even after many decades of research, the mode of growth of natural asbestos fibres remains largely a matter of conjecture". Certainly, the geology of the asbestos-bearing rocks and their overburden is too complex to fall within the scope of this thesis. However, a few facts are given below, taken from a 32-page guide to the geology of the mining region of South-Eastern Quebec, with more than a dozen maps, and many references (QAMA, 1972). The Quebec deposits of chrysotile lie in a belt a few miles wide and about 120 miles long. Chrysotile occurs mainly as cross fibre, but also as slip fibre and mass fibre. "Cross fibre occurs in veins with fibres occupying a position somewhere between normal and sharply oblique to the vein walls, whereas slip fibre is a stress mineral occurring as a coating on slip planes and orientated parallel to the direction of slip. Mass fibre is present in the ore of a few mines as aggregates of unoriented or semi-oriented leaves or bundles of chrysotile. At some locations the rocks have been completely or nearly converted into mass fibre." At Asbestos, Quebec, there is one mine (and a small factory), which has been in operation since 1881, producing mainly cross fibre but some mass fibre from rock that may contain as much as 50 per cent chrysotile. The present rating of this mine is 600 thousand tons of fibre a year. In the Thetford Mines area, there are presently four mines; deposits are made up principally of cross fibre, but an appreciable amount of slip fibre ore is being recovered. At one location, there were three deposits, one now worked out, one in production and the third not started. "Although difficult to explain, one interesting aspect of the fibre quality ... is the increasing

'softness' of the fibre with depth ... " Finally, much of the host rock is itself "serpentized".

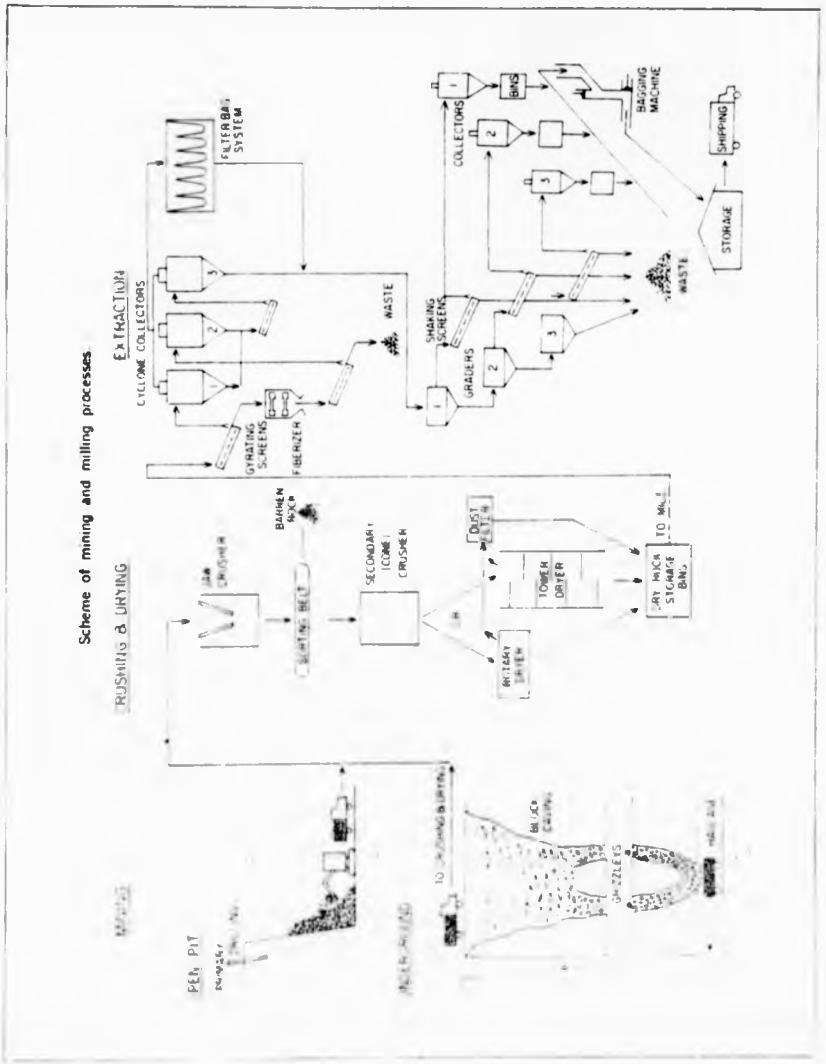
Ore, whether from an open pit or underground mine, often contains less than ten per cent asbestos, with ninety per cent or more waste rock. At the mine in Asbestos, Quebec, fifteen tons of ore are crushed to produce a ton of commercially disposable asbestos, but the total material moved is more than 60 tons (QAMA, 1972, p. 19). The ore is fed first to ore preparation plants, where it is crushed and separated. The larger fraction is further sorted by hand, but the smaller is first dried and then passed to mills in which operations are carried out in cycles of crushing, screening and then lifting by air (Lincoln, 1972). Figure 2.2, taken from Gibbs and Lachance (1972), gives a schematic illustration of the process, in principle.

2.5 Dust exposure and inhalation

Thus, milling is essentially a dusty operation, in which suppressive measures relying on wetting have not so far proved feasible. Mining, contrary to some expectations, is generally less dusty (Gibbs and Lachance, 1972). However, many jobs involving the handling of asbestos or asbestos products were, until quite recently, very dusty. Up to about 1950, baggers in the Quebec mills were exposed to concentrations of over 200 million (respirable) particles per cubic foot (of air), or 200 mpcf, and at that time the average concentration to which millers were exposed was approximately 75 mpcf (Gibbs and Lachance, 1972). In naval dockyards, many of the various processes involving asbestos were very dusty, and Harries (1971, p. 30) stated that "men are described as emerging from the compartments covered from head to

Figure 2.2

SCHEME OF MINING AND MILLING PROCESSES



foot in dust", and also that "although most of the fibre was blue, amosite fibre was used occasionally and was disliked because it was more dusty". In textile factories, carding has always been particularly dusty: Knox *et al.* (1968), reporting on a factory in Rochdale at which "steps [had been] taken to reduce exposure ... since the introduction of the Asbestos Industry Regulations in 1931", quoted yearly mean dust levels as high as 32 mpcf as late as 1952. Enterline *et al.* (1972b) reported historic dust levels (i.e. before 1947) in an American asbestos plant, in New Jersey, "in excess of 50 mpcf".

These figures of dust concentrations are, of course, meaningless in isolation - this matter is discussed in a later chapter - and, because they had been obtained by different technologies, they are not on a comparable basis. However, what is clear is that dust concentrations were, in each of the cases cited above, about two orders of magnitude worse than levels in the early 1970s, although these levels themselves were still much higher than today's "standards". The 1931 Regulations referred to by Knox and his colleagues will be mentioned later; at this point, it is only necessary to point out that they introduced dust suppressive measures in 1933. Thus, before that date dust concentrations were surely at least another order of magnitude worse than in the 1950s. Newhouse (1973a) cites anecdotal evidence to this effect in British factories, and Gibbs and Lachance (1972) corresponding evidence concerning Quebec mines and mills.

Such an insult to the lungs might well be expected to cause severe disease. Indeed, in *De Re Metallica*, published in 1556, Agricola (1494-1555) commented " ... the dust, which is stirred and

beaten up by digging, penetrates into the windpipe and lungs, and produces difficulty in breathing and the disease which the Greeks called asthma". Hunter (1975), who quotes this comment on p. 27, later (p. 918) added "It was here [Joachimsthal, in Bohemia] that Agricola strongly advocated the ventilation of mines, for he knew that dust entering the lungs caused disease associated with dyspnoea, and he spoke of corrosive dusts which ulcerated the lungs, producing consumption." Hunter (p. 27) considers Agricola's accounts of miners' ailments as rambling and without precision, and that the idea of diseases specifically caused by particular occupations had not in Agricola's day been conceived. Hunter believes that the conditions being described more than 400 years ago probably included silicosis, tuberculosis and lung cancer. Nevertheless, it would appear that Agricola was the first to recognize a "pneumoconiosis", albeit in hard-rock miners extracting gold and silver.

This term, coined as "pneumonokoniosis" by Zenker in 1876, is another collective noun, meaning literally lung-dust-condition, or better a health effect arising from the inhalation of dust. VanOrdstrand (1977) lists some 26 pneumoconioses, providing a personal classification into (A) the benign, (B) those that are usually reversible and (C) those which may be disabling and even fatal. It is unlikely that this grouping would be generally accepted by those working in this field, but there is little doubt that *asbestosis* may indeed be disabling and may lead to premature death; see below.

However, before reviewing the effects on health of the inhalation of dust, it is necessary to consider what happens to the dust itself.

This is a matter of great complexity, which has been the subject of theoretical and experimental investigations since the 1930s, but about which knowledge is still not complete. Reliance has to be placed on morphological and pathological evidence, which can, of course, be quite misleading. Experiments in man may be not only dangerous but unrewarding, because the present concern should be to examine in what *practical* circumstances particular amounts of fibre are retained in the lung and how these amounts differ and, even more difficult, why. For the present purposes, a very simplified statement must suffice: it is based on the following references:- McDonald, Gibbs *et al.* (1972); Muir (1972); Timbrell (1973); and Morgan (1975).

There appear to be two generally agreed facts. First, only those particles or fibres less than about ten micro-metres (μm) enter the respiratory system, and most of the larger even of this size are trapped in the nose or the upper regions of the main respiratory tract. It is also likely that the curly fibres of chrysotile have greater difficulty in penetrating deeply, because of interception in the branching respiratory passages, than the more rectilinear fibres of the amphiboles. Further, dust deposition is a complex process, involving sedimentation, inertial impaction and diffusion, each element of which depends on the sizes, shapes and density of the particulate matter, its concentration, electric charges and growth of hygroscopic particles due to humidity, and to ambient temperature and humidity which may reach extremes in some occupational environments, particularly mining. Thus only the very fine fibres penetrate to the alveoli, and the shortest of these to the periphery. The second fact is that a

high proportion of those fibres or particles that do penetrate to the small air cells is eliminated by cilial action and mucous escalation, to be swallowed and excreted *via* the gastro-intestinal tract. This process of elimination is usually highly efficient, and indeed is thought to be improved when heavily loaded. However, it may not be able to cope with truly massive quantities of retained particulate matter. Further, the macrophages, which have to engulf the fibres before they are eliminated are incapable of dealing with long straight fibres, common in crocidolite, and particularly in amosite.

The amount of fibre retained in the lung is an extremely small proportion of that inhaled, and that proportion may well vary, not only because of differences in factors already discussed, but also with factors of human variation. Such factors may be physiological or immunological; they may be altered by smoking habit or by respiratory disease, either acute or chronic; and they almost certainly include differences in breathing patterns, through nose or mouth, and in rate and depth of breathing dependent on physical effort.

Chrysotile fibres are more soluble than amphiboles and this plays an important part in determining what proportions are retained for long periods. Retained chrysotile fibres may break up into even smaller "fibrils", detected in the lung by electron microscopy *post mortem*. However, the fibres of crocidolite appear to retain their original form, and are often retained indefinitely, being easily recognized at autopsy. Retained fibres may migrate within the parenchyma, or to pleura or even peritoneum.

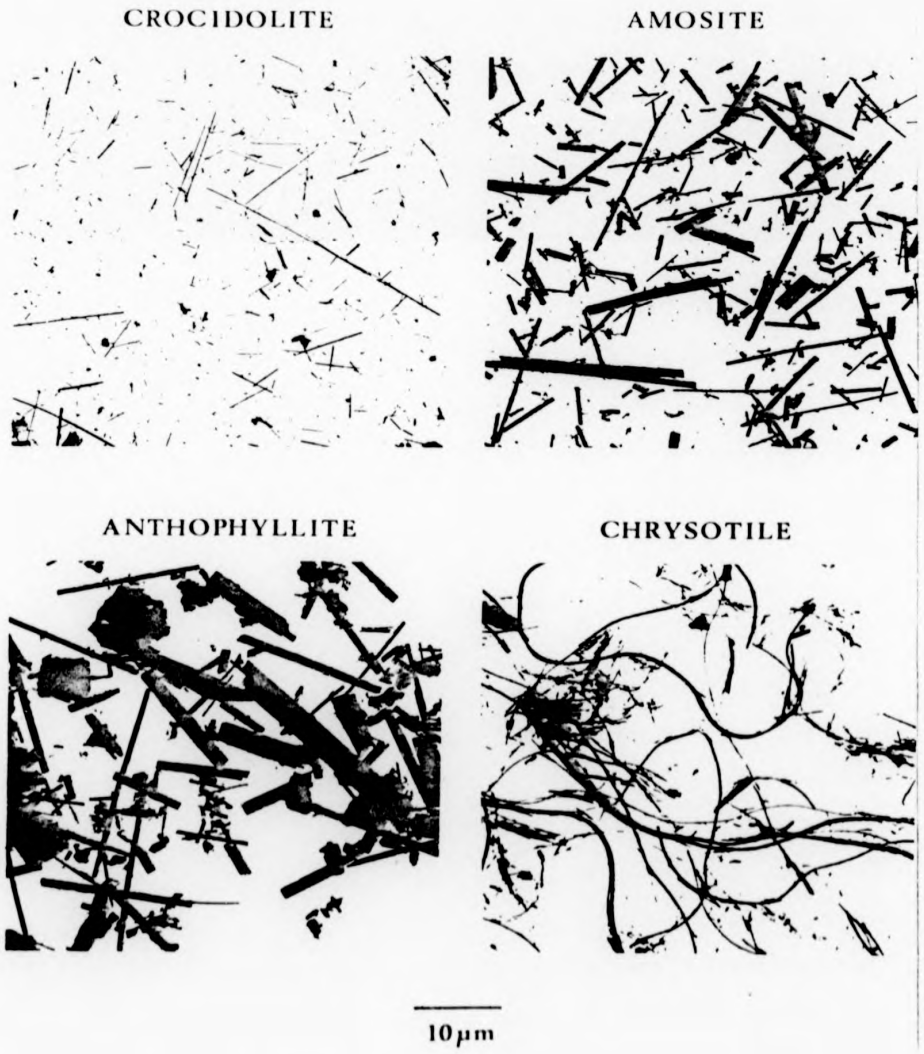
Figure 2.3 gives copies of electron micrographs of respirable fibres, taken from Timbrell (1973), who commented on the curly morphology of chrysotile fibres compared with the rectilinear shapes of the amphiboles, and that the diameters of crocidolite were of smaller order than those of anthophyllite, with amosite in between, and that this was a characteristic of such fibres from Cape Province, Finland and the Transvaal, i.e. whence all commercial supplies now come. He also emphasised the fragmentation of the chrysotile fibres, longitudinally, into fibrils of very small diameter, about 0.03 μm.

2.6 Pulmonary fibrosis; asbestosis

Those who have been heavily exposed to asbestos dust may develop asbestosis. Harries (1971, p. 11) reports Burton Wood's 1929 findings in 15 cases of asbestosis examined for symptoms, signs and x-ray changes. "The most important symptom was dyspnoea, which was progressive, and in the later stages of disease, extreme. Cough was often slight, and was a variable symptom. He noted a reduced chest expansion, cyanosis and finger clubbing often occurred. Dry crackling adventitious sounds were present, chiefly in the bases and superficially in the axillae. He thought the latter sound might have a pleural origin. The first patient described in the series is reported as having a vital capacity of 1,600 cc and this is the first report of a lung function test in asbestosis. The x-ray films were described, and Burton Wood mentioned that although some of the cases showed a 'ground glass' appearance over the lower zones, a fine mottling was seen on closer inspection. Basal pleurisy was seen in many films and also thickening of the apical pleural cap."

Figure 2.3

ELECTRON MICROGRAPHS OF ASBESTOS FIBRES



As a result of a report in 1928 of an asbestos worker with pulmonary fibrosis for which no occupational or infective cause, other than asbestos, could be found, Merewether and Price (1930) investigated, for the Factory Department of the Home Office, the effects of asbestos dust on the lungs (Harries, 1971, p. 11). They showed that, of 363 asbestos textile workers examined, 95 (over a quarter) had definite pulmonary fibrosis considered due to asbestos dust. These workers found a correlation between the incidence of fibrosis and the duration and intensity of dust exposure. Radiological examination of 133 workers revealed signs of diffuse fibrosis in 62 (nearly half), while radiological changes were suggestive in a further 25. Harries comments that radiographic technique was probably such as to lead to under-estimation of the prevalence of changes, but does not comment on how the workers subjected to x-ray examination were selected for this purpose. Merewether and Price found no particular tendency for respiratory tuberculosis to be present in the asbestos workers, with or without fibrosis.

The report of Merewether and Price suggested not only a dose-response relationship, but also that the interval between start of exposure and development of definite disease depended on the dust concentration. The latent period was as short as seven years in continuous high exposure, but could be as long as 25 years in lower concentrations. It was clear that dust suppression was needed if the disease was to be prevented. Merewether and Price further reviewed the dust concentrations in the various asbestos textile processes and made recommendations as to suitable methods of dust suppressive measures. It was these measures that formed the basis of the Asbestos Industry

Regulations 1931, in the United Kingdom. These were not implemented until 1933, but they included periodic medical supervision of persons working in certain prescribed asbestos occupations, while asbestosis was recognized as a condition covered by the Workmen's Compensation Acts.

The earliest studies of asbestos-related disease in the United States appear to have taken place in the mid-1930s, as described by Ayer *et al.* (1965), and a Threshold Limit Value was set "many years ago", according to Schall writing in 1964, by the American Conference of Government Industrial Hygienists, based on the findings of Dreessen *et al.* (1938). This standard was endorsed by the ACGIH in 1964, although Schall (1965) had some severe criticisms of it.

2.7 Diagnosis of asbestosis

Although it is nearly fifty years since these findings were presented, there is still no generally agreed definition of asbestosis. This is despite the introduction of a new international classification of radiographs of the pneumoconioses (to be discussed fully in a later chapter), which was prepared over the years 1966-71 with asbestosis well in mind; it had not been considered in the earlier ILO classifications. In 1971, Harries (p. 24) commented that there was disagreement over the presence and significance of the various symptoms and signs, including the radiological and lung function changes; in particular, the importance of pleural changes was not generally agreed. Harries felt that the most acceptable consensus of opinion was that asbestosis is a *progressive* pulmonary fibrosis resulting from exposure to asbestos dust, accompanied by fibrotic changes in the pleura which

may develop areas of calcification. He went on to state that little advance had been made in the matter of *early* diagnosis of asbestosis, pointing out that it still required intelligent assessment of all the relevant data. Lewinsohn (1977) stated that "asbestosis is still the *earliest* lung disease resulting from exposure to [asbestos] and its incidence in an exposed population is the most useful indicator of the degree of dust control exercised over a period of time". These views are not entirely compatible; the use of the terms "progressive" and "earliest" suggest different attitudes towards the condition. (The italics in this paragraph and the next are mine.)

Lewinsohn (1977) also stated that, for him, "the diagnosis [of asbestosis] depends upon: (1) An adequate occupational exposure history. (2) Physical signs of pulmonary fibrosis. (3) *Progressive* radiological changes. (4) Confirmatory measurements of altered lung function". The physical signs he was referring to were basal rales and finger clubbing, and he well recognized the subjectivity of their assessment. He made the point that the presence of asbestos bodies and fibres in sputum were of little importance in that they could only confirm exposure, which should already have been ascertained as part of the history. It was, of course, essential that the history should include all exposure, including any that may have occurred during, for example, home repairs. Lewinsohn did not explain what he meant by "progressive" x-ray changes. Further, his emphasis on the confirmatory nature of altered lung function is not echoed by all other workers. Thus, Keatinge (1973), reviewing the work of Ghezzi *et al.* (1972), stated that "even in the absence of radiographic changes, clinical examination and lung function tests will provide enough evidence to

demonstrate that the patient has been exposed to asbestos in the past". In this context, the evidence, concerning a specific patient, is required presumably for purposes such as compensation.

2.8 Is asbestosis progressive?

As to the presumed progressive nature of asbestosis, Gilson (1973) reported to the Working Conference held at the IARC, Lyon, that little progress had been achieved in the investigation of the effects of the removal from further exposure to asbestos dust. It had been one of the recommendations of the Union Internationale contre le Cancer (UICC) Working Group in 1964 that it was important to establish the subsequent morbidity and mortality from asbestosis, and the mortality from cancers associated with exposure to asbestos, in population groups no longer exposed to the dust. As a first step, Professor McDonald's team at McGill University assembled a group of 86 ex-employees of the Quebec chrysotile production industry; they were men born in the years 1901 through 1920 who had left the industry in the period 1950-61 and had been x-rayed before leaving. These men were x-rayed again in 1972, and seven separate assessments were made of the film pairs, all by experienced readers. Parenchymal change was recorded in at least four assessments for eight of the 66 men who had had substantial exposure before leaving, but for none of the 20 with only slight exposure. It was concluded (Becklake *et al.*, 1978) that the parenchymal changes observed after leaving the industry were attributable to the earlier occupational exposure to chrysotile; these changes were mainly in the nature of "attacks" rather than "progression". Pleural change was recorded for 15 of the 66 with heavier exposure and for 4 of the other 20, very similar proportions.

2.9 Asbestosis and mortality

Although there are still problems over definitions and whether or not asbestosis is essentially progressive, it remains clear that in those who have been heavily exposed to asbestos dust, there may be respiratory symptoms (usually breathlessness, first only on exercise, and later more severe, even seriously disabling), impaired lung function (particularly reduced Forced Vital Capacity, or FVC, and transfer factor), basal rales, finger clubbing, and changes on the chest roentgenogram; one, several or all of these signs and symptoms may be present, but there are also many heavily exposed workers who show none of them. X-ray changes include nowadays many that were incorporated into the international classification for the first time in 1968, and incorporated into the ILO U/C (1971) classification when this eventually appeared.

Asbestosis can undoubtedly lead to premature death. Indeed, the first description of the disease process, in 1906 by Dr. Montague Murray at the Charing Cross Hospital in London, was in an asbestos textile worker, with incapacitating breathlessness, but few other physical signs, who died at the age of 33; at autopsy, there was widespread pulmonary fibrosis, with no evidence of tuberculosis, together with marked pleuritic adhesions. "This patient was the sole survivor of 10 men working in the carding room when he commenced work [there]; the others had all died about the age of thirty" (Harries, 1971, p. 11). The second *post mortem* to direct attention to the possibility that silicates might cause extensive fibrosis was not until 1924, when Cooke demonstrated what have come to be called "asbestos bodies" in the lungs of a young tuberculous female textile worker (McVittie, 1965).

Three years later, according to McVittie, "a whole new literature relating to asbestos appeared in Britain", and in 1933, Merewether (cited by Harries, 1971, p. 13), while pointing out the difficulty of making the diagnosis of asbestosis, noted its insidiousness and reported the average age of death for men with asbestosis as 40.8 years, compared with 54.1 years for those with silicosis. Of the 100 men with asbestosis discovered in a Home Office enquiry in 1928, seventeen had died, ten with asbestosis, six from asbestosis with tuberculosis and one with asbestosis and carcinoma of the pancreas.

2.10 Lung cancer

Primary carcinoma of the lung has often been recorded in asbestos workers, frequently in association with pulmonary asbestosis. For example, Doll (1955) concluded that the average risk of cancer of the lung in workers who had been exposed to asbestos dust for twenty years or more was of the order of ten times that experienced by the general population. Although Braun and Truan (1958) criticised Doll's findings and "found no unusual risk to asbestos miners in the province of Quebec", their own conclusion was hardly justified; see below. Again, McCullogh in 1959 reported lung cancer in 65 out of 365 deaths (18 per cent) during 1924-55 in which *post mortem* had confirmed the presence of asbestosis (Harries, 1971).

McDonald (1973) reviewed cancer in chrysotile mines and mills, pointing out that Braun and Truan (1958; just referred to) had observed at least nine lung cancer deaths within a specified cohort, compared with six expected from Provincial rates. McDonald also referred to the findings of Kogan *et al.* (1966) in a ten-year study of

mine and mill workers from Sverdlovsk, a large Russian city on the eastern edge of the Ural mountains and fairly close to the world's most extensive chrysotile deposits, at Asbest: although materials and methods were not fully described, it was reported that mortality from lung cancer was increased by factors of 1.9, 2.3 and 3.1 for miners, factory workers and millers, respectively. The investigation of mortality in the Quebec chrysotile mines and mills carried out by McDonald and his colleagues has been reported *seriatim* (McDonald *et al.*, 1971; 1973; 1974; Liddell, McDonald and Thomas, 1977; McDonald and Liddell, 1978; and in preparation, but cited here as McDonald *et al.*, 1979). From the start, excess lung cancer mortality has been shown among the male workers. This study is particularly important because of its size (now 4,500 deaths in a defined cohort of nearly 11,000 men - and 440 women), because of the "purity" of the exposure, because it includes workers with very little exposure as well as those with heavy exposures, and because quantitative estimates of dust concentrations have been made. There appears to be a clear relationship between excess lung cancer mortality and total dust exposure.

An important co-carcinogen is, of course, cigarette smoke. The latest report by McDonald *et al.* (1979) will show that the data fit quite well a multiplicative model of risks, in which the two agents (asbestos dust and cigarette smoke) act in such a way that " ... addition of the second agent produces an effect which is proportional to the effect of the first. If smoking alone increases the natural incidence of lung cancer, say, 10 times (i.e. the relative risk due to smoking alone is 10) and asbestos alone increases it three times, the lung cancer incidence in those exposed to both asbestos and smoking

will be equal to 10 x 3 = 30 times the natural incidence". This quotation is from Saracci (1977), who was analysing the epidemiological evidence on the asbestos-smoking interaction, and concluded that this model stood as the most plausible interpretation of the interaction. The data from Quebec (not available to Saracci) suggest that exposure *for 50 years* to concentrations averaging one mpcf are roughly equivalent in their effect of generating lung cancer to the smoking of three cigarettes *a week* (McDonald and Liddell, 1978).

There have now been many investigations among other groups of workers, in all of which excess lung cancer mortality has been observed in comparison with expectations based on national, state or similar reference populations. There appears little doubt that all forms of asbestos can produce not only fibrosis but also lung cancer. However, Wagner *et al.* (1971), reviewing the epidemiology of asbestos cancers, stated that "The epidemiological evidence suggests that the use of chrysotile asbestos with efficient suppression carries only a small risk. The use of crocidolite should be avoided wherever possible. The position of amosite is probably intermediate, but dust control should be at at high standard". Enterline and Henderson (1973) were a little sceptical, although their own review tended to support the gradient in carcinogenicity indicated by Wagner and his colleagues. Further confirmation comes from a study by Weiss (1977).

Because the importance of fibre type cannot be overemphasised, an up-to-date review of the evidence on relationships between dose of asbestos dust and response in the form of lung cancer mortality is given in Chapter 6.

2.11 Mesothelioma

Although first suggested only in 1960, by Dr. J. C. Wagner and his colleagues, in South Africa, it is now generally accepted that asbestos dust can cause mesothelioma, i.e. malignant mesothelial tumours of pleura or peritoneum. This is an extremely rare tumour with an incidence around two per million population each year in many countries of the world (McDonald and McDonald, 1977). However, the incidence is often higher in cities with dockyards, and higher still in working populations exposed to asbestos. Important excesses occur in Turkey, particularly in two villages in central Anatolia, Karain and Tuzköy, where Baris *et al.* (1978) have reported extremely high incidence of mesothelioma, consistent with environmental exposure to a carcinogen since birth, although there are no asbestos deposits in the neighbourhood. Mineralogical studies have suggested that zeolite minerals may be the causative agents, and Pooley (1978) has shown that the zeolites near these villages, but not elsewhere in Anatolia, are of similar sizes and shapes as the fibres of crocidolite; see Figure 2.3.

McDonald and McDonald (1977) present proportional mortality rates for lung cancer and for mesothelioma from the main published cohort studies of male asbestos workers (Table 2.1). After allowance for differences in the general incidence of these diseases in the countries concerned, these authors commented as follows: "The findings for insulation workers, mainly obtained by Selikoff and his colleagues (1973), but supported by the studies of Elmes and Simpson (1971) in Belfast and Kleinfeld and others (1967) in New York State, were all very similar. Regardless of age, type of work or duration of observation period, about 20 per cent of deaths in insulation workers were

Occupational group	Location	Period	Deaths: all causes
Insulators			
All types	NY & NJ	1943-72	421
All types	US & Canada	1967-72	1,092
Construction	US & Canada	1967-72	446
Shipyards	US & Canada	1967-72	34
All types	NY	1945-65	46
All types	Belfast	1940-66	98
Asbestos factory			
Textiles	Rochdale	1916-67	125
Mixed products	London	1931-70	350
Mixed products	PA	1938-64	330
Mixed products	Cardiff	1936-62	133
Amosite insulation materials	NJ	1941-71	484
Mining and milling			
Chrysotile	Quebec	1936-73	3,749
Chrysotile	N. Italy	1932-70	270
Anthophyllite	Finland	1936-67	216
Talc & tremolite	NY	1940-65	91

Table 2.1

PROPORTIONAL MORTALITY FROM LUNG CANCER AND MESOTHELIOMA

(McDonald and McDonald, 1977, Table 12)

Per cent lung cancer	Per cent mesothelioma	Per cent pleural
20.0	7.6	25
19.5	7.1	34
17.7	5.2	43
26.5	8.8	66
21.7	6.5	33
24.5	7.1	57
17.6	3.2	100
20.6	6.9	38
10.6	2.4	0
8.3	0.8	100
15.1	1.4	43
5.7	0.2	100
2.2	0	-
9.7	0	-
9.9	2.2	50

caused by lung cancer and a third as many more by mesothelioma, predominantly of the peritoneum. The mortality of factory workers was more variable; the lung cancer ratios were generally lower and there were fewer mesotheliomas. The London factory investigated by Newhouse (1973b) which used chrysotile, crocidolite, and amosite seems to have fared worse than the predominantly chrysotile factories in Rochdale studied by Knox and others (1968), in Lancaster, Pennsylvania by Mancuso and El Attar (1967), and in Cardiff by Elwood and Cochrane (1964). The amosite plant in Paterson, New Jersey surveyed by Selikoff *et al.* (1973) occupied an intermediate position. In the asbestos production industry, experience was quite different, as shown by our own studies in Quebec and those of Ghezzi *et al.* (1972) in Northern Italy and also those of anthophyllite workers by Meurman *et al.* (1974) in Finland. In all these groups, lung cancer was a less frequent cause of death, and there were few pleural mesotheliomas and none of the peritoneum. This was in line with reports from Kogan *et al.* (1972) in the USSR of a similar moderate increase in lung cancer in the many thousand Russian chrysotile miners and millers, but no evidence of mesothelioma, though cases might have been missed."

Some more recent information follows. McDonald and his colleagues have discovered only eleven deaths due to mesothelioma among over 4,500 deaths in the years 1911-1975 in a defined cohort of more than 11,000 workers in the Quebec chrysotile production industry, some of whom were exposed very severely, with accumulated dust exposures amounting to over 4,000 mpcf.years, the equivalent of fifty years employment in dust concentrations averaging 80 mpcf. Of the eleven cases, one was a female known to have worked in a gas mask process -

see below - and a second was male employed only for a short time in the same factory at the relevant period. Of the other cases, all male, two were workers at Asbestos and seven at Thetford Mines, where dust concentrations were generally considerably higher that at Asbestos, and where men tended to be employed for longer periods. None of the cases had accumulated exposure greater than 1,000 mpcf.y, but there was some evidence of a dose-response relationship at Thetford Mines. Even here, the proportional mortality ratio (PMR), i.e. seven deaths from mesothelioma among a total of 2,284 deaths, was only 3.1 per thousand.

Selikoff and Hammond (1975) have recently updated three important series: 17,800 insulation workers in the USA and Canada, followed after at least twenty years since first exposure; 623 asbestos insulation workers in New York and New Jersey, followed similarly; and 933 amosite factory workers first employed 1941-45, followed after at least twelve years since first exposure. Deaths from mesothelioma in relation to those from all causes for the three populations were:- 87 in 1,109 (PMR = 78.3 per thousand); 35 in 444 (PMR = 78.8); and 10 in 429 (PMR = 23.3).

Peto *et al.* (1977) and Peto (1978) reported that, in all the workers studied at the factory in Rochdale, there had been some 34 deaths from mesothelioma. Five of these occurred among the 186 deaths (all causes) of workers with at least 10 years exposure, a PMR of 26.9 per thousand. Although most of the fibre used in this factory was chrysotile, some crocidolite has been processed; the amount is reported to be low, but there is considerable evidence (for example from a

former Medical Inspector of Factories) that "blue" asbestos was commonly used there in the 1950s.

Jones *et al.* (1976) and McDonald and McDonald (1978) have demonstrated enormous risks of deaths due to mesothelioma in workers exposed during World War II to crocidolite used in the filters of military gas masks, in England and Eastern Canada, respectively. In the Canadian study, a total of 199 persons had been employed in this process for a maximum of about two years, several at the factory in Asbestos, Quebec. Of the total, 56 were known to have died by the end of 1975; nine of the deaths were from mesothelioma, a PMR of 160.7 per thousand. Seven of the nine mesotheliomas were peritoneal.

All the mesotheliomas among miners and millers in Quebec and among factory workers in Rochdale were pleural, whereas the majority of those in other asbestos employment involved the peritoneum. Peto (1978) developed an argument based on these differences, which leads to incrimination of chrysotile. Undoubtedly, the PMR among Quebec miners and millers is rather higher than in the general population of Quebec, but it is very much less than in those known to have been exposed to amphiboles. Although no great reliance can be placed on the exact values of the PMRs, they do appear to give the order of magnitude of the risks, i.e. 3 per thousand for miners and millers at Thetford Mines; 23 for amosite workers; 27 for Rochdale factory workers; 78 for American and Canadian insulation workers; 104 for Barking factory workers; and 161 for Canadian crocidolite gas mask filter workers. These differences cannot be denied, despite the dismissal by Selikoff and Hammond (1975) of the proposal that "the

specificity for mesothelioma is greater for some kinds of asbestos than for others".

Thus it is difficult not to agree with McDonald and McDonald (1977) that the incidence of mesothelioma in insulation workers may be 100-200 times greater than in the general population, whereas in those engaged in chrysotile production it is perhaps three to six times greater. Indeed, it seems likely, as these workers suggested, that the potential for causing mesothelioma follows a gradient similar to that for lung cancer, *viz.* crocidolite is the worst hazard, chrysotile the least.

2.12 Cancer of the gastro-intestinal tract

Harries (1971, p. 231), in a review of evidence, remarked that several studies of asbestos workers had shown an increased risk of cancer of the gastro-intestinal tract. Bonser and colleagues had reported (1955) four abdominal cancers in 72 cases of asbestosis, and Keal, in 1960, one death from peritoneal neoplasm in 15 male deaths with asbestosis, and 9 female deaths from ovarian or peritoneal cancer in 15 similar female deaths. Mancuso and Coulter (1963) found an excess of peritoneal neoplasms, and Selikoff *et al.* (1964) excesses of the stomach, colon or rectum. Doll, in 1955, and with Knox and coworkers in 1968, showed no excess of cancers other than of the lung, while Meurman *et al.* (1974), studying anthophyllite workers in Finland, reported excesses of lung cancer and asbestosis, but not of cancers of the digestive system.

McDonald (1973), concerned with gastro-intestinal cancer in chry-

sotile miners and millers, reported no excess in Italy, but a slight excess, not always at the same sites, in each of his serial reports from Quebec. The excesses appeared only in those with exposures greater than 400 mpcf.y. In the latest Quebec material (McDonald *et al.*, 1979), case-control studies of all 154 deaths from cancer of the stomach or oesophagus and 88 deaths of cancer of the colon or rectum, each with two controls matched for age and mining district, have revealed that the relative risk for cancer of the stomach or oesophagus was normal for exposures up to 1,000 mpcf.y, but much enhanced at higher exposures. For cancer of the colon and rectum, relative risk was normal in men with exposures up to 300 mpcf.y, and then increased.

It would therefore appear that heavy exposure to asbestos may indeed lead to cancer at various sites in the gastro-intestinal tract. This is despite the statement by Gross (1974) that, because several laboratories in several countries had failed to induce cancer in experimental animals using fibres less than five μm in length, the concept that mesotheliomas and gastro-intestinal cancers arise from the ingestion of asbestos dust cleared from the lungs, should be abandoned.

2.13 Cancer of the larynx

From the patterns already discussed, it might seem likely that asbestos dust could also cause laryngeal cancer. However, there seems to be little reliable evidence in the recent literature. Shettigara and Morgan (1975) carried out a case-control study on 43 patients in Toronto, eliciting occupational and smoking histories. Both asbestos

exposure and cigarette smoking were considered "potent factors", with the "strength of association" higher for asbestos. Type of fibre was not considered, and the comment on relative potency is unwarranted in the light of the very small numbers on which it was based. Although their findings are claimed to be similar to those of Still and McGill (1973) in a hospital series, experience in the Quebec chrysotile industry was rather different. Here, McDonald and Liddell (1978) found no trace of a relationship between laryngeal cancer mortality and dust exposure, but a clear and convincing relationship with cigarette smoking. Only a small excess of deaths from laryngeal cancer was observed in the large series of asbestos insulation workers previously mentioned with regard to mesothelioma. Selikoff and Hammond (1975) considered deaths from this cause in those 11,656, nearly two-thirds of the complete cohort of 17,800, for whom smoking histories were available; four deaths due to larynx cancer were observed in the years 1967 through 1972, compared with 1.87 expected on the basis of US mortality rates for white males. The numbers were too small for the ratio of observed to expected (2.14) to be considered more than suggestive; all the cases had been smokers, the ratio being higher for those with a history of pipe and cigar smoking than for those who had smoked cigarettes.

2.14 Other health hazards

In their study of deaths to the end of 1975 in nearly 11,000 male Quebec asbestos workers, McDonald *et al.* (1979) used *inter alia* comparative composite cohort analysis (Case and Lea, 1955), sometimes called the man-years or the modified life-table method, to obtain numbers of deaths to be expected, for specific causes, on the basis of

rates for males in the Province of Quebec. The ratio of observed deaths to those expected can be seen to be a Standardized Mortality Ratio (SMR), adjusted for both age and era. The SMR for all causes was 1.1, and those for causes mentioned above were as follows:- Pneumoconiosis (asbestosis): 13.4; Lung cancer: 1.2; Cancer of oesophagus or stomach: 1.2; Cancer of colon or rectum: 0.7; Other abdominal cancers: 0.9; Laryngeal cancer: 1.0. The only unexpected finding is the low SMR for cancer of colon and rectum, despite the dose-response relationship mentioned above.

Other causes that produced SMRs noticeably above unity were Respiratory tuberculosis (1.3) and Accidents (1.2). The former has been eliminated as an effect of asbestos and appears to reflect the prevalence of the condition in the mining areas of Quebec, particularly Thetford Mines; the latter, although including work accidents, embraces all traumatic deaths and is clearly not a consequence of dust exposure. The SMRs for "Other" cancers, i.e. those not mentioned above, and for Cerebro-vascular diseases were both 1.1, but the usual test of statistical significance (Liddell, 1960) gave χ^2 values of only 0.8 and 0.7, respectively, despite the fairly large numbers involved (over 250 deaths from either cause). Thus, there would appear to be no additional health effect that could lead to premature death, at least in chrysotile mines and mills.

One minor effect is that asbestos corns may arise in those who get fibres into small open wounds (Harries, 1971). Finally, asbestos and asbestos products are mentioned several times in the index to the US Public Health Service's Guide to the Recognition of Occupational

Diseases (Gafafer, 1964), but in each instance not covered above it is a material with which the asbestos is mixed that is incriminated, e.g. benzene, cement or coal tar, not asbestos itself.

2.15 Summary; gaps in knowledge; thesis objective

It has been generally accepted for some years that inhalation of asbestos dust could lead to pulmonary fibrosis, carcinoma of the lung and the development of diffuse mesothelioma of the pleura and peritoneum (Bogovski *et al.*, 1973, p. xiii). There have also been several reports that asbestos exposure can lead to excess mortality from cancers of the gastro-intestinal tract and certain other sites, but the findings are not completely consistent.

There now seems little doubt that the different fibre types are associated with different risks. Risks of fibrosis, of lung cancer and of mesothelioma after exposure to crocidolite appear to be substantially higher than after corresponding exposure to chrysotile. The position of amosite may be intermediate, but too little information is available to place it accurately. There may also be differences in risk according to size of fibre, which may depend on the process.

The manifestations of pulmonary fibrosis include changes on the chest x-ray, respiratory symptoms, particularly breathlessness but sometimes also cough, increased sputum, or chest pain, impaired lung function, clubbing of the fingers, cyanosis and rales at the base of the chest (i.e. the adventitious dry crackling sounds described by Burton Wood, 1929). It is not clearly determined whether the fibrosis

is progressive after exposure has ceased.

The lapse period between the start of exposure and first signs or symptoms, some of which are also associated with cigarette smoking, is highly variable. The fibrosis, often called asbestosis but without consistent definition, can lead to premature death, which may not be coded, on death certification, to "pneumoconiosis" (codes 523-524 in the Seventh Revision of the International Classificaton of Diseases).

Lung cancers and mesotheliomas generally occur after long lapse periods. The former are also associated with cigarette smoking, and the risks appear to be multiplicative. The lapse period may be related to dose, age at first exposure, and amount smoked. An examination is in hand in the 245 lung cancer deaths within the 4,463 deaths up to the end of 1975 among the nearly 11,000 male Quebec chrysotile miners and millers being investigated by Professor McDonald and his colleagues; first results, in Table 2.2, provide no evidence of consistent variation in lapse period associated with dust exposure accumulated to age 45 or with smoking habit, but more detailed study is still required. The latent period between exposure to asbestos and appearance of mesothelioma is usually greater than 25 years, and even longer when the exposure was to chrysotile only (McDonald and Liddell, 1978).

The 1964 recommendations of the UICC Working Group (1965b) were for investigation of:- (1) fibre type; (2) dose-response relations; (3) effects of removal from further exposure; (4) all cases of mesothelioma; and (5) asbestos-exposed populations not so far studied. With regard to methods, the Working Group also recommended:- 1(a) col-

Table 2.2

LATENT PERIODS IN 245 DEATHS FROM CANCER OF THE CHEST

(Years from first asbestos exposure to death)

Smoking habit	Dust exposure accumulated to age 45 years (mpcf.y)					
	<30	30, <100	100, <300	300, <1,000	1,000 or more	All
Non-smoker	44.9 *(5)* *	42.8 *(2)*	40.5 *(3)*	33.8 *(5)*	45.9 *(5)*	41.5 *(20)*
"Undiff-erentiated"	38.6 *(43)*	35.9 *(12)*	35.0 *(11)*	41.2 *(22)*	43.2 *(12)*	39.0 *(100)*
15-25 cigarettes a day	35.7 *(28)*	38.0 *(19)*	40.8 *(16)*	39.5 *(22)*	40.8 *(7)*	38.4 *(92)*
26-50 cigarettes a day	36.4 *(13)*	40.6 *(7)*	38.2 *(3)*	39.6 *(7)*	44.9 *(3)*	38.9 *(33)*
All	37.7 *(89)*	38.1 *(40)*	38.6 *(33)*	39.7 *(56)*	43.2 *(27)*	39.0 *(245)*

* *Italic figures in brackets indicate number of deaths*

lection of full histories; 1(b) study of mortality and morbidity from *all* causes as well as from specific causes; and 1(c) consultation with statisticians at all stages from planning to analysis. 2 In all surveys:- (a) a standardized questionnaire should elicit symptoms (cough, sputum, dyspnoea and chest pain, as a minimum); and (b) when physical examination was possible, clubbing of the fingers, cyanosis and basal rales should be recorded. 3 A classification of chest radiographs should be developed. 4 Lung function tests should include FVC and Forced Expiratory Volume over 1 second ($FEV_{1.0}$).

By 1972, progress had been made on many of these recommendations. In a report to the Director of the IARC, the Advisory Committee on Asbestos Cancer could now make more specific epidemiological recommendations (Bogovski *et al.*, 1973, p. 341). A few of these were:-
(i) the need for investigation, within type of fibre, of differences in size of fibre, and past exposure, in the risk of excess cancers of the lung and of other sites; (ii) investigation of possibilities and efficacy of early detection of disease; and (iii) study of differences between the effects of low continuous exposure and intermittent high exposure. Others concerned such matters as *post mortem* studies of mesotheliomas, secular trends in the general population, talc, pleural plaques, and cost-benefit analysis.

One other recommendation is of particular interest in the present context. It was "to interrelate radiographic appearances, lung pathology, respiratory function, dust content and type, in asbestos workers".

Although the various symptoms and signs of asbestos-related disease are undoubtedly important, excess mortality is the outcome of prime epidemiological interest. In the Quebec studies, and elsewhere, radiographic appearances have been related to dust exposure; respiratory symptoms and lung function have been related to dust exposure and smoking habit; and x-ray changes have been related to function. There have been many studies of mortality in relation to exposure, but few have included quantitative assessment of both duration and dust concentration; smoking is an important concomitant variable.

However, the outcome of greatest interest has also to be related to the symptoms and signs, and to exposure and smoking. A limited study of mortality in relation to x-ray change was reported in McDonald *et al.* (1974), but the interrelationships of all these variables do not appear to have been studied, although of great potential importance. Investigation of these relationships in Quebec chrysotile miners and millers forms the general objective of this thesis; specific objectives are given in Chapter 7.

Chapter 3

RADIOLOGICAL CLASSIFICATION

3.1 Introduction

In describing the development of the classification of chest radiographs of the pneumoconioses sponsored by the International Labour Office (ILO) in 1958, Van Mechelen and McLaughlin (1962) stated that "The abnormal x-ray shadows seen in the pneumoconioses constitute various combinations of linear, reticular, nodular and massive shadows, and none of them is specific in the sense that similar shadows may not be found in other non-industrial pulmonary conditions". The first classifications appear to have been those drawn up in South Africa and America, in 1916 and 1917, respectively. The ILO have sponsored five, i.e. in 1930 at Johannesburg, in 1950 at Sydney, in 1958 at Geneva, in 1968 at Geneva again, and finally the ILO U/C (1971) classification (Gardner *et al.*, 1930; ILO, 1953; 1959; 1970; 1972).

3.2 International classifications up to 1958

The first international classification was adopted in Johannesburg in 1930. Its concern was, for all practical purposes, with silicosis only and it was based as firmly on clinical symptoms as on radiological abnormality; three stages of disease were recognized. Although there was a meeting sponsored by the ILO in 1939, no new classification was adopted. However, note was taken of an American classifica-

tion which included radiological changes, histological appearances, clinical findings, both objective and subjective, and degree of incapacity for work.

Meanwhile Irvine, in South Africa, had in 1934 produced what appears to have been the first attempt to classify in purely radiological terms. Ten years later, Eck and Hanaut put forward a strictly radiographic classification, which has clear reference to silicosis; four stages of abnormality were distinguished - linear, micronodular, nodular and pseudo-tumoral - with three grades in each. In 1948, Van Mechelen and Belayew used this as the basis of what was called the Hasselt classification.

An important change in development came in 1949, when the Medical Research Council's Pneumoconiosis Research Unit (PRU) published a classification of coalworkers' pneumoconiosis (Fletcher *et al.*, 1949). This distinguished simple and complicated pneumoconiosis, and introduced four quantitative grades of either. In Sydney in 1950, the ILO followed PRU closely, although the number of grades of simple pneumoconiosis was reduced to three (ILO, 1953). Also, a category was added for discrete opacities not falling into the quantitative grades. Quite soon, this was found unsatisfactory for most purposes, and at a meeting of British and French experts in 1951 a compromise between Eck and Hanaut (1944), PRU (Fletcher *et al.*, 1949) and ILO, Sydney, 1950, was introduced; this was called the Cardiff-Douai classification (Cochrane, *et al.*, 1951). It allowed for small opacities in simple pneumoconiosis to be categorized by both their size (p, m, n) and their number (grades 1, 2, 3).

In 1958, Van Mechelen and McLaughlin (as they reported in 1962) had been asked to find out how far the Sydney classification was being used, and discovered that it was employed in only two countries, and that for specific purposes. The Cardiff-Douai classification was used throughout Europe in coalworkers' pneumoconiosis, but for no other occupation. They found, nevertheless, a clear desire for an international agreement, and the ILO called experts together in Geneva. The conference decided on limitation to "persistent radiological opacities in the lung fields provoked by the inhalation of mineral dusts". Van Mechelen and McLaughlin commented that these opacities constituted the majority of pneumoconioses, but that asbestosis (and berylliosis) "are said to present features different from other pneumoconioses". The ILO, Geneva, 1958, classification (ILO, 1959) retained most of the Sydney and Cardiff-Douai features. Suspect pneumoconiosis was to be recorded as "Z", which related to "increased lung markings", small opacities had sizes p, m, n defined, with three grades of profusion, while the grading of large opacities (or complicated pneumoconiosis) was reduced to A, B, C. An interesting development was the introduction, or better re-introduction, of a class, "L", for linear opacities, i.e. "numerous linear or reticular opacities, the lung pattern being normal, accentuated or obscured". Meanwhile Van Mechelen and McLaughlin made the important point that "the number of small rounded opacities is relatively difficult to establish, because there is always a desire to combine two different aspects, i.e. their distribution in the pulmonary fields and their profusion".

This classification did allow for the optional recording of "the main complications of the pneumoconioses" by what were called "addi-

tional symbols"; these were as follows:-

(co) Abnormalities of the cardiac outline. To be replaced by (cp): cor pulmonale, if this condition is strongly suspected.
(cv) Cavity.
(di) Significant distortion of the intra-thoracic organs.
(em) Marked emphysema.
(hi) Marked abnormalities of the hilar shadows.
(pl) significant pleural abnormalities.
(px) pneumothorax.
(tb) opacities suggestive of active tuberculosis.

3.3 Classifications of asbestosis

As seen in the preceding section, asbestosis had been ignored in all the international classifications before 1958, and even in that could only be recorded in terms of "L" or certain "symbols". The remark that this condition was "said" to present radiological shadows differing from those in the other pneumoconioses appears rather naive in the light of Merewether's description, in 1930, of changes (Harries, 1971, p. 12) occurring in four stages:

I. increased linear striations
II. fairly definite fine dusty stippling
III. coarser mottling with increased linear striations
IV. grosser lesions with pleural changes and displacements due to the pull of fibrosing lesions.

In an important review, Bohlig (1965) pointed out that asbestosis did not present the same characteristics and uniform features as silicosis, and that several attempts had been made, from 1932 through 1956, to develop a classification, but always without international recognition. A three-stage scheme, originated by Saupe in 1938, had

been widely used but had the major drawback that the stages overlapped. Bohlig reported that he had tried to use the ILO classifications in asbestosis, and expressed the strong view that what was needed was development rather than a special, unrelated, scheme. The main difficulty lay in that most asbestoses (at least in Europe) showed linear marking and there was need to differentiate as to extent and density. He therefore proposed that the one class "L" be replaced by three grades of profusion (as for p, m, or n shadows), differentiated by the code "f" (for filamentum or fibra).

Sluis-Cremer and Theron (1965), at the same conference, pointed out the deficiencies of the ILO, Geneva, 1958, classification for use in asbestos workers, and also suggested three grades of profusion. They felt, however, that the linear markings could be classified as "fine" or "coarse" according as they were less or greater than 1 mm in width, and suggested the codes "Lf" and "Lc", respectively. These workers also proposed that pleural plaques should be recorded, and perhaps also pleural thickening, distinguishing whether diaphragm, mediastinum, walls or fissures were involved. They considered, too, that "symbols" should be added for bronchial carcinoma (Ca), mesothelioma (Me) and cystic changes (Cy).

3.4 The UICC/Cincinnati (or U/C) classification

These two proposals were modified into a system that was evaluated by a working group set up by the UICC (Union Internationale Contre le Cancer) Committee on Asbestos and Cancer in 1965. Meanwhile Professor Corbett McDonald and his colleagues working from McGill University had urgent need of a means of classifying several thousand

films from Quebec and had prepared proposals, and the US Public Health Service had expressed interest. The three groups met for four days in Cincinnati at the end of 1967, reviewed the results of the trials that each group had carried out, and eventually came to unanimous agreement on a new scheme designated as the UICC/Cincinnati (or U/C) classification. Its aims and principles were (Bohlig *et al.*, 1970):-

Aim 1. To provide a scheme for the systematic recording of the various radiographic appearances associated with exposure to asbestos and other dusts.

Aim 2. To provide a system in which the qualitatively different features of the film can be recorded separately and graded.

Aim 3. To provide a code that permits a brief description of the important radiographic features of the pneumoconioses.

Principle 1. As far as possible it is purely descriptive of radiographic appearances and does not use interpretive words such as "fibrosis" or "infection" which already have definitions in pathology.

Principle 2. It describes the natural history of the changes produced by asbestos and other dusts as far as is known, but it avoids the use of terms such as "early stages", "progressive" or "final".

Principle 3. It provides a system of recording semiquantitatively (with the help of verbal descriptions and standard films) features of the film which can be separately assessed with a reasonable degree of certainty.

Principle 4. It provides a means of describing the appearances seen in workers known to have been exposed to asbestos dust as well as to other dusts.

The U/C classification was an extension in several dimensions of the ILO, Geneva, 1958, scheme, and seems to have achieved most of its

intentions; however, Principle 1 (see above) was unfortunately not followed in its entirety. There is still a need to decide whether any radiographic changes are due to pneumoconiosis or not, and hence an element of diagnosis has been retained even in the latest developments; see page 6 of the present ILO U/C (1971) classification (ILO, 1972).

In the U/C classification, small opacities were subdivided into rounded and irregular; for rounded opacities, the "types" (or sizes) p, m, n, were replaced by p, q, r to reduce errors of transcription but without change of definition; for irregular opacities (replacing "L"), three "types" (or widths) s, t, u were introduced; each lung was divided into three zones, and a record was to be made of those in which the opacities appeared. Large opacities were subdivided into well-defined and ill-defined.

Perhaps the most important change was in what were called "other features". The "symbol" (pl) was replaced by (1) a record of whether either costophrenic angle was obliterated; (2) the grading of pleural thickening (or diffuse, uncalcified, plaques); and (3) the recognition of pleural calcification by site (diaphragm, walls or other), and by extent. In addition, when a diaphragm was ill-defined, this was to be indicated, as was the extent to which cardiac outline was ill-defined.

The number of "symbols", even excluding (pl), was increased to 18, of which six were obligatory, the rest optional.

Another major innovation was of the 12-point scale of profusion, developed within the National Coal Board by the present author and his

colleagues (Liddell, 1963; Liddell and May, 1966; Liddell and Lindars, 1969).

It had been agreed that, before the U/C classification was published, it should be subjected to tests of practicability and observer variation, both intra- and inter-observer. These tests, described briefly by Rossiter (1972a), were considered to show that the proposed scheme had reached a stage where it was likely to be a useful tool for epidemiological studies and it was therefore published (Bohlig *et al.*, 1970). It is fully described in the Appenidix to this thesis, bound at the end of Volume 1.

3.5 The ILO U/C (1971) classification

Shortly after the decision to publish the U/C classification, the ILO adopted it as the "extended" ILO, 1968, classification (ILO, 1970); this was expected to be particularly useful for epidemiological studies in which the radiographic features were to be related to other measurements, such as indices of dust exposure or lung function changes (Jacobson and Lainhart, 1972). At the same time, a "short" classification was authorised for clinical use and for purposes, such as compensation, to which the earlier (1950 and 1958) international classifications had been put. This 1968 short classification was very similar to that of the ILO in 1958, except that pleural thickening of the chest wall and diaphragm was to be recorded by means of the "symbol" (pl) and pleural calcification by (plc). Five further "symbols" were made obligatory, with twelve "optional extras".

The US Public Health Service and others, particularly the

group working from McGill University, conducted several studies using the U/C classification in asbestos workers, beryllium workers, coal-miners, dockyard workers, etc. Much varied experience was gained in a short time, and a meeting to exchange information was held at the MRC's Pneumoconiosis Unit at Penarth, in April 1971. Several changes were adopted, and the recommendation made that the ILO and U/C classifications should be identical. At the Fourth ILO International Pneumoconiosis Conference, at Bucharest in September 1971, these proposals were approved, and the classification became known as the "ILO U/C (1971) international classification of radiographs of the pneumoconioses" (ILO, 1972; Jacobson and Lainhart, 1972). (The Penarth meeting had not resolved all difficulties in the use of the U/C classification, as discussed by Jacobson and Gilson, 1972.)

As three years previously, there were two versions, this time called "complete" and "short". In the complete classification, the lung zones were defined more exactly than before, and there was a strong recommendation that the combined profusion of both rounded and irregular small opacities should be recorded - but without any guidance as to how this should be done. In the U/C, 1968, classification, the extent of pleural thickening had been coded on a four-point scale; in 1971, both width and extent were to be coded, each on to a three-point scale. The number of "symbols" was increased to twenty, and their use was made obligatory.

The short classification of 1971 made allowance for the recording of small irregular opacities, distinguishing type (width), s, t, u, but retaining the older four-point scales (0, 1, 2, 3) for profusion

of both forms of small opacity, and for "combined" profusion, again strongly recommended. Pleural thickening was to be recorded as in the 1968 short classification, but "symbols" were to be treated as in the complete 1971 classification.

In 1975, I carried out a trial, on behalf of the Royal Commission on the Health and Safety of Workers in Mines (Ontario), to evaluate the x-ray classification still in use at that time in Ontario; it had been based on the one adopted nearly forty years previously by the South African Miners' Phthisis Medical Bureau for use in cases of silicosis. Three experienced readers assessed 269 films, most of claimants for workmen's compensation but including a fair proportion of unexposed men. The report (Liddell, 1975a) recommended the adoption in Ontario of the latest international classification, not only for the obvious reasons, but also because a substantial proportion of films in the trial revealed irregular small opacities which could not be recorded in the Ontario scheme.

A further benefit of the trial was that it allowed a study of the use of the ILO U/C (1971) classification for films of men with varied exposures. Some of the difficulties in the recording of small opacities were discussed in a recent report (Liddell, 1977). The most serious were in the averaging of profusion over the affected zones of the lung, and, as expected, in the recording of combined profusion.

Jacobson and Lainhart (1972) emphasised that periodic reappraisal and further changes might be required, but that eventually a classification might be developed encompassing all diseases of the chest. In

the last few years, a group of experts in the USA, working under the auspices of the American Thoracic Society, has been examining ways of using or adapting the ILO U/C (1971) classification for radiographs of persons not occupationally exposed to mineral dusts. The American College of Radiology, in co-operation with the National Institute of Occupational Safety and Health and the ILO, is to hold a symposium in Washington in September 1978, to make a further appraisal of the present classification, and the ILO is calling experts to a meeting in Caracas to be held the following month.

3.6 Standard radiographs

As Van Mechelen and McLaughlin (1962) wrote, "it is generally accepted that it is practically impossible to use a radiographic classification accurately without a set of standard reference films". They discussed problems of finding a single set of films representing the various pneumoconioses, and then of making copies for use as reference. However, the final set was complete by May 1961, when these authors submitted their report.

These same standard films for small rounded opacities and for well-defined large opacities were also used in the U/C classification, but new films had to be provided as standards of small irregular opacities, of obliterated costophrenic angle, pleural thickening of the chest wall, irregular diaphragm and irregular cardiac border.

A reference set of films for use with the ILO U/C (1971) classification had to be considerably larger than ten years previously, in order to cover all the features, and Dr. J. C. Gilson in a paper

entitled "Choice of Standard Films, with particular reference to those for small rounded opacities", privately circulated in 1971, commented that, even for small rounded opacities, the factors to be taken into account on choosing standards had changed considerably from what they had been in 1958-61, but that there were strong grounds for maintaining the levels of standards of this radiographic feature. However, the methods of averaging profusion, when not evenly distributed over the lung fields, were changed in an important fashion in 1971; this is one of the problems referred to in Section 3.5 above, and can be seen to create further difficulties in the selection of standard films. Nevertheless, by the end of 1972, the ILO was announcing that standard films were available.

The set consists of 21 plates, approximately 350 mm square. Most are radiographs of both lungs in one person, but three show two or three portions of the lungs of different subjects. One full film is normal in all features, and each of the mid-category profusions 1/1, 2/2, 3/3 are illustrated for each type of small opacity (p, q, r; s, t, u). However, in none of the reference films for profusion 1/1 do the opacities affect all six lung zones, and the same is true for half the films for profusion 2/2 and even for the 3/3 of type u. Well-defined large opacities of each category (A, B, C) are shown, but only category B of ill-defined large opacities. Pleural thickening and diaphragmatic pleural plaques are illustrated on one plate (with parts of the radiographs from three persons), and all other features of the classification are seen in films chosen as reference for small opacities. About half the "symbols" are also illustrated in like manner. Rounded and irregular small opacities are stated to co-exist in one of

the films, but the details give no guidance on how the assessment of combined profusion was made (rounded: q, 1/1 with no zones marked as affected; irregular: t, 3/3 in all six zones; combined 3/3).

There is little difficulty in accepting Gilson's comment that levels of standards should not be changed unnecessarily, and the U/C, 1968, classification had of course made use of the existing standard films wherever possible. However, the principle on which the 12-point scales of profusion of small opacities were based ought to have been better served by the introduction of reference films on the borderlines between categories in the short classification. A reader might then have been able to decide, in direct comparison, whether a specific film showed less or more profusion than the standard, thus facilitating differentiation between, say, 1/2 and 2/1. It also seems likely that the "middling tendency" (Morgan *et al.*, 1974) – placing the majority of films in the middle of the short categories, i.e. at 1/1, 2/2 or 3/3, reducing the numbers placed close to but on one side or other of the category borderlines – observed in most studies of the use of the 12-point scales would be reduced, because there would not be a mid-category film against which to make direct reference.

3.7 Radiographic technique

No discussion of the classification of x-ray films of the pneumoconioses could be complete without at least a mention of radiographic technique. Despite many attempts at some degree of standardization, there is little agreement among experts on the criteria of film quality. Indeed, in one trial of the U/C, 1968, classification, it was reported by Rossiter (1972a) that agreement about technique was

worse than for any other radiographic feature: the complete range of assessments (from acceptable to very poor) was recorded by different observers on a quarter of the films in the trial. The effects of film quality on the actual readings are complex, as has previously been discussed by, for example, Liddell (1961). It seems likely that some, perhaps all, readers adjust their readings of films they consider of poor quality to what they believe would be seen in an acceptable film. Further complication arises because in recent years a new type of technique has been developed; this uses kilovoltages in the range 110 to 140 kV, with short exposure times, one thirtieth or one sixtieth of a second, whereas the earlier techniques had used 60 to 80 kV with longer exposures (0.05 to 0.08 s) in order to get adequate penetration. While good radiographs are possible from each method, with careful attention to detail (ILO, 1972), the films are not necessarily comparable, as has been appreciated recently in one particular study (Becklake *et al.*, 1978) where film pairs were assembled consisting of one film by each technique.

Further discussion of this subject is outside the scope of this thesis.

3.8 Summary

Although international classifications of radiographs of the pneumoconioses had been sponsored by the ILO since 1930, those up to and including that adopted in Geneva in 1958 had been concerned primarily with silicosis and coalworkers' pneumoconiosis. Even in 1930, Merewether had described the radiological appearances of asbestosis in rather different terms, and at the New York Academy of

Sciences conference in 1964 on the biological effects of asbestos strong dissatisfaction was expressed with the ILO classification for use in asbestos-exposed populations. The UICC Working Group agreed proposals in 1967, which with minor modifications were adopted by the ILO in 1968, and confirmed, after several more international meetings of experts in all the pneumoconioses, as the ILO U/C (1971) international classification of radiographs of the pneumoconioses (ILO, 1972).

One of the main driving forces behind the 1967 UICC Working Group meetings had been the need for a classification into which to assess many thousands of films of Quebec chrysotile miners and millers. The earliest agreed version of the U/C classification was adopted for that purpose (Rossiter *et al.*, 1972), and as these readings were used in the research reported here, full details of that classification are given in the Appendix to this thesis.

Chapter 4

QUEBEC CHRYSOTILE STUDIES I: POPULATIONS AND MEASURES OF EXPOSURE

4.1 Introduction

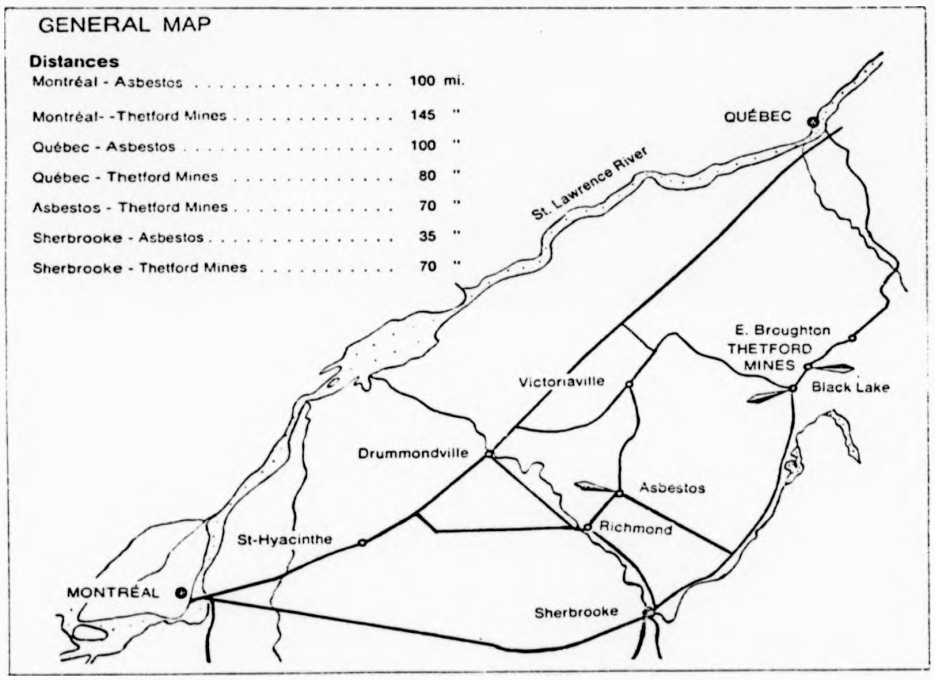
Although Quebec's share of the world market of asbestos has been falling in recent years, annual production of chrysotile during the 1970s has been fairly steady around 1.3 million short tons, and the labour force has numbered about six thousand. Mining is carried out in two neighbouring areas of the region known as les cantons de l'est (the Eastern Townships) of Quebec; see the map in Figure 4.1. The first workings, opened in 1878, were at Thetford Mines and work still continues in that town and in the nearby Black Lake and East Broughton. In 1881, one man opened a mine at Asbestos; the company has continued since, also operating a small factory in the town for the manufacture of mixed asbestos products. After mining, the rock containing asbestos is processed in adjacent mills, which crush the rock, extracting the fibre and preparing it for commercial disposal.

Those who have worked in the mines and mills of Quebec are, as mentioned in Chapter 2, of particular interest for several reasons. First, their exposure has been purely to chrysotile. In most industrial applications, different fibres are used, mixed in the one pro-

Figure 4.1

MAP OF THE ASBESTOS-MINING REGION

OF SOUTH-EASTERN QUEBEC



cess, or separately but within the same factory. Thus, although chrysotile dominates greatly, it is difficult to find other populations who have been exposed to a single fibre. Even where such a population has been found, it has been quite small; *cf.* Elwood and Cochrane (1964) and Weiss (1977). The second reason for the importance of the Quebec workers is that there have been so many of them - nearly thirty thousand by the end of 1966. Thirdly, many of these persons (predominantly male) worked for a very short period only and so had minimal exposure; on the other hand, there were substantial numbers who had had many years of employment often in the extremely severe dust conditions of the past. Thus, there are possibilities for comparison between heavily and lightly exposed.

Following the 1964 recommendations of the UICC Working Group (1965b) on problems requiring epidemiological study, Professor J. C. McDonald and his team based on McGill University started their investigations of the health effects of asbestos in this population, in 1966, at the request of the Canadian government and with encouragement from the Quebec government. There have been many facets of the investigation, which is still continuing, and some sixty reports. This multidisciplinary approach to such a large population exposed to a single fibre, with estimates of the dust concentrations to which they were exposed, has led Enterline (1976) to write: "Except for McDonald's study of asbestos miners, however, no really 'grand' studies of asbestos exposed workers have ever been undertaken in which exposure and response are related".

The remaining sections of this chapter concern the setting up of

the project with its many sub-projects, and findings are outlined and discussed in the following chapter.

4.2 Principles

The principles of this investigation, although never so far stated explicitly, can be summarized as follows:- (a) to identify each person who had ever been employed by an asbestos mining company in the Eastern Townships; (b) to obtain certain basic information about each such person - sex, date of birth and history of employment in the industry; (c) to formulate precise questions for study; (d) to select the appropriate sub-population on which each question could best be answered; (e) to collect the necessary additional information about each defined sub-population; and (f) to analyse the full data with the best available methods.

Principles (a) and (b) were attacked by getting the personnel department of each mining company to compile a register of all persons currently or previously employed, as at 1 November 1966. A "buff card" was made out for each such employee. On the face, a record was made of his or her name, date of birth and address (at the time of employment), and on the reverse a detailed work history was provided; see below. There were 44 mining companies in this enumeration, but records for several, all but one very small, had been destroyed. This meant that no buff card could be made out for 500 or more persons, and that the history was incomplete for some others, mainly older men, who had worked in one of the mines concerned but been transferred to another when it closed. For a small proportion of persons, again mainly older men, dates of birth were incomplete and in a few instances

missing.

Because of the inevitable movement of labour between companies or from asbestos work to another occupation and back, there was need to bring together all the buff cards for each individual who had more than one. In many instances, there was agreement in every particular of name and date of birth on more than one buff card, and there could be considerable confidence that the cards referred to the same person. However, in some personnel departments, names had been recorded by a clerk unfamiliar with the mother tongue of the workers concerned, often an English-speaking clerk entering the names of French Canadians, or of immigrants from central Europe; further, age at the time of first entry on the books was often recorded, rather than date of birth. Inevitably, therefore, there were cases where the probability was that more than one card referred to one person, but confidence could not be complete. In every individual case, the evidence was reviewed on at least three occasions before a final decision was made; a small investigation of the quality of matching is reported in Chapter 7. At a comparatively early stage, McDonald *et al.* (1971) reported that they had registered 27,669 men and women, of whom 6,415 were in employment on 1 November 1966. Of the total, a little over 1,000 had been employed by more than one company.

The broad aims of the study are to relate dust exposure assessments for each worker, from the start of the industry, to mortality, radiographic appearances, pulmonary function and respiratory symptoms (McDonald *et al.*, 1974). Specific aims will be introduced in relation to the findings discussed in the following chapter. However, it seems appropriate to mention here the basic selection of chest radio-

graphs for use in several sub-projects.

At both Asbestos and Thetford Mines, chest x-rays are used for two main purposes, pre-employment medical examination and annual medical examination. The procedures started in 1936 at Asbestos, but at Thetford Mines were not introduced until 1946 and were not universal until several years later. The interval between medical examinations, and hence between successive radiographs, naturally varied somewhat depending on each individual's circumstances.

Following the same principles as for the identification of all employees, it was decided to select a radiograph for everyone who had had one. The most recent, before November 1966, was identified, and those of 15,689 persons in all were collected from the two medical clinics.

4.3 Work histories

The work histories, on the reverse of the buff cards, contained a list of every job the employee had ever had in the company, with date of start, and, from the next succeeding record, date of termination. Any time employment ceased, a record of the date was provided. Where there was more than one card for any person, they were brought together and the work histories were combined; in a very few instances, discrepancies appeared between histories given by different companies, and had to be resolved.

The reliability of these histories has to be considered. There are two aspects: are the dates of starting and ending employment

correct? and are all the jobs listed for each man? The former has been checked in a current study that will not otherwise be reported here. For over 1,200 men selected for this study, the work histories were "edited" by computer program, written to my specification, and a Work History Verification form was printed out. This form listed all identifying information and also the recorded date of first employment, and by which company, dates of leaving any company, dates of rejoining or of starting employment at another company, and date of last recorded employment. Field enquiries have been made to determine whether (a) those concerned worked in the industry before the first recorded date or after that last recorded; (b) any gap in recorded history was, in fact, spent, wholly or partly, in the industry; and (c) during any recorded spell of employment the man was in fact working elsewhere. Enquiries are not yet complete, but it is known that for about two thirds of the men the enquiries were "negative", i.e. have validated the recorded work history. For a considerable further proportion, the only "positive" response was about employment after 1 November 1966, i.e. without casting doubt on the validity of the record up to that point. Most of the remaining discrepancies were to be expected: light has been cast on some doubtful cases where buff cards had been assumed to refer to the same man; some spells of long-term absence from employment, particularly in the armed services during World War II, had not been recorded at Asbestos, because such men were kept on the books until their return; the records destroyed at one mine can now be at least partially filled; etc. In general, the enquiries have indicated that the buff cards were of high reliability in regard to dates of starting and ending employment.

No means of checking the second aspect (specific jobs) has been found. Their importance is for the calculation of exposure (see below) and the clear-cut dose-response relationships that have been determined in various sub-projects suggests that this aspect, *together with* assessments of dust concentrations (also see below), has been satisfactory.

4.4 Dust measurements

The jobs entered on the reverse of the buff cards were listed, within each company, or mine within company, and a total of 13,346 names distinguished. However, in many cases, several names were used for the same work and exposure, and the number of distinct jobs was reduced to 5,783. Dust concentrations were required for each job as they may have fluctuated throughout its existence. The following account relies heavily on two reports by Gibbs and Lachance (1972; 1974).

Since 1949, Maurice Lachance, Ingénieur, had made annual measurements of dust concentrations at working places in the dryers, crushers and mills in all companies. From 1946, various investigators had made similar measurements in the small factory at Asbestos. All Mr. Lachance's measurements were made with a midget impinger, the others using a similar instrument and also several others. Records of concentrations were in millions of particles per cubic foot (mpcf). Well over 4,000 dust counts had been made by the end of 1966.

In mining and maintenance, and in certain other jobs, no dust assessments had been made by direct measurement, but dust levels were

estimated by investigating levels in the late 1960s and adjusting according to changes in operating procedures which had been reported by mining and maintenance personnel. Before 1949, few dust measurements had been recorded, and corresponding estimates were made, usually after interviews with employees of long service and in consultation with foremen, etc. In this way comparisons could be made with more recent conditions. All these estimates are necessarily approximate, but there was substantial agreement among those questioned. Nevertheless, for one reason or another, there is at least the possibility of some degree of overestimation (Gibbs and Lachance, 1972, p. 195).

The method of measurement used by Mr. Lachance was essentially that later approved by the Asbestos Textile Institute (1963). The count, sometimes referred to as "total dust" included both fibres and particles, and was carried out at a hundred-fold magnification. The dust counted was mostly below 7 μm in diameter (and so far from "total"); as fibre bundles exceeding 3 μm in width were rare, most of the dust counted was probably respirable.

Great variation was found between work places in dust levels, which were particularly high in certain areas of drying, milling, maintenance and waste disposal, and comparatively low in mining; differences were also found between mines, not only in different companies but also between those owned by the same organisation. Over the years 1949-66, when measurements had been taken, there was steady improvement in dust levels: the mean annual concentration in the mills fell from around 75 mpcf to about one-tenth that level. The mill at

Asbestos had the lowest concentrations throughout this period, reducing from about 30 mpcf in 1949 to a low level in 1956, which was fairly consistently maintained thereafter. In 1949-51, the mill with the worst experience each year had annual average concentrations above 200 mpcf, but in 1966, the average at the worst mill was below 15 mpcf.

Dust counts would probably have been substantially higher in the years before measurements were instituted. Thus they appear rather worse than in other historical studies (Knox *et al.*, 1968; Enterline *et al.*, 1972b) reported in Chapter 2.

Conversion to fibre counts would be desirable but has proved of doubtful value. Side-by-side sampling, using the midget impinger and a membrane filter device, has shown a poor degree of correlation between the two counts and shed little light on a means of conversion. The Quebec government's Comité d'étude, set up under Judge René Beaudry to study the health of asbestos workers in the Province, analysed up-to-date data supplied by Dr. G. W. Gibbs, and suggested that the relation: $(\text{fibres/ml}) = 10.97(\text{mpcf})^{0.68}$ would allow conversion (Dagbert, 1976). However, there was enormous variation in the ratio of the two counts: low counts on one instrument were, of course, often associated with low or medium counts on the other, but also occasionally with high values. Some extremes, estimated by eye from the figure in Gibbs and Lachance (1974), are:-

Midget impinger count (mpcf)	33	23	13	2
Membrane filter count (fibres/ml)	5	58	135	98
Ratio	0.2	2.5	10.4	49.0

This hundred-fold variation in ratios is mirrored in the very wide 95 per cent confidence limits provided by Dagbert on his conversion formula, i.e. $55.7(\text{mpcf})^{0.68}$ and $0.58(\text{mpcf})^{0.68}$; their ratio is $(55.7)/(0.58) = 96.0$

Over a realistic range of particle counts, i.e. at high levels, Dagbert's formula would suggest converting on the basis of five or so fibres/ml to unit mpcf, but Gibbs and DuToit (1978) stated that Dagbert had not adequately edited the data provided by Dr. Gibbs, and that the factor to convert historical particle counts should lie between one and five fibres/ml to unit mpcf. Converting on the basis of three fibres/ml to unit mpcf, the 1949 annual average concentration in the mills of Quebec, measured as 75 mpcf, may have been roughly equivalent to about 225 fibres/ml. Clearly, no great reliance can be placed on this figure, but there can be little doubt that dust levels were very much higher, by two orders of magnitude, than any modern "Threshold Limit Value" such as 5 or 2 or particularly 0.5 fibres/ml. Even in 1966, when the annual average concentration had fallen to around 7 mpcf, the fibre "equivalent" would appear to have been of the order of 20 fibres/ml, still unconscionably higher than today's standards.

4.5 Exposure calculations

When the National Coal Board's Pneumoconiosis Field Research (PFR) was set up in 1952 (Fay and Rae, 1959), a major concern was the calculation of indices of exposure to coalmine dust of each of the 35,000 miners under study. In the event, no attempt was made to assess historical exposures by more than a statement of the years worked at the coalface, elsewhere underground and on the surface, either at the

colliery where the man was questioned during a radiological survey or at any other mine. However, for the periods between surveys, detailed records were maintained of each man's work, job by job, day by day, taking account of overtime - which might be worked in another job - and absence. Dust measurements were made for each job and, thereafter, the length of time spent in each job was multiplied by the dust concentration there in the relevant period, and the cross-products were summed to obtain a single measure of exposure over the inter-survey period (usually about five years). The method of maintaining current histories was virtually identical to that introduced earlier for another purpose by Liddell and Finch (1958).

The Quebec studies required indices assessed over much longer periods, dating back to 1878. The method of obtaining them was similar to that of PFR, just described, taking the work histories on the buff cards as base; dates had been entered in terms of month and year only. However, the lack of the daily detail that had consumed such an enormous data processing commitment in PFR was much more than compensated for in Quebec by the vitally important estimates of early exposure.

For each of the 5,783 jobs, defined as above, and for each year of operation, an average dust concentration was estimated from the available dust measurements and approximations. The concentration was recorded on a 13-point scale, corresponding to:- 0.5, 2, 7, 12, 17, 22, 27, 32, 37, 42, 47, 70, and 140 mpcf. *Accumulated dust exposure*, or dust index, was originally calculated, as mpcf x years (or mpcf.y), by an adaptation of the method described above. An example

will illustrate the process:- consider a man who started in the industry during February 1965 in job 1 (dust code 3 in that year), moved to job 2 in September (code 5 in 1965, code 4 in 1966) and left in February 1966; the calculations are as follows.

Job	Year	Duration	Fraction of year	Concentration *	Cross-product
1	1965	Feb–Sep	7/12 = 0.58	(3) 7	7 x 0.58 = 4.06
2	1965	Sep–Dec	3½/12 = 0.29	(5) 17	17 x 0.29 = 4.93
2	1966	Jan–Feb	1½/12 = 0.13	(4) 12	12 x 0.13 = 1.56
				Accumulated dust exposure	=10.55

* The figures are (code) and corresponding concentration (mpcf).

For more recent studies, many of the exposure indices have been recalculated on an annual basis, and the proportion of each year worked has been recorded, a double-field being included on the computer record separately for each of the 63 years from 1904 (when the first employment of any of those involved in these particular studies started) through 1966. In the example above, the first 61 fields would be blank, and the entries for 1965 and 1966 would be:- 8.99 and 0.87; 1.56 and 0.13; respectively.

Several other indices of exposure were included in early studies. They were: accumulated dust exposure weighted for physical effort; accumulated dust exposure weighted for physical application; time-weighted dust exposure. This last is highly correlated with the first described (accumulated dust exposure, unweighted), and so little additional information can be gained from it. The others must be expected to have even higher correlations with the first, and have not in the event been found useful.

A fifth indication of exposure, useful because it does not take dust concentration into account, and so allows some form of comparison with other studies, is *years of exposure*. This can be expressed on a gross basis, counting from first employment to final departure from the industry, or on a net basis, omitting gaps in service from the index. These two measures have recently been termed gross and net service, respectively.

A correction to the accumulated dust exposure took account of variations in the length of the working week, which was generally 66 hours before World War II, but has been 40 hours in recent years. If the accumulated dust exposure is divided by length of service, the quotient will be only a rough indicator of average concentration, if the periods over which the averaging is done embrace changes in the length of the working week. To find true average concentrations, it would be necessary to re-calculate service, adjusting in such a way that the unit becomes a working year based on 40 hours a week, before finding the quotient. This would, however, mean that the working year could be as "long" as 66/40 = 1.65 years on the present basis.

Despite the shortcomings discussed above, the estimated exposures are of very great value. While it may be impossible to place each individual subject precisely on a continuum of dust exposure, the great width of the continuum means that it is possible to distinguish workers who have had varying degrees of exposure. For example, a ten-point scale of dust exposure accumulated by age 45 has been found useful in the most recent study of mortality (McDonald *et al.*, 1979); the boundaries between the classes were drawn at 1, 3, 10, 30, 60,

100, 200, 300 and 600 mpcf.y, and of 10,939 men so grouped the class size was never less than 573, nor more than 1,607. While one cannot be completely confident that every individual has been placed into the correct class, employees with short service (less than one year, say) were most unlikely to be correctly in any but the lowest classes of dust exposure, while those with long service (twenty years or more, say) could not be at the lower end of the scale of exposure. This is illustrated by the two-way distributions in Tables 9.2 and 9.13, below. Indeed, because the accumulated exposure for a man with long service, probably having had many jobs, is a sum of more-or-less independent terms, the coefficient of variation of his estimated exposure is certainly small, by virtue of the Central Limit Theorem, compared with the variation of exposures between subjects. There may remain certain biases, but they can have little effect on any internal comparisons; see the discussion by Fay and Ashford (1964) on corresponding problems in British coal mines.

4.6 The "main mortality cohort"

Because excess mortality is undoubtedly the most important health effect of asbestos exposure, the mortality study will be outlined first. It was based on employees thought likely to yield the most valuable information, i.e. those born in the years 1891 through 1920, who had been employed for one calendar month or more (McDonald *et al.*, 1971). Those born before 1891 would have been even more difficult to trace; those born after 1920 would, even by 1978, be less than 60 years old and would not have completed their working life-times, still not having reached an age of high mortality. The cohort defined in these ways is here called the "main mortality cohort".

A substantial proportion of this cohort had had less than a year's service, and many of these together with some others of longer service had not been exposed to appreciable quantities of dust. In their first report on mortality, McDonald *et al.* (1971) stated that the selected cohort comprised 11,788 persons who met the criteria.

Attempts were made, under the supervision of Professor Alison D. McDonald, to trace every person in the cohort, to establish whether he or she was alive or dead, and if dead to seek details of exact date and place of death, so that a copy of the death certificate could be obtained. The search for ex-employees was initiated in the mining towns, through relatives and friends, and was extended to parish registers, provincial death records, the Canadian Unemployment Insurance Commission (UIC) and eventually to many other areas and organisations. Many deaths were reported as having occurred in other provinces, in the United States, and overseas. Careful examination was made of the field information, and many additional enquiries were undertaken to ensure that the tracing was reliable. In particular, it was sometimes found necessary to alter an earlier decision, previously made in terms of information on the buff cards only, as to whether these cards referred to one or more persons. The quality of tracing is discussed briefly in Chapter 7.

Tracing was carried out to four points in time: 1 November 1966 (McDonald *et al.*, 1971); 31 December 1969 (McDonald *et al.*, 1973; 1974); 31 December 1973 (Liddell, McDonald and Thomas, 1977); and 31 December 1975 (McDonald *et al.*, 1979). The numbers reported were as follows:-

	Total cohort	Traced	Traced as per cent of total	Reported dead	Deaths as per cent of traced
1966	11,788	10,421 *	*88.4*	2,457	*23.6*
1969	11,572	10,120	*87.5*	3,270	*33.7*
1973	11,391	10,245	*89.9*	4,110	*40.1*
1975	11,379	10,257	*90.1*	4,547	*44.3*

* including 598 registered with the UIC between 1964 and 1 November 1966, and presumed alive.

The "loss" of 400 persons (3.5 per cent) from the originally defined cohort was for several reasons: buff cards thought to refer to different persons were in fact found to concern only one, in more instances than the reverse occurred; and stricter application of the criteria for selection revealed some originally included who should not have been. More stringent definitions for acceptability of tracing reduced the number reported as traced at end-1969 compared with three years earlier, but unceasing efforts have now improved the trace rate to over 90 per cent; see also below.

Cause of death, from the death certificate in the vast majority of cases, was coded according to the Seventh Revision of the International Classification of Diseases by a senior coder recently retired from the Department of Demography of Quebec. Some death certificates could not be obtained, because the death had occurred before registration was introduced in Quebec in 1926 or because it had taken place outside Quebec in regions where authorities would not provide copies. In some of these cases an acceptable cause of death was elicited from relatives.

A smoking history was sought from those alive and from relatives and friends of those who died after 1950. The information was coded in

four categories:- number of cigarettes smoked a day, age when started smoking, and if appropriate when quit smoking, and whether a pipe smoker or not. Unfortunately, the coding was found to have been carried out inexpertly, and care is needed in interpretation; see Liddell, McDonald and Thomas (1977, p. 477). In preliminary work for this last cited paper, I studied the records to the end of 1973, for males, and found that 84.2 per cent of losses, i.e. those not traced, were before 1936. Of 9,723 men known to be alive in that year, 5,797 were traced alive, 3,750 were known to have died, and only 176 were untraced (59.6 per cent, 38.6 per cent and 1.8 per cent, respectively). Of the 8,672 known to be alive in 1956, smoking questionnaires had been completed for 5,771 still alive in 1974, for 2,600 who died 1956 through 1973 and four who were alive in 1965 but were subsequently lost to view, i.e. for a total of 8,375 (or 96.6 per cent). Another way of expressing these findings is that for the 9,834 men traced to the end of 1973, smoking history was not known for 1,090, all but 26 of whom had died, mainly before 1950 so that smoking histories had not been sought.

4.7 The "physiology cross-section"

This name is given to an age-stratified sample of current male employees who were examined, in 1967 and 1968, using (a) a respiratory symptom (and smoking) questionnaire, (b) lung function measurements, and (c) the chest radiograph. The study was carried out primarily to determine the relationships between exposure to dust on the one hand and the various signs and symptoms that could be assessed by the tools listed above on the other hand. Because there were so few women in the industry, most of whom had been employed in low dust

concentrations, the study was confined to men; it was decided to include higher than representative proportions of older men.

From the register of persons ever employed in the industry up to 1 November 1966, it was determined that there were 6,415 current employees, of whom 6,180 were males. The 102 who were less than 21 at the time to which the register referred and the 37 who were 65 or more were excluded. A random sample was taken from each remaining five-year age group to yield a total of 1,027; the sampling fraction varied from 14 per cent in those of ages 21 through 25, to 47 per cent of those 61 through 65. The examinations took place in the summer of 1967, but only 885 men were seen and tested: 85 had died or left and 57 refused to participate. These losses were mainly among older men, and in the following summer all 241 men still at work and aged 61 through 65 but not already examined were called; a further 185 sets of tests were carried out.

A total of 1,069 men were tested, and included in a study of lung function in relation to radiographic changes (Becklake *et al.*, 1970). However, a serious deficiency in the questionnaire or the records of the lung function tests, or in the work history, led to the exclusion of 54 men, so that studies of respiratory symptoms and lung function in relation to dust exposure were confined to 1,015 men (McDonald, Becklake *et al.*, 1972; Becklake *et al.*, 1972).

The British Medical Research Council Questionnaire on Respiratory Symptoms (1966) was used with slight modifications and after translation into French, so that each man could be intereviewed in the lan-

guage of his choice. One fluently bilingual interviewer administered the questionnaire to all but 16 men (among the 54 excluded as noted above). Smoking history was recorded in the usual way during these interviews, and ages were verified; many required correction compared with the information on the buff cards.

In each of the two mining towns, a field laboratory was set up and the two technicians each tested eight men a day. Becklake *et al.* (1972) reported as follows: "The following tests of lung function were carried out in the order listed, using methods described fully elsewhere: (1) carboxyhemoglobin level measured by an oxygen rebreathing technique; (2) functional residual capacity (FRC) using a helium dilution technique; (3) vital capacity (VC) followed by three tests of forced vital capacity (FVC), the largest of which was also analyzed for the forced expiratory volume expired in 0.75 second ($FEV_{0.75}$) and in 1.0 second ($FEV_{1.0}$), for the $FEV_{1.0}$/FVC ratio expressed as a percent, and for the maximal midexpiratory flow (MMF); (4) diffusing capacity for carbon monoxide at rest measured by the single breath method ($D_{CO}[SB]$) as the product of Krogh's diffusion constant (K) and alveolar volume (TLC[SB]) calculated from helium dilution by the single breath; and (5) diffusing capacity for CO ($D_{CO}[SS]$) and the CO extraction rate (%) measured by the steady-state method at rest and at two levels of exercise (200 and 400 kg.meters/min) on a bicycle ergometer. Minute ventilation ($\dot{V}_1$) and oxygen consumption ($\dot{V}_{O_2}$) were also measured at the same time."

4.8 Procedures for reading 15,689 radiographs

As previously mentioned, the most recent radiographs of 15,689

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4.8 Procedures for reading 15,689 radiographs

As previously mentioned, the most recent radiographs of 15,689

persons had been collected (Rossiter *et al.*, 1972). They were to be used for several purposes, *viz:* (a) an examination of the U/C classification; (b) for relating to respiratory symptoms and lung function in the physiology cross-section; and (c) for relating to dust exposure measures. Six readers, all of whom had helped in the development of the U/C classification, participated in the reading which took place in three stages. First, a random sample of 600 films was read once each by all six readers, working independently, to allow an examination of inter-observer variation; so also were the films of the subjects in the physiology cross-section, to provide the most reliable basis for relating radiological findings to function measurements. All the films were then reassembled into thirty batches, by a quasi-random procedure of shuffling, and each reader read five batches. This second stage permitted some study of intra-observer variation, for each reader had now read a considerable number of films on two occasions. Finally, a complex procedure was adopted whereby all films which at the second stage had been recorded as in any way abnormal (by a single reader) were read by three others, and a sample of those assessed as normal at stage two were read by at least one other reader. The total number of assessments (excluding those on films considered unreadable) was 42,022; we have recently found that there were 1,868 sets of six readings, one per reader, from the first stage (a total of 11,208 assessments), 15,570 readings at the second stage (almost equal numbers by each reader, varying only between 2,553 and 2,611), and a further 15,244 at the third stage.

As a preliminary to the study of the relationships between radiographic findings and indices of dust exposure, the following films

were excluded: those of students employed in the summer only; those of applicants examined but not employed; those for men whose personnel records had been destroyed; a few for subjects with otherwise incomplete work histories; and a few films of very poor quality. This left 13,021 (or 83 per cent), of whom 12,174 were of males. Of these, 967 were listed as past or present employees of the small factory at Asbestos, leaving 11,207 production workers. In the report by Rossiter *et al.* (1972), relationships between radiological changes and indices of exposure were examined in the males aged 36 through 65 at the time of last recorded job in the industry (to November 1966); there were 6,129 production workers and 398 factory workers.

4.9 The "radiographic progression cohort"

In 1975, Eyssen reported on a study which had had the objectives: (1) to determine the most suitable method of assessing radiographic progression in the individual to allow the changes to be related to measures of his exposure; (2) to make a first examination of the relationship between radiographic progression and asbestos exposure; and (3) to describe changes over the years in the prevalence of radiographic change in the Quebec chrysotile producing industry. Eyssen stratified the 12,174 male subjects defined by Rossiter *et al.* (1972) - see previous section - by place of work (Asbestos or Thetford Mines), length of employment and whether they showed evidence on the latest film to 1966 of radiographic abnormality or not. Random samples were drawn from each stratum, with high sampling fractions for strata containing abnormal films and low fractions for strata with only normal films. In this way, 283 men were selected; for each man five radiographs were collected, including the latest available at that time,

i.e. up to 1971, and four previous films at roughly five year intervals spanning about 20 years.

Two methods of assessment were adopted by five readers, all of whom had been concerned in the main reading described in the last section. One method was side-by-side reading of the films in known temporal order, using the ILO U/C (1971) classification with further fourfold elaboration of the scales of (i) profusion of small opacities, both rounded and irregular; (ii) both width and extent of pleural thickening; and (iii) ill-defined cardiac outline. The other method was independent reading of all the films in randomized sequence into the (unmodified) complete ILO U/C (1971) classification (ILO, 1972).

This work was carried out before the mortality analysis to the end of 1973, and so the revised dust exposure calculations on an annual basis were not available even for those men in the present study who were in the "main mortality cohort". Calculations were carried out *ad hoc* of exposures in the quinquennia up to the earliest film for each man included in the study, and for the three periods between that film and the next three succeeding films included. Smoking histories were available for the men also in the "main mortality cohort" but not for the others. The two medical clinics, at Asbestos and Thetford Mines, provided what information they could from the subjects' medical histories on the smoking habits of these men, and so it was possible to classify all but three subjects as smoker or not; the three with unknown history were treated as smokers as the more likely habit.

For a more detailed examination of radiographic changes in relation to dust exposure, and other factors (Liddell, Eyssen *et al.*, 1977), closer study of the work histories revealed discrepancies, or employment outside the Quebec asbestos industry in work that involved exposure to asbestos or other dusts, in twelve men; these and four for whom there were important gaps in the radiological findings were excluded from the analyses, which were based on the side-by-side readings of four readers only, because one had not completed assessments on all films.

4.10 Summary

In 1967, Professor McDonald's team registered all workers, nearly thirty thousand, who had been employed in the Quebec chrysotile producing industry some time since its inception, in 1878, and November 1966. For various studies, the following populations were defined:-

(a) The "main mortality cohort" of those born in the years 1891 through 1920 who had been employed for one calendar month or more. Improving information over the years and stricter application of the criteria for inclusion in the cohort have reduced the numbers slightly from those first reported, and there are now 11,379 persons in the cohort, including 440 females (McDonald and Liddell, 1978).

(b) The "physiology cross-section", a sample of males at work in November 1966, weighted heavily towards the older age groups. A total of 1,069 men were interviewed, but data from respiratory symptoms questionnaires and lung function tests were complete for only 1,015 (McDonald, Becklake *et al.*, 1972;

Becklake *et al.*, 1972).

(c) The "radiographic progression cohort", a sample of 283 men for whom five chest radiographs were available spanning an average of twenty years. Because of some doubts about work histories, including exposure to possible airborne health hazards outside the Quebec industry, the cohort was reduced to 267 for further analysis (Eyssen, 1975; Liddell, Eyssen *et al.*, 1977).

(d) All persons, totalling 15,689, who had had a routine chest radiograph at one of the medical clinics in the Eastern Townships before November 1966. Exclusion of students employed in the summer only, applicants for work not taken on, some for whom personnel records had been destroyed and a few whose films were of very poor quality reduced the effective study population group to 13,021. However, the latest film for all 15,689 was collected and assessed.

As the main aims of the investigation were to relate dust exposure assessments for each worker to mortality and other biological responses to asbestos exposure, calculations were made of accumulated dust exposures, through work histories and estimates of dust concentrations, for all persons included in the various facets of the enquiries (Gibbs and Lachance, 1972; Liddell, McDonald and Thomas, 1977). Other indices of exposure were also obtained for certain specific purposes.

Chapter 5

QUEBEC CHRYSOTILE STUDIES II: FINDINGS

5.1 Introduction

In this chapter, I review the main findings from the studies by Professor J. C. McDonald and his colleagues of the health effects of asbestos exposure in the miners and millers of Quebec chrysotile. Sections 5.2 and 5.3 deal with radiological changes, respiratory symptoms and lung function, each in relation to asbestos dust exposure, and the next section with the interrelationships of signs and symptoms. Sections 5.5 and 5.6 concern mortality in relation first to dust exposure and then to radiographic changes. A brief summary follows, and in Chapter 6, the findings from other studies, both in chrysotile and in relation to other fibres, are discussed.

5.2 Radiological changes in relation to dust exposure

Of the two earliest reports relating radiographic appearances to exposure to asbestos dust (Rossiter *et al.*, 1971; 1972), the first was of a preliminary nature, concerning only 809 employees of one company at Thetford Mines. The authors' own summary (Rossiter *et al.*, 1971, p. 206) states that: "Small rounded opacities were related to total dust, but not to the other two indices, and ill-defined cardiac outline to total dust weighted by physical effort. Pleural thickening

and pleural calcification were related to age, but not to any dust index ... The distribution of pleural plaques in the Quebec asbestos mining areas was not uniform, the greatest concentration being in those who have worked in or near this mine." Their conclusion that their findings justified the use of the U/C classification could be accepted, but the results themselves needed generalization.

The main study (Rossiter *et al.*, 1972; also summarized by McDonald *et al.*, 1974) provided that generalization. The main concern was with all male production workers aged 36 through 65 who had ever had a chest radiograph; they numbered over six thousand. A further 398 male factory workers in the same age range were also considered. In each case, the radiographs examined were the latest available to 1966, and the workers' ages used in these reports were at 1 November 1966 for current employees, and at the end of employment for ex-employees. As radiographs were supposed to be taken annually, it might be expected that the age at x-ray was on average six months less than the reported age; it seems likely, however, that the average interval between x-rays was greater than a year, because of missed appointments and similar reasons, and the age at x-ray may have been on average rather more than six months younger than reported. However, the bias thus introduced into an individual's age is likely to balance out in the cross-sectional study reported here.

The men were classified by mining area, Asbestos or Thetford Mines, by five-year age-groups and separately by the five dust exposure indices outlined in section 4.5 above.

The procedures for reading the radiographs have also been described above; see section 4.8. Although it would have been simplest to derive an assessment for each film by averaging the category for each feature for the readers involved, up to six, it was thought that more information would be obtained by taking account of the distributions of categories for the individual readers in the random series of 600 films all had assessed, and that this would allow for inter-observer variability. To this end, radiological indices were calculated on scales whereon, for each factor, the change from the lower to the upper end of category (or grade) 1 was taken as the unit; these ends of the category were labelled 1 and 2 respectively. The distribution of categories for individual readers in the series of 600 films they had all read were used to "adjust" for differences between readers in their levels of reading. Thereafter, the scores were averaged for use directly in regression analyses; for prevalence statements however, the average scores were "discretized" and also often standardized for factors of less than immediate concern. Further, this scoring process was only possible for the graded features of the U/C classification; other features were taken as present if at least half the readers who had assessed a particular film thought so. Despite the unequal number of readers to a film, the authors considered that the reading pattern would have led to only small biases (Rossiter *et al.*, 1972); however, without full details of the adjustment procedure, it is not possible to examine its justification. At the worst, however, it could have affected only slightly the general patterns revealed in the analysis.

The main findings of this study illustrated in Figure 5.1 (taken from Rossiter *et al.*, 1972), can best be quoted from McDonald *et al.*

Figure 5.1

RADIOLOGICAL SCORES

Variation in the average score from cohort to cohort, with and without standardizing for exposure differences.

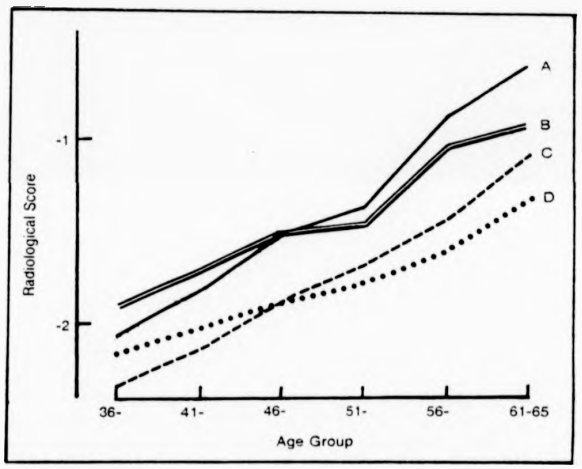
A Thetford Mines

B Thetford Mines, standardized for exposure

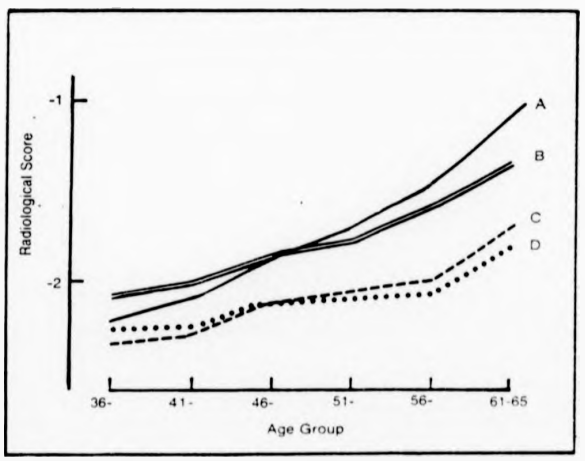
C Asbestos

D Asbestos, standardized for exposure

Irregular small opacities



Pleural thickening



(1974), *viz:* "The two main roentgenographic indices of response to asbestos exposure are irregular small opacities and pleural thickening. The prevalence of these, and of all other roentgenographic changes, were higher in the Thetford Mines area than in the Asbestos area of Quebec, although the two areas are only 80 km apart and the asbestos mined is geologically similar. The association of the changes with dust exposure was also stronger at Thetford Mines than at Asbestos. ... At Thetford Mines the prevalence of irregular small opacities of category 1 or more rose steadily with increasing dust exposure, whereas for category 2 or more the relation to dust exposure was less marked, except for the considerable rise in the most exposed workers. Even with allowance for differences in dust exposure, the prevalence of category 1 or more was markedly age-related, rising to 14.8% for those 61 to 65 years old. This age effect was much lower for category 2 or more, and amounted to only about one third of the change associated with dust exposure. At Asbestos, the prevalence of irregular small opacities of category 1 or more rose to 9.2% for those aged 61 to 65, and there was virtually no relation to dust exposure. With respect to only category 2 or more, the relation to age was much lower and prevalence increased among those with the highest dust exposure. For pleural thickening, the effect of age was slightly greater than the effect of exposure on the prevalence of grade 1 or more, and of grade 2 or more, for both mining areas.

"The biggest difference among all the roentgenographic features was in pleural calcification, for which the prevalence of grade 1 or more was 0.4% at Asbestos and 5.2% at Thetford Mines. In spite of the relationship to dust exposure, no correlation exceeded 0.3. This

is largely a reflection of the very high proportion of men who showed no roentgenographic change. Some of the differences between the two areas were not surprising, as overall dust exposure levels at Asbestos were considerably lower than at Thetford Mines, but others cannot be explained in terms of dust exposure or geology. If variation in fibre content of dust were an important factor, the results in mine workers, for whom the fibre proportion would be low, should differ from those for mill workers, in whom the fibre proportion would be higher. Altogether, there were 506 persons who had worked for ten or more years entirely in mining or entirely in milling. Analysis of roentgenographic changes in these men showed that mill workers had slightly higher prevalence of irregular small opacities, but the differences between Asbestos and Thetford Mines remained. No other consistent differences could be detected between the mine and mill workers.

Two further reports on radiological changes in relation to dust exposure were based on the "radiological progression cohort"; see section 4.9. The first of these (Eyssen, 1975) was concerned mainly with a choice between methods of assessing radiographic change in serial films. Correlations between measures of exposure and of radiographic progression had the first objective of determining whether either side-by-side reading of all five films in known temporal sequence into fine scales of profusion produced consistently higher correlations than independent randomized reading into the ILO U/C (1971) classification, without amendment; only a secondary objective was to elucidate any readily available epidemiological findings (Eyssen, 1975, p. 102). In the event, the correlations between radiographic progression, however assessed, and levels of dust exposure whether before the first

film or in the intervals between films were not substantial.

The other study (Liddell, Eyssen *et al.*, 1977) of the radiological progression cohort related to 267 men, excluding several from the complete cohort who had had exposure in the intervals between films or whose work histories were in considerable doubt. The analysis took place in three phases. Phase 1 was a "cross-sectional" study of all 267 men, to relate the x-ray status of the earliest selected film to potential stimuli. The men were then classified into two cohorts. The first cohort consisted of 150 men whose earliest film in the study contained no evidence of abnormality according to all four readers using both side-by-side and independent reading; Phase 2 was a study of "attacks" in this cohort. The remaining men formed the second cohort, and Phase 3 was a study of the progression of radiographic changes in these men from their first film which could be treated as "less-than-normal" over the rest of the period, averaging twenty years in total, covered by the available x-rays.

Phase 1 showed that abnormality in the earliest film was related mainly to the time since first employment, in close conformity with the findings earlier of Rossiter *et al.* (1972). Phase 2 revealed that, in 45 of the 150 men completely normal at first film, some radiological abnormality developed during the period of observation: incidence was weakly related to age; at Asbestos, the risk of developing small opacities was lower in smokers than in non-smokers. In Phase 3, there was an irregular pattern of correlation, more evident in Thetford Mines than in Asbestos, between radiological progression and various stimulus variables.

Liddell, Eyssen *et al.* (1977) concluded that associations between radiological responses and measures of asbestos exposure were, as always, weak, despite powerful methodology. They felt this indicated that effects were obscured by other factors (including susceptibility) uncorrelated with exposure. Several possibilities required consideration. First, the variables - response, stimulus or disturbing - chosen for analysis may not have been the most appropriate; or they may have been inaccurate. The x-ray shadows are only indirect signs of disease, and their assessment is seriously hampered by observer variation (despite attempts to minimise it by taking all four readings into account) and by the inevitable changes of radiographic technique over such a long period of study (again perhaps better allowed for in side-by-side reading than when films were read independently). The dust measurements were, of course, at best with imperfect instruments, sometimes only educated guesses, and not necessarily bearing much relationship to dust retention in the lung. Secondly, if asbestosis is progressive, radiographic change will be observed in men with long service, irrespective of recent exposure. Thirdly, there may well be differences in factors of social, psychological, behavioural, physiological or immunological nature that modify the amounts of dust inhaled, the proportion and constitution of what is retained, and the nature of the pathological response. In this study, 80 per cent of the men exposed for over 30 years, often to heavy concentrations, remained completely unaffected as far as their chest x-rays were concerned, whereas others were attacked after quite short periods of employment in the more recent eras when dust concentrations were comparatively low.

5.3 Respiratory symptoms and lung function in relation to dust exposure

As explained in the previous chapter, an age-stratified sample of 1,015 current male employees was examined, mainly in 1967, to explore the relationships between respiratory symptoms and lung function, on the one hand, and, on the other, dust exposure, taking age and smoking habit into account. There are two reports, which deal with symptoms (McDonald, Becklake *et al.*, 1972) and function tests (Becklake *et al.*, 1972) separately.

The symptoms studied were: winter cough; winter cough for three months of the year; phlegm in winter; phlegm in winter for three months of the year; cough and phlegm for three months of the year; wheezing most days; nasal catarrh for three months of the year; breathlessness compared with others of the same age; and disabling chest illness in the last three years. As far as possible, terms were used in the same way as others had used them, to allow comparison between studies. Smokers were classified in two ways, by current habits and in terms of cumulative numbers of cigarettes (or equivalent) smoked. The index of exposure to asbestos was the accumulated dust exposure.

The distributions of cough, phlegm and wheezing all followed a similar pattern: prevalence increased with age, and rates were much higher for smokers than for non-smokers or ex-smokers. However, they did not appear to be associated with dust exposure, except perhaps in men over 50. On the other hand, breathlessness, although increasing with age, was not clearly related to smoking but showed a definite, positive, association with dust exposure; this finding was not sensi-

tive to the degree of breathlessness being considered. Disabling chest illnesses were associated with age only, and nasal catarrh showed no important or consistent trends.

Attempts to disentangle the effects of the three factors (dust, smoking and age) were not entirely successful. However, the authors concluded that the prevalence of persistent cough and phlegm (bronchitis) was related to age and smoking habits and *perhaps* (my emphasis), in non-smokers and light smokers, also to dust exposure. Breathlessness on excercise was related to age and dust exposure but not to smoking. The age-corrected prevalences of breathlessness given by McDonald, Becklake *et al.* (1972, Table 4) have been further adjusted for smoking habit by the "equivalent average" method (Yule, 1934) in Table 5.1.

For the examination of lung function tests, the men were divided into only two groups according to their smoking habits, non-smokers being those who had never smoked even one cigarette a day for as long as a year. Thereafter, each group was divided into six classes according to accumulated dust exposure, and mean values of the various test measurements were calculated for each class, after "standardization" for age, height and weight, by means of a series of regression analyses. Many of the function measurements were associated with dust exposure; see Figure 5.2. The following paragraph is quoted from Becklake *et al.* (1972).

"*Lung volumes.* - In both non-smokers and smokers the standardized mean value for total lung capacity (TLC) fell with increasing dust ex-

Table 5.1

PREVALENCE OF BREATHLESSNESS
BY DUST EXPOSURE

1,015 men in the "physiology cross-section"

(Age-corrected prevalence percentages,
further adjusted for smoking habit)

Dust exposure accumulated to November 1966 (mpcf.y)	Prevalence of breathlessness (%)
Less than 10	5.0
10, less than 100	16.4
100, less than 200	23.8
200, less than 400	24.4
400, less than 800	28.8
800 or greater	38.0

Figure 5.2

LUNG FUNCTION, SMOKING AND DUST EXPOSURE

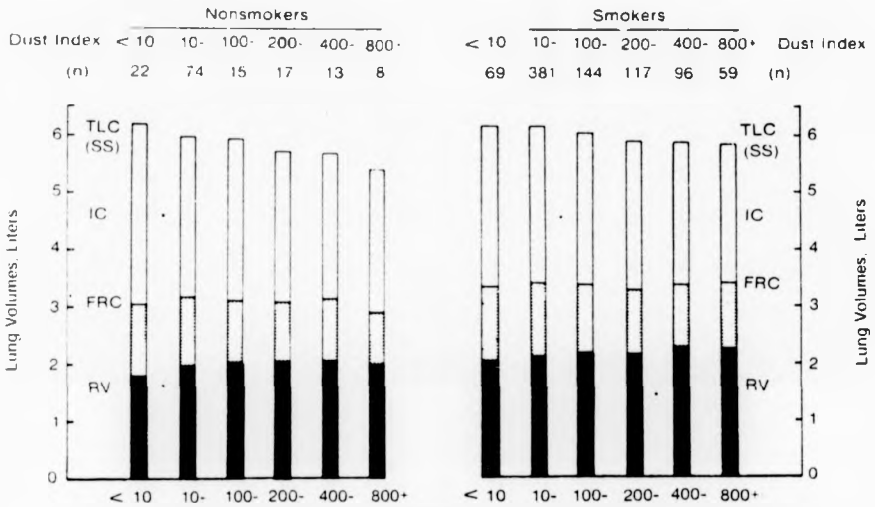
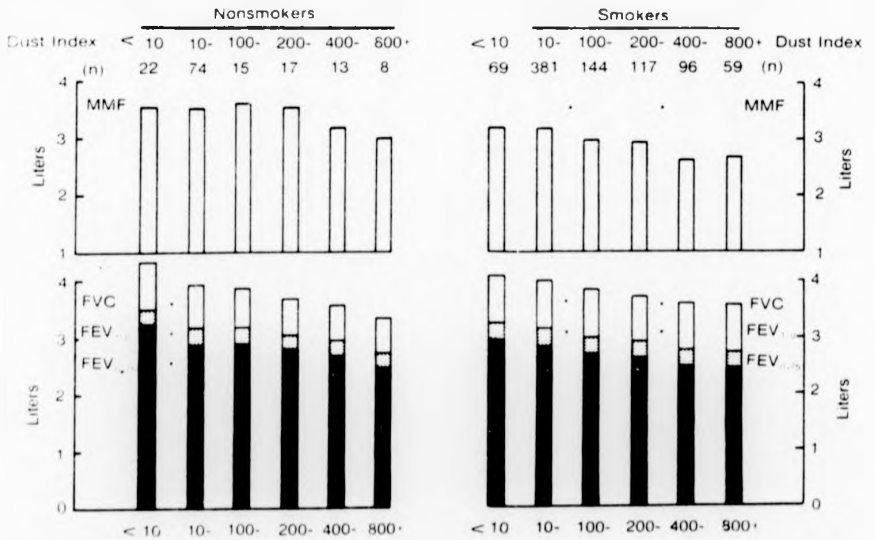


Fig 1.—Standardized mean values for subdivisions of lung volume (TLC[SS], IC, FRC, and RV) in nonsmokers and smokers divided by dust index. Number (n) of individuals in each subgroup is indicated.

Fig 2.—Standardized mean values for flow rates (MMF, $FEV_{.75}$, $FEV_{1.0}$ and FVC) in nonsmokers and smokers divided by dust index. Number (n) of individuals in each subgroup is indicated.



posure, due primarily to reduction in the inspiratory capacity (IC = TLC - FRC). Since IC is one of the components of VC, a fall in relation to increasing dust exposure was also seen in the FVC. In non-smokers the other subdivisions of lung volume, in particular FRC and residual volume (RV), did not show any evident trend with increasing dust exposure. In smokers, however, there was a progressive increase in RV. *Flow rates.* - Both in non-smokers and smokers, there was a progressive fall in FEV with time ... These decreases were probably attributable in part to diminished VC (or FVC) with increasing dust exposure. In contrast, MMF and $FEV_{1.0}$/FVC were reduced only in the higher dust index groups in non-smokers. Results of both tests were lower on average in smokers than non-smokers but were only reduced substantially in men with high dust exposure. *Diffusing capacity.* - Neither of the standardized measurements of diffusing capacity at rest ... showed any consistent trend in relation to increasing dust exposure in non-smokers or smokers. There was a fall in diffusing capacity on exercise ... with increasing dust exposure in the non-smokers, though because of the small numbers in five of the six non-smoker dust-exposure subgroups, this conclusion is tentative. However, the highest standardized values for D_{CO}(SS) both at 200 kg.meters/min and at 400 kg.meters/min were seen in the non-smokers with insignificant dust exposure [less than 10; and 10, but less than 100 mpcf.y]. Among the smokers, there was a fall in diffusing capacity on exercise ... but much less than that seen in non-smokers."

An important objective of the complete research programme was reported by Becklake *et al.* (1972) to be to determine methods of surveillance of workers exposed to asbestos but as yet unaffected accord-

ing to any clinical test. The authors gave their opinion that IC, FVC and FEV, which were sensitive to even low levels of exposure in non-smokers, and related to dust exposure over the full range in both non-smokers and smokers, would be suitable for health surveillance. However, the effects of dust exposure in smokers, although doubtless of statistical significance, were no larger than the standard errors of the variation about the regression lines, and even in non-smokers were not more than about half as large again as the corresponding standard errors. Thus, the mean standardized ICs for non-smokers exposed to less than 10 and more than 800 mpcf.y were 3.12 litres and 2.47 litres respectively, a difference of 0.65 litres, whereas the standard error was 0.58 litres. For smokers, the corresponding figures were: 2.81 and 2.43, with a difference of 0.38 litres and standard error 0.53.

5.4 Interrelationships of signs and symptoms

In the two foregoing sections, we have considered how various signs and symptoms were related to dust exposure. In this section, we examine their interrelationships. There seems to have been no study of how respiratory symptoms and lung function measurements were related in Quebec asbestos workers, and publications concerned with radiographic signs relate them only to function. A first report was made by Dr. M. R. Becklake and her colleagues to the 1968 Dresden conference; it was based on those members of the age-stratified sample for the physiology cross-section who were fully tested in 1967 (before its extension in 1968). This was superseded first by a summary report and then by a much fuller account with the same authorship (Becklake *et al.*, 1970).

The population studied consisted of all 1,069 men tested in 1967 and 1968. In other words, 54 men for whom data were incomplete were included, where possible, although they reduced the "physiology cross-section" to 1,015. This meant that the analyses were based on different numbers according to the function tests under review. The numbers of men included were as follows:- 1,062 for lung volumes; 1,059 for expiratory flows; and for diffusing capacities 840 (single breath), 1,049 (steady state), 932 (exercising at 200 kgm/min), 413 (at 400 kgm/min) and 149 (at 600 kgm/min).

It is implied (Becklake *et al.*, 1970, p. 641) that all 1,069 films for the complete population had been evaluated by each of the six readers, into the U/C classification. The subjects were themselves classified according to "the second highest reading" of small irregular opacities, the only parenchymal change of any importance because small rounded opacities were rarely recorded. Pleural change was taken as present if reported by at least two of the six readers.

As in the already discussed study of lung function measurements, these were "standardized" for age, height and weight by a complex regression procedure. The adjusted mean of each function measurement for the radiological group showing no parenchymal change (profusion of small irregular opacities 0/0; with six readers it would hardly occur that the second highest reading was 0/-) was taken as reference, and the mean for each other radiological group was expressed as a deviation from the reference. The effects of pleural changes were similarly assessed.

In those workers whose x-rays showed no sign of parenchymal disease, i.e. in the reference group, comparison with other populations suggested that VC and FVC might be slightly lower than "normal". In the presence of definite though minimal changes in the pulmonary parenchyma, VC and FVC were the only measurements to show "significant" reduction. Once more the word "significant" is being used in its statistical sense; for example, in those men whose profusion of small irregular opacities was, on the criteria given, 0/1, VC was over 95 per cent of that for the reference group. The authors write about function changes being "indicators of the progress of radiological disease" (Becklake *et al.*, 1970). The meaning is obscure, and what they are reporting is simply a matter of associations. Undoubtedly, VC was lower the higher the profusion of small irregular opacities, and there were several other tests which were similarly related, although they probably had substantially higher standard errors so that significance, in the statistical sense, was lower. Minute ventilation on exercise was selected by the authors as the most "sensitive" of the "indicators" other than VC. For any given grade of profusion of small irregular opacities, function was somewhat reduced when pleural thickening or calcification was also present.

5.5 Mortality in relation to asbestos exposure

Probably the most valuable of the Quebec chrysotile studies is the continuing evaluation of the effects of asbestos exposure on mortality. Tracing has been carried out to four points in time, and results have been presented in five reports, while one is in the final stages of preparation, *viz.*

Deaths to 31 October 1966	McDonald *et al.* (1971)
Deaths to the end of 1969	McDonald *et al.* (1973; 1974)
Deaths to the end of 1973	Liddell, McDonald and Thomas (1977)
Deaths to the end of 1975	McDonald and Liddell (1978); McDonald *et al.* (1979)

In a summary of the health of chrysotile mine and mill workers of Quebec, McDonald *et al.* (1974) wrote: "By the end of December 1969, 87.5 per cent of the 11,572 persons in the cohort [i.e. the "main mortality cohort"] and 99 per cent of those who had worked ten years or more had been traced. Of these, 3,270 had died, comprising 65.4 per cent of those born 1891 to 1895, but only 9.8 per cent of those born 1916 to 1920. The age-standardized mortality [i.e. deaths accumulated over the complete period of observation] per 1,000 men for certain causes of death are given in Table 3 [reproduced here as Table 5.2]. Cancer of the lung showed a rising rate with increasing dust exposure, particularly in the two highest dust exposure groups. Cancers of the gastro-intestinal tract also showed a rise in the two highest, and pneumoconiosis in the highest categories of dust exposure. Of 134 deaths in men from respiratory cancer, five were from pleural mesothelioma. These cases showed no clear relationship with dust exposure. There were no peritoneal mesotheliomas. Mortality from all causes fell with increasing dust exposure up to the highest dust group, probably because those who died young could not attain a high dust exposure. On the basis of Quebec death rates, the expected number of respiratory cancer deaths in the cohort was 139, or about 93 on the basis of estimated rates in the mining region. This suggests that mortality from respiratory cancer in the Quebec chrysotile industry as a whole has been at most 50 per cent above expectation and probably about

Table 5.2

MORTALITY TO END-1969
OF QUEBEC MINERS AND MILLERS

9,692 men in the "main mortality cohort"

(Cumulative mortality rates, per thousand men, standarized for era of birth)

Accumulated dust exposure (mpcf.y)	Cause of death			
	ALL CAUSES	Respiratory cancers *	Abdominal cancers	Pneumo-coniosis
Less than 10	365	10.3	18.0	1.6
10, less than 100	355	13.1	13.6	1.5
100, less than 200	354	13.4	18.7	0.8
200, less than 400	313	15.5	11.6	4.9
400, less than 800	323	21.4	26.3	4.9
800 or greater	395	32.1	28.7	23.6

* includes malignant pleural mesothelioma

25 per cent."

The earliest methods of analysis were known, from the start, not to have been ideal, and some improvements had already been incorporated in the reports of deaths to the end of 1969. In 1974, McDonald *et al.* stated that the first main weakness was that length of exposure might well have been related to length of survival - only those who survived say 30 years from first employment could have had 30 years of employment. The second lay in the use of deaths accumulated over many years (from 1910 to 1966) for a single calculation of mortality. An attempt had been made to overcome the first by use of a parametric approach due to Berry (1970), but this had not been entirely successful. One reason was that the two measures of exposure on which the analysis was based, i.e. years of employment and accumulated dust exposure, were so highly correlated that many of the 144 cells in the 4 x 6 x 6 array (four lengths of service, six groups of total exposure, six quinquennia of birth) were void or contained few men and hence no or very few deaths. The second weakness had been examined in 1973, by studying deaths 1967-69 separately - and finding essentially similar patterns. Nevertheless, an analysis based on man-years of exposure, what Case and Lea (1955) called "comparative composite cohort analysis", was planned. Meanwhile, a case-control analysis had already been adopted (Eyssen and Liddell, 1974), which, although only privately circulated because certain shortcomings were appreciated, suggested an important change in the approach to analysis.

So much importance was placed on finding the best methods of

analysis that it was felt desirable to seek advice from experts. This was done in two stages. First, a group met at McGill University in June 1974 to spend two days discussing methods of analysis. A full report (Liddell, 1974) was circulated; after it had been approved by all concerned, a brief summary was published (Liddell, 1975b). The second stage was to prepare an appraisal of methods of cohort analysis, using the Quebec mortality cohort data for applications, and to have this presented to a meeting of the Royal Statistical Society (RSS) where it could be discussed, and where paper, discussion and reply would be published (Liddell, McDonald and Thomas, 1977). Meanwhile, tracing was continued to the end of 1975, and what follows is a summary of these latest findings (McDonald and Liddell, 1978; McDonald *et al.*, 1979). They have been obtained using the methods discussed before the RSS, which appeared to find favour among the discussants who included several experts of impeccable reputation. There were two approaches, using argument *a priori*, from cause to effect, and *a posteriori*, from effect to cause. For the first, the cohort was divided into mutually exclusive sub-cohorts each subjected to Case and Lea's (1955) comparative composite cohort analysis, slightly amended in a Fortran program (PGM1A) written by Mario Rodrigues to my specification. This analysis accumulates man-years in view in each five-year square of the Lexis (1875) space, and calculates deaths expected on the basis of a reference population (here Quebec males) against which observed deaths can be compared. The ratio: (Observed deaths)/(Expected deaths) can be seen to be a Standardized Mortality Ratio (indirect method), or SMR (Liddell, 1960; Liddell, McDonald and Thomas, 1977). The second approach follows Miettinen (1969) in selecting a fixed small number of controls

for each case; this has been shown to have high efficiency (Ury, 1975) and the material can be used for analysis by regression models (Cox, 1972) if required.

According to McDonald *et al.* (1974) the "primary aim has been to define as accurately as possible the quantitative relationship between exposure to chrysotile asbestos and the incidence of lung cancer"; this aim will be examined first. Thereafter, we will consider the evidence on mortality from all causes, pneumoconiosis, other exposure-related diseases, and mesothelioma. The classification of causes of death, coded in the Seventh Revision of the International Classification of Diseases, used throughout our recent studies is given below, in Table 7.1.

For the *a priori* approach, we adopted two classifications of sub-cohorts. In the first, four classes were created according to length of service: less than 1 year (3,007 men); 1 year, but less than 5 years (2,324 men); 5 years but less than 20 years (2,503 men); 20 or more years (3,105 men). Each class was then divided into four (giving 16 subcohorts in all) according to total exposure accumulated up to a maximum of 20 years, and all men who had survived 20 years from first entry to the industry were followed thereafter to the end of 1975. The reasons for these definitions are fully discussed by Liddell, McDonald and Thomas (1977).

In the second *a priori* classification, ten sub-cohorts were formed according to dust exposure accumulated to 45 years of age, and follow-up was from that age onwards. In order to investigate the

interaction with smoking, the ten sub-cohorts were subsequently re-grouped into three, each subdivided by four levels of smoking.

In both uses of comparative composite cohort analysis the reference population was Quebec males. Full details of deaths annually by cause were available only from 1951 through 1973. The 1973 populations and deaths were reproduced as though they had occurred in 1974 and 1975 as well.

For the *a posteriori* approach, we chose four controls for each death from lung cancer. To be eligible, the control (a) had to be born in the same year as the case and to have survived into the year following the death of the case, and (b) had to have worked in the same mining area (Asbestos or Thetford Mines). One control for each case was selected as having similar smoking habit; three other controls without such restriction. Wherever choice was possible, it was made by strict application of a randomization procedure. Controls were found for all 245 lung cancer deaths (after excluding five mesothelioma deaths which had been coded 162-164 on the death certificate), the only relaxation in the criteria being that the first control was not always matched perfectly for smoking. The measure of exposure used in this analysis was dust accumulated up to nine years before the death of the case.

The findings from the three approaches to lung cancer mortality were in close conformity; they were also in very reasonable agreement with those reported earlier, even although those were on fewer deaths and by rather different methods. The Relative Risks in Table 5.3

Table 5.3

RELATIVE RISKS FOR RESPIRATORY CANCER AND ASBESTOS EXPOSURE AT FOUR STAGES OF ENQUIRY

Accumulated dust exposure (mpcf.y)	1966 *	1969	1973 †	1975	Accumulated dust exposure (mpcf.y)
less than 10	1.0	1.0	1.0	1.0	less than 10
10, less than 100	1.1	1.3	1.2	1.1	10, less than 100
100, less than 200	1.5	1.3	1.0	1.2	100, less than 200
200, less than 300	1.2	1.5			200, less than 300
300, less than 400			1.8	1.8	300, less than 400
400, less than 600	2.1	2.1			400, less than 600
600, less than 800			3.0	2.9	600, less than 800
800 or greater	3.2	3.1			800 or greater

* Relative Risks for 1966 and 1969 were derived from cumulative mortality rates, standardized for era of birth.

† Relative Risks for 1973 and 1975 were derived from SMRs. There were differences also in the definitions of exposure.

were all obtained by *a priori* analysis, but with differences in definitions of sub-cohorts and in methods of calculation, as already explained. However, a very similar dose-response relationship was demonstrated at each analysis (McDonald and Liddell, 1978).

Analysis of the case-control material, whether or not the matching was taken into account, indicated that a multiplicative model of the risk associated with smoking and with dust exposure fitted quite adequately. The corresponding *a priori* results are not open to such sophisticated methods of analysis, but do not suggest any alternative explanation.

The relationship of the response of lung cancer mortality by the end of 1975 to the accumulated dust dose in the 245 cases and their controls has kindly been examined by Mr. Geoffrey Berry, of the MRC Pneumoconiosis Unit. It is well fitted by the straight line:-

$$\text{Relative Risk} = 1 + 0.0012(\text{mpcf.y}).$$

The χ^2 statistic of 23.05 with nine degrees of freedom for differences between dust groups can be partitioned into a component of 19.61, with one degree of freedom, for the linear fit, leaving 3.43, with eight degrees of freedom, for departures from linearity. This last component is very small; further, there was no suggestion that any polynomial would improve the fit. Nevertheless, if only exposures up to 300 mpcf.y are considered, it is difficult to detect any excess lung cancer. The results from the 16 sub-cohorts, defined by length of service and average dust concentration, are compatible with either of these interpretations. Further analysis attempting to distinguish between possible models is in hand, but has so far produced no defini-

tive answer.

The exact form of dose-response relationship cannot be determined, but a linear relation is biologically plausible enough for malignant disease. Taking excess risk from respiratory cancer as linearly related to dust exposure in men who vary in their smoking habits, and to amount smoked in men from the same population who vary in their dust exposure, allowed McDonald and Liddell (1978) a rough comparison of the two hazards, by superimposing the relationships. This indicated that the Relative Risk of exposure to 1 mpcf for fifty years might be roughly equivalent to that from smoking three cigarettes a week during a similar period of life.

Relative Risks for a specific cause must, of course, be weighed in the light of overall mortality, otherwise they can be most misleading, as discussed by Liddell (1975c) in terms of a study by Sutherland (1959) of mortality from sinus cancer and lung cancer at a refinery in Port Colborne, Ontario. Sutherland's cohort was of 2,355 men employed for at least five years between 1930 and 1957, and he used the man-years approach of Case and Lea (1955), basing expectations on Ontario age- and cause-specific male death rates. He found Relative Risks of 36.8 for sinus cancer and 2.2 for lung cancer. However, the total number of deaths from these causes was only 26; the excess over expectation was 19. Meanwhile for all causes of death, the Relative Risk was 0.8; the cohort suffered in sum 63 fewer deaths than expected (245 compared with 308). On the more common causes of death (i.e. excluding those specifically studied) the Relative Risk was 0.7: the cohort suffered 80 fewer deaths than expected from these causes (219

compared with 299). The sinus and lung cancer deaths reduced this desirable shortfall by less than a quarter.

In the Quebec cohort, from 1951 to 1975, the number of deaths (all causes) expected, on the basis of provincial rates, was 3,328.2, almost equally divided between Asbestos (1690.2) and Thetford Mines (1638.0). There was a small excess, of about 39 deaths from all causes, at Asbestos and a rather larger one (of 182) at Thetford Mines. Respiratory cancer, in addition to showing the dose-response relationship discussed above, was also in excess of expectation, slightly at Asbestos and substantially at Thetford Mines (7 and 40, respectively). There was a total of 45 deaths from pneumoconiosis, among the 3,549 for all causes, and these are, at least in some senses, always an excess; 11 occurred at Asbestos and 34 at Thetford Mines.

The patterns with cancers of the abdomen were rather more complex. Those of the oesophagus and stomach were close to normal at Asbestos but in substantial excess at Thetford Mines (84 observed, compared with 54.41 expected). Cancers of the colon and rectum were only about three quarters of those expected, in both mining areas, but those of the organs in the abdomen were below expectation at Asbestos and above at Thetford Mines, although neither difference from expectation was very great. Other cancers were slightly above expectation in both places. Heart disease caused mortality very close to expectation in both places; so did stroke at Asbestos, although there was an excess of deaths from cerebro-vascular diseases at Thetford Mines. Tuberculosis deaths were close to expectation at Asbestos, but high at Thetford Mines (41 compared with 25.55 expected) while the deaths from

respiratory conditions not otherwise mentioned were slightly below expectation at Asbestos and slightly above in the other area. Accidental deaths were rather high at Asbestos and slightly above average at Thetford Mines; and deaths from other known causes below expectation at Asbestos and exactly as expected at Thetford Mines.

It was well known that dust concentrations had been considerably higher at Thetford Mines than at Asbestos and that service there had generally been longer. It was, therefore, to be expected that asbestos-related disease would be more common at Thetford Mines than at Asbestos. The dose-response relationship for lung cancer was very similar at the two places, and there were signs of dust relationships for cancer of the oesophagus and stomach, for respiratory tuberculosis and for other respiratory conditions, as well, as was to have been expected, for pneumoconiosis, these last being particularly clear.

Considerable effort has been given to the recognition of malignant mesothelial tumours not only in the cohort, but also in the population of the mining communities, either employees who did not meet the cohort criteria or those never employed by the mining companies. Ten male deaths in the cohort up to end-1975 were thought to be associated with malignant mesothelioma: only in three of these was it so stated on the death certificate; four were certified as "mesothelioma" and coded as benign; and three more were only discovered by study of autopsy findings. There is no easy way of arriving at an expected figure against which the ten can be compared, but from data obtained by McDonald and McDonald (1977), it was estimated to be about 2. In addition, there was one case in a female and, during 1976 and 1977,

four more cases in males. Thus, in all, we now know of 15 cases (all but one male) in the cohort, distributed as follows:- mine and mill workers: 10 at Thetford Mines and 2 at Asbestos; factory workers at Asbestos: 2 males and 1 female (McDonald and Liddell, 1978).

A final comment is required on female mortality. There were 440 women in the cohort; most had had short service, mainly in fairly low dust concentrations, so that very few had accumulated high dust exposures. Trace rates were higher even than for men, and there were fewer deaths than expected on the basis of provincial rates, i.e. 75 deaths from all causes in the years 1936-75 compared with 83.5 expected. By 1975, there had been two deaths ascribed to lung cancer (compared with one expected), but the excess was in fact a death due to pleural mesothelioma in a factory worker employed on the assembly of gas masks using crocidolite. Thus, there is no evidence of any excess mortality among female chrysotile workers, and they will not be discussed further in this thesis.

5.6 Mortality in relation to radiographic changes

A limited study was reported by McDonald *et al.* (1974). Among those traced up to the end of 1969 in the mortality study, nearly 10,000 were men; chest x-rays were available for a little over 5,000 of them, of whom 785 had died. Death rates were calculated by date of birth, accumulated dust exposure and the presence or absence of parenchymal or pleural radiographic changes.

For all causes combined, the death rate at Thetford Mines was increased in the highest dust exposure group but, after allowance for

exposure and date of birth, those whose radiographs showed parenchymal changes had higher mortality (220 deaths per thousand men traced) than those with normal x-rays (131 per thousand) or with pleural changes only (126 per thousand). A comparison was also made of deaths from respiratory disease, including tuberculosis and cancer. Similar methods of calculation showed 32 deaths in those with parenchymal change compared with only 8 expected, an excess of 24. Thus, 97 deaths in those with parenchymal changes on x-ray (with or without pleural changes) were observed, compared with 64 expected, an excess of 33, two thirds of them being attributed to respiratory causes. At Asbestos, the difference between the mortality of those with and without x-ray changes was less. There was a total of 431 deaths (all causes) but only 7 were associated with parenchymal change.

Further analysis of this material was not justified at that stage, because the number of deaths in those with chest x-rays was so small (785 out of 5,082, or only 15 per cent) and even at Thetford Mines, where dust exposure levels had been much higher than at Asbestos so that radiographic changes were to be expected more frequently, only 132 deaths were associated with x-ray change.

5.7 Summary

In male workers aged 36 to 65, the prevalence of radiographic changes rose with increasing dust exposure: of irregular small opacities (profusion 1/0 or more), from 2.2 per cent to 17.0 per cent; and of pleural thickening (grade 1 or more) from 1.4 per cent to 12.3 per cent. Small opacities or pleural changes were seen in 17 per cent of men; 3.2 per cent had both. Despite adjustment for higher dust

concentrations, the prevalence of radiologic changes was higher in the Thetford Mines area than in the Asbestos area. This was shown least by parenchymal changes, but was extreme with pleural calcification, which was common at Thetford Mines but virtually absent in Asbestos.

In 267 male miners and millers, each with five chest films spanning an average of 20 years, abnormality in the earliest film was related mainly to the time since first employment. In 30 per cent of the 150 men completely normal at earliest film, some radiological abnormality developed during the period of observation; in Asbestos, the risk of developing small opacities was lower in smokers than in non-smokers. There was an irregular pattern of correlation, more evident in Thetford Mines than in Asbestos, between radiological progression and various stimulus variables. The weak associations between radiological responses and measures of asbestos exposure indicated that effects were obscured by other factors (including susceptibility) uncorrelated with exposure.

An age-stratified random sample of 1,015 current male employees in the Quebec chrysotile mines and mills was subjected to a standard questionnaire and to lung function tests. The prevalence of persistent cough and phlegm (bronchitis) was related to age and smoking habits; in non-smokers and light smokers, it was perhaps also related to dust exposure. Breathlessness on exercise was related to age and dust exposure but not to smoking. In non-smokers, inspiratory capacity (IC), forced vital capacity (FVC) and forced expiratory volume ($FEV_{1.0}$) distinguished the lower two dust-exposure groups. In smokers, these tests were less sensitive.

The relationship between chest x-ray changes and pulmonary function was examined in basically the same sample. In workers whose x-rays showed no evidence of parenchymal disease, vital capacity (VC) and FVC may have been slightly reduced; in the presence of slight parenchymal changes, these measurements showed "significant" reduction. In addition, VC and exercise minute ventilation were related to radiological disease. For any given grade of parenchymal disease, function was somewhat more impaired when pleural thickening and/or calcification was also present.

Of 11,788 persons born between 1891 and 1920 employed in the Quebec asbestos mining industry, 88.4 per cent had been traced by the end of 1969, and 2,457 (23.6 per cent) had died. In the highest dust category, comprising 5 per cent of the cohort, the age-standardized rate was 20 per cent higher than in the other groups. Respiratory, cardiovascular and malignant disease accounted in equal proportions for the excess. There were 101 deaths from respiratory cancer (including three from mesothelioma), an estimated excess of about 15 deaths. The difference in rates of respiratory cancer between those maximally and minimally exposed was thought to be fivefold.

These patterns of mortality, exposure-response in particular, were essentially the same as those observed up to the end of 1975 (McDonald and Liddell, 1978). With almost half the men now dead and the survivors aged 55 to 84 years, it seems unlikely that further study will seriously modify the basic findings. As the latent period for malignant mesothelioma appears to be very long in these workers, we may expect the proportional mortality to rise somewhat from present

levels of 2.2 per 1,000 overall and 8.2 per 1,000 for men with 20 years or more exposure. However, the risk appears likely to remain very much lower than with crocidolite.

The major occupational hazards of chrysotile asbestos exposure are respiratory cancer and the complications of pulmonary fibrosis. Excess mortality from respiratory cancer has been shown to be directly related to accumulated dust exposure. The relationship may well be linear and a line also appears to describe the data for pneumoconiosis mortality and, indeed, for all causes.

Chapter 6

QUEBEC CHRYSOTILE FINDINGS AND OTHERS COMPARED

6.1 Introduction

The two other attempts to investigate health effects in Quebec chrysotile miners and millers are dealt with in section 6.2, followed by descriptions of other studies of workers in chrysotile only. Then we consider, in section 6.4, the relationships between dose (of exposure to asbestos of varying types) and response (of lung cancer mortality) and, in section 6.5, the interaction of asbestos dust and cigarette smoking. After a short discussion of mesothelioma, there are some summarizing comments on the health effects of asbestos exposure.

6.2 Other investigations in Quebec

In 1958, Braun and Truan published "An epidemiological study of lung cancer in asbestos miners". They attempted to collect data concerning all workers who had been "processed" through the clinic at Thetford Mines since its inception in 1947, and "similar" information at Asbestos. Because development of asbestosis in less than five years was considered rare, the cohort was defined as including every *miner* (my emphasis) who had a total exposure (? net service) of five or more years and was on the employment rolls in 1950; non-exposed

workers were excluded. Follow-up was over six years. The original cohort embraced 6,091 men, but 133 (2.2 per cent) were lost to view and "deducted". Of the 5,958 traced to presumably the end of 1955, a total of 187 had died (3.1 per cent), nine definitely and three possibly from cancer of the lung. After many tabulations and much discussion, the observed numbers of lung cancer deaths were compared with those expected from provincial rates on two bases, one ignoring age distributions but the other taking them into account and yielding an expectation of six deaths. Thus, the SMR was at least 9/6, or 1.5, and it is difficult to accept the statement of the authors that "it seems fair to conclude that the asbestos miners in the Province of Quebec do not have a significantly higher death rate from lung cancer than do comparable segments of the general population".

In 1974-75, a group from Mount Sinai Hospital, New York, made a study among long-term employees of some of the mining companies at Thetford Mines. The findings were presented by Dr.W. J. Nicholson, on 27 February 1976, to the Quebec Government's Comité d'étude sur la salubrité dans l'industrie de l'amiante (Président: René Beaudry, j.e.p.) and recently to the New York Academy of Sciences conference on Health Hazards of Asbestos Exposure (Nicholson *et al.*, 1978). As results had been provided to the unions who commissioned the study, and had been published by the media, they were available for comparison with his own team's findings by Professor J. C. McDonald, at an International Conference on Lung Diseases in Montreal, on 18 May 1975.

The Mount Sinai study was much more limited than the McGill investigation. It was concerned, primarily, with 1,214 men at Thetford

Mines (much the dustier of the two mining areas of Quebec) who, with a few exceptions, had worked there for at least twenty years. Table 6.1 compares the radiological findings by Mount Sinai and in the two McGill studies (Rossiter *et al.*, 1972; Eyssen, 1975) of men with long service. Although the three studies presented here are not strictly comparable, the findings are fairly similar except in the estimated prevalence of minor degrees of parenchymal change. The Mount Sinai readers clearly placed many films into category 1 of the profusion of small opacities which the McGill group would have considered normal or doubtful. Differences of this magnitude in the difficult area of the boundary between categories 0 and 1 are not surprising when research methods are not identical. The Mount Sinai rates were based on the *consensus* of four readers who were *aware* that the films were all from men with at least 20 years of employment, some having been first exposed up to 65 years previously. These two aspects of design were contrary to the recommendations of a 1973 National Heart and Lung Institute Workshop on the chest x-ray as an epidemiologic tool (Weill and Jones, 1975). In the McGill studies, on the other hand, both series of films included a high proportion from men with little or no exposure; the readers knew only that the subjects had been employed at some time in the Quebec industry and they recorded their individual findings independently.

The Mount Sinai group also carried out a small mortality study, following 544 mine and mill employees from 1961 through 1973. The workers were divided (without definition) into "low" and "high" dust exposure groups, and for all causes of death the two groups had observed and expected deaths, and ratios, as follows:-

Table 6.1

FINDINGS IN THREE RADIOLOGICAL SURVEYS
OF QUEBEC CHRYSOTILE MINERS AND MILLERS

Survey	Mount Sinai	McGill	McGill
Year	1974	1968	1974
Number of men	885	744	283
Mean years since first exposure	33	33	29
Prevalence (%) of:-			
small opacities			
profusion 1/0 or more	*52*	*18*	*26*
profusion 2/1 or more	*5*	*6*	*8*
pleural thickening			
grade 1 or more	*20*	*12*	*21*
pleural calcification			
grade 1 or more	*10*	*12*	*10*

Low dust exposure: 65 observed, 68.29 expected, SMR = 0.95

High dust exposure: 67 observed, 44.56 expected, SMR = 1.50

Deaths from specific causes are summarized in Table 6.2. One death from mesothelioma occurred - in the low exposure group - and has been included in "all other causes". The seven deaths from asbestosis in the "low" exposure group suggest that exposure was nevertheless substantial; a comparison of the expectations of deaths from all causes in relation to other material might indicate that the borderline between the two groups was taken somewhere around 1,000 (fibres/ml) × years. Whatever the exact borderline, many of those in the "low" exposure group had had severe exposure by present-day standards, as indeed must have been true because of the length of employment and the high historical dust concentrations. Thus, that the SMR for all causes was less than unity in this group is of at least as much interest as the Relative Risks, comparing the higher exposure group with the lower, of 4.33/1.55 = 2.79 from lung cancer, 1.11/0.72 = 1.54 for gastro-intestinal cancer and 4.24/2.09 = 2.03 for other respiratory conditions.

In Table 6.3, the Mount Sinai mortality findings are placed in context with those from the McGill investigation, where the latter figures relate to all men in the "main mortality cohort" with gross service of at least twenty years, divided into two sub-cohorts by average concentration (below and above 300 mpcf) over the first twenty years of employment. The larger numbers of the McGill investigation lead to much more reliable rates, and the Mount Sinai figures are seen to be in close conformity. This was only to be expected as the smaller study was essentially only a part of the McGill whole.

Table 6.2

MORTALITY AMONG 544 QUEBEC ASBESTOS
MINE AND MILL EMPLOYEES, 1961-73

Findings of Nicholson (1976)

(O = Observed deaths; E = Expected deaths; SMR = O/E)

	Low dust exposure			High dust exposure		
	O	E	SMR	O	E	SMR
Cancer:						
lung	7	4.52	*1.55*	13	3.00	*4.33*
gastro-intestinal	3	4.18	*.72*	3	2.71	*1.11*
other	4	6.75	*.59*	2	4.40	*.45*
Respiratory disease:						
asbestosis	7	*	*	11	*	*
pneumonia	1	2.01	*.50*	1	1.27	*.78*
other	2	2.79	*.72*	3	1.76	*1.70*
All other causes	41	48.05	*.85*	34	31.43	*1.08*

* Expectations low; SMRs high

Table 6.3

MORTALITY AMONG QUEBEC ASBESTOS
MINERS AND MILLERS

A: McGill study, 1910-75: 3,105 men with at least 20 years service
B: Mount Sinai study, 1961-73: 544 men

(O = Observed deaths; E = Expected deaths; SMR = O/E)

		Low dust exposure			High dust exposure		
		O	E	SMR	O	E	SMR
Cancer:							
lung	A	48	41.57	*1.15*	56	22.96	*2.44*
	B	7	4.52	*1.55*	13	3.00	*4.33*
gastro-intestinal	A	47	58.89	*.80*	45	33.02	*1.36*
	B	3	4.18	*.72*	3	2.71	*1.11*
Pneumoconiosis	A	11	*	*	25	*	*
	B	7	*	*	11	*	*
ALL CAUSES	A	620	657.88	*.94*	478	368.09	*1.30*
	B	65	68.29	*.95*	67	44.56	*1.50*

* Expectations low; SMRs high

6.3 Other studies of workers in chrysotile

In addition to the earlier reports from Russia, Cyprus, etc., there have been three recent accounts of mortality among workers exposed to chrysotile alone. The first discussed here concerns miners at Balengero, Piedmont, Italy, and was presented by Professor Giovanni Rubino and his colleagues at a meeting organised by the Royal Society of Medicine on 28 April 1977. The main concern was with 1,217 chrysotile miners followed from 1946 through 1975. All but 19 were traced (trace rate 98.5 per cent) and 336 were found to have died. The distribution of causes of death was similar to that in Quebec, except for excesses due to cirrhosis of the liver and cancer of the larynx, both comparatively common in Piedmont, and ascribed to heavy drinking of grappa. There had been one death due to mesothelioma.

Elwood and Cochrane (1964) described a follow-up study of workers from an asbestos factory near Cardiff, making mainly asbestos sheeting and pipe lagging, and using only chrysotile since 1935, although some crocidolite may have been used during 1933-35 in the manufacture of high-pressure pipes. Follow-up was of those alive fifteen years after exposure to asbestos dust. Deaths from all causes numbered 46 and those expected on the basis of mortality data for South-Eastern Wales were 47.87 (SMR = 0.96). For specific causes, the findings were:- neoplasm of lung, bronchus and pleura: 7 compared with 3.02 (SMR = 2.32); other neoplasms: 3 against 6.08 (SMR = 0.49); circulatory system 21 compared with 17.54 (SMR = 1.20); all other causes 15 when 21.23 were expected (SMR = 0.71). One death from mesothelioma was observed, but in a man who had probably worked with crocidolite. The authors concluded that chrysotile did not appear to be a

serious hazard as far as mesothelioma or abdominal tumours were concerned, though there was some evidence of an excess in the numbers of deaths from carcinoma of the lung and bronchus.

Weiss (1977) reported a thirty-year study of 264 men hired during 1939-45 who worked in a chrysotile products factory for at least a year and were alive at the start of 1945; follow-up was complete on 94 per cent. For all causes of death, the SMR was 0.61; for lung cancer and gastro-intestinal cancer it was 0.93 and 1.05, respectively. Although the SMR for all causes was higher for men who had worked at least five years in the factory compared with those who had had only one to four years employment, the difference in work duration had no effect on mortality from asbestos-related disease. Weiss concluded that men exposed to chrysotile alone showed a favourable mortality experience, but some caution in interpretation is necessary because of the very small numbers involved.

6.4 Asbestos dose related to the response of lung cancer

Although Schneiderman reviewed this subject in 1974, more data are now available, and this section attempts a synthesis.

Liddell, McDonald and Thomas (1977) published two analyses of the dose-response relationship of asbestos dose and lung cancer mortality in Quebec chrysotile workers. In certain ways, the most reliable estimate of the relationship was considered to be from an analysis in which each lung cancer death in the cohort was matched with five controls born in the same year as the case but who were alive at the end of the year in which the case died; dust exposures for cases and con-

trols were accumulated up to seven years before the death of the relevant case. Berry (1977a), using a minimum χ^2, iterative, program of his own, obtained the equation of the line of best fit as:

$$\text{Relative Risk} = 1 + 0.001869(\text{mpcf.y}) \qquad (1)$$

shown in the figure on p. 44 of Berry (1977b); a fit by eye had been made previously by the present author for other purposes, and this gave the regression coefficient as 0.001875. The χ^2 of 38.76 with nine degrees of freedom (Table 7 of Liddell, McDonald and Thomas, 1977) was partitioned by the fit of the line into 38.00 with one degree of freedom representing the linear trend and 0.76 with eight degrees of freedom for deviations from linearity; as Berry pointed out, this was almost too good to be true.

Because the dust exposure measures in these analyses were accumulated to seven years before the death of each case (and to the same age for his controls), there are some problems over treating the relations fitted in these terms as the most reliable. A more conventional approach, using *a priori* reasoning, i.e. arguing from cause to effect, grouped men according to their exposure and examined the SMRs of the groups. In Liddell, McDonald and Thomas (1977), 80 sub-cohorts had been defined in terms of length and place of service, accumulated dust exposure over twenty years, age at start of service and whether there had been substantial gaps in service. The SMRs for these 80 sub-cohorts clustered about the line given by (1), but the highest mean accumulated exposure in any sub-cohort was rather less than in the case-control approach so that the fit well away from the origin*

* The term "origin" is used as though the ordinate was excess risk, rather than relative risk.

could not be investigated.

McDonald *et al.* (1979) have used two definitions of sub-cohorts, allowing study of the corresponding relationships. The first grouping was into only 16 sub-cohorts, but using definitions exactly as previously for length of service and accumulated exposure; as was to be expected, the basic findings were closely similar to those found earlier. This time, however, the mean values of the accumulated dust exposures for each of the 16 sub-cohorts were obtained and it became possible to fit a line (by unweighted least squares)* to the SMRs, yielding the equation: SMR = 0.9262 + 0.001343(mpcf.y) or, expressed in terms of relative risk (RR),

$$RR = 1 + 0.001450(mpcf.y) \qquad (2)$$

and the coefficient of correlation was + 0.87.

As already discussed in section 5.5, the minimum χ^2 fit by Mr. Berry to the latest case-control data yielded the line:

$$RR = 1 + 0.001193(mpcf.y) \qquad (3)$$

and the χ^2 statistic with one degree of freedom for the linear fit was 19.61, leaving 3.43, with eight degrees of freedom, for departures from linearity. This last component is so small that it does not suggest any better non-linear fit, but it is now not "too good to be

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* Although weighted least squares would have been theoretically more suitable, the complications were not considered justified as the differences would arise far from the origin, whereas the region of particular concern nowadays is close to the origin.

true". The slopes of lines (2) and (3) are in good agreement, when the errors of estimation, particularly of (2), are taken into account. It is not possible to "prove" that the relationship is truly linear, but the goodness of the fits (low residual χ^2 values, and high coefficients of correlation) and inspection of the data provide no justification for a curve approaching the origin at a more shallow angle.

The use of accumulated dust exposure as the only factor studied can be criticised, for it would appear to require the assumption that the risk of lung cancer did not depend on how the dust was accumulated: 500 mpcf.y could mean 50 years of exposure to a steady average of 10 mpcf, or 10 years to widely fluctuating concentrations averaging 50 mpcf, or indeed an infinite variety of patterns of exposure. However, there are several indications that it is a satisfactory choice. First, of those measures discussed by Liddell, McDonald and Thomas (1977, p. 478), this yielded the closest relationship with excess lung cancer mortality. Secondly, a comprehensive study by Dr. D. C. Thomas (personal cummunication) has failed to reveal any improvement in relationships by consideration of other factors describing how the exposure was accumulated. Thirdly, although there was some early evidence (Merewether and Price, 1930) and some recent speculation, for example by Enterline (1978 and earlier), that the more severe the exposure in terms of concentration, the shorter the latent period, recent evidence provides no confirmation; see Table 2.2 above.

Other studies of workers for whom measures of the dose of asbestos dust are available are rare. Berry (1977b) quotes the work of Enterline and colleagues (1972a; 1972b; 1973) among retired asbestos

factory workers in the United States, but there are two problems only one of which Berry attacked. This first was that even those retirees who had virtually no asbestos exposure produced a lung cancer SMR well above unity; Enterline's group also gave information about men who had retired from a factory nearby where there had been no exposure to asbestos, and the SMR here for lung cancer was similarly high. Berry (1977b, p. 45) related the lung cancer SMRs for exposed workers, taking account of accumulated dose, to those unexposed, and found a satisfactorily linear relationship; further the slope of that fitted line, when the relative risk at zero dose was taken as unity, was quite similar to that of line (1) above. The second problem is more difficult: in one of their reports, Enterline *et al.* (1972b) stated that 1,026 of the retirees from the asbestos factory had been production workers, three-quarters of whom had been exposed only to chrysotile, whereas 438 had been maintenance or service workers and a fairly high proportion of these had been exposed to crocidolite. The dose-response relationships in the two groups were quite dissimilar, being virtually non-existent for the production workers when the device was adopted of comparing against unexposed workers from the non-asbestos plant. However, the other group showed a *curve* which was always much above the line fitted to the whole cohort, undoubtedly significantly so despite small numbers. Publication of a more detailed breakdown of the cohort according to the type of fibre to which they had been exposed (Enterline and Henderson 1973) confirmed differences between fibre type in the risks of lung cancer but did not provide information on dose to allow adequate investigation of the shapes and slopes of the relationships.

A recent mortality study of workers engaged in the manufacture of asbestos-cement products in Louisiana has so far been published by abstract only (Hughes *et al.*, 1978) and full detail is not yet available. However, 5,654 males with at least one month employment were followed for a minimum of twenty years. Tracing, through the Social Security Administration, was complete for only 76 per cent, and only 90 per cent of death certificates were obtained. It was reported that SMRs for total mortality were less than in the State of Louisiana, and that the only cause of death found to be dose-related was respiratory malignant neoplasms "with significantly increased SMRs (225, 187) in the two highest exposure categories (> 100 mpcf.y)". Men with service less than two years had no excess mortality, those with from two to ten years service had increased mortality only when average dust concentrations were greater than 20 mpcf, and those with ten or more years service had increased mortality when concentrations were greater than 5 mpcf. It is not stated in the abstract to which fibre(s) the cohort had been exposed.

There have been several reports on a factory at Rochdale (Knox *et al.*, 1965; 1968; Howard *et al.*, 1975; Peto *et al.*, 1977) but, although some environmental evidence has been given, it does not allow full examination of dose-response relationships. From the 1975 paper, it was possible to gain the impression that working conditions in this factory had been more-or-less in accord with present day standards for many years. The facts are far different, as could have been inferred from the 1968 paper and as now given in the latest (1977) paper. Men and women first exposed in 1951 or later numbered 347; of these six men died from lung cancer when only 3.2 such deaths were to have been

expected from national mortality rates; the SMR was thus 1.9. However, we are also told (Howard *et al.*, 1975) that five of the six lung cancer deaths were in men who had been working in areas where dust levels were high in 1952. Equal allocation of the man-years to higher and lower dust conditions provides the following:- lower dust concentrations: one death against 1.6 expected (SMR = 0.63); higher dust concentrations: five deaths against 1.6 expected (SMR = 3.13). Further all six cases were smokers.

Dr. M. L. Newhouse, in a series of papers with various colleagues, has reported *seriatim* on mortality of workers at the Cape Asbestos factory in Barking. Although dust concentrations were only classified in two ways (low-moderate; severe) and service likewise (less than two years; longer), there were large differences in SMRs among 1,414 men followed from twenty years after start of service (Newhouse, 1973a), *viz:-*

	Dust concentration	
	Low-moderate	Severe
Less than 2 years in the job	4/2.4 = 1.67	11/3.3 = 3.33
Longer in the job	5/1.6 = 3.13	17/2.3 = 7.39

Comparison with other experience is impossible without estimated concentrations, but the excess risks appear much higher than in Quebec. There can be no doubt that substantial amounts of crocidolite were used at this factory.

6.5 The interaction of asbestos dust and cigarette smoking

Saracci (1977) has reviewed the epidemiological evidence on the asbestos-smoking interaction, considering three models. In the first,

the excess incidence of lung cancer independently due to asbestos and to smoking add together when both agents are present (additive model). In the second model, the addition of each one of the two agents produces an increase in lung cancer incidence which is proportional to the effect of the other agent (multiplicative model). In the third, asbestos can increase lung cancer incidence only in the presence of smoking. Saracci did not have available at the time of writing the data from Quebec, but he reviews the rest carefully. As found by other investigators, he feels the additive model the least plausible of the three, and the multiplicative the most plausible.

The evidence from Quebec also supports the multiplicative model, and here the amount of data make the plausibility the greater. In studies based on *a priori* reasoning, there appeared little to suggest deviation from the multiplicative model. Thomas (1977) reported on his preliminary studies of the shapes and forms of relationships and interactions on lung cancer deaths to the end of 1973. His more recent work (Dr. D. C. Thomas, personal communication), using two methods of analysis, showed no evidence against the multiplicative model: in unmatched analysis of the case-control data (cases to end-1975 with four controls per case, one chosen matched for smoking habit, the other three not so matched), using multidimensional contingency table analysis, he found no significant improvement to the fit by varying the model from the simple multiplicative. In a matched analysis based on his own extensions of methods, following Cox (1972) and others, to be published (Thomas, 1978), he once more demonstrated a good fit of the multiplicative model to the data.

An important finding from Quebec is that of the 245 male lung cancer deaths, 20 were among non-smokers. While there remain some doubts about the quality of some of the smoking information obtained from the questionnaires, they relate mainly to the coding, the lack of information about men who died before 1950, and the inevitable problems about deciding who is an ex-smoker when the respondent may revert to smoking after being questioned. The exact amounts smoked may also be in doubt, but perhaps the most reliable piece of information is the statement that a man never smoked. In the non-smokers, the observed relationship between dose of asbestos and excess lung cancer was similar to that in smokers. This would seem to dispose of the third of Saracci's models.

Figure 6.1, following McDonald and Liddell (1978), takes excess risk from respiratory cancer as linearly related (a) to asbestos dust exposure in men who vary in their smoking habits and (b) to smoking in men from the same population who vary in their dust exposure, and superimposes the relationships, to allow a *rough* comparison of hazards.

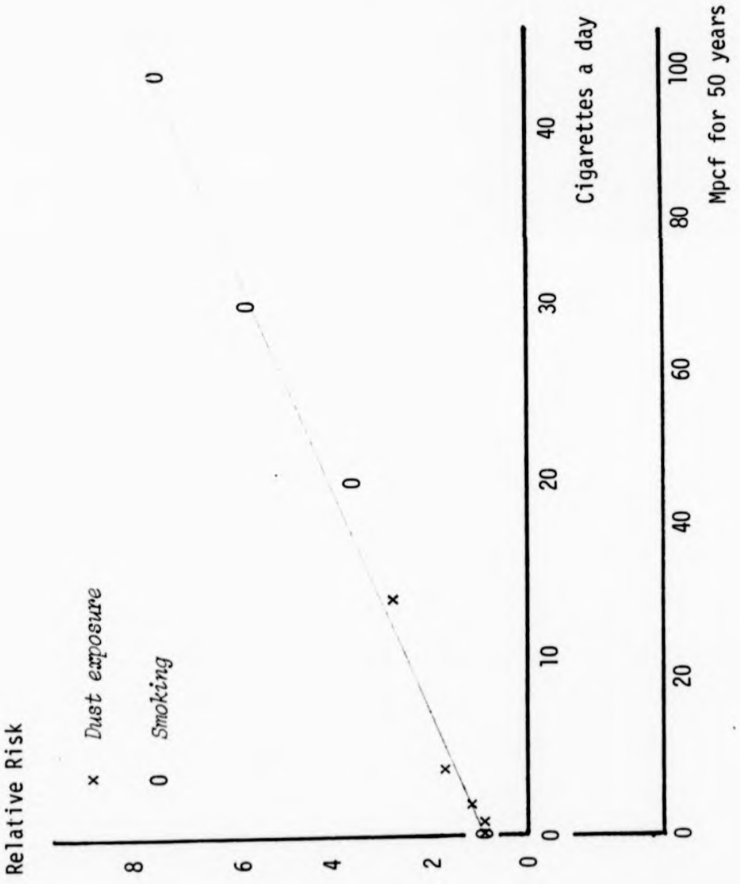
6.6 Mesothelioma

This thesis is not primarily concerned with mesothelioma, and there is a good review of the whole subject by McDonald and McDonald (1977). However, the importance of fibre type cannot be overemphasised and it seems necessary to discuss the incidence of this condition in those exposed to chrysotile alone.

In studies among Quebec workers, McDonald and Liddell (1978) reported a total of fifteen deaths (all but one male) due to mesothelioma, to the end of 1977. They were distributed as follows:- mine and mill workers: ten at Thetford Mines and two at Asbestos; factory

Figure 6.1

LUNG CANCER MORTALITY BY DUST AND SMOKING

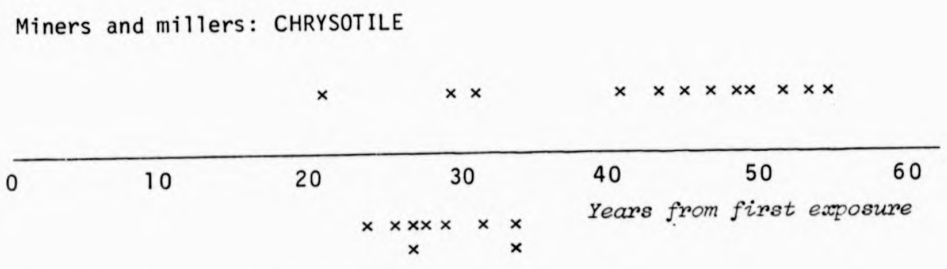


workers: three, including one female, all at Asbestos. Figure 6.2, also from McDonald and Liddell (1978), shows the twelve cases in miners and millers by interval from first employment, and nine cases in persons employed in Canada in World War II on the crocidolite filter pads for service gas masks (McDonald and McDonald, 1978). Two of the factory workers at Asbestos were involved in this process and are included in the lower part of Figure 6.2; the third factory worker may also have been exposed to crocidolite during the same period but is not included. This figure shows that, in the chrysotile mine and mill workers, the median period from first employment to death was nearly 20 years longer than in the factory workers exposed to crocidolite. Other differences were also impressive. Of the twelve "chrysotile cases", nine were in 3,105 men employed twenty years of more and three in 7,834 men employed less than twenty years; all twelve were pleural. The nine "crocidolite cases" were in total of only 199 persons, none of whom had worked in gas mask production for more than $2\frac{1}{2}$ years; six of the nine cases were peritoneal.

Of the nine mesothelioma deaths to the end of 1975 that might be considered due to exposure to chrysotile, seven were at Thetford Mines; none were in men with the highest dust exposures, but there was lower incidence among those more lightly exposed at Thetford Mines. In his study in the same area, Nicholson (1976) reported only one mesothelioma among 132 deaths, a rather higher proportional mortality, but still a rare occurrence. This death was not in the group that Nicholson designated as "high" dust exposure, but as already discussed his "low" dust exposure group surely included many men with substantial exposures by to-day's standards.

Figure 6.2

LATENT PERIODS IN MESOTHELIOMA



It is difficult to obtain an estimate of the number of mesothelioma deaths to be expected in the "main mortality cohort", particularly because of the high ascertainment rate in the Province of Quebec (McDonald and McDonald, 1977), but it seems not unreasonable to put it at about two for the miners and millers. Thus, there were four to five times as many deaths from mesothelioma as expected; it remains essential to keep the absolute number in mind, particularly in relation to the numbers of deaths from lung cancer (245 among men) especially those that might be considered asbestos-related (about 45 or 50).

Of the 336 deaths among Piedmont miners, Rubino reported one mesothelioma, a proportion quite close to that observed in Quebec. Elwood and Cochrane (1964) also reported one mesothelioma, but thought it was due to crocidolite exposure; Weiss (1977) observed no deaths from mesothelioma in his small cohort of chrysotile factory workers. Although Peto (1978) reported only pleural mesotheliomas from the Rochdale factory, crocidolite had been used there, although in what quantities is not known. All other occupational studies with high proportional mortality from mesothelioma have been in persons exposed to amphibole fibres. In all these studies the absolute numbers have been high, and there have been high proportions of peritoneal tumours.

6.7 Summary

In an important "State of the Art" review, Becklake (1976) listed (her Table 1) the pathological effects of asbestos exposure in man, differentiating between effects that are established, associated with a co-factor, possible and remotely possible, as follows:- established: asbestos bodies and/or fibres in the lung; asbestosis; pleural plaques

and calcification; pleural and peritoneal mesothelioma; and neoplasia of the gastro-intestinal tract; adding carcinoma of the lungs as a co-factor with cigarettes. As possible effects, she included carcinoma of the larynx, pleural effusion; as remotely possible, carcinomas of ovary and breast. For completeness, one should also mention asbestos corns, although they are not relevant to this thesis.

In her Table 4, Dr. Becklake listed studies that have demonstrated dose-response relationships between biological responses in man and estimated exposures, pointing out that most of the measures of exposure have been rather crude, including: years of exposure; years since first exposure; occupation; occupation and duration. The studies in Quebec chrysotile workers are nearly unique in having related responses to accumulated dust exposures, but there are two added studies by Weill and his colleagues (1973; 1975). Radiological changes both in the parenchyma and in the pleura have been found to increase with increasing dose, however measured. Lung function measurements, particularly FVC and $FEV_{1.0}$/FVC have often also been related to duration of exposure, and symptoms, particularly breathlessness on exercise, to dust exposure. These signs and symptoms are of course only "signposts" on the way to excess mortality, and this in turn has frequently been related to measures of exposure, with excesses especially noticeable for lung cancer, sometimes gastro-intestinal cancer, occasionally laryngeal cancer, and, where numbers or fibre type have made it possible, mesothelioma. However, the ability of the signs and symptoms themselves to predict excess mortality has not been the subject of such close study. In particular, the essential relationship between radiological change and excess

mortality has been examined so far only to a very limited extent in a general review of the health of Quebec miners and millers.

Chapter 7

THESIS OBJECTIVES AND MATERIALS

7.1 Introduction

Although there is no doubt that exposure to asbestos dust can, if severe enough, lead to premature death, little is known about how the various signs and symptoms make themselves apparent during the disease process. All have been shown to be interrelated, but there is little evidence of whether radiographic changes, depressed lung function or breathlessness are generally the earliest, or how any of them predict mortality. None of the various function tests and none of the respiratory symptoms can be considered specific to asbestos-related disease; on the other hand, x-ray changes that can be categorized into a "classification of the pneumoconioses" devised with particular concern for asbestosis must be taken as more specific. Thus one form of validation of a classification is in the extent to which radiological changes can predict mortality.

Other possibilities of validation are in relating *post mortem* pathology to radiographic appearances in an x-ray taken shortly before death; or, alternatively, in high correlations between appearances, say, in employed workers and measures of their exposure during relevant periods before the x-ray. However, correlations of this latter nature,

although positive in many studies in asbestos workers and in those exposed to other mineral dusts, have never been high enough to provide acceptable validation. In coalworkers, several attempts at validation of x-ray classifications in terms of *post mortem* findings have been made, but mainly in the same lungs (Liddell, 1972; Rossiter, 1972b and in several other reports). These authors concluded that the observed correlations between the profusion of small rounded opacities and the weights of various chemical dusts assessed in the lung were high enough to justify a claim that the ILO, Geneva, 1958, classification was of reasonable validity. However, this was only for coalworkers, and in any case was concerned with a now outdated radiological classification. As Rossiter (1972c) has pointed out, coalmine dust is relatively inert, and in considering the biological effects of asbestos in the lungs, pathological assessment would have to take into account the fibrosis rather than the dust itself. A study to this end has recently been put in hand in the Province of Quebec, but results cannot be expected for several months, and can at best be provisional only - because of the ways in which (a) cases have been selected and (b) assessments, both histological and radiological, have been made.

Although the National Coal Board's Pneumoconiosis Field Research (PFR), set up in 1952, had rather restricted objectives (Fay and Rae, 1959) concerned with the setting of dust standards in the light of radiological change (later extended by consideration of lung function), investigation of mortality was made feasible in the late 1960s, when National Insurance numbers for all men ever included in PFR were incorporated into the record-keeping system, to make it compatible with that introduced for the Board's Periodic X-ray Scheme (PXR) at its

inception in 1959 (Rogan, 1964; Liddell and May, 1966; Lee, 1966; Mead, 1966). Meanwhile Cochrane *et al.* (1964) had carried out a six-year mortality follow-up of males over 15 originally surveyed in the Rhondda Fach in 1952; an earlier study, with only two-and-a-half years of follow-up had been too short to carry much conviction. Although non-miners had lower SMRs than miners, there appeared to be no differences in the SMRs of those whose 1952 x-ray had been placed in any of the categories 0, 1, 2 or 3 of simple pneumoconiosis in the Cardiff-Douai or, for all practical purposes, the ILO, 1958, classification. Indeed, the only predictive value of the x-ray seemed to be that those with progressive massive fibrosis in 1952 had higher SMRs than those with normal x-rays or simple pneumoconiosis only. This was, at least to the present author, a rather surprising finding, which might have been due to several factors, including the rather short period of follow-up and the very special population examined. The former, but not the latter, was overcome in a follow-up of the same population for twenty years, reported by Cochrane (1973). However, the main finding was similar: "Survival rates for miners and ex-miners appear independent of the x-ray category of pneumoconiosis except for category B or C, where survival rates are much reduced". More recently, Jacobsen (1976) produced essentially the same result in a rather larger study, based on all 35,000 miners in the original PFR surveys; nevertheless, the yet larger, and completely representative, sources of PXR have still to be tapped.

These findings in coalworkers were not in accord with those reported by McDonald *et al.* (1974) in asbestos miners and millers; see section 5.6 above. The differences probably lie mainly in the dif-

ferent types of dust to which the subjects were exposed; the radiographic changes associated with asbestos exposure are not common in coalworkers, and certainly were not recognized in the classifications of radiographs used in both Cochrane's and Jacobsen's studies of coalworkers.

7.2 Objectives

In all these circumstances, it seemed of great importance to attempt to answer the question:

1. To what extent can radiological changes in those who have been exposed to asbestos dust predict mortality?

This, then, was the primary objective of the thesis research. Its justification was confirmed in the recent deliberations of eight distinguished film readers (from Germany, Italy, South Africa, the United Kingdom and the USA) who carried out a trial in Johannesburg during October 1977 of the ILO U/C (1971) classification for use specifically in asbestos workers (Irwig, 1978). Under the heading "x-ray abnormality and health impairment", these readers agreed that the morbidity and mortality associated with x-ray abnormality were largely unknown. They felt research in this area to be crucial, because the importance of radiology depends on its ability to predict illness and death, rather than on its sensitivity to asbestos exposure.

As some predictive ability was to be expected in the light of the findings reported by McDonald *et al.* (1974), two further questions arose:

2. Are the predictions of mortality from radiological changes improved or modified by other factors?
3. What are the best factors for predicting mortality?

7.3 Factors for study

The choice of factors for study, other than radiological changes, could only be made in the light of knowledge from mortality studies in exposed workers and in others, and in relation to the causes of death that it would be reasonable for radiographic change to predict.

The asbestos-related causes of death have been seen, from our own studies, to include pneumoconiosis and lung cancer, both of which might well be detected on the chest x-ray. Death certified as from respiratory tuberculosis and other respiratory conditions might also be radiologically diagnosable whether or not associated with dust exposure. Other diseases, including cancer of the oesophagus or stomach, or of the colon or rectum, and diseases of the heart, might well not be diagnosable on a chest x-ray, but if they were associated with heavy dust exposure, such exposure might have led to pneumoconiotic appearances in the radiograph. It was therefore felt necessary to differentiate all the above causes of death in the various analyses. It was also, of course, important to study deaths regardless of cause, while certain other groups (laryngeal cancer, "other" abdominal cancers, "other" cancers, accidents and cerebro-vascular diseases) were also differentiated, together with those where the cause was known but did not fall into any of the other defined categories. Inevitably, a precise cause of death would not be determined for a few deaths, which could not be placed into any of the thirteen defined groups of causes,

but were, of course, included in the counts of total deaths, or "deaths from all causes". Our classification of causes of death is given in Table 7.1.

As far as dust-related diseases were concerned, our own studies in Quebec had shown the undoubted importance of measures of exposure based on estimates of dust concentrations. Although no differences had been found between Asbestos and Thetford Mines in any of the mortality studies (except those concerning cancers of the gastro-intestinal tract) after due allowance for the substantially heavier and longer durations of exposure at the latter, there were some differences between the places in radiological findings. It was therefore felt it would be necessary to distinguish them again. Smoking was clearly important as far as lung cancer was concerned and might be expected to have associations with mortality from certain other causes.

No evidence was available from the Quebec studies about mortality in relation to respiratory symptoms and lung function, but these were known to be related both to dust exposure and to radiographic changes, while there was evidence from population studies, mainly in the USA, concerning mortality in relation to respiratory function and symptoms (see Chapter 14, below).

The remaining issue is of the length of follow-up. Were the objective to validate the x-ray classification against pathological findings *post mortem*, the x-ray should have been taken as late in life as possible, to leave the interval between x-ray and *post mortem* short. However, for a realistic test of the effectiveness of the x-ray in

Table 7.1

CLASSIFICATION OF CAUSES OF DEATH

(The codes are those of the Seventh Revision of the International Classification of Diseases)

Pneumoconiosis	523-524
Cancer of the chest	162-164
162: malignant neoplasm of bronchus and trachea, and of lung specified as primary; 163: malignant neoplasm of lung, unspecified as to whether primary or secondary; 164: malignant neoplasm of mediastinum.	
Tuberculosis of respiratory system	001-008
Other respiratory conditions	470-522; 525-527
470-475: acute upper respiratory infections; 480-483: influenza; 490-493: pneumonia; 500-502: bronchitis; 510-527: other diseases of respiratory system other than pneumoconiosis.	
Diseases of the heart	400-443
400-402: rheumatic fever; 410-416: chronic rheumatic heart disease; 420-422: arteriosclerotic and degenerative heart disease; 430-434: other diseases of heart; 440-443: hypertensive heart disease.	
Cancer of oesophagus or stomach	150-151
Cancer of colon or rectum	152-154
Malignant neoplasm of small intestine, including duodenum, of large intestine or rectum.	
Other cancers of the gastro-intestinal tract	155-159
Malignant neoplasm of biliary passages, liver, pancreas, peritoneum or unspecified digestive organs.	
Cancer of larynx	161
Other malignant diseases	140-148; 160; 165-205
Cerebro-vascular diseases	330-334
Vascular lesions affecting central nervous system	
Accidents, including poisonings and violence	800-999
Other known causes	All codes not listed above

predicting mortality in a working population, the follow-up period has to be rather longer. In diseases where the interval between diagnosis and death is very short, e.g. mesothelioma or to a lesser degree cancer of the lung, it then becomes unlikely that any direct validation of radiographic classification would be possible, as abnormal appearances specific of the terminal condition could hardly have appeared at the start of the follow-up period, usually substantially before diagnosis. In these circumstances, it becomes necessary to rely on indirect validation, i.e. prediction by means of radiographic changes not themselves typical of the condition in question but rather associated with the asbestos exposure, which at least in some of the cases may have led to the terminal disease.

7.4 Criteria for selecting a study population

The ideal criteria for choice of an acceptable study population can thus be listed as follows:-

(1) a chest x-ray towards the end of a person's employment; further, the x-ray should have been assessed, independently, by each of several readers (Weill and Jones, (1975), into a standard, but detailed, classification of pneumoconiotic appearances, including those of asbestos-related disease;

(2) a full work history, including estimates of dust concentrations, to allow calculation of measures of exposure;

(3) smoking history;

(4) lung function measurements and evaluation of respiratory symptoms close to the time of x-ray;

(5) complete follow-up for long enough that a substantial proportion of the population has died; and, for each death, date and certified cause.

No population could be ideal, and to carry out an entirely new study on an adequate scale would have been prohibitively expensive. However, it was possible to select two cohorts, "A" and "B", of Quebec miners and millers, both of which met many of these criteria.

Cohort "A" was intended to consist of all 1,015 men in the "physiology cross section" (see section 4.7 above) defined by both McDonald, Becklake *et al.* (1972) and Becklake *et al.* (1972): these men were still employed by the mining companies in Quebec in November 1966, full work histories to that point were available, and the last employment medical examination x-ray up to that time had been selected and read by six readers; smoking history, and respiratory symptoms had been elicited by questionnaire in 1967 or 1968, and lung function measurements made at that time; about two-thirds of the men had been born between 1891 and 1920, and so they were embraced in the "main mortality cohort" (see section 4.6 above) leaving only about one-third requiring to be followed. However, although the sample had been weighted (by stratification) towards older men, some of whom were close to retirement, most would have continued in employment, probably in the mining industry. Nevertheless, additional exposure beyond 1966 was to concentrations of dust at much lower levels than had been in existence for most of the men in earlier years, so that the increment was likely to have been at a low absolute level, although for many of the younger men it would not have been low in relative terms.

Cohort "B" consisted initially of all those persons in the "main mortality cohort" who had also had a radiograph assessed during the major reading studies of 1967. Well over 5,000 workers were expected to be in this group, but it was reduced for three reasons. First, to ensure the greatest degree of comparability, only those persons whose film had been successfully read in what Rossiter *et al.* (1972) called stage 2, i.e. by one of the six readers, after randomization into as nearly as possible equal sets, but excluding films considered "unreadable". Secondly, to preserve independence, the 603 men common also to Cohort "A" were excluded. Thirdly, the 231 women remaining at this point were excluded, after preliminary examination had confirmed (a) that they had in general had low exposures to asbestos, (b) that all but a few of their films had been assessed as completely normal, and (c) that the SMR, in relation to all women in the Province of Quebec, was considerably less than unity, in fact 0.78. In Cohort "B", we did not know the date of the last x-ray, but could assume that criterion (1) had been met as to the timing of the film, although only one assessment of each was available. Criteria (2) and (5) were met completely, by virtue of membership of the mortality cohort, and (3) partially so; there were no lung function measurements or assessments of symptoms.

Further details of the two study cohorts - definitions, sources of data, assembly of data, and numbers by follow-up status (alive at the end of the follow-up period, known to have died, or lost to view) - are given in the next two sections.

7.5 Cohort "A": definition, sources and numbers

The "physiology cross-section" described in section 4.7 above has been reported on by Becklake *et al.* (1970), who studied lung function in relation to radiological changes in 1,069 men, and by McDonald, Becklake *et al.* (1972) and by Becklake *et al.* (1972), studying respiratory symptoms and lung function in relation to dust exposure, who found it necessary to exclude 54 men for whom data discrepancies had been discovered, to leave a study group of 1,015. The magnetic tape files containing the function measurements, with heights and weights, and the questionnaire responses, were known as LUNF2F and QUESTF, respectively. Matching of LUNF2F against QUESTF indicated that all 1,071 persons on the first were also on the second, which contained records for eleven additional subjects who, presumably, had attended for interview but had not completed satisfactorily the tests in the mobile laboratories. The present concern was with males only, and so 40 females on LUNF2F were excluded. Thus, at that point, the population for study consisted potentially of 1,031 men.

The first stages of follow-up had been instituted early in 1977, making use of a list of 1,015 men, kindly supplied by Dr. Margaret Becklake, and purporting to relate to those included in the studies reported in 1972. The men were classified as to whether they were or were not in the mortality cohort, i.e. had been born in the years 1891 through 1920, or more recently - as all in this population had been at work in 1966, none could have been born before 1891. Arrangements were made for the tracing of those not in the mortality cohort. This work was carried out by Mrs. Proulx, at Asbestos, and Mrs. Beauchemin, at Thetford Mines, two ladies who had been specially trained for such

tasks over a number of years. However, the initial classification had been almost entirely by the date of birth given on Dr. Becklake's list, and that included any amendment made during interview in 1967 or 1968. Further, the list took no account of changes in the cohort since it was first prepared in about 1969, and so the classification required careful checking, which revealed several men who had been wrongly treated recently as part of the mortality cohort, i.e. who would now have to be traced; the field enquiries had also to be extended to cover those among the 1,031 men on LUNF2F who were not on Dr. Becklake's list. In fact, one of these could not be identified in the register either; we could find no trace of him in any of our data banks (except LUNF2F and QUESTF) and he was therefore eliminated from the study population at this stage. Meanwhile, every case in the "main mortality cohort" was positively identified: they totalled 624. This left 406 for follow-up, which was carried into the start of 1976, to bring it into line with that for the mortality cohort proper.

In the event, tracing was complete: as these men were all born after 1920, it was not surprising that most were alive. There had been eleven deaths, and dates and places, all in Quebec, were obtained. A request for copies of the death certificates was refused by the Quebec government, because of a new attitude to confidentiality; nevertheless, a satisfactory cause of death was obtained by further enquiry by Mesdames Proulx and Beauchemin. The eleven new causes of death were coded into the Seventh Revision of the International Classification of Diseases, in conformity with the practice for the "main mortality cohort"; see Table 7.1.

Several processes were needed to identify sets of x-ray readings for these 1,030 potential subjects, i.e. the 1,031 men on LUNF2F less one unidentifiable as described above. The identifying number was a 6-digit number which had been entered on the buff cards at the time of registration in 1966-67; for those men found to have more than one buff card, the number relating to his latest employment was on the LUNF2F record, but for most other purposes a special number, in less than six digits, had been used for the subject with appropriate cross-reference to the several six-digit numbers on the two or more buff cards recording his history. Usually, it was this shorter ID that had been used on the intermediate magnetic tape file, TRIPLF, from which the film reading number had to be extracted. Great care had to be taken to ensure that the search of TRIPLF was truly comprehensive; many checks were possible and all were instituted. Nevertheless, 21 men could not be traced on TRIPLF, which meant that it would not be possible to find any radiological assessments for them. This left 1,009 men in the study, the same number as had been come to in an earlier attempt at reconciliation (White *et al.*, 1974).

Next, the magnetic tape file (BASICX) containing over 42,000 x-ray readings had to be searched for any reading against the appropriate number for each of the 1,009 men still in the population; at least one reading was indeed found for every subject. In most cases, there was a set of six readings (one for each reader), together with a "stage 2" reading, and often additional readings from later stages of the radiographic study. However, for a total of 19 subjects, it was not possible to find or make up a set of six readings, one by each of the different readers. The potential study population was now

reduced to 990.

For these 990 men, we brought on to one magnetic disc file all the desired information from LUNF2F and QUESTF, together with the six radiographic assessments and, for men not in the mortality cohort, the tracing information, including, where appropriate, date and cause of death.

For those in the "main mortality cohort", it was comparatively simple to bring forward, from the magnetic tape records of that cohort, not only the tracing details, i.e. status (alive, dead or lost to view) and date of death (or loss) with cause of death, but also the fully detailed record maintained on what was called the Final Analysis File of the mortality study (Rodrigues, 1976). There remained the corresponding details of work history for men not in the mortality cohort. The details from the buff cards for all persons registered in 1966-67 but not included in the mortality cohort had been punched onto cards and transcribed, in "mirror image" without editing, on to what was called the Bonaventure magnetic tape. It might therefore have seemed a small task to obtain the work histories, for the 406 persons involved, in the same lay-out as for the remainder. However, this had been a laborious aspect of the original data processing (Rodrigues, 1976) and we needed access to many programs in the original suite, some of them used iteratively, and two more had to be written. Much checking was necessary, pinpointed by the computer programs, but rendered slow in execution because of fairly extensive clerical work, key-punching and verification, carried out by the author, while the basic records were held at an outstation. Some indication of the data processing involved

is given in Table 7.2; it was completed for all but one man whose buff card record was not on the Bonaventure tape, while the card itself could not be found in late 1977.

The detail carried forward to this stage was very long, so that costs for disc storage and for analytical computing would have been unnecessarily high. Three major savings were achieved. First, after record linkage had been successfully completed, unneeded identifying detail was eliminated; secondly, the environmental history was reduced to the totals of net service and of dust exposure accumulated to November 1966; and, thirdly, the information on symptoms and lung function measurements was reduced to a working minimum, in consultation with Professor Margaret Becklake and my supervisor. Other, smaller, savings would have been possible, but it was not felt they were worth the extra costs of rewriting the files in further shortened form.

The basic information retained on the working file (XMAFWF) is listed in Table 7.3. The symptoms and measurements recorded in 1967 or 1968 were as defined by McDonald, Becklake *et al.* (1972) and Becklake *et al.* (1972). Smoking was recoded into a single character six-point code as follows:- 0 = Non-smoker; 1 = Ex-smoker; 2 = Not a cigarette smoker; 3 = Smoker of 1-12 cigarettes a day; 4 = Smoker of 13-25 cigarettes a day; 5 = Smoker of 26 or more cigarettes a day. Net service and accumulated dust exposure were as described by Liddell, McDonald and Thomas (1977, p. 471); dates of start of first job were retained so that gross service could have been calculated to November 1966. Only the year of death (or of loss to view) had been recorded, and cause of death was coded (or recoded) into the Seventh Revision

Table 7.2

CALCULATION OF DUST EXPOSURES

for men in Cohort "A" not included in

Program or process	Input	Output
NEW.1	Bonaventure tape	Discs
WHLIST	Discs from NEW.1	Edit lists
NEW.2	Discs as above	Revised discs & cards
Manual/ Keypunch	Cards as above	Revised cards
WHLIST	Cards from above	Edit lists (clear)
UPDTEWH	Cards from above	Revised WH discs
PGM2	WH discs & Dust master	Error print-out
Manual/ Keypunch	Error print-out	Work History cards

"main mortality cohort"

Comment
373 men with single work histories, and 31 with mutiple, separately
404 men as above; 3,181 records
Adjustment to mine codes without alteration of numbers
(a) Removal of errors flagged by WHLIST; (b) Clearance of identity problems
Now 405 men, with 3,080 records
405 men, but redundant records removed, leaving 2,753
17 errors in work histories
17 errors corrected, entered on to punch document; complete revised work histories punched and checked

Table 7.2 (continued)

Program or process	Input	Output	Comment
UPDTEWH	WH disc & amended WH cards	Amended discs	Numerous errors in dust information
Manual/ Keypunch	List of dust errors	Revised dust cards	Preparation for correction of Dust master file
DUSTCHAD	Revised dust cards	Amended Dust master	Amendment of Dust master file
PGM3	Amended WH & Dust master discs	Exposures by job and year	Preparation for input to FAF; calculation of 7,006 annual concentrations by job
MORTMOD	ID and tracing cards	Mortmod disc	Final preparation for input to FAF
FAF	Mortmod disc	FAFile	Consolidation of exposure data, providing annual exposures for 405 men

BASIC INFORMATION FOR MEN IN COHORT "A"

Columns	Item and "format"	
7-12	Date of birth (day/month/year)	3I2
16-21	Identity number	I6
22-27	Date of test (day/month/year)	3I2
28-31	Height (cms)	F4.1
32-35	Weight (kgs)	F4.1
36-39	$FEV_{1.0}$ (litres)	F4.2
40-43	FVC (litres)	F4.2
44-47	MMF (litres)	F4.2
48-52	Persistent cough, persistent phlegm, breathlessness, wheezing, number of chest illnesses	I5
53-61	Smoking habit, recoded into a single character in column 362	I1
62-66	Clinic, and reference number	A1,I4
67-71	Reading sequence number	I5
72-114	X-ray readings: Reader A	
115-157	B	
158-200	C	43 columns laid out as
201-243	D	in p. 4 of the Annex
244-286	E	
287-329	F	
330	Status (Alive, Dead, or Lost to view)	A1
331-336	Date of birth as in "main mortality cohort"	3I2
337-338	Year of death, or loss (blank if alive)	I2
339-341	Cause of death (blank if alive or lost to view)	I3 *
342-345	Date of start of first job (year/month)	2I2
346-349	Recorded end of last job (year/month = 66/11)	2I2
350-353	Net service (years)	F4.2
354-360	Accumulated dust exposure (mpcf.y)	F7.2
361	Mining area (Asbestos or Thetford Mines)	A1

* First digit R or S for accidents at asbestos work

of the International Classification of Diseases.

The x-ray readings had been into the full U/C classification as adopted by the six McGill readers in 1967, i.e. well before its publication in 1970 - see section 3.4 above; however, the detail of which "additional symbols" had been recorded had not been punched, but only the count of the number of "symbols", and similarly there was a record as to whether or not any "comments" had been noted. These last "economies" were introduced in the preparation of the tape file containing the 42,022 readings that replaced one which, during the early 1970s, was found to have been incorrectly written on to tape a year or so earlier; the only way of repairing these deficiencies would have been by recourse to the original reading sheets. The readings for each particular reader were allocated a specific field on the complete record on XMAFWF.

Preliminary processing, partly to test the main analytical program, partly to check on the quality of the data, revealed that the recorded environmental history on a precursor of XMAFWF was inconsistent for one man: his last job was given in the detail as in the 1940s, but he had definitely still been in employment at the end of 1966. A search of the original register showed (a) that he was in the "main mortality cohort" and (b) that he had had employment at three companies, and hence three buff cards; no problems of identity had arisen, but his work since 1947, recorded on one of the buff cards, had inadvertently been omitted in the preparation of the work histories for the mortality cohort study. Although it will be necessary to make appropriate amendments on several files relating to that study before any further

analysis is undertaken, it would have been extremely costly to make the corresponding changes for Cohort "A"; further, this man was still living at the end of 1975 and so of less importance from an epidemiological point of view than a man who had died. He was, therefore, excluded from Cohort "A".

Thus, at the final count, this cohort consisted of 988 men. All were traced to the end of 1975, when 858 were still living (86.8 per cent). A total of 130 had died (13.2 per cent), and cause of death had been ascertained for all but two of the deceased. The attrition from the early count of 1,031 men was of twenty in the "main mortality cohort" and 22 others; none of the latter had died by the start of 1976. (It would have required several additional computer runs to determine the status - alive, dead or lost to view - of those two members of the "main mortality cohort" excluded because no film could be traced on the TRIPLF file or the 17 excluded because there was no complete set of six readings.)

7.6 Cohort "B": definition, sources and numbers

At the outset, it seemed that the enumeration of Cohort "B" would be a comparatively simple task involving only the manipulation of files on magnetic tape. Members of the "main mortality cohort" were defined on ASBMORT, whether an x-ray had been assessed was indicated on TRIPLF, which also gave the reading serial number, and the readings themselves were available on BASICX, indexed by that reading number. However, the complications were great, arising mainly because of men for whom there were now, or had ever been thought to be, more than one buff card in the original register of the complete working population

of the industry. The solution of all identity problems had to be confirmed, and additional difficulties arose for several reasons. First, a man's film might be indexed on TRIPLF under any, some or all of (a) the six-digit numbers identifying his various buff cards and (b) the special number, of less than six digits, allocated to all his records at the time TRIPLF was created (1969-70). Secondly, there might be more than one film for such a man, with increasing problems of accurate linkage. Thirdly, two further files created about the same time, i.e. 1969-70, IDCRDF and SAMASF, were brought into use in the expectation that they would aid the elucidation of the various problems of linkage; however, the first was found to be incomplete and to contain some further errors which, although few in number, rendered the file unsuitable for use as planned, while the latter was found to be even more out-of-date than had been anticipated and to contain certain information of which we could make no sense. Finally, the fact that we had failed to find a complete set of six readings for certain men expected to be in Cohort "A" meant that these men were now available for inclusion in Cohort "B", although initially they had been excluded on the grounds of *prima facie* membership of the other cohort.

Had these shortcomings and difficulties been fully appreciated at the start, it would have been possible to specify a simpler system of data processing, which would have been just as reliable. However, by the time the problems were fully manifest, the original plans were well advanced and there appeared nothing to be gained from abandoning them and starting afresh. Several contingency manoeuvres had to be developed, all devised to "fail safe", and so, at the end of the

process, we were certain that although longer and more complicated than it need have been it had been carried out correctly. In other words, no subject had been excluded from consideration unnecessarily, and none included unless there were no reasonable doubt that the x-ray reading was of a film of the person defined.

After the long process of matching and checking, we found a total of 5,425 cases on both ASBMORT and TRIPLF. Of these, 764 had more than one buff card, but only one film had been read for the majority of them. Indeed, there were only 22 persons for whom readings of two films were traced - too few for any study of differences in readings - and the later film was selected, after a careful study to be sure of its identity, in conformity with criterion (1) of section 7.4 above. We expected to exclude all 604 men from Cohort "A" who were also in the "main mortality cohort" but, because he was under different identity numbers in the two sets of data, one remained in Cohort "B", which at this stage comprised 4,822 cases.

The file BASICX contained 42,022 readings of the films for 15,689 persons. Almost all the films (15,570, or a shortfall of 0.8 per cent) had been read at Stage 2 and so were acceptable for Cohort "B", always provided the subject of the film was in the "main mortality cohort". The 4,822 cases as above were linked with the Stage 2 readings and films for all but 32 men were traced and the readings added to the record; the number of subjects now included was 4,790. The shortfall was 0.7 per cent of 4,822, very similar to that in the complete BASICX file; almost certainly, it was due to the films being

considered "unreadable". There seems no reason to believe that their exclusion could have led to bias in answering the questions of section 7.2. For reasons already given, we also excluded the 231 females, leaving a final count of 4,559 men in Cohort "B".

As before, the rather extensive basic file was cut down as far as reasonable to reduce costs of disc storage and of analysis, and the basic details on the working file (XMBFWF) are given in Table 7.4. Definitions of net service and accumulated dust exposure are as in Liddell, McDonald and Thomas (1977, p. 471). Smoking habit was re-coded from the record on the ASBMORT file as follows:- 0 = non-smokers; 1 through 5 indicate various categories of those believed not to be current cigarette smokers; 6 = 1-12 cigarettes a day; 7 = 13-25 cigarettes a day; 8 = 26-65 cigarettes a day; 9 = not known. This last category (9) included those for whom a question-naire had not been completed and others where interpretation was doubtful; the category was reduced by about twelve per cent by care-ful scrutiny of the latter group.

X-ray readings were complete in the same sense as for Cohort "A"; see previous section. To maintain "blindness" in study of inter-obs-erver differences, the readers' identity numbers were altered by ranking the readers according to the number of subjects whose films they had assessed. Once the specification for this process had been written, the re-labelling was carried out by Mr. Mario Rodrigues with-out further reference to the author, and the computer output contain-ing check totals etc. was verified by a means which meant that the

Table 7.4

BASIC INFORMATION FOR MEN IN COHORT "B"

Columns	Item and "format"	
1	Sex (M)	A1
2	Set (Reader)	I1
9-14	Identity number	I6
15-20	Date of birth (day/month/year)	3I2
30-34	Clinic and reference number	A1,I4
35-39	Reading sequence number	I5
40	Status (Alive, Dead, or Lost ot view)	A1
41	Quality of match in tracing	A/N
42-43	Year of death, or loss (blank if alive)	I2
45-47	Cause of death (blank if alive or lost to view)	I3 *
48-51	Date of start of first job (year/month)	2I2
52-55	Date of end of last recorded job (year/month)	2I2
56-59	Net service (years)	F4.2
60-66	Accumulated dust exposure (mpcf.y)	F7.2
67	Mining area (Asbestos or Thetford Mines)	A1
68-110	X-ray reading' 43 columns laid out as in p. 153 of the Annex	
137	Smoking habit, recoded	I1

* First digit R or S for accidents at asbestos work.

author was kept blind * to the identities of the six readers.

The status of the 4,559 men in Cohort "B" at the end of 1975 was as follows:- 2,957 known to be alive (64.9 per cent); 1,543 dead (33.8 per cent); with 59 lost to view (1.3 per cent). This last percentage is considerably less than in the entire "main mortality cohort", where the losses numbered 1,089 among 10,939 males, or 10.0 per cent. Most of these losses were after short service before 1936, the year in which routine radiology was introduced at Asbestos. Of the losses in Cohort "B", three-quarters were in men whose last employment was before 1951. Cause of death was determined for all but 16 cases (1.0 per cent), mainly among earlier deaths. In the entire "main mortality cohort", there were 125 deaths of unknown cause in a total of 4,463 male deaths (2.8 per cent), a difference again accounted for by the considerably higher proportion of deaths in the entire cohort before the 1950s.

It is usual in follow-up studies to accept the tracing, once achieved, as valid, without further investigation. For the main mortality enquiry, a code had been used to indicate the quality of the linkage of records from various sources. Table 7.5 illustrates how well the tracing had been carried out for the 4,500 men in Cohort "B" known to be alive in 1976 or to have died earlier. The code was devised to reflect the greatest amount of disagreement

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* The key, still not read by me, was sealed and has been lodged with Professor McDonald, as my supervisor.

Table 7.5

QUALITY OF RECORD LINKAGE

4,500 men in Cohort "B" known to have died or been alive in 1966

X: Surname and at least one forename identical or close match (i.e. phonetically compatible, accepted alternative or French/English equivalent.)

Y: Match of names less good than above, but considered adequate.

Z: Match of names themselves inadequate, but match confirmed by other evidence.

1: Full dates of birth available in all records, and identical.

2: Full dates of birth available in all records, but one day and/or one year out. Full dates of birth available in all records, but day and month interchanged. Worse than above, but year of birth identical or out by one only.

3: Worse than above.

Alive or Dead Work history	Alive Single	Dead Single	Alive Multiple	Dead Multiple	Total
Number of men	2,564	1,315	393	228	4,500
Cumulative percentages					
X 1	*91.0*	*57.5*	*45.3*	*29.4*	*74.1*
X 1 and X 2	*96.8*	*91.0*	*79.6*	*69.3*	*92.2*
X 1 through X 3, Y 1 and Y 2	*99.6*	*98.6*	*94.4*	*93.9*	*98.5*
Above and worse	*100*	*100*	*100*	*100*	*100*

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X 1 through X 3, Y 1 and Y 2	*99.6*	*98.6*	*94.4*	*93.9*	*98.5*
Above and worse	*100*	*100*	*100*	*100*	*100*

between sources; quality would thus inevitably appear worse the more documents were involved. The minimum was a single buff card and a smoking questionnaire for the living; however, there were many men with more than one buff card, and of course there was also the death certificate for the deceased. The first two lines of percentages reveal the expected trends in the quality of linkage, but they also indicate extremely high standards of matching records. The linkage indicated by the third line would still be generally considered quite acceptable, and the percentages in each column of this line are uniformly high.

7.7 Summary

In order to answer the following questions:-

1. To what extent can radiological changes in those that have been exposed to asbestos dust predict mortality?
2. Are the predictions of mortality from radiological changes improved or modified by other factors?
3. What are the best factors for predicting mortality?

two cohorts of Quebec chrysotile miners and millers were defined.

Cohort "A" comprised 988 men who were at work at the end of 1966 and had been subjected, mainly in 1967 but in some cases in 1968, to a questionnaire on respiratory symptoms and smoking habits and to lung function tests. The latest chest radiograph to November 1966 had been assessed by each of six readers into the U/C classification, later published by Bohlig *et al.* (1970); see the Appendix at the end of Volume 1 of this thesis. Information available about each man is listed in Table 7.3. By the end of 1975, there had been 130 deaths;

no member of the cohort had been lost to view.

Cohort "B" comprised all 4,559 men who (a) were born in the years 1891 through 1920 and had worked in the chrysotile producing industry for at least a month (and so were included in the "main mortality cohort"), (b) had had an x-ray, the latest to 1966, assessed by one of the six readers mentioned in the previous paragraph, and (c) were not in Cohort "A". A list of the information collected about each man is in Table 7.4. By the end of 1975, there had been 1,543 deaths and 59 men had been lost to view.

The leading information about each man is given in parts "A" and "B" of the Annex to this thesis, bound as Volume 2. The reasons for the sequences in which the men are listed are given in section 8.4, below.

Chapter 8

M E T H O D S

8.1 Introduction

Methods of cohort analysis have for long been a matter of controversy, and will probably remain so despite a recent review by the present author and his colleagues (Liddell, McDonald and Thomas, 1977). This review came about because, from the beginning of their studies of mortality in Quebec chrysotile workers, Professor McDonald and his team were aware of imperfections in the methods they were forced to use in the initial analyses. Several improvements were introduced into later reports (McDonald *et al.*, 1973; 1974), one of them being an approach by taking several controls for each case which I had proposed unaware of Miettinen's (1969) suggestion. The selection of controls and the analysis were carried out by Dr. Gail Eyssen (*cf.* Eyssen and Liddell, 1974), but the method was not fully described in the literature at that time because it had not been adequately explored. There were still possibilities of improvement, and a small group of statisticians met at McGill University in June 1974 to discuss them. Many ideas were exchanged, and a full report (Liddell, 1974) was circulated to all concerned; after agreement, a brief summary was published by Liddell (1975b). Nevertheless, there still remained the need for the McGill group to employ the best possible means of analysis on their important data, and although it was now fairly clear how the basic

problems were to be handled it seemed appropriate to have the principles presented to a wide circle of experts for open discussion which would be published.

Now, only a year after the review was presented to the Royal Statistical Society, there have been further developments in analysis, as mentioned in the discussion and reply (Liddell, McDonald and Thomas, 1977, pp. 485-491) and as explained in a paper by one of the authors of the review (Thomas, 1978). This chapter discusses briefly the need for distinguishing modes of reasoning for analysis from what the study design may appear to imply; thereafter, the main methods readily available with either form of logic are outlined, with advantages and disadvantages, and, finally, the methods used to analyse the data from Cohorts "A" and "B" are described.

8.2 Modes of reasoning

The expression "cohort study" is being used increasingly to describe an investigation which takes a population of known characteristics and works forwards to the affected persons in that population; because of the direction of development, such studies have long been termed "prospective", in the epidemiological literature. However, that term has an everyday meaning, involving placing the enquiry in time, which is frequently misleading. Thus, both Cohort "A" and Cohort "B" were not set up until 1977, and much of the work on them has been, in the temporal sense, retrospective. This is not an unusual situation, and a study of the present nature has sometimes been called a "retrospective cohort" study, which denies the traditional, epidemiologic, use of the adjective (see, for example, Taylor and

Knowelden, 1957; McMahon *et al.*, 1960).

Because a cohort study is prospective, in the sense first used above, it can be analysed by an argument that also works forward, i.e. from cause to effect; such an argument has, since 1710, been styled *a priori*, according to the Oxford English Dictionary. The fundamental application to cohort studies such as the present is as follows:- (1) the population is classified into sub-cohorts according to known characteristics (such as normal or abnormal radiological findings; light or heavy dust exposure; non-smokers, light smokers or heavy smokers); (2) the sub-cohorts are followed through an appropriate "study interval"; (3) the numbers of deaths in each sub-cohort are counted; and (4) comparison of sub-cohorts is made in terms of selected measures of mortality. The mode of argument has clearly been from cause (abnormality of x-ray, dust exposure, smoking) to effect (mortality), i.e. *a priori*.

However, this is not the only form of argument that can be used in a cohort study; it remains possible to compare the characteristics of those who have died against those of the survivors. Here the mode of argument is a working backwards from the fact of death (or survival) to the factors which may have led to the differences. It is equally clear that this style of argument is from effect to cause, designated *a posteriori* (also since 1710 according to the OED). There are two rather different applications of this mode of reasoning. In one, each death, as it occurs, is compared with all still surviving; methods of analysis have been put forward by Cox (1972), based on regression models, and developed by others, including for example

Thomas (1978). In large cohort studies, comparisons of this nature are extremely costly, even with efficient programs for fast computers, and so sampling of the survivors is desirable. If a fixed number of survivors, now called "controls", is selected for each death (or "case"), the material is exactly equivalent to that of a "case-control" study. The method of Cox (1972) and its successors can be applied, but they are rather complex; simpler methods are available in many circumstances. The principle of sampling the living was recommended by Liddell, McDonald and Thomas (1977), and endorsed in the discussion.

The case-control design (previously called "retrospective" in the epidemiological sense, but quite possibly carried out prospectively in the everyday sense) has often been criticised because:- (a) it may be subject to severe bias in data collection; (b) it does not provide estimates of risk, but only of relative risk; and (c) in practice the principles by which the controls have been selected have left much to be desired. It is therefore important to appreciate that the use of *a posteriori* reasoning in a cohort study is of a rather different nature; in particular, criticism (a) will normally not apply (Armitage, 1977). On the other hand, there is no denying that the criteria by which controls are selected are just as important here as in other case-control situations.

8.3 Comparative composite cohort analysis

By far the most generally used applications of *a priori* analysis in the study of mortality in a cohort have been based on "comparative composite cohort analysis", the method that Case and Lea (1955) used for investigating the effects of mustard gas poisoning during World

War I. The cohort is divided into a number of sub-cohorts, who are followed over an appropriate "study interval" (see below). The mortality experienced by each sub-cohort is compared with the experience expected for that group on the basis of some "reference population" (see below). Sex-, age-, year-, cause-specific death rates for the reference population are applied to the "man-years in view" accumulated by the sub-cohort, the man-years being found by summation, over all persons in the sub-cohort, of the years between the start of the study interval and the earliest date of: death, last trace for those lost to view, end of study interval or, when the study interval extends beyond the period of follow-up, the end of follow-up of those alive at that time. In theory, this can be done for each one-year square (or one-year triangle) in the relevant diagonal band of the Lexis (1875) diagram, which has age and time as orthogonal axes. However, the practice has been to use five-year squares to reduce the bulk of the computations.

The choice of *study interval* is of importance, particularly to avoid the fallacy, which appears to have been pointed out first by Enterline and Henderson (1973), that long exposure tends to improve life expectancy; in other words, only those who live to a considerable age have the opportunity to acquire long exposure. It is also important that the classification of sub-cohorts be made on the values of the appropriate characteristics at the start of the study interval, or earlier, but never later.

In Cohort "A", all men were known to be alive at the time of physiological testing, and this has been taken as the start of the

study interval. In Cohort "B", where one set of factors of sub-cohort definition comprised the radiological changes, these must have preceded the start of the study interval. However, as the men were also known to be alive at least until the end of their last recorded job, the study interval could not start earlier than that for each man; otherwise, man-years would be accumulating in the denominators of mortality rates while the numerators remained void until the recorded date of last employment. This date has, then, been taken as the start of the study interval for Cohort "B".

The choice of *reference population* is also not without difficulty. The usual selection has been of national mortality, following Case and Lea (1955), or mortality for other major populations, such as all males in the Province of Quebec (e.g. McDonald and Liddell, 1978) or white males in the United States (e.g. Enterline *et al.*, 1972 etc.) However, these may not be relevant to any specific occupational cohort, because of geographic and socio-economic factors, to say nothing of factors of selection into and out of the cohort. Even rates of a more localized nature may fail to overcome the socio-economic difficulties, and there is no reason to believe that they will improve the situation over selection. Some workers, for instance Dr. I. J. Selikoff's group at Mount Sinai School of Medicine, New York (Selikoff *et al.*, 1978), amend the officially coded cause of death whenever they consider this justified by study of the death certificate or by autopsy findings. However, there would appear to be no way in which corresponding changes could be made for any external reference population, so that it is not possible to find appropriate expected numbers of deaths against which to compare the adusted observed numbers.

Fortunately, these issues are not of great importance if the aim is to make comparisons between sub-cohorts, without trying to obtain absolute risks for each sub-cohort separately. Indeed, for this aim, any reference population will be satisfactory if its mortality experience is not too widely different from that in the cohort under review. Nevertheless, there are often advantages in an internal reference, i.e. comparing sub-cohorts each against the experience of the entire cohort. In particular, the difficulties over selection may be at least reduced, no special data collection is required, and no restrictions get imposed on the study interval by any lack of availability of material for specific years, or causes, for the reference population, while for normally rare conditions that are nevertheless not uncommon in the cohort, e.g. pneumoconiosis in those exposed to dust, the expectations will be more realistic for internal comparisons.

Liddell, McDonald and Thomas (1977) also discussed various ways in which observed and expected deaths could be compared. These workers had hoped that the potential of a flexible program for linear modelling, GLIM (Nelder, 1975), could be explored in the Quebec asbestos mortality study. However, Dr. I. D. Hill, who volunteered to carry out this exploration, has been unable to do more than preliminary analysis. Despite its drawbacks, the use of the simple ratio between observed and expected deaths, which can be seen to be a Standardized Mortality Ratio (SMR), is still widely used if only for its simplicity and universality; it has a further advantage over other ways of relating observations to expectations in the ease with which it can be partitioned in analysis by χ^2, in the ways described in general by Armitage (1971, p. 362 *et seq.*) and with particular reference to mor-

tality studies by Liddell, McDonald and Thomas (1977).

A major drawback of comparative composite cohort analysis is in the conflicting aims for the definition of sub-cohorts, which have to remain reasonably large if comparisons between them are to be in any way reliable. If there are several factors of interest, and particularly if their interactions may be important, they cannot easily be examined together, in mutually exclusive sub-cohorts, as is theoretically essential. Instead, they have to be examined one at at time, or in pairs, and this leads to unresolvable problems in the disentangling of "effects" when the factors themselves are correlated.

8.4 *A posteriori* methods of cohort analysis

The approach of comparing each death with all surviving to that time is theoretically possible, but is not feasible in large cohorts; further, it usually requires modification (of the sort described by Thomas, 1977) from the original proposals, which were devised for use in clinical trials, with rather different constraints (Cox, 1972). This section will therefore be concerned with situations in which the living have been sampled. Further, we will consider only purposive sampling, e.g. to find a set of "controls" against which certain "cases" can be compared; the number of controls for each case will be taken as fixed. This is simpler, and so more practical, than when differing numbers of controls are taken for each case, and there seems no need to extend the discussion further, although Thomas (1978) claims that the methods of analysis he has developed cope with all case-control situations.

Two important aspects of *a posteriori* analysis should be mentioned at the outset. First, all comparisons are internal; this has clear advantages, with the corollary that absolute risks cannot be calculated (except when a method such as that of Cox, 1972, is applied to a cohort in which there has been no sampling). However, unless the reference population in *a priori* analysis is truly relevant, such methods are also unable to produce reliable absolute risks. The second aspect is that, in all but the most exceptional circumstances, all cases of interest can be included. Even if some deaths are of unknown cause, the likelihood is that only a trivially few cases will be missed, while the effect on the controls is only that the pool from which they are to be drawn is a little smaller than otherwise.

There is much current argument (see, for example, Miettinen, 1970; McKinlay, 1975; 1977) about the value of matching in the sampling of controls. Sometimes matching is essential for validity, and then it would appear from a seminar sponsored by the *Journal of Chronic Diseases* in April 1978 (Drs. G. E. Eyssen and D. C. Thomas, personal communications) that the matching should be taken into account in the analysis. However, there are strong indications that results are often virtually unaffected by whether or not the analysis is performed treating the data as matched or unmatched. For example, it is difficult to distinguish the results obtained by Dr. Thomas (personal communication) of the two forms of analysis when applied to the lung cancer deaths to the end of 1975 and their controls, as described by McDonald and Liddell (1978). When controls have been matched with cases for disturbing variables (using the nomenclature of Cochran, 1965), no analysis can assess the effects of such variables, although interactions with

the variables of direct interest (which therefore have not been included in the matching) can be evaluated; they can, of course, be evaluated along with the effects of the disturbing variables themselves, if such variables are not matched for. Thus, a strong argument is being developed for reducing matching to that essential for validity.

As in all forms of case-control investigation, the criteria for the selection of controls are vital. In the Quebec mortality studies, the fundamental criteria were that selection would be strictly at random from among men born in the same year as the case and known to have survived at least into the year following that in which the case died.

Although discussion up to this point has been confined to case-control situations, this is no restriction on *a posteriori* argument. The foundation of such methods is some form of discrimination - so far between the characteristics of the dead and those of the living. The same mode of argument can be applied to comparison between any groups of cases, without reference against "controls". An example would be a comparison of exposure factors in smokers and in non-smokers all of whom had died from lung cancer. It would not then be reasonable to match, in the same way as before, for time variables, but rather to see whether one of the important differences between smoking and non-smoking lung cancer cases was, for instance, age at death. Nor need the groups compared be only two in number. Thus it has recently been possible to examine "latent periods" (i.e. the intervals between first exposure and death) in the 245 lung cancer deaths within the Quebec "main mortality cohort", classified *post hoc* according to dust exposure accumulated to age 45; the first results are in Table 2.2.

Exploitation of such methods of reasoning leads to great flexibility in hypothesis formulation.

8.5 Methods for *a priori* analysis of Cohorts "A" and "B"

Whatever advantages lie with *a posteriori* reasoning, there can be no doubt that the traditional approach to the analysis of a cohort has attractions, although the simplicity frequently claimed for it (for example by Doll, 1964; Fox, 1977) is more apparent than real. It remains important to carry out certain basic analyses using *a priori* reasoning, and for the moment the best approach appears to be the comparative composite cohort analysis of Case and Lea (1955) - with the modification that Case and Lea used probabilities of dying, while most computer programs, such as MYCL (Hill, 1972) or PGM1A, written by Mario Rodrigues, to my specification, for the McGill analyses, use death rates.

For both cohorts, "A" and "B", we have used as reference the mortality of males in the Province of Quebec. Annual mid-year populations and deaths from all causes (i.e. total deaths), both by five-year age groups, have been published by Statistics Canada, and were punched from the latest revisions. The years covered were 1926 through 1974, and the material for the last year was reproduced to provide approximations for 1975. This has been called Quebec Deck 1. Cause-specific information was sought from Statistics Canada, who were able to supply details of every death, either sex, all ages, for the causes we had specified by codes from the Seventh Revision of the International Classification of Diseases, for the years 1951 through 1973. Programs were written (a) to assemble this material for males, in five-year age

groups, annually, for each of the 12 groups of specific causes listed in Table 7.1, and (b) to calculate the numbers of deaths for causes not specified in that table, by subtraction from the data in Quebec Deck 1. The material for 1973 was reproduced twice to provide approximations for 1974 and 1975, and this has been styled Quebec Deck 2.

Quebec Deck 2 was adequate for Cohort "A"; as already explained, the start of the study interval was, for each man, his date of test and this was never earlier than mid-1967. However, for Cohort "B" there were 116 deaths before 1951 and for these cause-specific death rates for Quebec over the period 1926-50 would have been desirable. Attempts had been made to assemble this material, which would of course have been useful in the main mortality studies, but great difficulties had been encountered. Despite the expenditure of much effort and many dollars, a substantial proportion of error was found in what had been punched, and it would have been necessary to spend much further effort if the material were to be made reliable. After discussion with my supervisor, it was decided not to proceed with this work, and so the only Quebec mortality rates available for 1926-50 were for all causes, i.e. Quebec Deck 1.

The data of Cohort "A" are considerably more extensive than those of Cohort "B", but on rather limited numbers. Nevertheless, because they include respiratory function and symptom details, they are of particular interest. It remained desirable to find, if possible, a sub-division of Cohort "B", as closely comparable as possible to Cohort "A", if only to ensure that the exclusion of the latter from those initially available for the former had not led to any unforeseen

biases. A sub-division to resemble Cohort "A" would consist of those still at work in November 1966 who were known to be alive at the end of 1967 - bearing in mind that no detail of the date of death or loss to view was available other than the year in which the event had occurred. The number of men meeting these criteria was 1,732, and it seemed appropriate to divide the remaining 2,827 into two further classes according to date of last job. The obvious choice was to conform with the dates of Quebec Decks 1 and 2: it was found that 1,422 men had left employment before 1951, and they were designated "Era 1"; 1,405 men had left employment some time from the start of 1951 to October 1966, or were still employed at November 1966 but died before the end of 1967, and they were taken to comprise "Era 2". The previously determined group of 1,732, as like as possible to Cohort "A", was termed "Era 3".

For Cohort "A", each man's study interval began at the time of the physiological test in 1967 or 1968. For Cohort "B" as a whole, the intervals started when the men left employment. However, when this cohort was split by Era, and when regard was taken of which Quebec Deck was available for use, the study intervals had to be adjusted as follows:-

Era		Start	End
1	Before 1951	End of last job	End of 1950
	After 1950	Start of 1951	End of 1975
2		End of last job	End of 1975
3		Start of 1968	End of 1975

It will be appreciated that the man-years accumulated for the three Eras separately do not sum to those for Cohort "B" in its entirety: the interval from end of job (November 1966) to the end of 1967 for

men in Era 3 has been excluded. Most analyses were carried out Era by Era, but as will be explained later it is inappropriate to present them in this way. The observed and expected numbers of deaths were therefore totalled, over the three Eras, to obtain corresponding values, and SMRs, for the entire Cohort "B". The numbers observed were correct, but the expectations were slightly low. One analysis was repeated on the complete cohort, and this allowed some study of the effects. Man-years from 1951 through 1975 accumulated for the entire cohort were 55,970, compared with 54,021 when the separate figures for the three Eras were added, a shortfall of 3.5 per cent. The corresponding figures for numbers of deaths expected were 990.3 and 965.5, a shortfall of 2.5 per cent; SMRs would of course be overstated by the same amount, i.e. 2.5 per cent. As was to be anticipated, the shortfall in expectations was consistent over each group of causes of death. Further, in the two sub-cohorts which could be compared on both bases, there was no suggestion of differences in the *relative* effects. While it would have been possible to repeat all the *a priori* analyses on the entire Cohort "B", this would have been expensive and could have gained little. It was therefore decided, again in consultation with my supervisor, that this additional work could not be justified.

A rather simplistic form of the first question to be answered (see Chapter 7, above) is: do those whose chest radiographs are assessed as completely normal have lower mortality than others? This has been answered, directly, in both cohorts, by classifying each subject as radiologically "normal" or "less-than-normal". A reading was taken as normal if there were no record of parenchymal change (profusion of small opacities 0/- or 0/0 for both rounded and

irregular; no large opacities), no record of pleural thickening (whether of the walls or affecting the costophrenic angles, the diaphragm or the cardiac border), no record of pleural calcification, and no "additional symbols". There were 302 subjects in Cohort "A" (30.6 per cent) in whom all six readings were normal. In Cohort "B", where there was only a single reading of each film, the criteria of normality were much easier to meet: 67.7 per cent of subjects were "normal". In both cohorts, mortality in the "normals" has been compared with that in the others, i.e. the "less-than-normals". In Cohort "B", the analysis was carried out separately for each reader.

The instructions for the use of the U/C (and later) classifications of radiographs include provision for recording 0/- for "barndoor" normal films, i.e. those whose parenchyma are so exceptionally normal that the reader has no doubt that all other readers would also call them normal (*cf.* Liddell, 1963). Not all film readers make substantial use of this coding and even for those who do it is rare in a working population. However, when it is used, it is most unusual for other abnormality to be recorded, and it is seldom indeed that other readers rate the parenchyma as other than normal. For a few of the "normal" men in Cohort "A", at least one reader had recorded 0/- for both rounded and irregular small opacities. In Cohort "B", the number of subjects recorded as "barndoor" normal differed from reader to reader, as was to have been expected. In both cohorts, these exceptionally normal cases were compared in terms of mortality with the rest of the "normals", by reader separately in Cohort "B".

A closer examination of the first question was possible from five

further analyses, for both cohorts, after sub-division according to a score of:- (1) small opacities, rounded and irregular combined; (2) large opacities; (3) pleural changes, other than calcification; (4) pleural calcification; and (5) "additional symbols". These analyses were not independent of those by "normality"; nor were they independent of each other. For each radiographic feature, the subjects were classified afresh and the "less-than-normal" subjects may have appeared on several occasions with positive scores for the various features.

The method of scoring the x-ray readings is given in Table 8.1, the first part summarizing the detail of the second. In Cohort "A", the six scores of each feature, one score per reader, were added together to give a summary score. For small opacities, rounded and irregular combined (SSC in Cohort "A" and SC in Cohort "B"), three classes were distinguished, i.e. "absent", "slight" and "present". Three similar classes of pleural changes (PL) were also distinguished in Cohort "B". All other features were dichotomized into "absent" and "present".

In preliminary attempts to answer the second question, about whether radiographic or other factors are important in predicting mortality, three further analyses were also carried out for each cohort, again without regard for independence. The factors were:- (1) mining area (two sub-cohorts in each case: Asbestos and Thetford Mines); (2) accumulated dust exposure (four sub-cohorts in each case); and (3) cigarette smoking (two sub-divisions of Cohort "A", with a third, "unknown smoking habit", in Cohort "B").

Table 8.1

TRANSLATIONS OF X-RAY READINGS

Part 1: Summary

PARENCHYMAL CHANGES

Small opacities: rounded		(profusion) x (# zones)
irregular		(profusion) x (# zones)
combined		SC
Large opacities		LO

PLEURAL CHANGES

Pleural thickening		PT = "Extent" x Grade
Costophrenic angle(s)	CP	
Irregular diaphragm	ID	
Irregular border	IBG	
Pleural, other, "total"		PO
Pleural changes, other than calcification		PL
Calcification (grade)		PCG
SYMBOLS		SY

Part 2.1: Detail (SCALING)

Small rounded opacities:	profusion	Scale AR *
	zones affected	Count (BR)
Small irregular opacities:	profusion	Scale AI *
	zones affected	Count (BI)
Large opacities		Scale LO †
Costophrenic angle		Scale D1 §
Pleural thickening:	diffuse	Scale D2 §
	plaque	Scale D3 §
	grade	0, 1, 2, 3 (PTG)
Irregular diaphragm		Scale D4 §
Irregular cardiac border		0, 1, 2, 3 (IBG)
Pleural calcification:	grade	0, 1, 2, 3 (PCG)
"Additional Symbols"		Count (SY)

* Scales AR and AI: Notional scale of Liddell and May, (1966, p. 9) except 0/- → 0

† Scale LO: (Well-defined) A → 1, B → 2, C → 3.
(Poorly defined) D → 1, E → 2, F → 3.

§ Scales D1 through D4: L → 1, R → 1, B → 2.

Part 2.2: Detail (SCORING)

Small opacities	SC = (AR x BR) + (AI x BI)
Large opacities	LO
Pleural changes, other than calcification	PL = D1 + (D2 +D3)(PTG) + D4 +IBG
Pleural calcification	PCG
"Additional Symbols"	SY

In Cohort "A" only, a further eight analyses were possible; these were again based on subdivision of the cohort for one factor at a time, not in combination. For each of persistent cough, persistent phlegm and wheezing, the classification was into those in whom the symptom was absent or present; the same subject may well have appeared more than once as showing symptoms. For breathlessness, the breakdown was into three grades: absent, slight and more definite, corresponding to the codes 0, 1 and 2 + 3, recorded on tape.

Three lung function measurements were considered, *viz:-* FVC, the $FEV_{1.0}/FVC$ ratio and MMF. In each case, we calculated deviations from the regression line relating the measure to height and age, and negative deviations greater in absolute value than one standard deviation of the scatter about the regression line were treated as "impaired". Adiposity was assessed in terms of $(weight)/(height)^2$, i.e. Quetelet's index (Billiewicz *et al.*, 1962), and a subject was considered overweight if his index was more than one standard deviation greater than the mean.

In all cases, Relative Risks were calculated for all causes (total deaths) and for each specific group of causes, and differences between sub-cohorts were examined by a modification of the usual χ^2 procedure. Where there were only two sub-cohorts for any factor under examination, there was only one degree of freedom for the χ^2 calculation. Otherwise, we partitioned the χ^2 orthogonally to allocate one degree of freedom to each partition in what appeared the most logical way. The procedures are illustrated in Chapters 10 through 12 on actual findings.

8.6 Methods for *a posteriori* analysis of Cohorts "A" and "B"

No detailed planning was possible before most of the results from *a priori* analysis had been obtained. It was then clear that, although the "normals" did indeed have lower SMRs than the "less-than-normals", and although important relationships had been found, one of the most interesting findings was of the failure of radiology *always* to predict mortality even where it might have been expected to. These phenomena required investigation, which involved design of the type discussed in section 8.4. But first, a conventional form of case-control analysis has to be described.

Case-control studies could, in theory, have been carried out in relation to all reported deaths. Certain considerations, however, reduced the cause groups of interest to six, which can best be discussed in three pairs: pneumoconiosis and respiratory cancer; accidents and strokes; respiratory tuberculosis and diseases of the heart. One would certainly expect pneumoconiosis to be detected on x-ray, provided that, after the x-ray had been taken, there had been neither significant progression of the disease, i.e. a late "attack", nor substantial additional exposure to a dust hazard (not necessarily asbestos). There appear to be two carcinogens for lung cancer - asbestos dust and, much the more powerful, cigarette smoking - which may act multiplicatively. There might well be what could be termed "smoking cancers", without undue exposure to asbestos, and these might not be detected on the radiograph; equally, there might be "dust cancers" in non-smokers which, although comparatively rare, would be detected, one might expect, through the pneumoconiotic appearances associated with the dust exposure.

As far as the second pair of causes, accidents and cerebro-vascular diseases, are concerned, the aim of case-control studies would be essentially to "prove a negative" - a thankless task, however important in theory. As for the third pair, both respiratory tuberculosis and deaths from heart disease might be linked in some way with dust exposure. However, tuberculosis, if seen on the x-ray, should certainly have been recorded, probably as an "additional symbol"; it might, of course, have been mis-diagnosed, perhaps as large opacities, or indeed the diagnosis on death certification might be incorrect. There might be cases where death was certified as due to a disease of the heart in which the underlying cause of death was dust-related, but there would certainly be many cases, probably the majority, where death recorded as due to heart disease was in no way related to asbestos exposure, and so could not be detected as pneumoconiotic change on the x-ray.

In the light of these considerations, the case-control studies were confined, after consultation with my supervisor, to pneumoconiosis and lung cancer. Further, largely because of the small numbers of such deaths in Cohort "A", the more formal case-control studies were restricted to Cohort "B".

For the two selected conditions, one control was chosen in respect of each case, 31 of pneumoconiosis and 118 of lung cancer. Ideally, the control would have been born in the same year as the case and have survived him, but ensuring that the control's survival was long enough made it necessary on a few occasions to relax the first of these criteria, but never by more than a year or at most two.

This form of matching is considered essential for the validity of the case-control comparison. Additionally, the control was selected from among men whose radiograph had been assessed by the same reader, but no other form of matching was felt necessary, or even desirable, in the light of recent debate on this issue. Where, as usually, there were several men from amongst whom a control could be selected, the choice was made by the author, by strict use of a process of randomization.

Two of the most important questions left unanswered by the *a priori* analyses were why some cases of dust-related disease which one would have expected to be detected on x-ray were not; and what was seen on the radiograph in the "less-than-normal" who later died. To these ends, we studied the readings of all cases of pneumoconiosis, lung cancer and mesothelioma, in both cohorts, and examined the provenance of those with "normal" and "less-than-normal" films.

The simplest form of analysis was applied in each of the *a posteriori* studies.

8.7 Summary

Traditional methods of cohort analysis have used *a priori* reasoning, i.e. from cause forward to effect, classifying the cohort into sub-cohorts which differ in the factors of prime interest, such as radiological features, and comparing sub-cohorts in terms of their mortality experience. However, the aims for defining sub-cohorts are conflicting: if they are to be large enough that measures of mortality will be reliable, it becomes extremely difficult to maintain

them mutually exclusive, and so the examination of more than one or two factors, and particularly of the interactions between several factors, is virtually impossible. Nevertheless, these methods *appear* simple in concept - and of explanation - and so they have been adopted here, at lest for exploratory purposes.

Both cohorts have been divided into sub-cohorts, *one factor at a time,* for each of the following factors: radiological normality or otherwise; presence of several radiographic features; mining area; dust exposure; and smoking habit. In addition, Cohort "A" has been correspondingly subdivided according as the subjects had symptoms or depressed lung function or were overweight, or not. A two-way breakdown of Cohort "B" was possible by radiological normality and reader.

Methods using *a posteriori* reasoning, from effect back to cause, are much more flexible, even in a cohort, or "prospective", design. Two causes of death, pneumoconiosis and cancer of the chest, have been singled out for detailed study by such methods, which have included more-or-less conventional case-control studies and also examination of the reasons for the apparent "failures" of the radiological system to predict mortality.

The leading data for each man are listed in two parts, one for each cohort, in the Annex of this thesis, which is bound as Volume 2. The order of presentation of the material, which should aid any approach of an *a posteriori* nature, either on the same causes of death but in greater detail or to other causes of death, and the lay-outs, are described in the Annex.

Chapter 9

DESCRIPTION OF THE STUDY COHORTS

9.1 Introduction

In this chapter, the study cohorts are described and compared. The first section deals with Cohort "A", and then Era 3 of Cohort "B" is examined, showing how little like Cohort "A" it turned out to be, despite the method of its selection. The three Eras of Cohort "B" are then compared, before the entire cohort is described, in as nearly as possible the same terms as for Cohort "A". Finally, it is shown that the six groups of men within Cohort "B" whose radiographs were assessed by the different readers were in fact, as was to have been expected from the method of randomization described by Rossiter *et al.* (1972), closely similar in all measured features.

9.2 The men of Cohort "A"

To recapitulate, these workers were the great majority, 97.3 per cent, of the "physiology cross-section", which has been fully described by McDonald, Becklake *et al.* (1972) and Becklake *et al.* (1972). All 988 men were still at work in November 1966, but the sampling had been heavily weighted towards older men, excluding altogether some of the youngest (i.e. less than 21 years old) and utilising small sampling fractions, except for the oldest of those still under 65 in 1966. All

men were interviewed in 1967 (about 85 per cent of them) or 1968, the earliest interview being in April 1967 and the latest in June 1968. As follow-up was from the date of the physiological testing, age at test is an important consideration: ages ranged from 22.1 years to 66.5, with a mean of 49.3 years. All of the 988 men were traced to the end of 1975; 130 had died.

Table 9.1 gives the distribution of the 988 men by age at test, and shows that the proportion coming from Asbestos (30.1 per cent, overall) was similar in all age groups. Net service, to November 1966, was closely associated with age at test, suggesting that the average age of first employment was about the same in each age group. Accumulated dust exposure was also associated with age at test, as were average dust "concentrations"; even after re-adjustment for the length of the working week, there can be no doubt that the older men had been subjected to much higher dust concentrations, on average, than the younger. The proportion of smokers of at least 13 cigarettes a day was largely independent of age, except for those aged 65 or more at the time of test.

Table 9.2, showing the two-way distribution of the 988 men, by net service and accumulated dust exposure, reveals the expected close association of these two factors; it is also compatible with the previously observed relationship between length of exposure and average "concentration".

The prevalence of each feature of radiographic change is shown, by age at test, in Table 9.3. There was, in most cases, a steady

Table 9.1

MINING AREA, SERVICE, EXPOSURE AND SMOKING: COHORT "A"

	Age at Test					
	<35	45-44	45-54	55-64	≥65	Total
Number of men	149	201	243	378	17	988
Percentage from Asbestos	26.8	27.9	30.5	33.6	-	30.1
Net service (years)	5.3	14.8	19.0	28.3	32.0	19.9
Accumulated dust exposure (mpcf.y)	22	128	215	429	687	259
Average dust "concentration" (mpcf)	4.1	8.7	11.3	15.2	21.5	13.0
Percentage smoking at least 13 cigarettes a day	68.5	73.6	66.3	62.7	41.2	66.3

Accumulated dust exposure (mpcf.y)	Net		
	<1	1, <5	5, <10
<1	5	4	
1, <3	5	54	
3, <6		25	4
6, <10		28	1
10, <30		23	60
30, <100		4	30
100, <300			
300, <600			
600, <1,000			
1,000, <2,000			
2,000 or more			
Total	10	138	95

Table 9.2

SERVICE AND EXPOSURE: COHORT "A"

service (years)				
10, <15	15, <20	20, <30	≥30	Total
				9
				59
1				30
4	5			38
33	5	10	8	139
40	73	70	17	234
19	55	101	61	236
1	16	49	51	117
	5	22	47	74
1	3	6	26	36
		5	11	16
99	162	263	221	988

Table 9.3

PREVALENCE OF RADIOGRAPHIC CHANGES: COHORT "A"

(Percentages of readings)

	Age at Test					
	<35	35-44	45-54	55-64	≥65	Total
Number of readings (six per subject)	894	1,206	1,458	2,268	102	5,928
Small opacities: rounded						
0/1 or greater	1.6	2.2	5.1	4.2	5.9	3.7
1/0 or greater	0.4	0.7	2.5	2.0	2.9	1.7
Small opacities: irregular						
0/1 or greater	6.2	11.3	17.8	25.2	34.3	17.8
1/0 or greater	1.7	2.2	8.0	12.5	19.6	7.8
Large opacities: present	-	0.4	0.3	0.8	-	0.5
Costophrenic angle involvement	0.1	3.6	5.8	10.3	5.9	6.2
Pleural thickening						
Grade 1 or greater	0.2	2.2	4.8	7.7	12.7	4.8
Irregular diaphragm: present	0.2	1.2	2.2	4.9	4.9	2.8
Irregular cardiac border	-	0.3	1.0	3.2	3.9	1.6
Pleural calcification	-	0.6	1.6	7.3	16.7	3.6
"Additional symbols"						
At least one recorded	2.5	5.2	4.1	11.2	15.7	7.0

gradient with age, often distorted only by the figure for the oldest group which contained only 17 men. It was to be expected that the older men, who had also had the heavier exposure to asbestos dust, would show more dust-related changes on x-ray than the younger men. The corresponding gradient for "additional symbols", which are largely unrelated to dust exposure, was again not unexpected, as chest diseases are known to be age-related. The small opacities seen were mostly irregular, there were very few large opacities, but substantial proportions of the men had pleural changes of one sort or another. Thus, reading into the earlier ILO, Geneva, 1958, classification, devised mainly for silicosis and coalworkers' pneumoconiosis and excluding pleural changes, would have produced a very much lower prevalence of radiographic abnormality. The first line of Table 9.4 shows how few men, particularly in the older groups, had films assessed as normal by all six readers. The remainder of this table gives the average values of the scores adopted for use in the *a priori* analyses. That the trends revealed here are closely similar to those of Table 9.3 is confirmation that the scoring system was acceptable for the purposes for which it was devised.

Table 9.5 indicates the prevalence of symptoms, age group by age group. The very low overall prevalence both of persistent cough and of persistent phlegm, and the lack of a gradient with age for the former, are not consistent with the data in Table 3 of McDonald, Becklake *et al.* (1972). This indicates clearly that the answers to the relevant questions, as recorded on the tape QUESTF, have been incorrectly interpreted recently. Although these symptoms are included in the analyses, they cannot be considered reliable.

Table 9.4

RADIOGRAPHIC SCORES: COHORT "A"

	Age at Test					
	<35	35-44	45-54	55-64	≥65	Total
"Less-than-normal" (%)	37.6	60.7	70.8	84.7	94.1	69.4
Small opacities (combined)	1.9	3.0	8.4	12.8	20.8	8.2
Pleural changes *	0.0	0.5	1.1	2.1	3.4	1.2
Pleural calcification	-	0.0	0.1	0.6	1.5	0.3
"Additional symbols"	0.2	0.3	0.3	0.8	0.9	0.5

* other than calcification

Table 9.5

RESPIRATORY SYMPTOMS: COHORT "A"

	Age at Test					
	<35	35-44	45-54	55-64	≥65	Total
Prevalence of symptoms (%)						
Persistent cough	8.1	5.5	6.2	6.3	5.9	6.4
Persistent phlegm	2.7	4.5	4.5	7.1	-	5.2
Breathlessness:						
Grade 3	0.7	1.5	4.9	8.5	17.6	5.2
Grades 3 and 2	9.4	12.4	19.3	28.8	35.2	20.3
Grades 3, 2 and 1	32.2	45.3	54.3	68.5	76.5	55.0
Wheezing						
Grade 2	2.0	6.5	12.8	16.4	-	11.0
Grades 2 and 1	24.2	36.3	42.4	45.8	47.1	39.8
Chest illnesses						
Average number of illnesses per hundred men	8.7	26.4	29.6	30.7	47.1	26.5

Fortunately, these are the only inconsistencies revealed for either cohort, when compared with published material, after allowance for the differences in selection and presentation. Thus, breathlessness, although in some sense standardized for age by the form of the questions, showed a clear and not unexpected gradient with age, as did the prevalence of wheezing. The average number of chest illnesses reported is given for the sake of completeness; this average also showed a clear gradient with age, a reflection of the increasing incidence of chest illness with ageing.

Mean values of height, weight and adiposity (assessed by Quetelet's index; see Billiewicz *et al.*, 1962), and of lung function measurements, are given in Table 9.6, by age at test. As frequently in a cross-section, the older men were shorter than the younger. Although weight was also negatively associated with age, adiposity varied only slightly with age, tending to be a little higher for the older than for the younger men. The three direct measures of lung function ($FEV_{1.0}$, FVC and MMF) all showed strong age-gradients, but the effects of differences in height between the age groups has not at this stage been allowed for. The ratio $FEV_{1.0}$/FVC tended to be slightly less in older men than in younger.

Adiposity and the four measures of lung function were each, separately, related to age at test and height, various combinations of age and height, and weight, by means of standard multiple regression analyses (Armitage, 1971, Chapter 10), using a program in the SPSS package (Nie *et al.*, 1970). Table 9.7 gives the coefficients of regression of each variable on age at test and height (units in Table),

Table 9.6

HEIGHT, WEIGHT AND LUNG FUNCTION: COHORT "A"

	Age at Test					
	<35	35-44	45-54	55-64	≥65	Total
Anthropometry						
Mean height (m)	1.72	1.70	1.68	1.66	1.64	1.68
Mean weight (kg)	73.4	73.0	73.4	71.5	67.8	72.5
Adiposity (1,000)W/H^2	2.48	2.54	2.58	2.59	2.52	2.56
Lung function						
$FEV_{1.0}$ (l)	3.93	3.45	2.94	2.45	2.08	2.99
FVC (l)	4.75	4.25	3.76	3.20	2.74	3.78
MMF (l)	4.32	3.75	2.93	2.34	1.92	3.06
Ratio: $FEV_{1.0}/FVC$	.830	.814	.781	.764	.766	.788

Table 9.7

LUNG FUNCTION AND ADIPOSITY RELATED TO AGE AND HEIGHT

(Cohort "A")

"Dependent" variable	Regression coefficients			Standard error	
	of age at test (years × 10^2)	of height (m)	constant term	after regression	before
$FEV_{1.0}$	- 4.0370	3.9582	- 1.6780	0.4976	0.7898
FVC	- 3.9676	5.4212	- 3.3868	0.5489	0.8688
MMF	- 5.8869	3.1807	0.6129	1.0323	1.2999
$FEV_{1.0}/FVC$	- 0.2209	-	0.8974	0.0806	0.0847
$(1,000)W/H^2$	-	-	2.5597	-	0.3701

together with the constant term in the regression equation, and the standard errors before and after regression analysis.

As had been hoped, and expected from the findings of Billiewicz *et al.* (1962), adiposity was virtually independent of height. It was also only slightly related to age at test; although the F-statistic was 8.09 (degrees of freedom 1 and 985) and so of enormous "statistical significance", the partial correlation coefficient was only 0.0903. Even after all age and height adjustments, the standard error was reduced only trivially, i.e. from 0.3701 to 0.3687. Both $FEV_{1.0}$ and FVC were clearly related to age at test and height, but to no other combinations of these variables, nor to weight. The multiple correlation coefficients were 0.7771 and 0.7757, respectively, with F-statistics of 750.6 and 744.0. The standard errors were considerably reduced by the fitting of the appropriate equation. Preliminary discussion with Professor Becklake had suggested that there might have been some errors in the recording of MMF, and slight confirmation appeared in the rather lower multiple correlation coefficient and F-statistic, compared with those above, i.e. 0.6089 and 290.1. However, these may do no more than reflect the greater inherent errors in the MMF test compared with that on which both FEV and FVC are based. In any case, the analysis confirmed that only age at test and height needed to be taken into account, and that there was considerable reduction in the standard error by doing so. The ratio $FEV_{1.0}$/FVC was virtually unrelated to height, as might have been expected from the close relationship with height of both numerator and denominator of the ratio. Adjustment for age appears justified, but could reduce the standard error only slightly, while

the coefficient of multiple correlation and F-statistic were only 0.3096 and 104.4, respectively.

A simple demonstration of the extent to which certain of the various factors were interrelated is given in the correlation matrix of Table 9.8. No great reliance should be put on the values of the actual coefficients, particularly as "statistical significance", at the five per cent level of tradition, is associated with a coefficient as low as 0.062. Most of the larger coefficients in this matrix, i.e. those greater than about 0.25 in absolute value, simply reflect phenomena already discussed. Thus, there were substantial correlations between all the radiographic scores (variables 1 through 4), and between them and:- age at test (variable 8), dyspnoea (variable 9), dust exposure (variable 6) and lowered FVC (variable 10, negative values). Dust exposure (variable 6) was directly related to age at test (variable 8) and to dyspnoea and depressed FVC (variables 9 and 10), whereas these last two were not closely correlated. Not shown, but not unexpected, were associations between certain measures of lung function; by far the largest (.80) was that between deviations from the regression predictions of MMF and the $FEV_{1.0}$/FVC ratio.

Finally, Table 9.9 shows the distribution by cause of the 130 deaths observed in Cohort "A", against expectations based on Quebec Deck 2. Also given in this table are the values of the χ^2 statistics obtained from the Observed and Expected numbers of deaths as $(O - E)^2/E$. These values were high, indicating considerable excesses, for pneumoconiosis and lung cancer; there was also a slight excess of deaths from laryngeal cancer.

Table 9.8

MATRIX OF CORRELATIONS: COHORT "A"

		Variable number									
		1	2	3	4	5	6	7	8	9	10
Radiographic scores											
"Abnormality"	1	·	.51	.31	.34	.05	.24	.00	.37	.13	-.09
Small opacities	2		·	.35	.19	-.01	.25	.03	.32	.23	-.17
Pleural changes *	3			·	.19	.05	.18	-.00	.28	.18	-.18
"Symbols"	4				·	-.01	.10	-.09	.22	.13	-.11
Environmental factors											
Mining area †	5					·	.04	.03	-.05	-.12	-.04
Dust exposure §	6						·	.04	.49	.23	-.21
Social factors											
Smoking	7							·	-.03	.11	-.03
Age at test	8								·	.28	·
Symptomatology											
Dyspnoea	9									·	-.19
FVC ¶	10										·

* other than calcification

† a positive sign means higher value of correlate at Thetford Mines

§ square root of accumulated dust exposure

¶ deviation from regression "prediction"

Table 9.9

DEATHS IN COHORT "A"

From date of test to the end of 1975

(O = Observed deaths; E = Deaths expected from Quebec Deck 2; SMR = O/E)
$\chi^2 = (O - E)^2/E$

	O	E	SMR	χ^2
Pneumoconiosis	3	0.14	*21.48*	58.43
Cancer of the chest	22	10.61	*2.07*	12.23
Respiratory tuberculosis	0	1.00	*0*	1.00
Other respiratory conditions	6	7.71	*.78*	0.38
Diseases of the heart	44	52.36	*.84*	1.33
Cancer of oesophagus or stomach	5	3.88	*1.29*	0.32
Cancer of colon or rectum	3	4.26	*.71*	0.37
Other abdominal cancers	5	3.17	*1.58*	1.06
Cancer of larynx	2	0.70	*2.87*	2.41
Other malignant diseases	11	9.49	*1.16*	0.24
Cerebro-vascular diseases	5	8.64	*.58*	1.53
Accidents	6	8.96	*.67*	0.98
Other known causes	16	18.05	*.84*	0.23
Cause not known	2	-	-	-
ALL CAUSES	130	128.95	*1.01*	0.01

9.3 Era 3 of Cohort "B"

It will be recalled that this subdivision of Cohort "B" was made with a view to comparisons with Cohort "A". Here are the percentage age distributions of the two groups:-

	Cohort "A"	Era 3 of Cohort "B"
	(Age at test)	(Age at Nov. 66)
Less than 35	15.08	-
35 - 44	20.34	-
45 - 54	24.60	69.69
55 - 64	38.26	27.31
65 and over	1.72	3.00
	100	100

The mean age at test in Cohort "A" was 49.3 years and at November 1966 in Cohort "B" was 52.9 years; adjustments to the start of the study interval in that cohort increased the difference between the mean ages to 4.7 years. Further, and perhaps more important, because there was a substantial proportion of the men in Cohort "A" who were born after 1920 but none, by definition, in Cohort "B", the *shapes* of the distributions are quite different. In addition, net service to November 1966 was 24.8 years in Era 3 compared with 19.9 years in Cohort "A", and dust exposure accumulated to November 1966 was 333 mpcf.y against 259, although this is a reflection of the length of employment difference rather than of differences in dust concentrations.

Although smoking habits had been assessed on rather different bases, there did not appear to be great dissimilarity between the two groups, and, after due allowance had been made for the number of

readers, the scores of radiological change tended to be a little higher in Era 3, no doubt because of the higher average exposure to asbestos. Finally, Table 9.10 gives the distribution by cause of the 241 deaths in Era 3, in the same layout as for the previous table, and shows a tiny shortfall of total deaths, and marked excess only for deaths due to pneumoconiosis.

Despite some of the similarities of these two groups of men, the differences in age distributions and in exposure are considered large enough to render further comparisons between them unrewarding.

9.4 The three Eras of Cohort "B"

Some important differences between the three Eras of Cohort "B" are illustrated in Table 9.11. The confounding of the restriction on dates of birth, to the years 1891 through 1920, and the definitions of the eras in terms of dates of last employment in the industry, meant that Era 1 was much the youngest of the three at the start of their study intervals, and Era 3 much the oldest, while the average length of study interval was much greater in Era 1 than for the others. Although there were only comparatively small differences between the Eras in their distributions by dates of birth, tracing problems had been much worse for men in Era 1. Another important difference arose from the much earlier start of routine chest radiography at Asbestos than at Thetford Mines; this led to a very much higher proportion of men from this town in Era 1 than in the other Eras, when the proportion reflects much more accurately the numbers employed in the two mining areas. One reason for the comparatively low rate of tracing in Era 1 was the short net service; many men would have had employment of very

Table 9.10

DEATHS IN COHORT "B", Era 3

1968-75

(O = Observed deaths; E = Deaths expected from Quebec Deck 2; SMR = O/E)
$\chi^2 = (O - E)^2/E$

	O	E	SMR	χ^2
Pneumoconiosis	11	0.25	*43.65*	462.25
Cancer of the chest	27	21.72	*1.24*	1.28
Respiratory tuberculosis	0	1.84	*0*	1.84
Other respiratory conditions	13	14.07	*.92*	0.08
Diseases of the heart	97	104.43	*.93*	0.53
Cancer of oesophagus or stomach	7	7.47	*.94*	0.03
Cancer of colon or rectum	4	8.03	*.50*	2.02
Other abdominal cancers	6	6.58	*.91*	0.05
Cancer of larynx	1	1.45	*.69*	0.14
Other malignant diseases	19	17.90	*1.06*	0.07
Cerebro-vascular diseases	11	15.40	*.71*	1.26
Accidents	16	15.51	*1.03*	0.02
Other known causes	26	34.78	*.75*	2.22
Cause not known	3	-	-	-
ALL CAUSES	241	249.42	*.97*	0.28

Table 9.11

DIFFERENCES BETWEEN ERAS IN COHORT "B"

	Era 1	Era 2	Era 3
Number of men	1,422	1,405	1,732
Distribution of man-years by age	%	%	%
Less than 45	*40.8*	*13.6*	-
45-64	*52.9*	*56.0*	*89.0*
65 and over	*6.4*	*30.4*	*11.0*
	100	*100*	*100*
Man-years accumulated per man	25.0	9.7	7.6
Average age on survival to the end of 1975 (years)	65.5	69.6	62.0
Lost to view (per cent of total)	3.1	1.1	-
Smoking histories not available (per cent of total)	11.9	2.8	0.9
Percentage of men from Asbestos	78.3	45.8	38.3
Net service (years)	6.4	21.8	24.8
Accumulated dust exposure (mpcf.y)	156	459	333
Average dust "concentration" (mpcf)	24	21	13

short duration only, and such persons are notoriously difficult to trace. Accumulated dust exposures in the three Eras represent the resultant of differences both in net service and in dust concentrations.

One other difference is even more dramatic: because all deaths in 1966 and 1967 had been excluded from Era 3, in the attempt to make it similar to Cohort "A", the proportional mortality was affected, as it was also by secular trends, particularly in the almost complete eradication of respiratory tuberculosis, and the growing incidence of lung cancer; some of the larger differences between Eras are illustrated below:-

	Era 1	Era 2	Era 3
Number of deaths (all causes)	532	770	241
Percentage due to:-			
Pneumoconiosis	*0.4*	*2.3*	*4.6*
Cancer of the chest	*5.5*	*8.4*	*11.2*
Respiratory tuberculosis	*7.1*	*2.7*	-
Other respiratory	*4.1*	*5.1*	*5.4*
Cerebro-vascular diseases	*4.5*	*8.7*	*4.6*
Accidents	*13.7*	*7.5*	*6.6*
Other causes	*64.6*	*65.2*	*67.6*
	100	*100*	*100*

As noted in section 8.5, the *a priori* analyses were, for the most part, carried out on each Era separately, but the findings reported below are for the complete cohort. No important differences between Eras were noted.

9.5 The men of Cohort "B"

To recapitulate, men were included in Cohort "B" if (a) they were born in the years 1891 through 1920 and had worked for at least

a month in the Quebec chrysotile producing industry, i.e. were in the "main mortality cohort", and (b) a radiograph taken during employment had been collected in 1967 and read at Stage 2 (as described by Rossiter *et al.*, 1972) by one of six readers, into the forerunner of the U/C classification (Bohlig *et al.*, 1970). The study interval for each man started at the end of his last recorded employment in the industry, and so "age at last job" is an important consideration: ages ranged from 15.1 years to 74.0 years, with a mean of 47.2 years. All but 59 were traced to the end of 1975; there had been 1,543 deaths.

Table 9.12 corresponds to Table 9.1, except that the distribution of men is by age at last job, as distinct from age at test, but for both tables age has been calculated from the start of the study interval. The proportion from Asbestos varied with age, because of the interaction between this factor and the earlier start of radiology in that area. As in Cohort "A", net service and accumulated dust exposure showed the expected trends. The average dust "concentration", however, is less easy to interpret, because of further interactions with time and place. The smoking percentage in this cohort was dependent on age at last job, perhaps reflecting habits varying with era of birth.

The two-way distributions, by net service and accumulated dust exposure, are shown for the two mining areas in Table 9.13. The diagonal patterns in both sections of this table are similar to that in Table 9.2. The differences between Asbestos and Thetford Mines are, of course, complicated by the varying proportions from the two areas by era of employment, bearing in mind that dust concentrations were

Table 9.12

MINING AREA, SERVICE, EXPOSURE AND SMOKING: COHORT "B"

	Age at Last Job					
	<35	35-44	45-54	55-64	≥65	Total
Number of men	901	738	1,702	878	340	4,559
Percentage from Asbestos	80.8	67.9	41.2	45.2	27.1	53.1
Net service (years)	4.2	8.4	21.3	28.6	34.0	18.2
Accumulated dust exposure (mpcf.y)	104	162	320	506	709	317
Average dust "concentration" (mpcf)	25	19	15	18	21	17
Percentage smoking cigarettes	67.9	63.5	65.1	54.3	40.7	61.3

Accumulated dust exposure (mpcf.y)	Net		
	<1	1, <5	5, <10
<1	137	7	
1, <3	109	30	1
3, <6	37	83	15
6, <10	21	74	7
10, <30	24	105	109
30, <100	5	87	65
100, <300		29	54
300, <600			33
600, <1,000			1
1,000, <2,000			
2,000 or more			
Total	333	415	285

Table 9.13 (A)

SERVICE AND EXPOSURE: COHORT "B" (ASBESTOS)

service (years)				
10, <15	15, <20	20, <30	≥30	Total
				144
				140
2				137
8	8			118
43	16	10	8	315
59	154	178	62	610
42	48	161	99	433
53	25	103	83	297
15	23	48	82	169
	8	12	35	55
		1	1	2
222	282	513	370	2,420

Accumulated dust exposure (mpcf.y)	N e t		
	<1	1, <5	5, <10
<1	31	8	
1, <3	33	27	3
3, <6	15	13	6
6, <10	8	23	3
10, <30	16	67	52
30, <100	7	48	42
100, <300	1	17	36
300, <600		3	14
600, <1,000		3	3
1,000, <2,000			5
2,000 or more			
Total	111	209	164

Table 9.13 (TM)

SERVICE AND EXPOSURE: COHORT "B" (THETFORD MINES)

service (years)				
10, <15	15, <20	20, <30	≥30	Total
				39
				63
5				39
3	3			40
31	7	12	17	202
70	67	95	35	364
62	72	212	144	544
22	50	148	110	347
6	12	124	87	235
6	8	63	97	179
1	4	18	64	87
206	223	672	554	2,139

substantially lower at Asbestos than at Thetford Mines, but in both areas decreased greatly over the years.

The prevalence of each radiographic feature is given in Table 9.14 (*cf.* Table 9.3). Fairly similar trends with age can be discerned, as well as a broadly similar pattern of the sorts of abnormality observed. Because there was only one reader of each film, the proportion of "less-than-normals" was much lower in Cohort "B" than in Cohort "A"; however, over half the men aged 65 or more at the time of last job (shortly after the x-ray in most cases) were assessed as "less-than-normal" - see Table 9.15 (*cf.* Table 9.4) which again reveals trends similar to those in the previous table, and confirms the utility of the scoring system.

The correlation matrix in Table 9.16 is of rather smaller dimensions than those for Cohort "A" (Table 9.8); it revealed bigger differences between mining areas than in the other cohort, particularly in dust exposure, but neither this nor any other association was unexpected.

Table 9.17 gives the numbers of deaths observed since 1950 against expectations based on Quebec Deck 2, and adds SMR and χ^2 as before. There was a large excess of deaths from all causes; in addition to the clearly significant excesses from pneumoconiosis and lung cancer, which were to have been expected, there were major excesses of deaths due to many other causes. This experience, rather different from that in the "main mortality cohort" of 10,939 men (McDonald *et al.*, 1979) from which Cohort "B" was drawn, requires explanation. Net service

Table 9.14

PREVALENCE OF RADIOGRAPHIC CHANGES: COHORT "B"

(Percentages of readings)

	Age at Last Job					
	<35	35-44	45-54	55-64	≥65	Total
Number of readings (one per subject)	901	738	1,702	878	340	4,559
Small opacities: rounded						
0/1 or greater	1.1	1.2	2.3	4.7	5.6	2.6
1/0 or greater	0.8	0.8	1.3	3.3	5.0	1.8
Small opacities: irregular						
0/1 or greater	4.1	9.3	16.9	27.1	30.6	16.1
1/0 or greater	1.4	5.1	7.1	15.9	22.4	8.5
Large opacities: present	1.2	0.4	1.5	2.7	3.2	1.6
Costophrenic angle involvement	4.3	2.6	8.6	11.8	17.9	8.1
Pleural thickening						
Grade 1 or greater	2.2	1.8	6.0	10.3	17.9	6.3
Irregular diaphragm: present	1.0	1.4	3.8	7.3	9.4	3.9
Irregular cardiac border	0.3	0.5	2.2	5.0	8.5	2.6
Pleural calcification	1.6	1.5	3.8	6.6	12.4	4.2
"Additional symbols"						
At least one recorded	6.1	5.6	8.1	13.1	22.6	9.3

Table 9.15

RADIOGRAPHIC SCORES: COHORT "B"

	Age at Last Job					
	<35	35-44	45-54	55-64	≥65	Total
"Less-than-normal" (%)	14.5	18.6	34.4	47.6	59.1	32.3
Small opacities (combined)	0.4	0.8	1.3	3.3	3.7	1.6
Pleural changes *	0.1	0.1	0.3	0.5	0.9	0.3
Pleural calcification	0.0	0.0	0.1	0.1	0.2	0.1
"Additional symbols"	0.1	0.1	0.1	0.2	0.2	0.1

* other than calcification

Table 9.16

MATRIX OF CORRELATIONS: COHORT "B"

		Variable number						
		1	2	3	4	5	6	7
Radiographic scores								
"Abnormality"	1	·	.58	.54	.46	.24	.23	.02
Small opacities	2		·	.37	.16	.16	.20	-.01
Pleural changes *	3			·	.16	.19	.18	.01
"Symbols"	4				·	.07	.09	-.03
Environmental factors								
Mining area †	5					·	.26	.04
Dust exposure §	6						·	-.02
Social factor								
Smoking	7							·

* other than calcification

† a positive sign means higher value of correlate at Thetford Mines

§ square root of accumulated dust exposure

Table 9.17

DEATHS IN COHORT "B"

1951-75

(O = Observed deaths; E = Deaths expected from Quebec Deck 2; SMR = O/E)
$\chi^2 = (O - E)^2/E$

	O	E	SMR	χ^2
Pneumoconiosis	31	0.91	*34.07*	994.95
Cancer of the chest	119	67.16	*1.77*	40.01
Respiratory tuberculosis	40	11.85	*3.38*	66.87
Other respiratory conditions	67	52.89	*1.27*	3.76
Diseases of the heart	566	415.93	*1.36*	54.15
Cancer of oesophagus or stomach	53	31.10	*1.70*	15.42
Cancer of colon or rectum	28	32.35	*.87*	0.58
Other abdominal cancers	21	23.19	*.91*	0.21
Cancer of larynx	4	5.02	*.80*	0.21
Other malignant diseases	93	71.60	*1.30*	6.40
Cerebro-vascular diseases	101	70.02	*1.44*	13.71
Accidents	110	61.36	*1.79*	38.56
Other known causes	183	146.92	*1.25*	8.86
Cause not known	11	-	-	-
ALL CAUSES	1,427	990.28	*1.44*	192.60

in Cohort "B" was 18.2 years compared with only 10.5 years in the complete cohort, and accumulated dust exposure was 317 mpcf.y compared with 202 mpcf.y. However, these are not the major reasons for the high excess mortality in Cohort "B"; rather the explanation lies in death at work or which followed retirement on grounds of ill-health. Except for Era 3, either of these events would have led to the inclusion of the man concerned in the study group. Indeed, in the whole of Cohort "B", a high proportion of deaths (27.9 per cent) occurred before the end of the year following that in which employment terminated; any such deaths among men still employed in November 1966 would move them from Era 3 to Era 2 and helps to account for an extremely high proportion of such deaths, 46.2 per cent, in Era 2. It is possible to infer from a statement on p. 472 of Liddell, McDonald and Thomas (1977) that in the entire "main mortality cohort", less that about a fifth of deaths to the end of 1975 occurred within twenty years of first employment, a startling contrast when it is borne in mind that the average period of employment in Cohort "B" was less than twenty years.

Although so many early deaths in Cohort "B" might suggest a denial of the so-called "healthy worker effect" (see for example Fox and Collier, 1977), evaluation would require a rather different analysis, in which all men in the cohort at work at a given time be followed from that point. However, what is clear is that Cohort "B" would not have been suitable for an investigation of mortality in relation to environmental factors. On the other hand, it in no way detracts from the data for the present purposes; indeed, the comparatively large number of early deaths allows the possibility of examining whether or not they could be predicted from a radiograph taken only a fairly

short time before death.

9.6 Randomization of men in Cohort "B" to film readers

The method by which the films for all 15,689 subjects were placed into thirty batches, five of which were to be assessed by each reader, was described by Rossiter *et al.* (1972); it was one of shuffling by dealing, common among card players as one of the best modes of randomization. In practice, even on the enormous numbers of large films, it seems to have worked extremely well. The 4,559 men in Cohort "B" had been classified by reader into what will be called "sets" varying in size from 727 to 787, against the average of 760; there is no trace of inequality in this allocation, particularly when it is recalled that one reason for differing sizes would be the proportion of films considered "unreadable", a proportion determined by the subjective judgement of the individual reader.

The total number of men lost to view was 59, distributed between sets as follows:- 5, 7, 9, 9, 14, 15. Again, there is no suggestion of factors other than chance at work. Of those traced, 34.3 per cent had died: the proportion varied between sets from 32.5 per cent to 36.4 per cent, again without any trace of other than chance effects: a test of the equality of proportions yielded $\chi^2 = 4.57$ with five degrees of freedom, a value that would be at least equalled in more than 60 per cent of allocations. Examination of the differences between sets in the numbers of deaths by specific groups of causes suggested once more that all differences could be accounted for by the effects of randomization. Similarly, the six distributions of subjects by quinquennium of birth, within set, were tested for equality; here the χ^2

statistic has 25 degrees of freedom and was calculated as 30.47, with associated P-value of a little less than 20 per cent.

Finally, three other variables were examined:- age at last job; net service; and accumulated dust exposure. A one-way analysis of variance was carried out on each to test the hypothesis that the means were equal in the six sets. This led in each case to an F-statistic with 5 and 4,553 degrees of freedom; the values of F were 1.08, 1.45 and 0.54, respectively, all without trace of statistical significance - on the null hypothesis, the median value of F would be close to 4.4, and statistical significance at the traditional five per cent level would require F as large as 11.1.

It would therefore seem that the randomization achieved its purpose in equalizing the sets, and hence that differences that did exist between the six sets of subjects in terms of radiographic assessment or relations of such assessments to mortality or other factors were almost certainly due to differences between the readers. In fact, the percentage assessed "less-than-normal" by each reader is given, by age at last job, in Table 9.18. Although, within reader, a gradient with age is obvious, its severity depended on the reader: the prevalence of "less-than-normals" in those 65 and over, expressed as a ratio against the corresponding prevalence in those less than 35 on last employment, varied from 67.7 / 18.5 or 3.66:1, for Reader 6, to 60.0 / 11.3 or 5.31:1, for Reader 3. More important is the very large difference between readers in their assessments of overall prevalence of abnormality, varying from 20.9 per cent (Reader 1) to 39.9 per cent

Table 9.18

RADIOGRAPHIC ABNORMALITY, BY READER

(Percentages of readings assessed as "Less-than-normal")

	Age at Last Job					
	<35	35-44	45-54	55-64	≥65	Total
Reader 1	11.1	11.2	19.9	32.8	45.6	20.9
Reader 2	11.5	11.4	24.4	33.1	47.2	23.8
Reader 3	11.3	14.7	43.1	60.9	60.0	37.1
Reader 4	16.5	28.8	44.1	54.4	70.6	39.9
Reader 5	17.4	24.8	37.2	46.8	67.9	35.0
Reader 6	18.5	20.9	37.2	53.1	67.7	36.2
ALL READERS	14.5	18.6	34.4	47.6	59.1	32.3

(Reader 4). These differences are of undoubted significance: χ^2 was calculated as 105.85, with five degrees of freedom, and the P-value is extremely low.

9.7 Summary

Although the 988 men in Cohort "A" were considerably older than the population at work in November 1966, the period of follow-up was short - no more than seven-and-a-half years - and only 130 had died by the end of 1975, almost exactly the number expected on the basis of Quebec male mortality. However, there were excesses due to diseases known to be asbestos-related, i.e. pneumoconiosis and cancer of the chest; excluding these causes, there were 105 deaths compared with 118.20 expected, giving a Standardized Mortality Ratio of 0.89. As was to have been anticipated, both from first principles and from previous publications, the older men in this cohort had had longer service, at heavier dust concentrations, than the younger. The older men were a little shorter, but of similar adiposity, and they had fairly similar smoking habits; they reported more respiratory symptoms, and exhibited more signs of pulmonary abnormality, both radiological and physiological. Errors in data processing meant that the symptoms listed as persistent cough and persistent phlegm were not reliably transcribed; it is possible they refer to particularly severe manifestations of bronchitis.

Cohort "B" did not overlap with Cohort "A", and the 4,559 men in it contained much more than a representative proportion from Asbestos, Quebec, because of the earlier start of routine radiology there. The

method of selection also meant that mortality was high, especially from sudden death, including accidents, myocardial infarction and stroke. There were also large excesses from pneumoconiosis and chest cancer. As in Cohort "A", men who were older when they left employment had had longer service; however, because they might have left at any time in the period from the start of radiology to the end of 1966, there was little variation in average dust concentration with age at leaving. Those older at leaving included a lower proportion of cigarette smokers than the younger. Again as was to be expected, the older showed more radiographic change, both parenchymal and pleural, and of "additional symbols".

Chapter 10

F I N D I N G S I

10.1 Introduction to Chapters 10 through 13

Chapters 10 through 13 present the findings from both study cohorts. Those from Cohort "A" are given before those for Cohort "B", but even although the radiological assessments were made by six readers the many fewer deaths in Cohort "A" (130 compared with over 1,400) render the findings in this cohort less reliable than those in Cohort "B"; further there are several tables in which it would be impossible or unrewarding to provide results for the smaller cohort.

In Chapter 10, the first concern is with total mortality, i.e. that from "all causes", in relation to whether the radiograph was assessed as "normal" or "less-than-normal", with consideration of differences between readers for Cohort "B". The study is then extended to examine, one after the other:- deaths due to specific groups of causes; various radiographic features; and other factors, environmental, physiological, etc.

Pneumoconiosis, cancer of the chest and mesothelioma are all asbestos-related diseases and should be associated with x-ray change, although the rapid progress of the malignant conditions may make direct association with radiographic change tenuous. These three causes of

death are studied in considerable depth in Chapter 11. Deaths from respiratory tuberculosis and other respiratory conditions, and some deaths recorded as due to heart diseases may also be asbestos-related; they might be manifest on the chest radiograph either directly or perhaps indirectly through the pneumoconiotic changes associated with the dust exposure. Relationships between x-ray changes and deaths from these causes are also described in Chapter 11.

Whether or not deaths from the other groups of causes of Table 7.1 are dust-related, any x-ray changes could surely only be pneumoconiotic and related at best indirectly to the disease process. Deaths from these causes (cancer of oesophagus or stomach; cancer of colon or rectum; other abdominal cancer; cancer of the larynx; other cancers; strokes; accidents; and known causes not already included) form the subject of Chapter 12. A brief summary of findings is in Chapter 13.

In many of the tables, the following headings:- O, E, SMR, RR and χ^2 are used, with the meanings indicated below.

O represents the number of deaths observed, from 1951 or later start of the study interval to the end of 1975.

E represents the number of deaths expected on the basis of mortality in Quebec, 1951-75, i.e. calculated by comparative composite cohort analysis (Case and Lea, 1955) using Quebec Deck 2 as reference; note that estimates have had to be made for the last two years - see section 8.5, above.

SMR is the ratio O/E, which can be shown to be a mortality ratio standardized, by the "indirect method" (*cf.* Liddell, 1960), for both age and year of death.

RR, or Relative Risk, is the ratio of the two SMRs it stands beside.

χ^2 designates a statistic which, asymptotically, has the χ^2 distribution with one degree of freedom. The various methods of calculating this statistic are discussed as each is introduced.

The values of Relative Risk, RR, measure the size of an effect, those of χ^2 give an indication of its "statistical significance". The critical values of the theoretical distribution are as follows (calculated from Diem, 1962):-

Probability	10%	5%	1%	0.1%	0.01%	0.001%
χ^2	2.71	3.84	6.64	10.83	15.14	19.51

There are several reasons why this brief table does not give reliable estimates of the significance of Relative Risks quoted in these chapters. First, many are based on expectations so small that the distribution of the statistic is not close to the asymptotic form. Secondly, the statistics have not been determined independently. Thirdly, many statistics have been calculated for both cohorts, and the problems of simultaneous inference arise (see, for example, Miller, 1966). A fourth difficulty, of interpretation, derives from the fact that the χ^2 statistic, which has the form $(O - E)^2/E$, is of dimension E (or O), in other words depends on the numbers of deaths in the test. This means that the same Relative Risk in Cohort "B" as in Cohort "A" would lead to a value of χ^2 about ten times larger in the cohort with over 1,400 deaths than in that with only 130.

Nevertheless, the χ^2 statistics can be used as a guide to the relative importance of an observed Relative Risk, although no P-value

can be associated with it. With this proviso, and with particular caution, guidance can still be obtained even when expectations are very small. It is important not to make the best (i.e. an accurate probability statement) the enemy of the good (i.e. a less reliable but still revealing approximation). Many statistical texts state that χ^2 statistics should not be calculated in any situation where an expectation falls below some arbitrary value (often 5), although Cochran (1954), among others, has suggested considerably more lenient rules*.

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* An illustration of what can be lost by rigid application of a rule of thumb can be found in considering the incidence of mesothelioma in Cohort "B". From the data of McDonald and McDonald (1977), adjusting the proportional mortality of mesothelioma in Canada (i.e. 25 male deaths in 1970 in a total of 82,052 deaths from all causes in men over 24 years of age, or 0.305 per thousand) by a factor of two to allow for the higher ascertainment rate of mesotheliomas in the Province of Quebec, the number of mesotheliomas to have been expected in Cohort "B" may be estimated as 2 x 0.305 x 1,427/1,000 or 0.87, whereas five were observed; the value of $\chi^2 = 19.62$, with associated probability a fraction less than 0.001 per cent. While this P-value is in no sense precise, it does suggest that as many as five mesotheliomas in Cohort "B" did not arise from purely chance factors. In Cohort "A", the corresponding figures are:- O = 1; E = 0.08; $\chi^2 = 10.70$, with probability, from the tables, a little greater than 0.1 per cent. Here, with only one observed death, the finding *by itself* would have to be treated with great caution; nevertheless, in conjunction with that in Cohort "B", it provides convincing evidence of a serious mesothelioma risk.

10.2 Total mortality in men with "less-than-normal" radiographs

Mortality from all causes is summarized for both cohorts in Table 10.1, in relation to whether a subject had been assessed as "normal" or "less-than-normal". The χ^2 statistics have been calculated by multiplying each tabled value of E by a factor (all observed deaths) / (total of expected deaths). In this way, expectations were obtained for the calculations which were, in total, the same as the total of deaths observed, and which then allowed an internal comparison of the two sub-cohorts defined in terms of radiological findings. It can be shown, by simple but tedious algebra, that the statistic can be calculated as

$$\chi^2 = (RR - 1)^2 \, O_1 \, O_2 \, / (RR)(O_1 + O_2)$$

where RR and O have the usual meanings and the suffixes indicate the two groups being compared.

In every case of Table 10.1, the Relative Risk of the "less-than-normals" compared with the "normals" was greater than unity. As indicated by the values of χ^2, the effect was rather less significant for Cohort "A" than for Era 3 of Cohort "B", although similar in magnitude (RRs of 1.24 and 1.33, respectively), at least in part due to the differences in the numbers of deaths. The effects were undoubtedly large for the other two Eras of Cohort "B" (RRs of 1.59 and 1.72). One reason for these differences may lie in the exclusion of deaths before the end of 1967 from the two groups recently at work and with comparatively low Relative Risks. In the two earlier Eras of Cohort "B", the intervals between x-ray and death were short for substantial proportions of the men, and "prediction" of mortality from radiographic changes may thus have been improved.

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Table 10.1

RADIOLOGICAL ABNORMALITY AND TOTAL MORTALITY

		O	E	SMR	RR	χ^2
Cohort "A"	"Normal"	21	24.91	.84		
	"Less-than-normal"	109	104.04	1.05	*1.24*	0.83
Cohort "B"						
Era 1 *	"Normal"	306	301.49	1.01		
	"Less-than-normal"	110	68.20	1.61	*1.59*	17.67
Era 2	"Normal"	354	205.73	1.72		
	"Less-than-normal"	416	140.66	2.96	*1.72*	57.49
Era 3	"Normal"	131	153.03	.86		
	"Less-than-normal"	110	96.41	1.14	*1.33*	4.97
All Eras †	"Normal"	791	675.15	1.17		
	"Less-than-normal"	636	315.12	2.02	*1.72*	106.88

* From 1951

† The figures for Eras 1, 2 and 3 do not total to those for All Eras (except for observed deaths): expectations for Era 3 are low because the study interval when this Era was considered separately did not start until 1968; and the Relative Risks and χ^2 statistics are not, in any case, additive.

Table 10.2 gives distributions of man-years, accumulated in the comparative composite cohort analyses, by broad age groups, for "normals" and "less-than-normals" in Cohorts "A" and "B", the latter divided by Era. Those for Era 1 were for the period after 1950 only, and so for the complete study interval would have been weighted even more to the younger groups. The distributions of the four groups of "normals" were completely different, one from another, and those for the "less-than-normals" almost equally so. An important, but expected, finding was that the "less-than-normals" were consistently older on average than the "normals"; further, this was repeated for each radiological feature examined. It emphasises the importance of some form of age-standardization in order to facilitate comparisons, and confirms the necessity for ensuring that effects are maintained over all portions of a cohort's ageing through the diagonal band of a Lexis (1875) diagram.

In Table 10.3, the six readers are compared in terms of the "sensitivity" and "specificity" of the predictions of mortality possible from radiological findings; the usual definitions, as for example in Armitage (1971, p. 434), have been used, together with the index J (or sensitivity plus specificity, expressed as percentages, less 100 per cent), proposed by Youden (1950). This is rather arbitrary in giving equal weight to sensitivity and specificity which, in this instance, were at quite different levels - less than half the deaths were predictable, whereas nearly three quarters of survivors were among "normals" - but ranks the readers in an order similar to that suggested from a two-way plot. At first sight, an index J = 16.7 per cent is disappointing, but it must be appreciated that the radio-

Table 10.2

DISTRIBUTIONS OF MAN-YEARS BY AGE

	Cohort "A"	Cohort "B"		
		Era 1 *	Era 2	Era 3
"Normals"				
Less than 45	49.9	36.2	19.7	-
45 through 64	43.1	60.8	65.9	93.3
65 and over	6.9	3.0	14.4	6.7
	100	100	100	100
"Less-than-normals"				
less than 45	18.4	17.8	16.8	-
45 through 64	58.9	73.5	60.1	91.4
65 and over	22.6	8.8	23.1	8.6
	100	100	100	100

* From 1951

Reader	Outcome	Radiological finding			Sensitivity (%)
		Normal	Other	Total	
5	Alive	369	145	514	
	Dead	125	126	251	*50.2*
6	Alive	343	148	491	
	Dead	146	135	281	*48.0*
4	Alive	335	176	511	
	Dead	124	132	256	*51.6*
1	Alive	411	75	486	
	Dead	158	76	234	*32.5*
3	Alive	317	154	471	
	Dead	142	123	265	*46.5*
2	Alive	386	98	484	
	Dead	176	80	256	*31.3*
ALL	Alive	2,161	796	2,957	
	Dead	871	672	1,543	*43.6*

Table 10.3

"SENSITIVITY" AND "SPECIFICITY", BY READER

(Cohort "B")

Specificity (%)	Youden's J (%)
71.8	22.0
69.9	17.9
65.6	17.2
84.6	17.1
67.8	14.3
79.8	11.1
73.1	16.7

logical aim was not the prediction of mortality but the description of pneumoconiotic appearances. Further, it would not be unexpected were the relationships to be quite different when individual radiological features are evaluated in terms of deaths from specific causes; see the sections below on pneumoconiosis and lung cancer.

In the ILO U/C (1971) classification of radiographs (ILO, 1972) category 0/- is introduced in terms almost identical to those used by Bohlig *et al.* (1970) in publishing the U/C classification, except that they are called "very obviously normal" films where earlier the phrase "barndoor" normal of the original proposal (Liddell, 1963) had been retained. Table 10.4 differentiates subjects with such films among the "normals". The χ^2 statistics are calculated as for Table 10.1 i.e. on a purely internal basis. For Cohort "A", the SMRS were very similar. In assessment of films for Cohort "B", only Readers 1 and particularly 5 made substantial use of the 0/- category, and for both these readers the SMR for the "barndoor" normals was less than for the remainder of "normals", leading to Relative Risk for the latter of 2.75 for Reader 1 and 1.24 for Reader 5. Although there is no trace of statistical significance in the findings in Table 10.4, as indicated by the values of χ^2 reaching only 2.27, the tendencies for Readers 1 and 5 are suggestive that more common use of the category could lead to clearer differences. Bohlig *et al.* (1970) stated that "barndoor" normal films were usually from young subjects, but there has been some fear that the category 0/- could mask emphysema. The lower SMRs, which of course take age differences into account, might indicate that this last concern is not well founded.

Table 10.4

"BARNDOOR" NORMAL FILMS AND TOTAL MORTALITY

		O	E	SMR	RR	x^2
Cohort "A"	"Barndoor" normal	2	2.41	.83		
	Other normal	19	22.50	.84	*1.02*	0.001
Cohort "B"						
Reader 5	"Barndoor" normal	17	19.43	.87		
	Other normal	98	90.43	1.08	*1.24*	0.67
Reader 1	"Barndoor" normal	2	4.81	.42		
	Other normal	143	125.16	1.14	*2.75*	2.19
Others	"Barndoor" normal	4	2.43	1.65		
	Other normal	527	432.89	1.22	*.74*	0.36
All readers	"Barndoor" normal	23	26.67	.86		
	Other normal	768	648.48	1.18	*1.37*	2.27

10.3 Cause-specific mortality in the "less-than-normals"

Table 10.5 gives the numbers of deaths in each of 13 cause groups, together with the risk in the "less-than-normals" relative to the "normals" and the associated χ^2 statistic, calculated as previously. In Cohort "B", among those with radiographic changes, there were greatly enhanced risks (all with χ^2 values greater than that associated with the 0.1 per cent "significance level" in an independent test) of deaths from:- pneumoconiosis (RR = 11.75); respiratory tuberculosis (RR = 6.28); "other" respiratory conditions (RR = 2.29); cancer of the chest (RR = 3.24); and diseases of the heart (RR = 1.53). There also appeared to have been excesses in the "less-than-normals" of deaths from (Relative Risks in brackets):- cancer of oesophagus of stomach (1.58); cancer of colon or rectum (2.34); "other" cancers (1.44); accidents (1.58); and "other" known causes (1.53).

Few of these findings were repeated in Cohort "A": all three deaths from pneumoconiosis were indeed in the "less-than-normals", leading to a Relative Risk expressed as infinity (∞), because the SMR for "normals" was zero, but there were so few deaths that the value of χ^2 was small. Similar estimates of "infinite" Relative Risks, of accidents and of "other" known causes, were associated with higher values of χ^2, although neither reached a conventional level of statistical significance.

10.4 Total mortality and specific radiographic changes

The first two lines of Table 10.6 are repeated from the first table in this chapter, except that the expected numbers of deaths (E) for Cohort "B" were obtained by summation over the three Eras, and so,

	Cohort "A"	
	$O_1 + O_2$	RR
Pneumoconiosis	3	∞
Cancer of the chest	22	*.78*
Respiratory tuberculosis	-	
Other respiratory conditions	6	*1.03*
Diseases of the heart	44	*1.03*
Cancer of oesophagus or stomach	5	∞
Cancer of colon or rectum	3	∞
Other abdominal cancers	5	*.91*
Cancer of the larynx	2	*.23*
Other cancers	11	*.40*
Cerebro-vascular diseases	5	*.82*
Accidents	6	∞
Other known causes	16	∞

Table 10.5

RADIOLOGICAL ABNORMALITY AND CAUSE-SPECIFIC MORTALITY

		Cohort "B" (1951-75)		
	χ^2	$O_1 + O_2$	RR	χ^2
	0.50	31	*11.75*	41.24
	0.24	119	*3.24*	44.63
		40	*6.28*	37.28
	0.00	67	*2.29*	12.17
	0.01	566	*1.53*	25.25
	1.08	53	*1.58*	2.79
	0.66	28	*2.34*	5.34
	0.01	21	*1.34*	0.43
	1.31	4	*2.21*	0.66
	2.23	93	*1.44*	3.02
	0.03	101	*1.17*	0.59
	2.59	110	*1.58*	5.24
	3.85	183	*1.53*	8.12

Table 10.6

		Cohort "A"					Cohort "B" (1951-75)				
		O	E	SMR	RR	χ^2	O	E	SMR	RR	χ^2
"Normal"		21	24.91	.84			791	660.25	1.20		
"Less-than-normal"		109	104.04	1.05	*1.24*	0.83	636	305.27	2.08	*1.74*	110.72
"Less-than-normal" with:-											
Small opacities (a)	absent	56	63.82	.88			253	143.52	1.76		
	present	53	40.21	1.32	*1.56*	3.44	383	161.75	2.37	*1.98*	120.30
Large opacities	absent	103	100.09	1.03			589	296.42	1.99		
	present	6	3.94	1.52	*1.81*	1.56	47	8.85	5.31	*4.43*	99.94
Pleural changes *	absent	56	65.03	.86			331	169.47	1.95		
	present	53	39.00	1.36	*1.61*	4.01	305	135.80	2.25	*1.87*	83.69
Pleural calcification	absent	90	91.48	.98			544	257.92	2.11		
	present	19	12.55	1.51	*1.80*	3.73	92	47.35	1.94	*1.62*	16.59
"Additional symbols"	absent	68	65.84	1.03			418	223.10	1.87		
	present	41	38.19	1.07	*1.27*	0.79	218	82.17	2.65	*2.21*	104.67

* Other than calcification (a) For Cohort "A" includes $1 \leq SSC \leq 6$.

as explained in section 8.5, were slightly lower, both for "normals" and for "less-than-normals", than in Table 10.1; the SMRs are correspondingly a little higher, but the Relative Risk virtually unaffected.

For the remainder of this table the "less-than-normals" were subdivided according to the absence or presence of specific radiographic appearances, treating each feature one at a time. The Relative Risks and χ^2 statistics were calculated comparing those in whom a feature was present against the "normals"; on this basis, the Relative Risks were all higher than when presence of a feature was compared with its absence, whether or not another feature rendered such a film "less-than-normal". The procedure leads to a partition of that χ^2 statistic with two degrees of freedom which compares all three groups; the quoted χ^2 statistics, as before with one degree of freedom, were larger only when the value of RR was increased sufficiently to compensate for the smaller numbers obtained by the exclusion of those "less-than-normals" in whose films the specific feature was not recorded.

For each radiographic feature, the SMRs for those in whom it was present was higher than in the "normals", in both cohorts. This effect was clearly significant in Cohort "B" for all features. Even for pleural calcification, where the Relative Risk was only 1.62, the χ^2 statistic was large by conventional standards (16.59); χ^2 statistics were enormous, varying from 120.30 to 83.69, for small opacities (RR = 1.98), "additional symbols" (RR = 2.21), large opacities (RR = 4.43) and non-calcified pleural changes (RR = 1.87), respectively. (RR and χ^2 are not more closely correlated because of the differing numbers of deaths associated with each type of x-ray change.) For

Cohort "A", with many fewer deaths, χ^2 values were much smaller, but Relative Risks were not inconsiderable; they could probably be taken as "significant" for pleural changes without calcification (1.61) and with calcification (1.80) and for small opacities (1.56).

10.5 Total mortality and non-radiographic factors

The next three tables (10.7 through 10.9) deal with factors other than radiographic. For each factor separately, each cohort was divided into two sub-cohorts and the SMR in the second was related to that in the first. Although the initial classification by accumulated dust exposure had been into four sub-cohorts, the three with lowest exposures were found to differ only slightly in SMRs, and so they were re-combined to yield a comparison of those whose accumulated exposure was at least 300 mpcf.y against those with lower exposures. Correspondingly, those with only grade 1 breathlessness were grouped with those who did not report this symptom, to allow comparison against those with definite dyspnoea. Thus, in each section of the three tables, there were only two sub-cohorts to be contrasted, and the calculations of χ^2 could be on the same basis as for Table 10.1.

The SMR of men at Thetford Mines was a fraction lower than of those at Asbestos, in Cohort "A", but slightly higher in Cohort "B" (Table 10.7), while those with higher dust exposures had higher SMRs in both cohorts, RR = 1.21 in Cohort "A", and 1.59, with χ^2 = 75.37, in Cohort "B". The lack of independence must be emphasised; dust exposures at Thetford Mines were on average greater than at Asbestos, and it seems likely that the observed higher risk at Thetford Mines in Cohort "B" was caused mainly by the heavier exposure rather than by

Table 10.7

TOTAL MORTALITY BY MINING AREA, DUST EXPOSURE AND SMOKING HABIT

	Cohort "A"					Cohort "B" (1951-75)				
	O	E	SMR	RR	χ^2	O	E	SMR	RR	χ^2
Asbestos	41	39.98	1.03			728	536.63	1.36		
Thetford Mines	89	88.97	1.00	*.98*	0.02	699	428.87	1.63	*1.20*	12.04
Accumulated dust exposure (mpcf.y)										
less than 300	77	82.08	.94			853	678.53	1.26		
at least 300	53	46.86	1.13	*1.21*	1.10	574	286.95	2.00	*1.59*	75.37
Smoking habit:										
non-, ex-, light	33	50.33	.66			494	459.65	1.07		
cigarettes *	97	78.61	1.23	*1.88*	10.18	885	493.42	1.79	*1.67*	84.99

* For Cohort "A", at least 13 cigarettes a day
For Cohort "B", known cigarette smokers

"neighbourhood" or other environmental differences. Cigarette smokers had substantially worse SMRs in both cohorts compared with the remainder; that the Relative Risk was higher in Cohort "A" (1.88) than in Cohort "B" (1.67) may have something to do with the different classification of smoking habits indicated in the footnote to the table. This was much the most "significant" effect noted so far in Cohort "A", χ^2 = 10.18, and was also associated with very large χ^2, i.e. 84.99, in Cohort "B".

Tables 10.8 and 10.9 can only refer to the one cohort, "A", because corresponding information was not obtained for the other. Of symptoms, only breathlessness was definitely associated with enhanced mortality (RR = 1.49, χ^2 = 4.70), but there were suggestions of positive effects associated with persistent cough (despite the unreliability of data transcription) and with wheezing. Mortality in those with sub-normal FVC was rather worse than in the others, but the other two lung function measurements were unrelated to mortality. Those who were overweight in 1967 or 1968 did not have an elevated risk.

Table 10.8

RESPIRATORY SYMPTOMS AND TOTAL MORTALITY

(Cohort "A")

	O	E	SMR	RR	χ^2
Breathlessness					
Absent or slight	87	96.89	.90		
Definite	43	32.05	1.34	*1.49*	4.70
Persistent cough *					
Absent	120	120.47	1.00		
Present	10	8.47	1.18	*1.19*	0.27
Persistent phlegm *					
Absent	124	120.33	1.03		
Present	6	8.62	.68	*.68*	0.89
Wheezing					
Absent	69	72.58	.95		
Present	61	56.36	1.08	*1.14*	0.55

* As explained in relation to Table 9.5, these symptoms cannot be considered reliable.

Table 10.9

LUNG FUNCTION, ADIPOSITY AND TOTAL MORTALITY

(Cohort "A")

	O	E	SMR	RR	χ^2
Forced Vital Capacity					
Normal	103	110.04	.94		
Sub-normal *	27	18.90	1.43	*1.53*	3.88
Ratio: $FEV_{1.0}/FVC$					
Normal	112	111.09	1.01		
Sub-normal *	18	17.85	1.01	*1.00*	0.00
Maximal mid-expiratory flow					
Normal	112	110.62	1.01		
Sub-normal *	18	18.32	.98	*.97*	0.01
Adiposity					
Normal	109	107.77	1.01		
Overweight *	21	21.17	.99	*.98*	0.01

* At least one standard deviation from "adjusted" mean

Chapter 11

FINDINGS II

11.1 Introduction

As explained in section 10.1, the present chapter is concerned with deaths from disease which might be expected to have been detected on the chest radiograph, either directly or through changes arising from the retention of asbestos dust. Sections 11.2 and 11.3 examine in some depth radiological findings in men whose deaths were certified as from pneumoconiosis and from cancer of the chest, respectively. The next section deals with mesothelioma, and the chapter ends with consideration of deaths from respiratory tuberculosis, other respiratory conditions and heart diseases.

11.2 Pneumoconiosis

Although fibrosis of the lung due to the inhalation of asbestos dust may lead to premature death which is not certified as due to pneumoconiosis, deaths which are coded to that cause remain those that one would expect to be best predicted by a classification of radiographic appearances considered to be due to the retention of dust in the lungs. This is confirmed by the high risks of death from pneumoconiosis in the "less-than-normals" relative to the "normals" seen in Table 10.5, and the ability of the presence of parenchymal and pleural changes to predict total mortality (Table 10.6). The present section

is concerned with deaths coded to pneumoconiosis; there were three such deaths in Cohort "A" and 31 in Cohort "B". Table 11.1 gives Relative Risks, for both cohorts, associated with the presence of each radiographic feature, and with mining area, comparing Thetford Mines against Asbestos, higher accumulated dust exposure and cigarette smoking. χ^2 calculations for the former were on the same basis as for Table 10.6 and for the latter on the same basis as for Table 10.1. Corresponding figures for those with symptoms, depressed lung function, or overweight are also given for Cohort "A"; χ^2 basis again as for Table 10.1.

Because there were only three deaths recorded as due to pneumoconiosis in Cohort "A", it would have been most unlikely that any value of χ^2 reached a conventional level of significance, even although for all three their films were "less-than-normal"; small opacities were recorded in all three films, and the χ^2 statistic is about as high as it would be possible to achieve, and so is at least suggestive, particularly when considered in conjunction with the very high value (86.60) for Cohort "B", wherein the Relative Risk was 21.58. In that cohort, there were also clear associations of high mortality and the presence of pleural changes other than calcification (RR = 21.07) and of "additional symbols" (RR = 19.17), and with high levels of accumulated dust exposure (RR = 8.78). Only four of the 31 deaths were in men in whom large opacities had been recorded, but the expectation was too small to be computed to two decimal places; this led to unmeasurably high SMR (and hence RR and χ^2), but the actual numbers suggest that too much reliance should not be placed on these facts. The higher SMR at Thetford Mines was again probably no more than a reflection of the higher dust exposures there. There were slight

Table 11.1

DEATHS FROM PNEUMOCONIOSIS
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	3		31	
	RR	χ^2	RR	χ^2
"Less-than-normal"	∞	0.50	11.75	41.24
Small opacities	∞	2.40	21.58	86.60
Large opacities	-	-	†	†
Pleural changes *	∞	1.07	21.07	71.62
Pleural calcification	∞	1.17	2.44	0.18
"Additional symbols"	∞	0.39	19.17	39.55
Thetford Mines *cf.* Asbestos	.80	0.03	3.37	10.64
Accumulated dust exposure	3.00	0.89	8.78	37.35
Cigarette smoking	1.50	0.11	1.11	0.08
Breathlessness	0	1.09		
Persistent cough	0	0.23		
Persistent phlegm	0	0.23		
Wheezing	.67	0.11		
Depressed FVC	3.00	0.89		
Depressed ratio $FEV_{1.0}/FVC$	0	0.50		
Depressed MMF	0	0.50		
Overweight	3.00	0.89		

* other than calcification † see text

signs of a dust effect in Cohort "A", but the three cases were largely free of respiratory symptoms.

The next two tables (11.2 and 11.3) describe the radiographic changes seen in the films of the men who died from pneumoconiosis (codes 523 and 524 of the Seventh Revision of the International Classification of Diseases); for Cohort "A", the 18 readings, six per case, are presented in one-way distributions. From Table 11.2, it can be seen that parenchymal change was recorded in the majority of the readings, 14 (or 77.8 per cent) and 23 (or 74.2 per cent) in the two cohorts, but other changes in the absence of parenchymal change were rare. There were two and five assessments as normal (11.1 per cent and 16.1 per cent) in Cohorts "A" and "B".

Table 11.3 illustrates the recording of parenchymal change in the pneumoconiosis cases. In Cohort "A", only small opacities were recorded, all irregular (types s, t, u) rather than rounded; the profusion averaged 3.5 on the notional scale of Liddell and May (1966, p. 9), corresponding to a reading between 1/1 and 1/2, considerably higher than the average in other cases where small opacities were seen. The pattern in Cohort "B" was not dissimilar, but among the 23 readings of small opacities, there were three where rounded opacities (p, q, r) predominated, and four in which large opacities were seen, each time in association with small opacities of profusion at least 2/2. The average profusion of small opacities in the 23 films was 5.6 on the notional scale, close to 2/2 and even higher than in Cohort "A".

For further investigation, a control was selected for each case

Table 11.2

RADIOGRAPHIC CHANGES IN PNEUMOCONIOSIS DEATHS

	Cohort "A"	Cohort "B"
Number of deaths	3	31
Number of readings	3 x 6 = 18	31
Type of change		
Parenchymal and other	9	17
Parenchymal only	5	6
	14	23
Pleural only	2	1
	16	24
Other *	-	2
	16	26
No change	2	5
	18	31

* One case of left costophrenic angle obliteration, with one "additional symbol"; one case of a single "additional symbol".

Table 11.3

PARENCHYMAL CHANGES IN PNEUMOCONIOSIS DEATHS

Entries in square brackets concern large opacities, A, B and C well-defined, D, E and F ill-defined.

Profusion of small opacities	Cohort "A"	Cohort "B"
(0/-)	-	-
(0/0)	-	-
0/1	2 (2t)	2 (1t, 1u)
1/0	2 (2s)	-
1/1	4 (3t, 1u)	4 (3t, 1u)
1/2	-	2 (1p, 1u)
2/1	-	1 (u)
2/2	2 (1t, 1u)	7 (1r, 2s, 1t, 3u) [2 (1A, 1E)]
2/3	1 (t)	1 (u) [1(D)]
3/2	1 (t)	2 (1q, 1u)
3/3	2 (1t, 1u)	4 (1s, 3u) [1(D)]
3/4	-	-
	14	23

in Cohort "B", as described in section 8.6, and some results from the case-control pairs are given in Tables 11.4 and 11.5. The former shows, reader by reader, the number of pairs in which pneumoconiotic change, i.e. small opacities, pleural thickening or ill-defined diaphragm or cardiac border, was:- (a) present in both members of the pair; (b) present only in the case; (c) present only in the control; and (d) absent in both. The appropriate test for association is that due to McNemar (1947), although the correction for continuity usually used should be halved (Pirie and Hamdan, 1972). This test is based solely on the disparate pairs, (b) and (c), and leads to yet another form of χ^2 statistic, i.e.

$$\chi^2 = \{ |(b) - (c)| - \tfrac{1}{2}\}^2 \div \{ (b) + (c) \}$$

again with one degree of freedom. Numbers are too small for a test for each reader separately; pooling yields $\chi^2 = 11.07$, which can be taken as providing some confidence that the excess of pairs in (b) over those in (c), common to five of the readers, was not due to chance.

Table 11.5 classifies cases and controls, independently, by accumulated dust exposure and by smoking habit. For the one case with exposure apparently less than 30 mpcf.y, the last recorded job ended in September 1930 at Thetford Mines. Clearly this man could not have been x-rayed at that time, and there is obviously a gap in the recorded work history; it is not unlikely that he had had substantial exposure in the period between 1930 and the time of x-ray that would have placed him in a higher exposure category. However, even including him with exposure as recorded gives the Relative Risks as in the table, showing the clear dose-response relationship expected, as these men

	Pneumoconiotic change:		
In case:	Present	Present	Absent
In control:	Present	Absent	Present
	(a)	(b)	(c)
Reader 1	-	2	-
Reader 2	-	2	-
Reader 3	-	4	-
Reader 4	3	1	1
Reader 5	3	3	-
Reader 6	1	5	1
ALL READERS	7	17	2

Table 11.4

RADIOGRAPHIC CHANGES IN PNEUMOCONIOSIS DEATHS, AND CONTROLS

(Small opacities or uncalcified pleural changes)

Absent Absent (d)	Number of case-control pairs
2	4
1	3
-	4
1	6
-	6
1	8
5	31

Table 11.5

ASBESTOS DUST EXPOSURE AND SMOKING HABIT
IN PNEUMOCONIOSIS DEATHS AND IN CONTROLS

Accumulated dust exposure (mpcf.y)	Cases	Controls	RR
Less than 30	1 *	7	*1*
30, less than 100	-	7	*0*
100, less than 300	6	7	*6.0*
300 or greater	24	10	*16.8*
	31	31	

Smoking habit	Cases	Controls	RR
Non-smoker	5	4	*1*
Not current cigarette smoker	9	11	*0.6*
Cigarette smoker	17	16	*0.8*
	31	31	

* See text

formed part of the "main mortality cohort" (McDonald and Liddell, 1978). Again the lack of a relationship between smoking and pneumoconiosis is confirmation of the findings in the entire "main mortality cohort" (McDonald *et al.*, 1979).

Finally, we wished to discriminate between those pneumoconiosis deaths in which the x-ray had shown pneumoconiotic change (as defined for Table 11.4) and the others. Table 11.6 shows that the 23 cases with pneumoconiotic change on the x-ray differed little from the other seven either in accumulated dust exposure or in interval between last recorded job and death.

11.3 Cancer of the chest

The investigation of cancer of the chest (i.e. deaths coded in the Seventh Revision of the International Classification of Diseases to: 162, malignant neoplasm of bronchus and trachea; 163, malignant neoplasm of lung, unspecified as to whether primary or secondary; or 164, malignant neoplasm of mediastinum) followed the same lines as that for pneumoconiosis deaths. The tables follow the same broad pattern as those illustrating section 11.2, and the same definitions have been used.

Table 11.7 shows that in Cohort "B", the presence of each radiographic feature was associated with an enhanced risk of chest cancer, the strongest association being with large opacities: RR = 20.73; χ^2 = 142.98. There were also increased Relative Risks in the presence of "additional symbols", 5.27 with χ^2 = 55.24, uncalcified pleural

Table 11.6

PNEUMOCONIOSIS DEATHS WITH AND WITHOUT RADIOGRAPHIC CHANGES

(Excluding one death with exposure less than 30 mpcf.y)

Accumulated dust exposure (mpcf.y)	Changes:-	
	Present	Absent
Less than 30	-	-
30, less than 100	-	-
100, less than 300	3	3
300 or greater	20	4
	23	7

Interval between last recorded job and death (years)	Changes:-	
	Present	Absent
Less than 5	13	4
5 or greater	10	3
	23	7

Table 11.7

DEATHS FROM CANCER OF THE CHEST
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	22		119	
	RR	χ^2	RR	χ^2
"Less-than-normal"	.78	0.24	3.24	44.63
Small opacities	1.41	0.66	2.88	19.46
Large opacities	1.20	0.03	20.73	142.98
Pleural changes *	1.21	0.16	3.73	34.80
Pleural calcification	.75	0.13	2.82	5.89
"Additional symbols"	.25	2.11	5.27	55.24
Thetford Mines *cf.* Asbestos	1.21	0.16	1.59	6.48
Accumulated dust exposure	2.04	2.90	2.87	36.03
Cigarette smoking	4.11	6.10	3.35	30.33
Breathlessness	.87	0.07		
Persistent cough	.71	0.12		
Persistent phlegm	.68	0.14		
Wheezing	1.82	1.98		
Depressed FVC	1.69	1.10		
Depressed ratio $FEV_{1.0}/FVC$	.61	0.44		
Depressed MMF	1.32	0.25		
Overweight	1.11	0.04		

* other than calcification

changes (RR = 3.73, χ^2 = 34.80), small opacities (RR = 2.88, χ^2 = 19.46), and perhaps pleural calcification (RR = 2.82, χ^2 = 5.89). Higher accumulated dust exposure and cigarette smoking also led to higher SMRs (RR = 2.87 and 3.35, respectively, with χ^2 = 36.03 and 30.33), but the high Thetford Mines risk relative to Asbestos was, once more, probably due to the higher levels of dust exposure there, and related to the high risk in those with pleural calcification. In Cohort "A", the risk in comparatively heavy cigarette smokers relative to others was great enough, 4.11, to yield a high value of χ^2, 6.10; the Relative Risks in those with higher dust exposure and in those whose films showed parenchymal or pleural change, without calcification, were also greater than unity, as were those in men who reported wheeze or had low FVC.

The pattern of radiographic change in chest cancer cases (including two in Cohort "B" which occurred before 1951, but excluding three mesotheliomas in that cohort whose deaths had been coded 162 or 163), shown in Tables 11.8 and 11.9, was quite different from that in pneumoconiosis (Tables 11.2 and 11.3). First, there were much higher proportions of normal readings, i.e. 65 out of 132, or 49.2 per cent and 48 in 118, or 40.7 per cent, in the two cohorts, compared with 11.1 per cent and 16.1 per cent in pneumoconiosis. Secondly, the proportions of the chest cancer cases showing parenchymal changes were rather lower, i.e. 90 readings out of 250, or 36.0 per cent, than of pneumoconiosis cases, where there were 37 readings of parenchymal change out of 49, or 75.5 per cent. Thirdly, such parenchymal changes as were recorded were of substantially lower profusion of small opacities (averages corresponding to profusions of less than 1/1 and

Table 11.8

RADIOGRAPHIC CHANGES IN CHEST CANCER DEATHS

	Cohort "A"	Cohort "B"
Number of deaths	22	118 *
Number of readings	22 x 6 = 132	118
Type of change		
Parenchymal and other	29	26
Parenchymal only	22	13
	51	39
Pleural	15	18
	66	57
Costophrenic angle only	1	2
	67	59
"Additional symbol" only	-	11
	67	70
No change	65	48
	132	118

* Consisting of 119 cases in the years 1951 through 1975, plus two before 1951, less three mesotheliomas coded 162 or 163 (see Table 11.14).

Table 11.8

RADIOGRAPHIC CHANGES IN CHEST CANCER DEATHS

	Cohort "A"	Cohort "B"
Number of deaths	22	118 *
Number of readings	22 x 6 = 132	118
Type of change		
Parenchymal and other	29	26
Parenchymal only	22	13
	51	39
Pleural	15	18
	66	57
Costophrenic angle only	1	2
	67	59
"Additional symbol" only	-	11
	67	70
No change	65	48
	132	118

* Consisting of 119 cases in the years 1951 through 1975, plus two before 1951, less three mesotheliomas coded 162 or 163 (see Table 11.14).

Profusion of small opacities	Cohort "A"
(0/-)	-
0/0	-
0/1	16 (1p, 2q, 2s, 1(ps), 10t)
1/0	10 (1p, 1(qs), 8t)
1/1	19 (1p, 1q, 3s, 11t, 3u) [1(D)]
1/2	3 (2t, 1u)
2/1	-
2/2	3 (2t, 1u)
2/3	-
3/2	-
3/3	-
3/4	-
	51

Table 11.9

PARENCHYMAL CHANGES IN CHEST CANCER DEATHS

Cohort "B"

-	
6	[6 (1A, 3B, 1D, 1E)]
6 (5t, 1u)	[2 (1D, 1E)]
6 (1q, 1s, 3t, 1u)	[1(B)]
5 (1p, 1q, 3t)	
4 (1p, 1s, 2t)	[2(D)]
2 (1p, 1q)	
7 (1s, 5t, 1u)	[1(D)]
1 (p)	[1(A)]
-	
2 (1p, 1s)	[1(B)]
-	

39

a little more than 1/1 in Cohorts "A" and "B", respectively), while the opacities were more frequently rounded (p, q, r), i.e. in 15 out of 84 readings of profusion at least 0/1 (17.9 per cent), compared with 3 out of 37 (8.1 per cent) in pneumoconiosis. Finally, in Cohort "B", there were 15 cases in which large opacities had been recorded, only three associated with small opacities of profusion 2/2 or greater; see also Table 11.12.

The numbers in Table 11.10 are just large enough to allow tests, of the sort described in relation to Table 11.4, reader by reader; for each reader, the number of case-control pairs in which radiographic change was present in the case but not in the control, column (b), was greater than the number in which the reverse was true, column (c), but only for Readers 6 and 2 was χ^2 substantial (7.52 and 5.56, respectively). However, pooling over all readers led to $\chi^2 = 16.54$, a little higher than for pneumoconiosis - because of the larger numbers rather than better discrimination.

Table 11.11 reveals the expected dose-response relationships for both dust exposure and smoking, but these two factors do not discriminate well between those cases whose x-rays were normal and others; see the upper part of Table 11.12. However, the shorter the interval between last recorded job (usually less than a year after the x-ray had been taken) and death, the lower the probability of the film being assessed as normal: for the four intervals in the lower part of Table 11.12, the proportions with normal films were 18.8 per cent, 44.4 per cent, 64.3 per cent and 70.0 per cent, as the intervals increased. In 11 of the 14 chest cancer cases in whose films "large

	Radiographic change:-			
In case:	Present	Present	Absent	Absent
In control:	Present	Absent	Present	Absent
	(a)	(b)	(c)	(d)
Reader 1	2	6	1	6
Reader 2	-	11	2	10
Reader 3	7	6	4	5
Reader 4	4	7	2	2
Reader 5	4	5	4	5
Reader 6	7	11	1	6
ALL READERS	24	46	14	34

Table 11.10

RADIOGRAPHIC CHANGES IN CHEST CANCER DEATHS, AND CONTROLS

(Any radiographic changes)

Number of case-control pairs	
15	
23	
22	
15	
18	
25	
118	

Table 11.11

ASBESTOS DUST EXPOSURE AND SMOKING HABIT
IN CHEST CANCER DEATHS AND IN CONTROLS

Accumulated dust exposure (mpcf.y)	Cases	Controls	RR
Less than 30	18	25	*1*
30, less than 100	20	24	*1.16*
100, less than 300	17	21	*1.12*
300 or greater	63	48	*1.82*
	118	118	

Smoking habit *	Cases	Controls	RR
Non-smoker	6	8	} *1*
Not current cigarette smoker	17	45	
Cigarette smoker	91	61	*3.44*
	114	114	

* Excluding four pairs in which habit was not known for case

Table 11.12

CHEST CANCER CASES WITH AND WITHOUT RADIOGRAPHIC CHANGES

Smoking habit	Accumulated dust exposure (mpcf.y)	Changes:- Present	Changes:- Absent
Not cigarettes	Less than 300	3 (2)*	3
	300 or greater	9 (6)	9
Cigarettes	Less than 300	30 (11)	16
	300 or greater	27 (14)	17
		69 (33)	45

* Numbers of cases in which small opacities were recorded

Interval between last recorded job and death (years)	Changes:- Present	Changes:- Absent
Less than 5	39 (11) ¶	9
5, less than 10	20 (1)	16
10, less than 20	5 (1)	9
20 or greater	6 (1)	14
	70 (14)	48

¶ Numbers of cases in which large opacities were recorded

Table 11.12

CHEST CANCER CASES WITH AND WITHOUT RADIOGRAPHIC CHANGES

Smoking habit	Accumulated dust exposure (mpcf.y)	Changes:- Present	Changes:- Absent
Not cigarettes	Less than 300	3 (2)*	3
	300 or greater	9 (6)	9
Cigarettes	Less than 300	30 (11)	16
	300 or greater	27 (14)	17
		69 (33)	45

* Numbers of cases in which small opacities were recorded

Interval between last recorded job and death (years)	Changes:- Present	Changes:- Absent
Less than 5	39 (11) ¶	9
5, less than 10	20 (1)	16
10, less than 20	5 (1)	9
20 or greater	6 (1)	14
	70 (14)	48

¶ Numbers of cases in which large opacities were recorded

opacities" had been assessed, the interval was less than five years; in the six cases with "large opacities" but no small opacities, the intervals were all short, three dying in the same year as that of last employment, two in the following year, and one in the year after that.

11.4 Mesothelioma

A total of six deaths within Cohorts "A" and "B" had been recognized, during the course of the main mortality study, as due to malignant mesothelioma, all of the pleura; one was in Cohort "A" and had been coded to cause 227 in the Seventh Revision of the International Classification of Diseases, and in Cohort "B" two had been coded to 162, one to 163 and two to 212. Table 11.13 gives the six readings of the film for the one case in Cohort "A". He died in 1972, aged 62; his first employment in the Quebec asbestos industry was in January 1928, giving an interval between first exposure and death of 44 years. At November 1966, his net service was 39 years, but the dust exposure accumulated until then was only 227 mpcf.y. The five cases in Cohort "B" are described in Table 11.14. The first six lines indicate the radiographic changes, and there follows a brief summary of other facts about each case.

One of the most interesting findings about asbestos-induced mesothelioma is the long latent period, i.e. interval between first exposure to asbestos and death, probably even longer for chrysotile than for crocidolite; see Figure 6.2 above. A total of twelve cases exposed to chrysotile only have now been ascertained among Quebec miners and millers; the six not reported here had had rather longer latent periods, ranging from 41 years to 56, with a median of 52 years.

Table 11.13

MESOTHELIOMA: ONE CASE IN COHORT "A"

Death in 1972, at age 62 years, coded "227"

Interval from first exposure to death: 44 years

Net service, at Thetford Mines: 31 years; 227 mpcf.y accumulated.

Cigarette smoker

		ROUNDED SMALL OPACITIES			IRREGULAR SMALL OPACITIES			LARGE OPACITIES			PLEURAL THICKENING			ILL DEFINED DIAPHRAGM	ILL DEFINED CARDIAC BORDER	PLEURAL CALCIFICATION				14917	
Rdr	Quality	Type	Profusion	Zones	Type	Profusion	Zones	(a)	(b)	Costo-phrenic angle	(c)	(d)	(e)			Diaph	Wall	Other	Grade	Symbols	Comments
A	+		/			/										R L			1		
B	+		/			/								R,L		R L	L		1		
C	+		/			/										R L	R L		1		
D	+		/		u	1/0	✓ ✓ ✓ ✓ ✓									R L	R L		1		✓
E	+		/		t	0/1	✓									R L			1		
F	+	p	0/1	✓		/										R L	L		1		
	+ ± ± U/R	p q r	0/– 0/0 0/1 1/0 1/1 1/2 2/1 2/2 2/3 3/2 3/3 3/4	Check zones involved	s t u	0/– 0/0 0/1 1/0 1/1 1/2 2/1 2/2 2/3 3/2 3/3 3/4	Check zones involved	O A B C	O D E F	O R L	R L	R L	O 1 2	O R L	O 1 2 3		R L		O 1 2 3	O Cn Es Od	Co Cp Ho Np Tb

(a) Well defined (c) Diffuse

(b) Poorly defined (d) Plaques

(e) Grade

Table 11.14

MESOTHELIOMA: FIVE CASES IN COHORT "B"

	Case number				
	1	2	3	4	5
Parenchymal change (type and profusion)	-	-	p: 1/0 t: 0/1		-
Thickening of pleural walls	-	-	-	-	-
Ill-defined diaphragm	-	R	-	-	-
Ill-defined cardiac border	-	-	-	-	-
Pleural calcification (grade)	-	3	-	-	-
"Additional symbols" (count)	1	-	-	-	-
Coded cause of death	162	162	212	212	163
Year of death	1956	1964	1967	1967	1975
Age at death (years)	40	57	73	57	64
Interval from first exposure to death (years)	21	45	30	31	52
Mining area (Asbestos or Thetford Mines)	TM	TM	TM	A	TM
Year of last employment	1955	1963	1962	1966*	1966*
Net service (years)	15	44	19	30	31
Accumulated dust exposure (mpcf.y)	71	353	52	622	684
Cigarette smoker	Yes	NK	Yes	Yes	Yes

* Still employed at November 1966

Only for one man in Cohort "B" was the latent period as long as the median for the complete group of twelve cases. This difference between the cases in the studies now being reported and the others is undoubtedly due to these others having completed their employment in the chrysotile mines and mills before radiology started there, and so being excluded from either study cohort.

11.5 Respiratory tuberculosis

Each of the next three sections is concerned with deaths from the three groups of causes mentioned in section 11.1 not already dealt with. Each group is the subject of a single table, with the same lay-out and definitions as for Table 11.1.

Although respiratory tuberculosis (Table 11.15) used to be a fairly common cause of death in Quebec, there were no deaths from this cause in Cohort "A", nor in Era 3 of Cohort "B". Not surprisingly, the risks of the "less-than-normals" were very much higher than those of the "normals". It is well-known that the radiological appearances of tuberculosis can be confused with pneumoconiotic change, particularly with large opacities (RR = 51.21), while the "additional symbols" (RR = 12.80) include means of recording diagnoses of tuberculosis. As the dust effect (RR = 1.64) was not nearly as large as that associated with abnormality of x-ray (RR at least 6.28), it seems possible that the high Relative Risks for small opacities (7.58) and for pleural changes (9.63 and 7.49) were artefactual rather than the results of heavy exposure to asbestos dust. The difference between Thetford Mines and Asbestos is probably a reflection of differences between the areas in the prevalence of tuberculosis in the general population, for

Table 11.15

DEATHS FROM RESPIRATORY TUBERCULOSIS
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	0		40	
	RR	χ^2	RR	χ^2
"Less-than-normal"			6.28	37.28
Small opacities			7.58	33.64
Large opacities			51.21	159.98
Pleural changes *			9.63	51.89
Pleural calcification			7.49	10.96
"Additional symbols"			12.80	64.88
Thetford Mines *cf.* Asbestos			2.06	5.41
Accumulated dust exposure			1.64	2.29
Cigarette smoking			1.85	3.22
Breathlessness				
Persistent cough				
Persistent phlegm				
Wheezing				
Depressed FVC				
Depressed ratio $FEV_{1.0}/FVC$				
Depressed MMF				
Overweight				

* other than calcification

it was observed also in the main mortality study (McDonald *et al.*, 1979) for all male workers regardless of exposure. The smoking effect was so small compared with all the others that it should probably not be given much weight.

11.6 Other respiratory conditions

Deaths from other respiratory conditions (Table 11.16) also seem to have been predictable from radiographic changes and once more there appears to have been some misdiagnosis, either on the x-ray or on death certification, where "large opacities" are concerned. In Cohort "B", the dust effect was considerable (RR = 1.85, χ^2 = 6.47), and so the high Relative Risks for the men with pneumoconiotic changes (RRs 2.99 and 3.27 for parenchymal and pleural changes; χ^2 values 18.21 and 20.61) may well have been reflections of heavier exposure. The Relative Risk for smokers may have been because of chronic bronchitis.

In Cohort "A", only pleural calcification, among radiographic features, was associated with high risk; no explanation has been found. The most marked findings in this cohort were Relative Risks of 5.95 and 6.21 for depressed FVC and MMF: they are both eminently reasonable, as are the high Relative Risks, even if without high values of χ^2, associated with breathlessness (2.87), persistent phlegm (2.60), wheeze (1.26) and $FEV_{1.0}$/FVC (3.10).

11.7 Heart disease

Although relative risks of death from heart disease (Table 11.17) were not generally as great as for the conditions just discussed, the

Table 11.16

DEATHS FROM OTHER RESPIRATORY DISEASES
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	6		67	
	RR	χ^2	RR	χ^2
"Less-than-normal"	1.03	0.00	2.29	12.17
Small opacities	1.05	0.00	2.99	18.21
Large opacities	0	0.16	11.82	34.04
Pleural changes *	1.07	0.00	3.27	20.61
Pleural calcification	3.30	1.94	1.58	0.55
"Additional symbols"	1.64	0.26	1.90	2.10
Thetford Mines *cf.* Asbestos	.92	0.01	.98	0.00
Accumulated dust exposure	.31	1.28	1.85	6.47
Cigarette smoking	.68	0.23	1.39	1.74
Breathlessness	2.87	1.83		
Persistent cough	0	0.43		
Persistent phlegm	2.60	0.82		
Wheezing	1.26	0.08		
Depressed FVC	5.95	6.17		
Depressed ratio $FEV_{1.0}/FVC$	3.10	1.90		
Depressed MMF	6.21	6.25		
Overweight	0	1.19		

* other than calcification

Table 11.17

DEATHS FROM HEART DISEASE
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	44		566	
	RR	χ^2	RR	χ^2
"Less-than-normal"	1.03	0.01	1.53	25.25
Small opacities	1.62	1.87	1.71	27.92
Large opacities	2.25	1.76	1.12	0.05
Pleural changes *	1.44	0.93	1.54	13.96
Pleural calcification	1.64	1.11	1.34	2.25
"Additional symbols"	1.32	0.50	1.98	30.46
Thetford Mines *cf.* Asbestos	.96	0.01	1.13	1.96
Accumulated dust exposure	1.42	1.36	1.57	27.90
Cigarette smoking	1.38	1.00	1.78	41.95
Breathlessness	2.26	7.58		
Persistent cough	1.42	0.46		
Persistent phlegm	0	3.17		
Wheezing	1.39	1.20		
Depressed FVC	1.29	0.42		
Depressed ratio $FEV_{1.0}/FVC$	.79	0.23		
Depressed MMF	.60	0.94		
Overweight	1.12	0.08		

* other than calcification

values of χ^2 are larger because they are based on so many more deaths. The dust and pneumoconiotic effects may be explicable by diagnosis of heart disease in deaths accelerated by pneumoconiosis in those heavily exposed to asbestos. The "additional symbols" associated with high risk may have included those relating to heart conditions, e.g. cor pulmonale or enlarged heart. The smoking effect was to have been expected from other studies in asbestos workers (e.g. McDonald *et al.*, 1979) and in other populations (see several reports summarized by Holman, 1976), as was the high Relative Risk for those who reported breathlessness (see, for example, Higgins and Keller, 1970).

Chapter 12

F I N D I N G S I I I

12.1 Introduction

It seems most unlikely that any manifestation of the remaining groups of causes of death could be discernible on an x-ray of the chest. However, in dust related conditions, there might be pneumoconiotic changes as indirect signs of disease. As in the previous chapter, each group of causes of death is the subject of a table on the same lines as Table 11.1.

12.2 Cancer of the gastro-intestinal tract

The pattern of relationships between dust and *cancer of the oesophagus or stomach* (Table 12.1) are far from clear. In Cohort "B", the effect of exposure to asbestos dust (RR = 2.76, χ^2 = 14.79) was rather larger than was to have been expected from the main mortality study (McDonald and Liddell, 1978), but most of the pneumoconiotic x-ray appearances with high Relative Risks probably arose from the dust exposure. The high Relative Risk for pleural calcification (3.45), together with the high risk at Thetford Mines (2.06), where most of the calcification is concentrated, might suggest a common aetiology, conceivably in the drinking water supplies. It is not impossible that the high risks in Cohort "A" for the overweight (RR = 3.38) and those with persistent cough (RR = 3.63) reflect true underlying relation-

Table 12.1

DEATHS FROM CANCER OF OESOPHAGUS OR STOMACH
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	5		53	
	RR	χ^2	RR	χ^2
"Less-than-normal"	∞	1.08	1.58	2.79
Small opacities	∞	0.88	2.06	5.87
Large opacities	-	-	4.70	4.82
Pleural changes *	∞	0.92	2.44	9.42
Pleural calcification	-	-	3.45	10.85
"Additional symbols"	∞	0.96	0.76	0.17
Thetford Mines *cf.* Asbestos	1.81	0.29	2.06	6.76
Accumulated dust exposure	0	3.08	2.76	14.79
Cigarette smoking	∞	3.29	1.56	2.47
Breathlessness	0	1.73		
Persistent cough	3.63	1.52		
Persistent phlegm	0	0.37		
Wheezing	.31	1.20		
Depressed FVC	0	0.86		
Depressed ratio $FEV_{1.0}/FVC$	1.55	0.16		
Depressed MMF	1.52	0.14		
Overweight	3.38	2.01		

* other than calcification

ships.

For *cancer of the colon or rectum* (Table 12.2), the dust effect in Cohort "B" was quite small and could hardly account for the Relative Risks of those with pneumoconiotic changes on the x-ray. However, the χ^2 values were not large, whereas those in Cohort "A" for breathlessness and depressed FVC were. It is not clear why cancer of the lower gastro-intestinal tract should cause respiratory signs and symptoms.

Table 12.3 deals with *other causes of the abdomen*, i.e. malignant neoplasms of:- biliary passages and of liver, stated to be primary site (155); liver, secondary or unspecified (156); pancreas (157); peritoneum (158); or unspecified digestive organs (159). In view of the comparatively low values of the χ^2 statistics, particularly in relation to dust exposure, it would seem that the chest x-ray cannot materially affect prediction of mortality from these causes.

12.3 Cancer of the larynx

Laryngeal cancer (Table 12.4) has been cited (see Chapter 2) as one response to asbestos exposure, but here the only sign of a dust effect in the admittedly very few cases in either cohort was in the reverse direction. On the other hand, McDonald and Liddell (1978) showed no excess of cancer of the larynx, but a very clear dose-response relationship with cigarette smoking, which is in no way contradicted by the present information. Nor is it surprising that this disease should be associated with dyspnoea. However, the lack of any other important association of laryngeal cancer was according to ex-

Table 12.2

DEATHS FROM CANCER OF COLON OR RECTUM
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	3		28	
	RR	χ^2	RR	χ^2
"Less-than-normal"	∞	0.66	2.34	5.34
Small opacities	∞	1.51	1.76	1.07
Large opacities	-	-	0	0.12
Pleural changes *	∞	0.39	2.42	3.32
Pleural calcification	∞	2.11	2.94	2.47
"Additional symbols"	∞	1.62	2.31	1.81
Thetford Mines *cf.* Asbestos	.23	1.76	1.85	2.60
Accumulated dust exposure	3.29	1.06	1.44	0.89
Cigarette smoking	∞	1.96	.74	0.62
Breathlessness	∞	8.81		
Persistent cough	0	0.21		
Persistent phlegm	0	0.22		
Wheezing	.64	0.14		
Depressed FVC	11.71	6.53		
Depressed ratio $FEV_{1.0}/FVC$	0	0.48		
Depressed MMF	0	0.49		
Overweight	0	0.59		

* other than calcification

Table 12.3

DEATHS FROM OTHER CANCERS OF THE ABDOMEN
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	5		21	
	RR	χ^2	RR	χ^2
"Less-than-normal"	.91	0.01	1.34	0.43
Small opacities	1.18	0.02	1.27	0.16
Large opacities	0	0.16	0	0.16
Pleural changes *	1.22	0.03	2.28	3.22
Pleural calcification	0	0.37	3.34	4.15
"Additional symbols"	1.24	0.04	1.89	1.01
Thetford Mines *cf.* Asbestos	∞	2.24	1.12	0.07
Accumulated dust exposure	.43	0.61	1.20	0.15
Cigarette smoking	.97	0.00	2.70	4.03
Breathlessness	0	1.69		
Persistent cough	0	0.35		
Persistent phlegm	3.52	1.45		
Wheezing	.84	0.04		
Depressed FVC	0	0.87		
Depressed ratio $FEV_{1.0}/FVC$	0	0.81		
Depressed MMF	0	0.83		
Overweight	0	1.00		

* other than calcification

Table 12.4

DEATHS FROM LARYNGEAL CANCER
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	2		4	
	RR	χ^2	RR	χ^2
"Less-than-normal"	.23	1.31	2.21	0.66
Small opacities	.59	0.28	4.10	2.73
Large opacities	0	0.36	0	0.02
Pleural changes *	0	1.66	2.55	0.57
Pleural calcification	0	0.94	0	0.09
"Additional symbols"	0	1.66	0	0.16
Thetford Mines *cf.* Asbestos	∞	0.92	3.71	1.49
Accumulated dust exposure	0	1.18	.81	0.03
Cigarette smoking	∞	1.26	∞	3.58
Breathlessness	∞	5.78		
Persistent cough	0	0.15		
Persistent phelgm	0	0.15		
Wheezing	∞	2.52		
Depressed FVC	0	0.33		
Depressed ratio $FEV_{1.0}/FVC$	0	0.33		
Depressed MMF	0	0.33		
Overweight	0	0.41		

* other than calcification

pectation.

12.4 Other causes

Again, with *other cancers* (Table 12.5), it is not surprising that none of the tabulated Relative Risks appears of significance.

It is difficult to see how *cerebro-vascular diseases* (Table 12.6) could be associated with exposure to asbestos dust, but the Relative Risk in Cohort "B" was not trivially enhanced; there was also an elevated risk, in both cohorts, among those in whose films large opacities had been recorded. These films require careful study to see whether there is any possibility of predicting strokes. The Relative Risks for certain respiratory signs and symptoms in Cohort "A" may well appear important only because they are based on so few deaths due to cerebro-vascular disease.

In considering Table 12.7, it is important to appreciate that the *accidents* included all deaths coded 800–999; only a minority were accidents at work, and by no means all of these occurred while the man was employed in the asbestos industry. Compared with the causes of death discussed in the last few pages, there is even less reason to believe that the x-ray could predict mortality from accidents. Fortunately, there are few high values of χ^2; on the other hand, there are several contradictions, e.g. in the high Relative Risk for small opacities without a corresponding effect of higher dust exposure. Nor can the high risks for smokers be adequately reconciled. The pattern is only confused by the differences between the two cohorts.

Table 12.5

DEATHS FROM OTHER CANCERS
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	11		93	
	RR	χ^2	RR	χ^2
"Less-than-normal"	.40	2.23	1.44	3.02
Small opacities	.15	3.52	1.50	2.42
Large opacities	1.48	0.26	1.34	0.07
Pleural changes *	.46	1.40	1.52	2.33
Pleural calcification	0	1.15	.96	0.01
"Additional symbols"	.16	3.40	1.13	0.09
Thetford Mines *cf.* Asbestos	.79	0.14	.84	0.70
Accumulated dust exposure	.64	0.45	1.14	0.34
Cigarette smoking	1.73	0.67	1.42	2.71
Breathlessness	.30	1.50		
Persistent cough	3.13	2.36		
Persistent phlegm	1.38	0.10		
Wheezing	.48	1.22		
Depressed FVC	0	1.87		
Depressed ratio $FEV_{1.0}/FVC$	2.34	1.67		
Depressed MMF	.61	0.23		
Overweight	0	2.17		

* other than calcification

Table 12.6

DEATHS FROM CEREBRO-VASCULAR DISEASES
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	5		101	
	RR	χ^2	RR	χ^2
"Less-than-normal"	.82	0.03	1.17	0.59
Small opacities	0	0.77	1.23	0.69
Large opacities	5.25	3.40	4.43	9.90
Pleural changes *	1.07	0.00	.98	0.00
Pleural calcification	0	0.45	1.08	0.03
"Additional symbols"	1.08	0.00	1.65	3.18
Thetford Mines *cf.* Asbestos	.30	1.90	1.34	2.17
Accumulated dust exposure	1.03	0.00	1.63	6.07
Cigarette smoking	2.70	0.85	1.09	0.17
Breathlessness	4.29	3.02		
Persistent cough	0	0.37		
Persistent phelgm	0	0.39		
Wheezing	5.06	2.61		
Depressed FVC	9.02	8.56		
Depressed ratio $FEV_{1.0}/FVC$	1.57	0.16		
Depressed MMF	1.58	0.17		
Overweight	1.26	0.04		

* other than calcification

Table 12.7

DEATHS FROM ACCIDENTS
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	6		110	
	RR	χ^2	RR	χ^2
"Less-than-normal"	∞	2.59	1.58	5.24
Small opacities	∞	1.54	1.97	8.51
Large opacities	-	-	1.29	0.05
Pleural changes *	∞	0.43	1.39	1.17
Pleural calcification	∞	2.08	.64	0.34
"Additional symbols"	∞	1.66	1.41	0.86
Thetford Mines *cf.* Asbestos	2.10	0.48	1.00	0.00
Accumulated dust exposure	3.02	2.02	1.16	0.49
Cigarette smoking	1.07	0.01	1.80	7.46
Breathlessness	1.99	0.66		
Persistent cough	0	0.41		
Persistent phlegm	0	0.34		
Wheezing	1.52	0.26		
Depressed FVC	1.15	0.02		
Depressed ratio $FEV_{1.0}/FVC$	1.28	0.05		
Depressed MMF	1.12	0.01		
Overweight	1.15	0.02		

* other than calcification

The last table (12.8) deals with deaths from *causes known but not included in any group already discussed.* Although the Relative Risks for the higher dust exposure sub-cohorts were not particularly high, those for several radiographic changes were, and that for small opacities in Cohort "B" is inexplicable. The high risks, in both cohorts, among those whose films were read as having "additional symbols", might be explained by careful consideration of the particular "symbol" and the exact code of the cause of death. The smoking effect, present in both cohorts, could possibly be elucidated by similar detailed enquiry.

Table 12.8

DEATHS FROM OTHER KNOWN CAUSES
RELATED TO RADIOGRAPHIC CHANGES AND OTHER FACTORS

(Relative Risks, and χ^2 statistics)

	Cohort "A"		Cohort "B"	
Number of deaths	16		183	
	RR	χ^2	RR	χ^2
"Less-than-normal"	∞	3.86	1.53	8.12
Small opacities	∞	1.24	1.86	13.44
Large opacities	-	-	2.75	3.78
Pleural changes *	∞	6.61	1.22	0.73
Pleural calcification	∞	6.88	1.80	3.98
"Additional symbols"	∞	4.12	2.16	13.74
Thetford Mines *cf.* Asbestos	.75	0.32	1.17	1.08
Accumulated dust exposure	1.06	0.01	1.15	0.76
Cigarette smoking	2.76	2.74	1.51	7.31
Breathlessness	1.01	0.00		
Persistent cough	.94	0.00		
Persistent phlegm	.93	0.01		
Wheezing	.43	2.26		
Depressed FVC	1.35	0.22		
Depressed ratio $FEV_{1.0}/FVC$	.89	0.02		
Depressed MMF	.86	0.04		
Overweight	1.18	0.07		

* other than calcification

Chapter 13

S U M M A R Y O F F I N D I N G S

13.1 Introduction; total mortality

This chapter summarizes briefly the findings described in the preceding pages. Synthesis and discussion are in Chapter 14.

Those whose chest radiographs revealed abnormality of any feature (small opacities, large opacities, pleural changes with or without calcification, or additional symbols) had Relative Risks of total mortality greater than unity, substantially so far all features in Cohort "B"; in Cohort "A", the Relative Risks were generally rather smaller, but only the comparatively few deaths appeared to prevent "significance".

In Cohort "A", cigarette smoking was associated with the most significant Relative Risk (1.88, χ^2 = 10.18), more so than for any of the radiographic changes and considerably greater than that for the men most heavily exposed to dust (RR = 1.21, χ^2 = 1.10). Relative Risk of death from all causes in cigarette smokers was slightly less pronounced in Cohort "B" (1.67), but the dust effect was almost as large (1.59), and both were associated with very large values of χ^2. The dust effect in Cohort "B" probably accounted for the higher SMR at Thetford Mines than at Asbestos, in that cohort. In Cohort "A",

those with dyspnoea and depressed FVC also had enhanced Relative Risks.

13.2 Asbestos-related diseases of the respiratory system

The radiographs of those who died from *pneumoconiosis* generally showed parenchymal change, often well advanced. The Relative Risk of pneumoconiosis in those more heavily exposed to asbestos dust was high in both cohorts, but considerably less than the Relative Risk of those with pneumoconiotic changes. Smoking was not a factor, and the three cases in Cohort "A" were largely asymptomatic.

The great majority of the men who died from *cancer of the chest* were cigarette smokers, or had had substantial exposure to asbestos dust, or both. In both cohorts, cigarette smokers had a higher Relative Risk than those with heavier exposure, markedly so in Cohort "A" (RRs of 4.11 and 2.04, respectively), where none of the Relative Risks for radiographic change were very great. Perhaps because of the larger dust effect in Cohort "B", some of the Relative Risks for radiographic changes were considerable, with that for "large opacities" dominating. This last may well have been recognition, on the x-ray, of the malignancy itself; for those in whom "large opacities" were diagnosed, the interval between x-ray and death was usually short. In both cohorts, only about half the x-ray readings were of pneumoconiotic change, and it was generally not advanced.

There were only six *mesotheliomas*, one in Cohort "A", whose film at least six years before death showed bilateral calcified plaques, and five in Cohort "B", with highly variable x-ray findings. The very short duration of this illness, after diagnosis, means that the changes

on a routine x-ray taken during employment are likely to be largely irrelevant. The latent periods, between first asbestos employment and death, were rather shorter than in the remainder of those in the "main mortality cohort" who died from this malignancy.

13.3 Other respiratory diseases; heart disease

There were forty cases of *respiratory tuberculosis* in Cohort "B", all before 1967. Very high Relative Risks were found for all radiographic features, particularly for "large opacities" (perhaps again with some element of mis-diagnosis) and "additional symbols" (probably including those specific for tuberculosis, i.e. tba and tb).

The Relative Risks of *other respiratory diseases* in Cohort "A" were high for men with depressed lung function (MMF: 6.21; FVC: 5.95; $FEV_{1.0}$/FVC: 3.10) and dyspnoea (2.87); however, there was a low Relative Risk for those with heavy dust exposure and little excess risk associated with x-ray change. In Cohort "B", on the other hand, there was a considerable dust effect, and, probably as a result of this, high Relative Risks for large opacities, pleural changes (without calcification) and small opacities.

Death at work, or shortly after ending employment, was a criterion for exclusion from Cohort "A", but not from Eras 1 and 2 of Cohort "B". Some deaths ascribed to *heart disease* may well have been due to the after-effects of dust retention, and this is substantiated by high Relative Risks both among the heavily exposed and associated with small opacities, in both cohorts. Smokers also had high Relative Risks of heart disease, as has been shown in many other studies (see review by

Holman, 1976). Those in Cohort "A" who reported breathlessness also had high risk of death from heart disease.

13.4 Other cancers

The patterns with cancers of the *gastro-intestinal tract* are confused. For the upper tract (oesophagus and stomach), there was a major dust effect in Cohort "B" and some high risks associated with changes on chest x-ray, for which no explanation has been found. For the lower tract (colon and rectum), there were dust effects in both cohorts, together with high risks associated with pleural changes and, in Cohort "A", with breathlessness and depressed FVC. For other abdominal cancers, there were no clear-cut findings.

There were only six *cancers of the larynx*, all among smokers, confirming the dose-response relationship found by McDonald *et al.* (1979), while no marked excess, nor relation to dust exposure or x-ray changes, again confirmed earlier findings. The two cases in the smaller cohort reported breathlessness and wheeze.

No consistent findings were observed with *cancers of other sites.*

13.5 Strokes; accidents; other causes

Strokes and accidents should surely be unrelated to asbestos exposure or to x-ray change. However, for *cerebro-vascular diseases*, the Relative Risks for those with large opacities were high in both cohorts; in Cohort "A", so were the risks of those who were breathless, wheezed or had depressed FVC. Again, Relative Risks for *accidents* (including, but by no means dominated by, accidents at work in

the asbestos industry) were high for those with small opacities and, in Cohort "B", for smokers.

The Relative Risks for the *other causes* of death not so far summarized were high, in both cohorts, for men whose radiographs showed "additional symbols", small opacities, and pleural calcification, and for smokers.

13.6 Conclusion

Most of the findings summarized in sections 13.1 through 13.3 could be taken as validation of the system of radiological classification. Some of those in sections 13.4 and 13.5 are difficult to explain and require further investigation.

Chapter 14

D I S C U S S I O N , S Y N T H E S I S
A N D R E C O M M E N D A T I O N S

14.1 Introduction

This has been the report of research aimed at answering specific questions about the prediction of mortality in asbestos workers from changes observed in radiographs of men while still employed. The next sections examine the weaknesses and strengths, both of materials and of methods, for these purposes. Section 14.2 shows how the present study cohorts would not have been suitable for a general investigation of mortality, but were of great strength for the current objectives. Section 14.3 emphasises the limitations of the commonly used *a priori* form of reasoning and highlights the need for the adopted strengthening by use of *a posteriori* methods.

Premature death is the end-point of a disease process which may have been initiated or accelerated by exposure to agents such as inhaled asbestos dust or cigarette smoke, and which may have been manifest, during life, by symptoms or signs. Severe exposure to asbestos dust has long been known to be associated with increased prevalence of dyspnoea, and of radiographic changes in the parenchyma of the lungs, and of their pleura, and with excess mortality from pneumoconiosis, cancer of the chest, malignant mesothelial tumours of the pleura (and sometimes of the peritoneum), and certain other causes. Cigarette

smoking can lead to respiratory symptoms, depressed lung function, and increased mortality from lung cancer, coronary heart disease, and some other causes; such effects may be synergistic with those of asbestos exposure. Section 14.4 considers total mortality in relation to various signs and symptoms.

The successes and failures of radiology as a predictive tool are discussed in the following sections, 14.5, 14.6, 14.7, dealing with pneumoconiosis and related conditions, neoplasia, and other causes of death, respectively. The next sections review the validation of the current radiographic classification of dust-related chest conditions and discuss its use for the surveillance of the asbestos worker.

The chapter ends by considering oustanding gaps in knowledge, with recommendations as to how they might be filled.

14.2 Mortality in relation to dust exposure, smoking and mining area

The continuing study of the "main mortality cohort" has shown clear excesses of deaths from certain causes, with dose-response relationships, as summarized by McDonald and Liddell (1978). The two present study cohorts, "A" and "B", were selected to achieve specific objectives; they were not intended to provide any substitute for a general investigation of mortality in relation to factors such as accumulated dust exposure and cigarette smoking. Nevertheless, basic findings of this nature were very similar to those seen in the entire cohort of nearly eleven thousand workers. Had they not been so, serious doubts would have been cast on the utility of the present

research materials. In fact, both cohorts had strengths and weaknesses; particular concerns are with the periods of follow-up and the definitions of study intervals.

Cohort "A" was comparatively small and the period of follow-up was short. Deaths from all causes numbered only 130, but many other studies have been based on even smaller numbers. A more important deficiency was that, by definition of this cohort, any deaths occurring within a short interval after the last available radiograph were excluded; see below. Total mortality was close to expectation, but there were excesses due to pneumoconiosis and cancer of the chest. The three deaths from pneumoconiosis were among men with heavier dust exposure; the 22 deaths from chest cancer almost entirely in men who had been cigarette smokers, or had had heavier dust exposure, or both.

Cohort "B" was almost half the size of the "main mortality cohort"; however, relative to that cohort, there were certain selection biases. First, a rather higher than representative proportion of men had worked at Asbestos, because radiology had started there several years earlier than at Thetford Mines; thus some of the highest dust exposures were excluded. Secondly, in order to have had an x-ray while employed, the men were considerably younger, on average, than those in the "main mortality cohort"; see Table 14.1. Thirdly, as discussed in section 9.5 above, the selection procedure led to the inclusion of all men who died at work or shortly afterwards; this was the reverse of the situation in Cohort "A", and is also discussed below. Not least because of these earlier deaths, mortality from all causes was higher than expected in Quebec; there were excesses

Table 14.1

DATES OF BIRTH: COHORT "B" AND "MAIN MORTALITY COHORT"

	Cohort "B"	"Main mortality cohort"
Number of men	4,559	10,939
Percentage distribution by quinquennium of birth		
1891 through 1895	*7.2*	*13.8*
1896 through 1900	*9.3*	*17.7*
1901 through 1905	*10.2*	*18.0*
1906 through 1910	*15.7*	*16.7*
1911 through 1915	*25.6*	*16.0*
1916 through 1920	*32.0*	*17.7*
	100	*100*

from pneumoconiosis and related conditions (respiratory tuberculosis, "other" respiratory conditions and heart diseases) and from cancer of the chest and other sites for which asbestos has been implicated, particularly of oesophagus and stomach. Relations with dust exposure and smoking were found more-or-less as could have been forecast from the main studies.

14.3 Limitations of *a priori* argument

The main methods of analysis followed convention in that they used *a priori* reasoning, classifying the men into sub-cohorts according to the values of certain factors, and following each sub-cohort separately. Because of the many factors to be examined, each classification (with a few exceptions) had to be carried out without regard for any other, so that when factors were interrelated it became impossible to disentangle effects. This is a well-known disadvantage of the procedure, and there is no reliable means of overcoming it when cross-classification by two or more factors creates sub-cohorts so small that there are too few deaths within them to be reliable, particularly when any specific cause of death is under consideration. Methods of analysis such as GLIM (Nelder, 1975) attempt to deal with the problem by fitting models to the re-grouped sub-cohorts, and then seeking signs, in the full detail, of divergence from the model; however, this cannot be very revealing for the same reason, i.e. small unreliable cells in the multi-dimensional array.

The use of *a posteriori* methods - examining factors of interest among the cases, often in comparison against controls - can be more revealing and was adopted for the investigation of the more important

causes of death. Only where case-control studies have been carried out can we make statements on the sensitivity and specificity of the radiological system in predicting mortality.

One example of the problem of disentangling effects is afforded by the apparent differences between the two mining areas, Asbestos and Thetford Mines, in mortality from pneumoconiosis. There were 31 such deaths in Cohort "B", 22 of them from Thetford Mines, a disproportionally high number. However, this was undoubtedly the more dusty area and the need was to determine whether area or dust was the causal factor. When a control was chosen for each case, it was found that 18 of the controls were also from Thetford Mines; the number of cases from Thetford Mines with a control from Asbestos was eight, and the reverse occurred four times, so that, in the McNemar test based on the disparate case-control pairs, the test statistic was $\chi^2 = 1.02$, which with one degree of freedom has an associated P-value of about thirty per cent, i.e. without trace of significance. In other words, it would seem likely that the differences between pneumoconiosis cases and their controls were in the dust exposures (see the large differences in Table 11.5) and not in the areas where the men had worked.

14.4 Total mortality in relation to signs and symptoms

Review of the literature relating to non-exposed populations suggests that certain symptoms and lung function tests may predict mortality from respiratory conditions and also from heart diseases and all causes. There have been many studies of morbidity and mortality in patients with chronic bronchitis or emphysema (recent examples are by Cole *et al.*, 1974, and Petty *et al.*, 1976), but

what is relevant in the present context is mortality related to respiratory symptoms and signs in general populations. Higgins and Keller (1970) pointed out that little was known about the prognostic implications of what they called the "less severe but very common manifestations of chronic respiratory disease"; they then described a study of the adult population of the American town of Tecumseh. Here, 2,452 men over 16 years of age were examined in 1959-60, and 1,759 (or 72 per cent) were re-examined about four years later. The number known to have died in the interval was 113 and the deaths were classified according to:- symptoms at the earlier examination; diagnosis then of chronic bronchitis, emphysema or asthma; ventilatory capacity; and certain combinations. The method of mortality analysis, particularly in determining expected numbers of deaths, is not entirely clear. The sign or symptom with the highest SMR (2.5) was shortness of breath: it would appear that 63 deaths had been observed compared with 25 expected. The SMRs for cough and phlegm grade II and for chronic bronchitis were both 1.8, while men with $FEV_{1.0}$ below 2 litres had an SMR of 1.4. As *a priori* arguments had been used, without attempt to make the subcohorts mutually exclusive, the usual lack of independence of findings arose and has to be noted.

Kauffmann *et al.* (1975) examined the prognostic value of chronic cough, sputum and spirometry in a ten-year mortality study among 1,487 men working in factories near Paris. Mean indices of lung function, adjusted for age, were worse for those who died than for those who survived, but the differences were small and patterns were not consistent from one age group to another, the youngest, i.e. those aged 30 through 34 years, conforming least well. The "statistical signific-

ance" claimed for the differences lay largely in the numbers of subjects, and provided no grounds for prediction.

Ashley *et al.* (1975) related pulmonary function to ageing, cigarette habit and mortality, as part of the Framingham Study. In two medical examinations, approximately ten years apart, the numbers of men studied were 1,937 and 1,127. It is not clear whether all those examined on the later occasion were part of the cross-section from the former, but the proportion is at best 58 per cent. The only tabular material on mortality provided "standardized univariate logistic risk function coefficients for risk of death in ten years for FVC and $FEV_{1.0}$ per cent in an eighteen year follow-up" but no details are given of the methods. From a figure giving the "*smoothed* (my italics) probability of death in ten years by FVC at [the earlier] examination in those then aged 50-59" (without explanation of the reason for choosing this group) it might appear that there was a dose-response relationship.

This evidence on prognosis in general populations is suggestive but not particularly convincing.

In Cohort "A", where respiratory symptoms and signs had been assessed, dyspnoea and depressed FVC were associated with high Relative Risks of mortality from all causes, 1.49 and 1.53, respectively, but other symptoms and other function measurements had low predictive value. In the same cohort, total mortality was predicted especially well by cigarette smoking (RR = 1.88, with $\chi^2 = 10.18$), and also, but less well, by parenchymal changes on the x-ray (RRs of 1.56 for small

opacities and 1.81 for large opacities), by pleural changes (RRs of 1.80 for calcification and 1.81 for other pleural changes); the Relative Risks quoted in this sentence were higher than for the one respiratory symptom and the one lung function test that had fair predictive value. Accumulated dust exposure was associated with enhanced risk, but only to the extent of RR = 1.21.

In Cohort "B", all features of radiographic change predicted excess mortality from all causes (RRs of parenchymal changes: 1.98 for small opacities and 4.43 for large opacities; RRs of pleural changes: 1.87 for un-calcified and 1.62 for calcified; and RR = 2.21 for "additional symbols"); so did cigarette smoking (RR = 1.67) and comparatively severe dust exposure (RR = 1.51).

In all these "comparisons" of effects, it is important to bear in mind their lack of independence, and the different methods by which they have been assessed. However, it seems not unreasonable to infer that respiratory symptoms and lung function changes are not better predictors of excess mortality than radiographic changes, nor than cigarette smoking; asbestos dust exposure by itself does not appear a particularly good predictor of mortality.

14.5 Pneumoconiosis and related conditions

Pneumoconiosis is surely due to retention of dust in the lungs and so death from this cause should be associated with x-ray change. There are other conditions which may well be associated, directly or indirectly, with dust retention, and so should be detectable on the

radiograph, again either directly or indirectly, *viz:*- respiratory tuberculosis, "other" respiratory diseases and heart diseases.

Pneumoconiosis. The effect of asbestos dust exposure was obvious, but there was no effect of smoking. These findings were clear in the main mortality study, summarized by McDonald and Liddell (1978) and equally so in both cohorts of the present research; further they were confirmed by case-control methods in Cohort "B". Most of the radiographs of men who died from pneumoconiosis showed radiographic change, usually parenchymal, with or without pleural change. "Sensitivity" was found as 77 per cent, i.e. 24 men with changes in 31 deaths. "Specificity", assessed in the controls, was 71 per cent: 22 men without parenchymal change among 31 controls. The small opacities in the radiographs of men who died from pneumoconiosis were nearly always irregular; their profusion was comparatively high, all but four of the 37 readings, i.e. nearly nine-tenths, being of category 1/0 or greater. Large opacities were present only rarely and only when the background small opacities were of profusion at least 2/2.

In view of the causal association with dust exposure, some pneumoconiotic x-ray change was to be expected in every case of death from pneumoconiosis. However, there were seven men who died from this condition, all of whom had been exposed to at least 100 mpcf.y (four of them to more than 300 mpcf.y), in whom no radiographic change was noted. The explanation for this "failure" of the radiological system does not lie in a long interval between x-ray and death; the distribution of intervals was almost identical in those men whose films were

normal and others. However, one or perhaps two of the "failures" might conceivably have had time for a radiological attack as they did not die until 1975 and 1974, having been still at work in November 1966. Three of the seven "failures" had accumulated dust exposures by the time of x-ray of 987 mpcf.y, 1,036 mpcf.y and 2,798 mpcf.y. Although their deaths were attributed to pneumconiosis, it might be that the symptoms were non-specific but that known heavy exposure led to this cause being included at death registration. On the other hand, there may well have been some fibrosis of the lung generated by such large quantities of dust to which the men had been exposed, but the fibrosis was either not radio-opaque or had been missed in the radiographic assessment; three different readers were involved. Another factor that can be eliminated as an excuse for the "failures" is film quality: four of the seven films had been scored "+" and the other three "+/-".

Respiratory tuberculosis. Although this cause of death was common before 1965, much more so at Thetford Mines than at Asbestos, there were no deaths from this disease in Cohort "A" nor in Era 3 of Cohort "B". However, there were 40 deaths from tuberculosis in the earlier Eras of this latter cohort; Relative Risks were high for each type of radiographic change, particularly "large opacities" and "additional symbols". These shadows may have been of the tuberculosis itself, while the other recorded changes may have been indirect manifestations.

The generally accepted view has been that tuberculosis is not related to asbestos exposure, although it is well-known to interact with silica (see, for example, the review by Liddell, 1975c). Table 11.15 shows that men at Thetford Mines had a risk of death from respiratory

tuberculosis twice as high as that at Asbestos, while the Relative Risk associated with heavier dust exposure was rather less (1.64). This might suggest that for this disease, the causative factor lay in the socio-economic environment of Thetford Mines, rather than in the asbestos dose; that dust exposures were heavier there may well have been only coincidental.

"Other" respiratory diseases. Relative Risks of these conditions were very high for men in Cohort "B" with large opacities, un-calcified pleural changes and small opacities; they were also high for "additional symbols", and in men with heavier dust exposures. In Cohort "A", on the other hand, the factors with the highest Relative Risks were depressed MMF and FVC. It may be that the "mixes" of diseases included under this heading were rather different in the two cohorts, because of the different eras in which the deaths occurred, i.e. only after 1967 in Cohort "A", but as far back as 1951 in Cohort "B".

Heart disease. Because of the definition of Cohort "A" that men had been interviewed and tested at least six months and often over two years after the x-ray had been taken, sudden death closely following that event would exclude from this cohort, but not from Cohort "B". Thus, for this cause of death also, different patterns would not be surprising. In Cohort "A", the highest Relative Risk was for dyspnoea; all radiographic features were also associated with enhanced risk, as were dust exposure and cigarette smoking. The lung function measurements showed little association with mortality. In Cohort "B", the most significant Relative Risk was for cigarette smokers, but heavy accumulated dust exposure, small opacities, pleural changes and "add-

itional symbols" were all associated with excess mortality.

Holman (1976) states that: "Numerous epidemiological studies have indicated that cigarette smokers have increased mortality ratios for Coronary Heart Disease (CHD); that is, cigarette smokers show significantly increased death rates compared with non-smokers. The risk incurred by cigarette smoking increased with increasing dosage and, as measured by mortality ratios, is more marked for men in the younger age groups, under age 60, although the absolute increment in death rates experienced by smokers over that of non-smokers continues to increase with increasing age." Her most reliable sources appear to be Hammond and Horn (1958), Doll and Hill (1964), Best (1966), Kahn (1966), Hammond and Garfinkel (1969) and Weir and Dunn (1970). The studies varied in size, from 41 thousand British doctors to 359 thousand US males, in ages at entry, in length of follow-up (three-and-a-half years to ten years) and in categorization of smoking habits. This categorization is not always entirely clear in Holman's Table 3, but the highest category was never less than 20 cigarettes a day. The Relative Risks of death from CHD for those who smoked most, compared with non-smokers, were, for the six studies in the order given above:- 2.41; 1.43; 1.78; 2.00; 1.77; and 1.74. The basic finding that cigarette smokers have increased mortality ratios compared with non-smokers is fully supported by Relative Risks of 1.38 and 1.78, in our two cohorts (Table 11.17).

However, CHD accounts for by no means all the mortality ascribed to diseases of the heart; a substantial proportion of deaths of this nature are in fact secondary to lung disease, and there is likely to

be some mortality in an asbestos-exposed population that is coded on death certification as due to heart disease which has arisen as a consequence of the dust exposure. This is confirmed by the high Relative Risks in both cohorts for men with heavier dust exposure and for each radiographic feature, although the "additional symbols", so prominent in Cohort "B", may be to some extent at least a direct record of heart disease unrelated to dust exposure.

14.6 Neoplasia

Chest cancer is undoubtedly associated with smoking and dust exposure. The illness, once diagnosed, has short duration. Thus, if the interval between x-ray and death is short, the cancer itself might be detected; if the interval is longer, it is possible that there would be signs of dust retention.

Parenchymal changes were seen in about a third of the chest cancer cases in both cohorts, with pleural thickening and calcification in a further eighth; in Cohort "B", one-tenth more showed only "additional symbols". The parenchymal changes were quite different compared with those in pneumoconiosis deaths; there were rather more rounded opacities, profusion was rather lower and there were several cases of "large opacities", mainly poorly-defined, often on a low or even non-existent background of small opacities. In the 118 cases, some radiographic change was seen in 70 (sensitivity 59 per cent); among 118 controls, films were normal for 80 (specificity 68 per cent).

It must be counted a "failure" when the radiograph was assessed as normal despite substantial dust exposure, say greater than 300

mpcf.y. In 62 men who died from chest cancer after exposure to at least this amount of dust, only 36 showed any radiographic change and in only twenty of these cases was the finding of small opacities. Indeed, the proportion of cases whose films showed small opacities was independent of smoking habit and dust exposure (Table 11.12). However, there is insufficient evidence at this stage to answer the question whether cancer of the lung is a complication of asbestosis or an independent reaction to the retention of asbestos dust, i.e. unrelated except by chance to fibrotic reaction.

However, some of what have been called "failures" were due to long intervals between x-ray and death. When this interval was substantial, the proportion of "normals" was considerably higher than when the interval was short (see section 11.3). Nevertheless, there was another form of "failure" of the radiological system when death was not long after the x-ray: a considerable proportion of the radiographic changes that were recorded were of "large opacities", i.e. an apparent recognition of the malignancy itself, but classifying it as though it were pneumoconiotic.

Mesothelioma. Intervals between x-ray and death were either greater than five years (three deaths) or a year or less (the other three deaths). In the former situation, only indirect evidence could be found, because of the very short duration of the disease; the three films were recorded as (1) with pleural calcification (by all six readers for the one case in Cohort "A"); (2) with small opacities of type "p" and profusion 1/0; and (3) normal. In the shorter intervals, it would not have been impossible that the tumour itself was recog-

nized, but the readings were (4) pleural calcification associated with irregular diaphragm; (5) small opacities type "t" of minimal profusion, 0/1; and (6) an "additional symbol" - the only reading that might have been a correct diagnosis of the mesothelioma.

Cancer of oesophagus or stomach is thought to be dust-related but could not be seen directly on a radiograph of the chest; there were, however, pneumoconiotic signs in a substantial proportion of cases. With *other cancers of the gastro-intestinal tract*, the signs were less marked, being weak for cancer of colon or rectum and particularly so for other abdominal cancers.

There is little evidence that *laryngeal cancer* is related to chrysotile exposure, and there were no indications to that effect in this study. There were very few deaths from this cause and all the men had been smokers; there had been too few to provide any measure of statistical confidence in the association with smoking from this study alone, but the main mortality investigation gave clear evidence of a smoking effect.

With *other cancers*, there was little indication of association with radiographic change.

14.7 Other causes of death

There is little reason to believe that the remaining cause groups could be realistically related to asbestos exposure, and so the effects that were observed are surprising and difficult to rationalize. Deaths from strokes appeared to be predictable from depressed FVC, the pres-

ence of large opacities on the radiograph and heavy dust exposure; accidental death from small opacities and smoking. As to "other" causes, higher risks in both cohorts in relation to radiographic changes could only be elucidated by examination cause by specific cause, even although it had been thought that all *relevant* causes had been included in one or other of the groups already discussed.

14.8 Validation of the radiographic classification

This research has provided clear validation of the U/C classification for those diseases which are both asbestos-related and distinguishable on x-ray. There was similar validation for those conditions only indirectly detectable on the radiograph.

The importance of *small irregular opacities* and *pleural changes*, introduced into radiographic classifications for the first time in 1967, requires emphasis. The reason for including these features was that they were frequently seen in the x-ray films of asbestos workers although no need for a classification of them had arisen for those exposed to silica or coalmine dust. Nevertheless, three recent studies have to be mentioned.

Amandus *et al.* (1976) stated that Carilli *et al.* (1973) had demonstrated much more frequent occurrence of "small irregular opacities" in smoking than in non-smoking females. However, the radiographic classification in the earlier study was non-standard, having been devised with the aim of seeing whether two particular readers could identify which subjects were smokers and which not in terms of x-ray appearances. A little later, Theriault *et al.* (1974) claimed to have shown

an association between smoking and small irregular opacities in granite shed workers. Here, a routine chest x-ray film of each of 784 workers had been assessed into the U/C classification (Bohlig *et al.*, 1970); but, unfortunately, only one reader was involved. He found small opacities in 233 films (29.7 per cent), mostly rounded (166 films); the prevalence of irregular small opacities of profusion category 1 or more was 67 of 784 men, or 8.5 per cent. Interpretation of the findings is difficult, but the so-called direct relationships between smoking and both the profusion and type of small irregular opacities appear to be no more than a "confounding" with an effect of age. Finally, it is particularly hard to disentangle the results of Amandus *et al.* (1976), because of oddly selected samples, of smoking and non-smoking coal miners, chosen in terms of *earlier* radiographic assessments, once more by a single reader; in particular, subjects in whom profusion of 0/1 had been read were excluded. In both smokers and non-smokers, just 87 per cent of the selected films had been placed in category 0 of small opacities. Among the 4,479 smokers included in the study, irregular small opacities, with or without rounded opacities, had been seen in 287, or 6.4 per cent; in the 1,687 non-smokers, 59 had had irregular small opacities (3.5 per cent).

Whether any of these findings would have been repeated with other film readers remains a matter of speculation. The findings from the present research were much more positive, and it must be stressed that they are based on the radiological assessments of six readers who had helped to devise the classification they used. Nevertheless, they showed the usual degree of inter-observer variation, and there was some evidence of differences between them in their ability to predict

mortality (see, for example, Table 10.3).

Although only one reader made substantial use of the *0/- category of profusion* of small opacities, there was some suggestion that it indicated a truly exceptional normality associated with good prognosis. More attention should be paid to this category; it does not appear to be purely a reflection of youth, nor is it a mask for emphysema which has obscured lung markings.

Instructions on *averaging the profusion of small opacities*, particularly when those of both types co-exist, have not remained standard with the various classifications. The 1967 notes in the Appendix state that: "As in the case of rounded small opacities, the profusion of the irregular small opacities is based on an averaging over *the whole lung* and by comparison with the standard films", but ILO (1972, p. 9) has it that: "The category of profusion of both types of small opacities is determined by averaging the profusion in *the affected zones*", and appears to add confusion by continuing: "When there is marked difference in profusion in different zones of the lung, the *zones predominantly affected* are the ones over which averaging is made". Although almost immediately there is an exhortation to state the method of averaging in publications using the classification, such a statement seems never to have been made. It is now not possible to determine exactly how averaging was carried out in 1967 during the reading described by Rossiter *et al.* (1972) and used as the basis for the present research. Thus, whether the scoring system described in Table 8.1 was the best that could have been devised cannot be known.

However, it did allow some *grading of the numbers of small opacities*, rounded and irregular combined, in both cohorts. In Cohort "B", the SC scores were placed in three groups:- 0 ("absent"); 1 through 3 ("slight"); and 4 or greater ("positive"). Bearing in mind the method of obtaining SC, the "slight" group had no more than profusion 0/1 in half the lung zones. In Cohort "A", the sums of the 6 SC scores were also placed in three groups:- 0 ("absent"); 1 through 6 ("slight"); and 7 or greater ("positive"). Here, the "slight" group represents even lower profusion averaged over the six readers. The SMRs for all causes of death in the two cohorts were:-

Small opacities	Cohort "A"	Cohort "B"
"Absent"	.78	1.30
"Slight"	.96	2.08
"Positive"	1.32	2.48

Although not of "statistical significance" at a conventional level, the gradient in both cohorts is suggestive. It was maintained for several causes of death, indicating that even the very slight parenchymal changes ("normal" in the *Short* U/C or ILO U/C classifications) *may* have some clinical significance.

Further investigation is required of methods of scoring, as well as of averaging; advice is also needed on how to record *combined profusion* when both rounded and irregular forms exist together. The possibility mentioned by Liddell (1977, p. 94) for each lung zone to be treated separately for purposes of categorization would appear to deserve exploration. Although, when it was first proposed, it was thought unlikely to recommend itself to readers, several experts have since expressed interest in such a procedure. This would probably

require, first, scrutiny of the whole film to aid identification of the changes seen zone by zone; thereafter, it should be possible to record *for each zone as a unit* whether small opacities exist and, if so, of what type(s) (p, q, r; s, t, u) and profusion(s), with a final assessment of how much of each zone is affected *in toto*.

It is also desirable to reduce the "middling tendency" (Morgan *et al.*, 1974) and help might well be given by the use of *standard films on the border-lines* between categories, instead of films representing the mid-points of categories, as at present.

There are, no doubt, other ways of improving the present classification, but change should not be made for one purpose without considering the effects for other purposes. It now appears (Dr. Robert N. Jones, personal communication) that the meeting to be held in Washington in September 1978, to make recommendations to the ILO's meeting of experts in Caracas the following month, is to be concerned mainly with the classification for purposes of compensation. Such purposes are undoubtedly important, but differ for each compensation system, even within one country's administration, depending on the mineral to which the claimants have been exposed; they may differ greatly from one country to another. It was a great strength of the ILO U/C (1971) classification that it could be used for compensation, clinical and epidemiological purposes, with variation only of emphasis. A change of the classification to facilitate its use within compensation schemes that is not compatible with its use in other circumstances, with concern also for historical comparisons, would be a grave error.

14.9 Surveillance of the asbestos worker

Sensitivity and specificity of various tests which might be used for surveillance of workers are, as always, conflicting aims. Smoking, dust exposure, some function tests, respiratory symptoms, early radiographic changes can all be made sensitive, but in certain conditions it would appear that radiographic changes are the most specific in relation to the forecasting of mortality in asbestos-exposed populations. There follows an example of the way in which these measures of a test vary with the level at which a decision is taken: it deals with small irregular opacities in predicting death from pneumoconiosis. Using profusion of category 0/1 as the cut-off point yielded sensitivity 64.5 per cent and specificity 80.6 per cent, and there would have been 714 false positives among a total of 734, or 97.3 per cent. However, with 1/0 as cut-off, sensitivity was reduced to 58.1 per cent, while specificity increased considerably to 96.8 per cent, and the proportion of false positives was reduced to 95.4 per cent.

Little is yet known about progression of radiographic appearances while workers are still employed or after withdrawal from exposure, when consideration is concentrated on the problems of prognosis. Serial assessments are required on radiographs and of symptoms, particularly breathlessness, perhaps assessed by a simple standardized exercise test to replace the reliance on answers to a questionnaire. Study should also be made of how the reliability of predicting mortality is changed by variation in the length of the study interval.

Specific recommendations would be to extend the tracing of men in Cohort "A" for several more years, perhaps in the first place to the

end of 1978; by this time there will have been substantially more deaths and it should be possible to make more reliable statements on the relative importance of radiological and other signs and symptoms, particularly if there have been enough deaths to justify case-control studies, or other *a posteriori* methods. Further, there were 277 men in this cohort who were subjected in 1974 to a second set of measurements, including chest x-ray. Follow-up of these men for a rather longer period - to allow adequate mortality for investigation - might cast light on the problems.

Further possibilities arise in another group of men, those in the "radiographic progression cohort" described in section 4.9. Although they numbered only 283, and almost all were still alive at the end of 1975, in a few years there should have been sufficient mortality to justify detailed study. One advantage of this group is that x-ray films spanning an average of twenty years have already been assessed by a group of experienced readers into the ILO U/C (1971) classification by two methods, independent randomized reading and side-by-side reading, the latter with certain scales of abnormality elaborated four-fold beyond even the twelve points of the complete classification of profusion in the current international classification.

14.10 Other possible developments

The recent (U/C, 1968) classification of the pneumoconioses has been almost completely justified in its ability, when used by the six readers who formed the McGill University panel, to predict mortality from asbestos-related disease and even from other causes. That is not to say that sensitivity and specificity were perfect; they were far

from that ideal, but very much higher than in studies of the earlier (ILO, Geneva, 1958) classification in relation to the mortality of coal miners. Nor is it to be thought that the present, ILO U/C (1971), classification is itself ideal. However, the principal need is to build from this essentially valid system.

In the first place, the important questions concern generalizability: can other readers do as well, or better, than the six whose assessments were used in this research? can their reading be improved for use in the prediction of mortality, either generally or by bringing the less successful up to the levels of the more successful?

In our own cohorts, some of these questions could be answered as follows:-

1. In Cohort "A", two hundred or so men of those originally selected for clinical examination but who were not included because of death, illness, refusal, etc., should be incorporated.

2. The date on which each x-ray was taken should be ascertained, so that information could be gleaned on asbestos exposure to that point and afterwards, particularly for those men who continued employment after November 1966.

3. The study intervals should be divided into several periods, short to start with and then of increasing length, in order to learn more about the effects of length of follow-up on

prediction of mortality. If the follow-up is too short, not only will there be few deaths but there would be little evidence to assist in surveillance. When the period of follow-up is long, there will be many deaths, but only indirect effects are likely to be apparent on the x-ray.

4. Work histories should be reviewed, probably in case-control studies, to reduce the workload substantially; such studies could be extended to as many cause groups as might be considered of interest.

5. The radiographs of all men included in the case-control studies just mentioned should be re-examined by the same six readers, still working independently, and, more importantly, by other readers, to allow generalization. In this regard, it would be essential to follow the principles enumerated by the Workshop on the Chest Roentgenogram as an Epidemiologic Tool (Weill and Jones, 1975) by having at least three readers, working independently and "blind" to the provenance of the films, which should include some of unexposed workers; possibilities of repeat reading should also be examined.

6. Purposive study should be made of films which showed abnormality not "justifiable" by the provenance of the film. E.g. what are the appearances recorded as "large opacities" which arose in lung cancer, stroke, etc.? is there any way that stroke, for example, could possibly be predicted from

x-ray?

It might be argued that a radiological classification which has been so well validated in chrysotile miners and millers would not require further study where both radiographic and health effects are known to be much more dramatic. However, it would seem important to extend the questions, asking whether the system works as well in populations exposed to other fibres, either chrysotile in the manufacturing processes, or amphibole in production or manufacture, or in mixed situations.

Postscript

ORIGINALITY; IMPORTANCE; RESPONSIBILITIES; ACKNOWLEDGEMENTS

My interests in relationships between radiology and mortality arose while I was developing the NCB elaboration of the profusion of small opacities and studying the morbidity and mortality of coal miners - remaining disappointed that the prediction of mortality from x-ray findings among the miners of the Rhondda Fach had been so poor. In mid-1976, when Professor McDonald's team reviewed gaps in epidemiological knowledge, the research described in this thesis became feasible: the question that had intrigued me for so long had also been posed by Dr. George Wright, chairman of the Scientific Committee of the Institute of Occupational and Environmental Health; appropriate information could be obtained from the population already under study in Quebec; and I could take day-to-day responsibility for the project.

There was never any doubt about the importance of the project, but the recent confirmation from the 1977 Johannesburg Asbestos Symposium was particularly encouraging. The research has substantially validated the current international radiological classification, but generalization is still required. Some light has been cast on the use of the chest x-ray in the surveillance of the asbestos worker, but here too further research is needed.

The "buff card" register of employees was initiated by Mr. Charles Rossiter, of the MRC Pneumoconiosis Unit, during secondment to McGill University, and he and Dr. Graham Gibbs devised means for calculating the various indices of exposure. The tracing of subjects and, almost as difficult, the ascertainment of causes of death were carried out under the supervision of Professor Alison McDonald; credit for the very few losses and the excellent standard of linkage is due first to her, and also to her two devoted colleagues, Mesdames

Beauchemin and Proulx, in the Eastern Townships. Most of the tracing to the end of 1975 had been completed by the time the present research was under way, but some was still in hand, while men not in the "main mortality cohort" had to be traced and causes of death found; these tasks were completed by the same team, to whom I am extremely grateful.

Although the radiological studies reported by Mr. Rossiter and his colleagues had been largely completed before I started work at McGill University, the 42 thousand readings had been the first major use of the NCB elaboration outside the National Coal Board itself. Mr. Rossiter's contributions, not only of the statistical nature reflected in his publications, but also, and perhaps more important in the present context, of data processing, were quite invaluable. I also gained greatly from discussions with all six film readers, particularly during the project reported by Dr. Gail Eyssen; especial thanks are due to Dr. John C. Gilson, CBE, FRCP, formerly Director of the MRC Pneumoconiosis Unit, who has been a source of great stimulation over many years.

Because so many changes had become necessary for the main mortality investigation, it was decided in 1975 to devise a new system of data processing, rather than attempt to up-date files and programs that had already been extensively patched. The many necessary programs for this new, more flexible, system were written to my specifications by Mr. Mario Rodrigues, who also applied them conscientiously to the several vast bodies of data, requiring many iterations before we could consider the data files to be truly "clean". It is a pleasure to record the gratitude of the entire team to Mr. Rodrigues for this work, which led to a particularly meritorious M.Sc. At all stages, the problems of record linkage have been great; my intimate knowledge of the procedures and practices adopted in the main mortality enquiry made it possible to apply the same standards in the present research.

My concern with methods of cohort analysis has been intensive since shortly before the meeting of statisticians at McGill University

in June 1974, through the presentation to the Royal Statistical Society in June 1977, to the present. My thanks are due to many statistical colleagues who have helped, a few "formally" in 1974, but most quite informally over the years. They include: Professor Peter Armitage; Mr. Geoffrey Berry; Professor David Cox; Dr. Gerald Draper; Professor Phil Enterline; Dr. Gail Eyssen; Professor Michael Healy; Dr. David Hill; Dr. Larry Muenz; Dr. David Oakes; Dr. Peter Oldham; Mr. Charles Rossiter; Dr. Peter Smith; Mrs. Joyce Snell; Dr. "Babs" Solari; and Dr. Duncan Thomas.

Without access to the current findings of the main mortality investigations, this thesis would have been much less satisfactory. I supervised Mr. Rodrigues, in 1976-77, in the analysis of deaths to the end of 1973, and thereafter in the up-date and re-analysis to end-1975. However, in interpretation, my contribution has been essentially co-operative. In particular, the recent review paper (McDonald and Liddell, 1978) was prepared principally to show how results had been essentially unchanged since the outset, and frequent reference to it should not be construed as suggesting that I had any role other than that indicated here. However, this paper has provided many useful up-to-date results and has already been presented, so that it should appear in print before "McDonald *et al.* (1979)", which is still in preparation. Nevertheless, it is important to emphasize that the results of our main mortality enquiries are the fruits of team effort.

I wish to thank all members of the research team at McGill University, i.e. Professor Margaret Becklake; Dr. Gail Eyssen; Dr. Graham Gibbs; Professors Alison and Corbett McDonald; and Dr. Duncan Thomas, for permission to use data and for willingly-given advice. Data processing and other computing, all to my specifications, were entrusted to Mr. Rodrigues, and I owe him an enormous debt of gratitude which I am very pleased to acknowledge; I also thank Ms. Marielle Olivier who assisted from time to time. Thanks are also due to the Institute of Occupational and Environmental Health for their financial support to Professor McDonald for this research.

Two others deserve especial mention. The first is Mrs. Ann Yaxley, who has assisted cheerfully, often well "beyond the call of duty". It would be impossible to describe all the ways in which she has helped, and I am enormously grateful to her.

Finally, Professor Corbett McDonald, friend and colleague for many years, has been an inspiring supervisor. He has criticized constructively from his encyclopaedic knowledge of the subject, yet without interfering, and has shown me how to extend my reading much more widely than would otherwise have been possible. He is the Principal Investigator of "the only really grand study" of which my project has formed but a part, and it was only with his permission and under his aegis that I was able to use the data and conduct this research. The gratitiude I owe him is clearly unrepayable.

In conclusion, I must take full responsibility for any errors in this thesis (in particular for those concerning taped information on certain symptoms), and for all opinions expressed.

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Appendix

INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION

The following pages (368 through 374) are reproductions of the earliest known notes on the use of the U/C classification, later published in slightly amended form by Bohlig *et al.* (1970). These instructions bear the "imprint" JCG/MAR, the reference 67/27 and the date 29th December, 1967.

The "Appendix II" referred to at the foot of the first page of the instructions is reproduced here as page 376; a reduced version of the reading sheet of "Appendix I", referred to on the second page of the instructions, is on page 375 of the thesis.

The list of Symbols (see page 374) was not attached to the present copy of the original instructions.

CT/2/

INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION

29th December, 1967.

Technique

Inspection for technique:

if good or acceptable, record "plus" +

if poor, record "plus/minus" +/-

if very poor but just readable for some features record +-- and add comments on type of defect

if unreadable record "minus" -

Only use unreadable if you think the reading is likely to be too doubtful to convey any appreciable information. If unreadable, make a note of why in "comments".

Classification

Look at the film and decide if any of the changes seen in the pleura or the parenchyma are sufficiently characteristic of pneumoconiosis (including that seen in asbestosis) to be recorded in the classification. If it is probable that all the changes seen are the result of some other aetiology do not classify, but record opinion using symbols, e.g., Tb, Np (neoplasm), etc., and by other notes in the "comments" column. If the changes might be due in part to pneumoconiosis record on the classification scheme but make note in "comments" what other aetiology is likely.

Note: there is no provision in this classification for doubtful pneumoconiosis ('0' used in the I.L.O. 1958 classification and the modification of this by the U. S. Public Health Service). The reader is required to make a decision one way or the other, but there is provision within the classification for recording changes which are less than the first category of "small rounded" and "small irregular opacities".

Small Rounded Opacities

These are classified as on the I.L.O. 1958 scheme as modified by the U. S. Public Health Service (see Appendix II).

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INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION, 2

Profusion: The profusion is determined using the I.L.O. standard films and the verbal definitions, with the N.C.B. elaboration to give extra information about where the film lies within the I.L.O. categories 0, 1, 2 & 3. The instructions for using the N.C.B. elaboration are: "Record the I.L.O. category into which you would classify the film following the normal convention. If, in the course of that classification, you considered an adjacent category as a serious possibility, record this after the formal category".

The system of coding is shown at the bottom of the reading sheet (Appendix 1) and is a 12-point scale from 0/- to 3/4.

Notes:

(i) regard the I.L.O. 1958 films as typical (mid-category) films showing 1, 2 and 3.

(ii) within category 0 record as 0/- those films which are indubitably normal at first glance (barn-door normals)*. Those which are normal after a brief inspection are recorded as 0/0. Those in which you seriously considered category 1 before deciding that they were category 0 are recorded as 0/1.

(iii) although there is no formal category 4, record those films which you regard as being above category 3-standard as 3/4.

Type: On the basis of "averaging" for both lung fields record the predominant type as follows -

p = pin-head (I.L.O.) small opacities up to 1.5mm.

q = micronodular (m) (I.L.O.) - opacities with diameters 1.5 to 3mm.

r = nodular (n) (I.L.O.) - diameters 3 to 10mm.

Notes:

(i) 'p', 'q', 'r' which should be in small case not capitals, are used in preference to 'p', 'm' and 'n' on the I.L.O. 1958 scheme, because 'm' and 'n' may get confused in writing and in speaking when recording the classification. Also, with 's', 't' and 'u' they form an alphabetic sequence of coding of the type of small opacities.

* (Barn door, "used joc. of a target too large to be missed" Oxford English Dictionary).

INSTRUCTIONS FOR USING ILO/CINCINNATI CLASSIFICATION, 3

Small Irregular Opacities

This group in the classification is used to record those films in which there is a filling of the rib spaces with irregular opacities of various thicknesses, shapes and densities, but which are not clearly rounded. As in the case of the small rounded opacities, the irregular small opacities tend, particularly when present in profusion, to obscure the normal lung architecture. Hence obscuration of the normal lung pattern is a feature of both those films with small rounded and small irregular opacities.

Small irregular opacities may be present alone or with small rounded opacities. Both should be recorded if it is thought both can be convincingly seen on the same film. It may, for example, be possible to see small rounded opacities in the upper zones and small irregular opacities in the lower zones, or vice versa. If only one type of opacity is present, record this and record 0/0 for the other type.

Profusion: As in the case of rounded small opacities, the profusion of the irregular small opacities is based on an averaging over the whole lung and by comparison with the standard films. However, unlike the small rounded opacities, the profusion of the irregular small opacities is judged purely by reference to the standard films and there is no verbal definition of the extent (number of rib spaces) affected for each category.

Type: The irregular small opacities are divided into three types -

s = fine, irregular or linear opacities

t = thick irregular or linear opacities

u = coarse (blotchy) opacities (not obviously linear)

Notes:

(i) The type should be decided on the basis of predominant feature of the film taken as a whole. Thus, only one type - 's', 't' or 'u' will be recorded for a film. This is the same convention used for classification of 'p', 'q' or 'r'.

(ii) On account of the variation in shape and size of the irregular small opacities no dimensions are specified (cf., 'p', 'q', 'r'). Classification of type depends largely on comparison with the standard film. In general, if in doubt record 't'. 's' is used for films in which the pattern is definitely fine and more linear, 'u' for films in which the shadows are 'blotchy' with little evidence of linearity.

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INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION, 4

Site of Small "Rounded" or "Irregular" Opacities: in order to provide information about the predominant site (lung zone) in which the small rounded or small irregular opacities are present the reading sheet provides a box with six partitions for recording which zones the opacities are best seen. These boxes should be completed by putting a tick in the appropirate space.

Notes:

(i) The provision of boxes for recording the zones most affected has been made because, whereas in many types of Pneumoconioses the upper zones are usually affected first, this is not thought to be so in Asbestosis. The added information about the lung zones most affected may help to separate the changes in the lung appearance associated with different types of dusts.

Large Opacities

In order to cover a wider range of features of large opacities than is described in the I.L.O. 1958 scheme, the large opacities are divided into two types: (i) well-defined, (ii) poorly-defined.

Well-defined: These are the large opacities of the I.L.O. 1958 scheme. Their size is classified according to the largest diameter using the standard films, A, B and C, and the verbal and pictorial definitions in the U. S. Public Health Service extension of the I.L.O. scheme (Appendix II).

Poorly-defined: D, E and F, are used to record large opacities in which the edge of the shadow is markedly ill-defined and yet the appearance is one of a large shadow in the lung parenchyma. The size is recorded using the same definitions as A, B and C opacities.

Notes:

(i) It is not certain how useful or frequently recorded this type of ill-defined opacity will be. It was introduced to cover the appearances occasionally seen in the Asbestos-exposed workers where the large opacities may be much less clearly demarcated from the surrounding lung than in other types of Pneumoconioses, but the system may be useful to record the ill-defined larger opacities also seen in other types of Pneumoconioses and which are not well covered by the I.L.O. 1958 scheme.

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INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION, 5

Pleural Thickening

The type, severity, and site are recorded using standard films, but in order to simplify the classification and improve its specificity certain conventions are used.

Type: Two types are recognised. "Diffuse" and "plaques". When both are present both are recorded (see Appendix I).

A "plaque" is recorded only when an area of pleural thickening has a sharply defined edge and is plate-like but uncalcified. Plaques are usually limited to the wall but if definite should be recorded when present on the diaphragm or elsewhere.

"Diffuse" pleural thickening covers all other types including the narrow linear bands running parallel to the chest wall.

Standard films are provided to demonstrate the appearances and the grading (severity) of pleural thickening.

Site and Severity

Costophrenic angle: Record this as present or absent, right and/or left. A standard lower limit film is provided. No upper limit film is provided, but the convention is to report "C.P." alone where there is approximately equal length of change along the surface of the diaphragm and up the chest wall. If the thickening extends appreciably further up the chest wall, or along the diaphragm, then the film should be classified as "C.P." and Pleural Thickening (diffuse or plaques) on the wall and diaphragm.

Notes:

(i) Loafing of the diaphragm should not be recorded as 'C.P.' even though it leads to the obscuration of the costophrenic angle.

Walls: Pleural thickening on the chest wall is recorded in three grades. Lower limits of these, 1, 2 and 3 are provided in standard films. The grade is averaged for both lung fields together and also for diffuse and/or plaques. The site is recorded as right and/or left.

Diaphragm: Where there is a definite plaque on the diaphragm, this should be recorded as present, right and/or left, and graded as part of a general pleural thickening, affecting the whole film.

INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION. 6

Irregularity of Diaphragm and Cardiac Border

There is uncertainty whether the irregularity of the diaphragm and cardiac border which are not uncommon in pneumoconiosis is due to pleural thickening and/or super-imposition of other shadows. The need to record the appearance and severity of change has been provided for as follows:-

Irregular Diaphragm: This is recorded simply as present or absent, right and/or left, using the standard film as lower limit.

Notes:

(i) The lower limit is intentionally set fairly high as small irregularities of the diaphragm are not thought to be very important epidemiologically.

(ii) The classification is purely descriptive and therefore, the diaphragm is recorded as irregular whatever the cause, whether this is actual thickening of the pleura, or distortion (if marked), or fuzziness, or if the contour is markedly altered as a result of pleural calcification.

(iii) Irregularities, considered to be due to leafing, are not recorded.

Irregular Cardiac Border: This is graded using the lower limit standard films 1, 2 and 3 which are based on extent of irregularity rather than degree of fuzziness.

Notes:

(i) Special attention is given to the left cardiac border.

(ii) The abnormality recorded is fuzziness of the outline whatever the likely cause (as for irregular diaphragm).

(iii) Irregularity, thought simply to be due to cardiac fat casting a double shadow along the border is not classified as cardiac irregularity.

Pleural Calcification

This is graded 1, 2 and 3 using the lower limit standard films provided, and recorded right and/or left on the diaphragm and/or the wall. If present in other sites, for example, the mediastinum or the fissure, this is noted in the appropriate column. The grading is averaged over the whole lung -

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INSTRUCTIONS FOR USING WHO/CINCINNATI CLASSIFICATION, 2

Pleural Calcification (continued)

Grade 1 : less than 3 square inches (approx. 16 sq.cms.)
Grade 2 : less than 9 square inches (approx. 260 sq.cms.)
Grade 3 : 9 square inches and above (approx. 260 sq.cms.)

Notes:

(i) Pleural calcification should be assessed, using both the sharpness of definition of the shadow, as well as its density. The comparison of density with neighbouring rib structures may help to decide whether or not it should be graded as a pleural calcification as opposed to a pleural plaque (uncalcified).

(ii) The total area is estimated by summating the areas as seen on films and allowance is not made for the larger actual area of plaque when it is seen edge on.

(iii) Calcification thought to be in the wall of the aorta is not recorded.

(iv) Avoid recording artefacts due to scratches on the screens etc.

Symbols

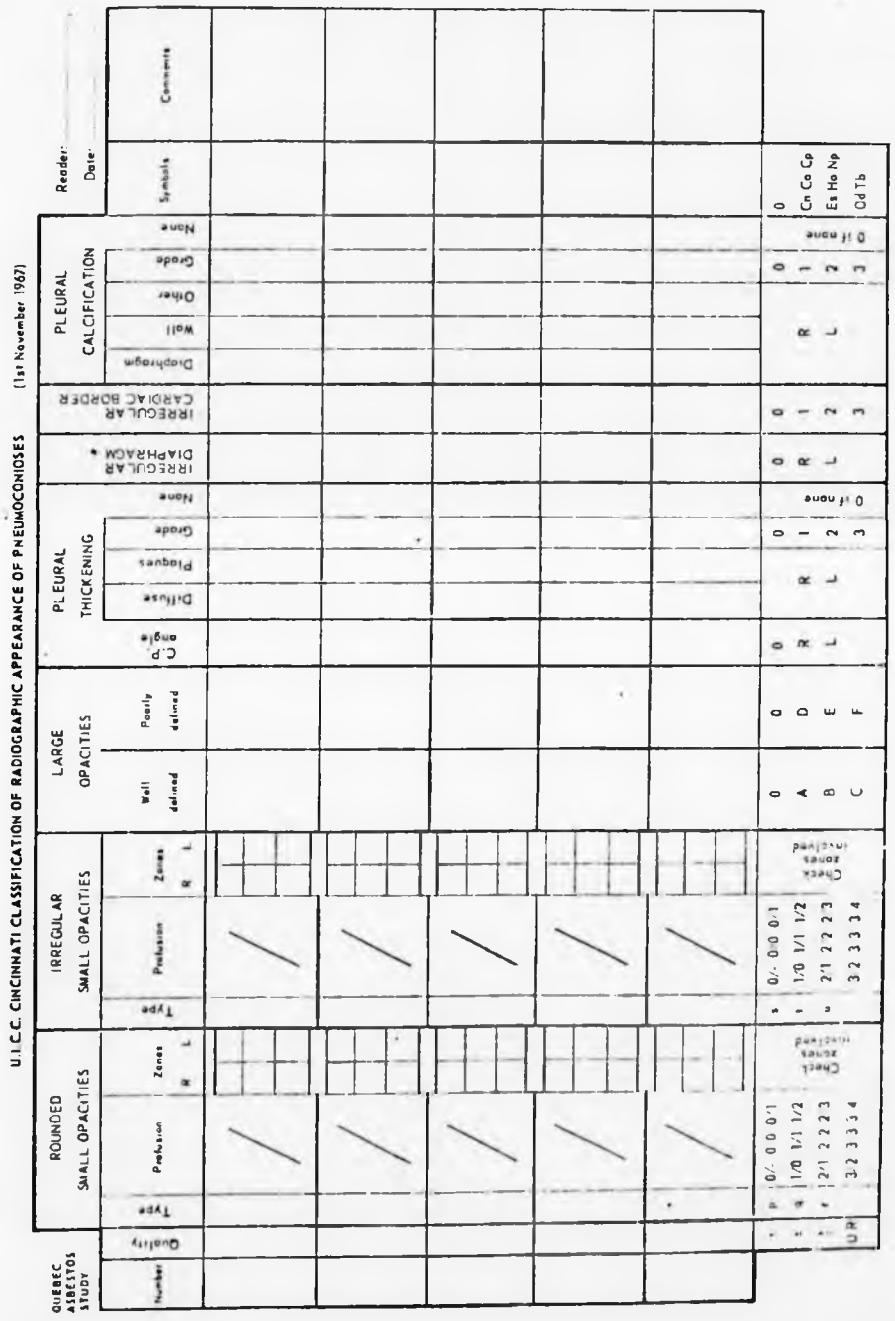
Certain additional features in the film can be recorded in code, using the list of symbols (see attached list). It is impracticable to record systematically all the symbols, but if a selected few are recorded systematically, this should be noted when reporting results, so that valid comparisons may be made with other surveys.

Symbols of special interest in asbestos workers are likely to include:-

Mp - neoplasm, either bronchial tumour, or mesothelioma.
Cn - calcification of nodules.
Co - cardiac abnormality (other than irregularity of cardiac outline reported in the main classification).
Cp - cor pulmonale.
Es - eggshell calcification.
Ho - honeycomb appearance. Note: This is a relatively specific type of appearance which will have already been recorded in the irregular small opacities column under 's', 't' or 'u', but if present to a marked degree may be usefully noted in the symbols.
Od - other diseases.
Tb - clinically significant tuberculosis, but excludes primary complex.

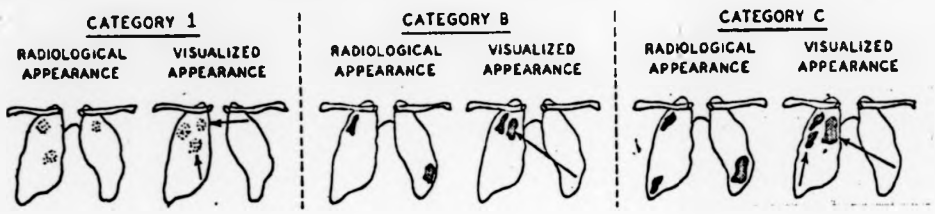
"Appendix I" to

INSTRUCTIONS FOR USING UICC/CINCINNATI CLASSIFICATION



PNEUMOCONIOSIS		
SMALL OPACITIES	THE CATEGORIZATION DEPENDS ON THE EXTENT AND PROFUSION OF THE OPACITIES. 1 A NUMBER OF OPACITIES IN AN AREA EQUIVALENT TO AT LEAST THE SECOND AND THIRD ANTERIOR RIB SPACES OF EITHER SIDE, AND AT THE MOST, NOT GREATER THAN ONE THIRD OF THE TWO LUNG FIELDS COMBINED. 2 OPACITIES MORE DIFFUSE THAN IN CATEGORY 1 WHICH MAY BE DISTRIBUTED OVER THE WHOLE OR NEARLY THE WHOLE OF THE LUNG FIELDS. 3 VERY NUMEROUS OPACITIES DISTRIBUTED OVER THE WHOLE, OR NEARLY THE WHOLE OF THE LUNG FIELDS.	THESE ARE CLASSIFIED ACCORDING TO THE GREATEST DIAMETER OF THE PREDOMINANT OPACITIES AND DENOTED BY THE FOLLOWING SYMBOLS: p - GREATEST DIAMETER UP TO AND INCLUDING 1.5 mm. q - GREATEST DIAMETER FROM 1.5 mm UP TO AND INCLUDING 3 mm. r - GREATEST DIAMETER FROM 3 mm UP TO AND INCLUDING 1 cm 
LARGE OPACITIES	A - AN OPACITY HAVING A GREATEST DIAMETER EXCEEDING 1 cm. AND UP TO AND INCLUDING 5 cm. OR SEVERAL OPACITIES EACH GREATER THAN 1 cm., THE SUM OF WHOSE GREATEST DIAMETERS DOES NOT EXCEED 5 cm. B - ONE OR MORE OPACITIES LARGER OR MORE NUMEROUS THAN THOSE IN CATEGORY A WHOSE COMBINED AREA DOES NOT EXCEED ONE THIRD OF THE VISIBLE RIGHT LUNG. C - ONE OR MORE OPACITIES WHOSE COMBINED AREA EXCEEDS ONE THIRD OF THE VISIBLE RIGHT LUNG FIELD.	

* THE BACKGROUND OF SMALL OPACITIES SHOULD BE SPECIFIED AS FAR AS POSSIBLE.



MORTALITY OF QUEBEC CHRYSOTILE WORKERS IN RELATION TO RADIOLOGICAL FINDINGS WHILE STILL EMPLOYED

Francis Douglas Kelly Liddell

Thesis submitted for the degree of

PhD

Volume 2: Annex

TUC Centenary Institute of Occupational Health
London School of Hygiene and Tropical Medicine

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CONTENTS OF VOLUME 2 (ANNEX)

This Annex contains the leading data for all subjects in Cohorts "A" and "B". The contents are as follows:-

RECORD LAY OUT: COHORT "A"

#	Length	Item, including units and "format"
1	6	Identity number
2	4	Height (cm) (F4.1)
3	4	Weight (kg) (F4.1)
4	4	$FEV_{1.0}$ (litres) (F4.2)
5	4	FVC (litres) (F4.2)
6	1	Wheezing
7	1	Number of chest illnesses
8	5	Film reading sequence number
9	1	Status (Alive, Dead or Lost to view)
10	2	Year of death, or loss (blank if alive)
11	3	Cause of death * (blank if alive or lost to view)
12	4	Date of start of first job (year/month)
13	4	Recorded end of last job (year/month = 66/11)
14	2	Net service (years: truncated) (I2)
15	5	Accumulated dust exposure (mpcf.y: truncated) (I5)
16	1	Mining area (Asbestos or Thetford Mines)
17	1	Smoking code (see p. 172)
18	1	"Normal" or "Less-than-normal"
19	6	Date of birth (day/month/year)
20	4	Age at test (years) (F4.2)
21	2	Persistent cough/phlegm (unreliable)
22	1	Breathlessness
23	43	X-ray readings (see next page)

* First character R or S for accidents at asbestos work

LAY-OUT OF X-RAY READINGS: COHORT "A"

Each line (columns 87-129) refers to a specific reader

#	Length	Print cols.	Item
1	1	87	"6"
2	1	88	Film quality (+; +/-; +/--)
3	9		Small rounded opacities
		90-92	type; profusion
		93-98	zones (RU; RM; RL; LU; LM; LL)
4	9		Small irregular opacities
		100-102	type; profusion
		103-108	zones (RU; RM; RL; LU; LM; LL)
5	2		Large opacities
		110-111	well defined; poorly defined
6	1	113	Costophrenic angle(s)
7	3		Pleural thickening
		114-116	diffuse; plaques; grade
8	1	118	Irregular diaphragm
9	1	120	Irregular cardiac border
10	4		Pleural calcification
		122-125	diaphragm; wall; other; grade
11	1	127	"Additional symbols" (count)
12	1	129	"Comments" (1 = Yes)

COHORT "A"

"Normals": Alive

pp. 6 - 46

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10433	1646	530	307	357	00	14467	A		4601	6611	9		20	T	S
10496	1791	792	408	504	00	6254	A		4706	6611	12		9	T	D
10556	1587	569	355	396	00	15568	A		5404	6611	12		25	T	T
10527	1605	796	330	352	00	11775	A		4735	6611	14		213	T	S
10653	1429	834	454	553	01	6464	A		5035	6611	9		44	T	S

10690	1595	702	259	347	00	10615	A	4505	6611	17	45	1	1	8
10709	1753	990	321	337	00	11236	A	5105	6611	9	57	1	5	8
10406	1691	693	262	769	00	4732	A	5509	6611	11	47	1	0	8
10409	1727	766	463	527	00	9993	A	5605	6611	4	34	1	1	8
10410	1676	524	275	167	00	1316	A	4114	6611	21	5111	1	5	[illegible]
10417	1749	740	411	530	00	12537	A	5205	6611	14	41	1	4	[illegible]
10495	1691	693	243	760	00	4600	A	5034	6611	24	1282	1	1	[illegible]

4

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90101	1707	653	276	411	20	7331	A		2704	6611	17		106	T	1
90112	1707	777	347	340	10	11520	A		3703	6611	3		23	T	5
90234	1562	581	246	335	00	3314	A		4305	6611	23		157	T	0
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93256	1513	584	263	317	01	4911	A	2603	6611	21	135	T	T	T
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100034	1903	643	416	567	00	8826	A	6436	6611	2		S	A	S
100075	1476	871	324	432	10	11517	A	5806	6611	2		S	A	S
100087	1664	613	355	438	00	2572	A	5605	6611	10	21	A	A	T
100132	1722	683	394	534	00	12[illegible]	A	6308	6611	2		S	A	S

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100523	1740	650	471	524	10	15073	A		4507	6611	1	2	A	4	1
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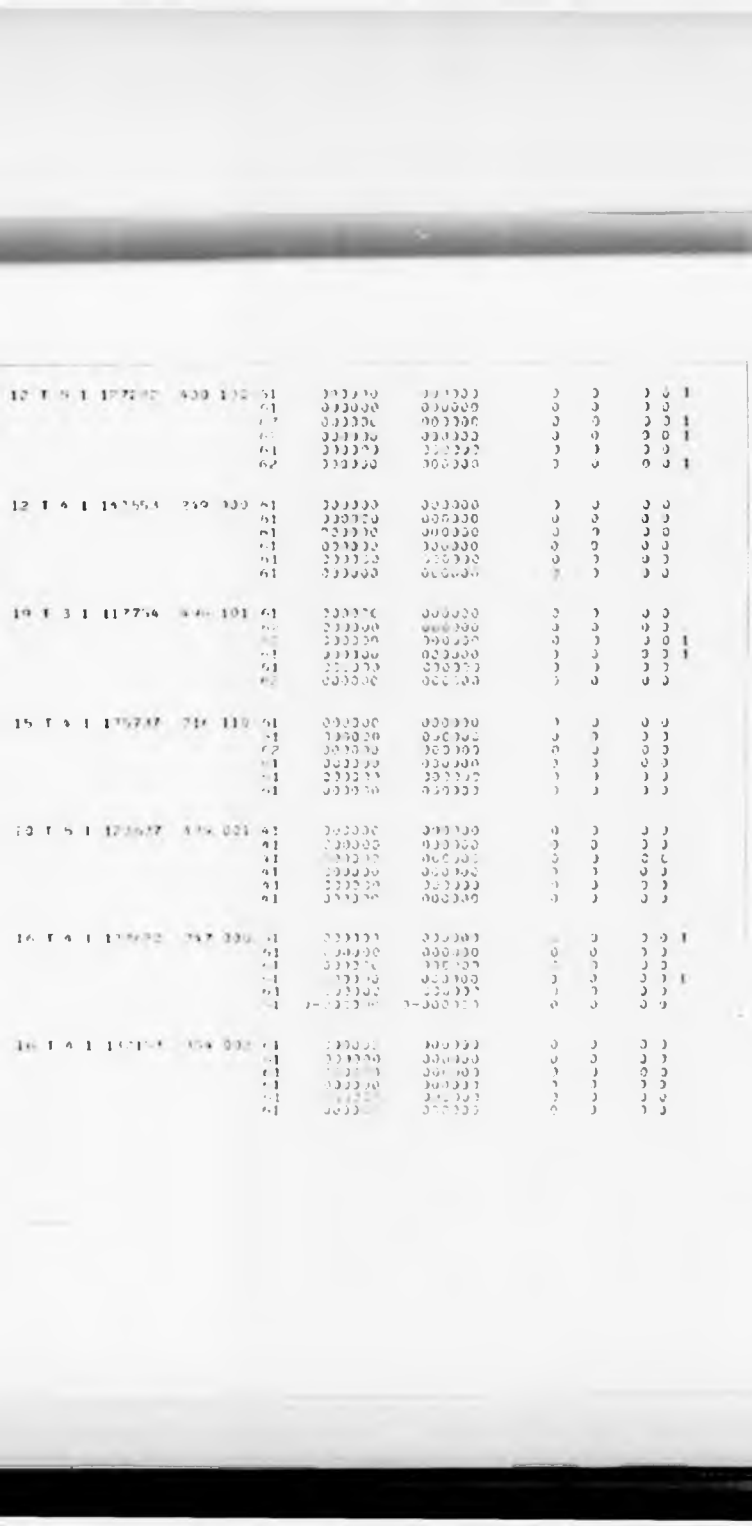

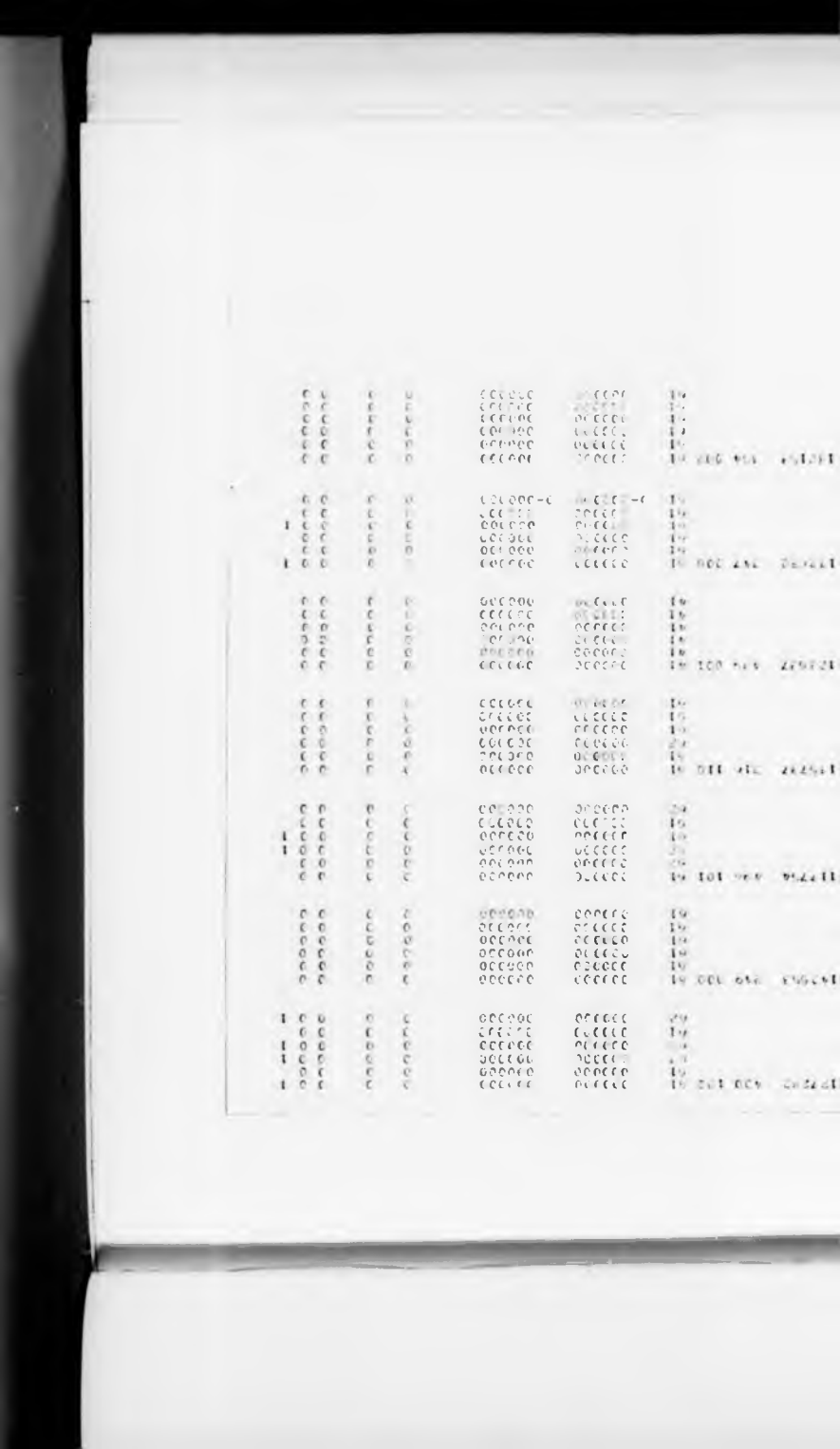

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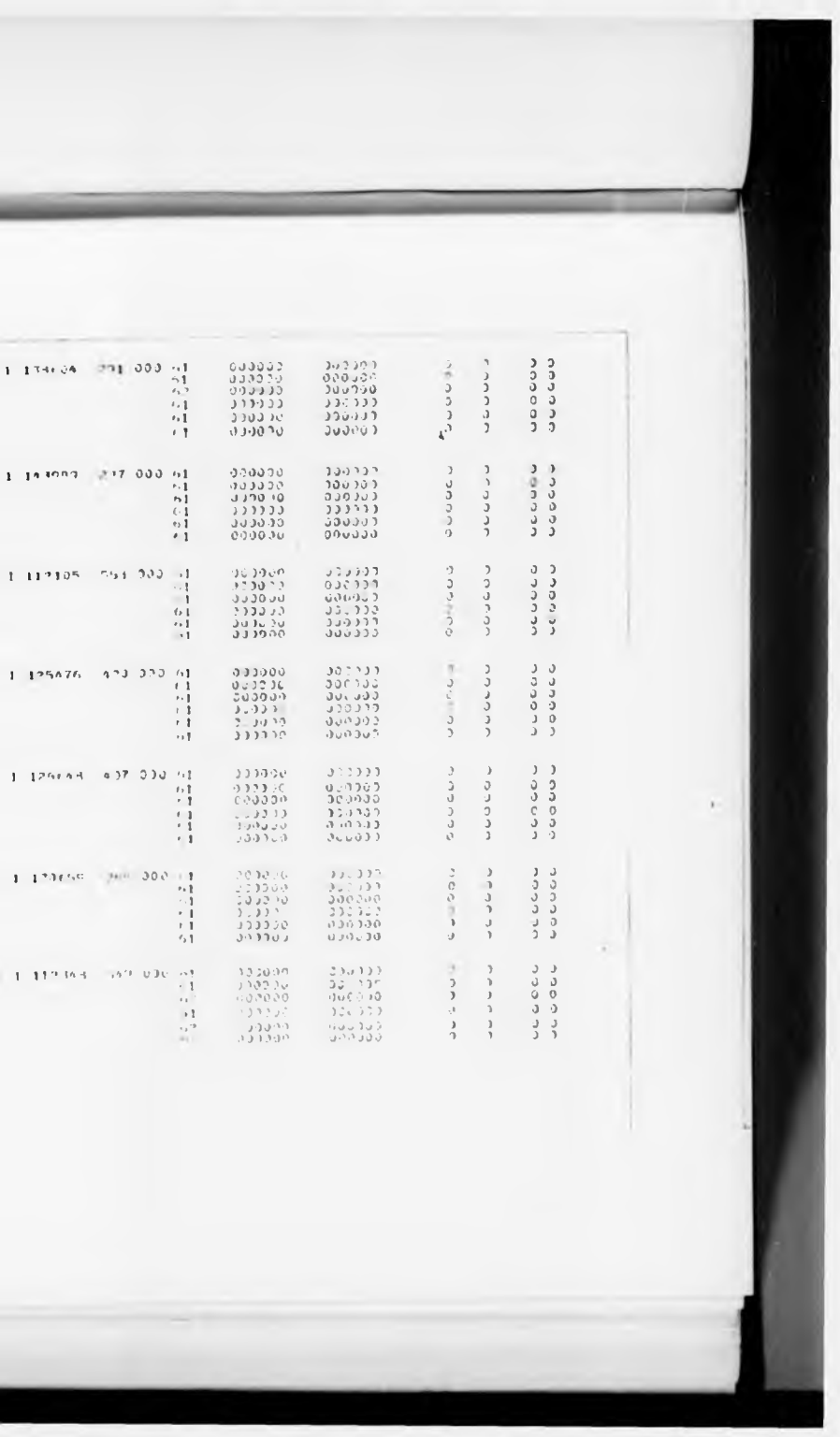

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833397	1703	555	351	424	00	6576	A		5303	6611	7	26	T
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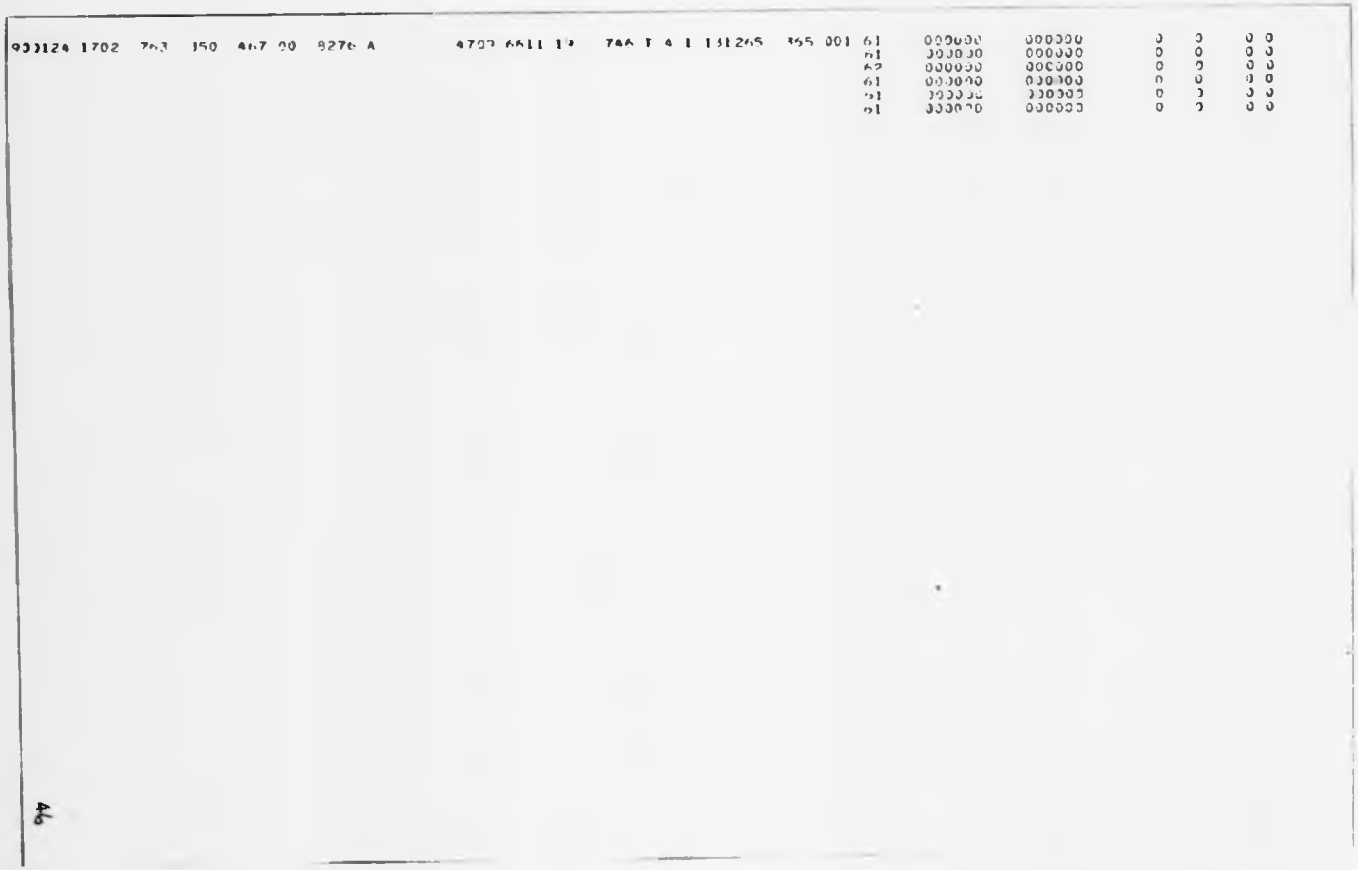
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COHORT "A"

"Normals": Dead

pp. 48 - 50

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330305	1651	753	276	365	00	11319	0	68	420	3603	6611	23
600046	1753	393	329	447	00	11317	0	74	420	5738	6611	1
302155	1731	747	417	502	00	10??	0	73	4??	????	6611	?

COHORT "A"

"Less-than-normals": Alive

pp. 52 - 134

54	1664	655	223	373	00	14855	A	2707	6611	16	185	T	4	2	107262	633	110	61	000000	000000		0	0		0	0
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52

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10293	1686	702	233	359	10	7604	A		2036	6611	39	480 T 5
10299	1753	940	249	342	00	2210	A		2436	6611	33	373 T 3
10311	1664	735	340	406	00	2917	A		4207	6611	23	98 T 3
10341	1765	600	233	378	00	12129	A		4713	6611	33	249 T 5

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13881	1681	738	266	319	23	14846	A		4401	6611	22	210 T
10882	1714	802	262	353	22	13577	A		1302	6611	23	1455 T
13883	1722	789	349	452	00	13923	A		5132	6611	15	45 T
13991	1737	739	287	391	00	4240	A		3733	6611	21	900 T
10301	1727	725	230	355	10	7595	A		5204	6611	14	152 T
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131365	1441	1127	371	444	13	6263	A		4135	6611 23
131375	1723	916	333	332	02	14609	A		4211	6611 23
131412	1526	685	180	385	00	173	A		2334	6611 09

101430	1732	949	239	340	22	3486	A	4713	6611	19
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131453	1753	819	300	351	33	9665	A	5011	6611	19
131461	1613	812	217	263	10	13832	A	4709	6611	19
131465	1732	849	261	354	33	2939	A	2235	6611	91
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131509	1727	952	353	408	00	12566	A		5537	6611 2
131534	1671	837	249	318	20	9619	A		4136	6611 34
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131535	1689	728	256	432	00	13728	A		5210	6611 14

78

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233001	1605	935	296	322	20	9188	A	5011	6611 15
233004	1676	785	275	454	10	9937	A	4229	6611 24
233014	1551	749	329	392	00	5965	A	5012	6611 15
233017	1748	549	249	194	00	4163	A	4311	6611 26

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191733	1597	777	349	489	00	774	A	3933	6611 26
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230001	1605	935	296	322	20	9188	A	5011	6611 15
230004	1576	785	275	364	10	9937	A	5239	6611 24
230014	1551	749	329	382	30	5565	A	5012	6611 15
230017	1748	549	269	39[illegible]	00	6164	A	4311	6611 26

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200349	1621	716	295	381	01	13641	A	2634 6611	27
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200123	1681	865	257	356	00	3602	A		1836	6611 17
200131	1740	875	296	410	00	5770	A		2611	6611 21
200139	1707	722	414	492	00	4251	A		5604	6611 13
200148	1646	679	295	403	21	15644	A		4503	6611 21

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75 A 0 2 128656  321 001 62   000000 T01001001        0  0    0 1
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38 A 0 2 107240  625 300 61   000000 S01011011        0  0    0 1
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233154	1702	725	385	461	00	8343	A	5007	6611	16
233174	1773	865	334	398	10	13141	A	4535	6611	21
233177	1603	663	333	481	02	5625	A	4506	6611	14
233178	1732	677	300	431	11	12511	A	3539	6611	11
233183	1714	757	273	324	00	2379	A	2812	6611	37
233185	1737	638	248	361	03	5836	A	4717	6611	13

200199	1562	556	136	229	23	9319	A		2002	6611	36
200196	1451	728	167	246	00	5433	A		2409	6611	42
200198	1758	706	347	447	03	5459	A		5103	6611	14
200204	1671	698	186	227	12	12765	A		4704	6611	27
200207	1758	704	293	349	00	12973	A		4136	6611	20
200219	1631	690	197	247	02	3014	A		2333	6611	37
200224	1748	736	288	336	13	8355	A		3513	6611	24

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148 A 4 2 107551   600 000 31   000000 S11011011 D   0 0   0 0
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119 A 1 2 106400   620 000 31   000000   000000     0 0   0 0
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                       32 C00000000   000000000     0 0   0 0 1
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 19 A 4 2 139006   207 000 61 P01010010   000000     0 0   0 0
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 46 A 5 2 120224   476 003 61   000000 T01011110     0 0   0 0
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200223	1707	858	312	325	00	10116	A	4302	6611	23
200252	1753	701	295	434	00	10872	A	4705	6611	39
200266	1714	802	277	433	00	8061	A	4135	6611	25
200276	1740	671	331	346	00	14081	A	2506	6611	41
200285	1701	734	372	511	00	5530	A	5509	6611	11
200291	1671	562	181	372	10	6675	A	4613	6611	20
200296	1778	674	444	591	00	5490	A	4601	6611	20

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200306	1656	779	318	372	30	10260	A		3705	6611 24
200311	1546	540	141	432	00	10800	A		4105	6611 25
200314	1644	611	261	346	00	3876	A		4702	6611 19
200318	1638	524	332	418	02	9186	A		5101	6611 15
200319	1723	699	334	493	10	5140	A		4504	6611 21
200327	1580	657	214	390	00	2293	A		2613	6611 33
200333	1664	722	360	446	10	7000	A		4613	6611 18

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 54 A 4 2 125323  525 102 61  000000  T010000011    0  0   0 0
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203 T 3 2 100002  528 001 61  000000  000000  F 1  0  0H 2 0
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1343 T 5 2 110436  530 002 61  000000  000000    0  0   0 1
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200365	1464	804	288	171	20	4577	A		3701	6611 25
200367	1613	562	252	292	12	15651	A		4539	6611 21
200368	1740	943	371	453	00	14444	A		2605	6611 29
200374	1581	799	319	426	10	11586	A		5337	6611 16
300309	1575	589	263	319	00	11405	A		1434	6611 28
333318	1613	547	271	363	13	2176	A		3513	6611 13

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271 T 4 2 112231  551 000 61    000000    000000        0    0    0 0 1
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                          62    000000 0101010101  D   L  1    0    0 1 1
                          61 001010000  1010001001        0    0    0 1 1
                          61    000000    000000        0    0    0 0 1

592 T 3 2 105697  616 000 61    000000    000000        0    0    0 0
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1024 T 4 2 107694  646 111 61    000000 S1000001001    B   0 F  0    0 0
                          61    000000 S0100001001      L  I B  0 F  1 0
                          61    000000    000000      H   0 H  0    0 0 1
                          61    000000    000000      F   0 F  0    0 0
                          61    000000    000000      HH  I F  0    0 0
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1030 T 3 2 105726  627 001 31 P11010010 S1100001001    F   I    0    0 0
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                          31 0000000000 1010001001    F   0    0    0 0
                          31    000000 1011011011    F   0    0    0 0
                          31 0000000000 1010100101    F   0    0    0 0
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143 T 4 2 125320  423 001 61    000000    000000        0    0    0 0
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261 T 3 2 128403  349 001 61    000000    000000        0    0    0 0
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300336	1689	727	192	260	10	3648	A		3109	6611	31
300142	1529	494	264	313	02	5571	A		4202	6611	23
300217	1580	466	262	325	10	8608	A		2315	6611	43
300225	1605	515	162	228	00	14263	A		2711	6611	33
300229	1691	760	350	307	10	14647	A		5408	6611	13
300229	1714	686	288	337	30	12409	A		4407	6611	21

300232	1765	726	460	577	00	8458	A	6205	6611	2
300238	1681	670	289	398	01	245	A	4205	6611	24
300240	1554	742	258	300	00	11599	A	4805	6611	18
300255	1400	907	171	248	20	681	A	2309	6611	30
300297	1758	658	370	484	00	1387	A	4737	6611	19
300355	1959	807	430	507	00	5670	A	4706	6611	19
300357	1732	961	383	372	02	423	A	3610	6611	23

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303396	1722	758	325	348	00	10741	A	5406	6611	13	62	T
300411	1740	589	374	457	10	367	A	4602	6611	23	123	T
303415	1676	887	316	417	00	13933	A	4336	6611	26	571	T
303460	1732	738	429	456	00	9672	A	5301	6611	8	17	T
300482	1629	619	126	229	20	11196	A	1613	6611	20	226	T
300483	1603	710	252	331	00	7732	A	1704	6611	20	260	T
300487	1651	744	248	282	12	12682	A	2635	6611	33	117	T

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273 T 4 2 128726  386 001 61   000000   000000   0 0    0 0
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                  61 P01 111000   000000   0 0    0 1 1
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49 T 5 2 102682   648 001 61   000000   000000   0 0    0 0
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418 T 4 2 104674  638 001 61   000000   000000   0 0 BR 1 0
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497 T 0 2 126674  400 000 61   000000   000000   0 0    0 0
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1837 T 4 2 103204 651 002 62   000000   000000   0 0    0 0
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290 T 4 2 100417  574 000 61   000000   000000   0 0    0 0
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300535	1575	545	328	380	00	9520	A		4505	6611	20
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300564	1611	473	220	281	00	15688	A		2301	6611	37
300566	1740	810	403	517	00	13851	A		4209	6611	21
300593	1646	557	148	311	10	15575	A		2309	6611	43
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333583	1707	642	244	297	13	696	A	3739	6611	25	837	T
333609	1676	435	252	332	10	12253	A	4235	6611	24	158	T
333704	1681	726	352	468	20	13176	A	5906	6611	14	41	T
333732	1613	612	165	209	23	10674	A	4736	6611	18	100	T
333748	1664	771	275	333	13	2613	A	3336	6611	27	351	T
333751	1656	572	236	342	20	6171	A	2601	6611	18	287	T

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300757 1605  627  214  286 00 10536 A    4209 6611 24   633 T 1 2 106682  619 002 31    CC0000 T01001001   FS I F 0    0 0
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300766 1651  589  170  269 00 14295 A    2102 6611 31  3019 T 5 2 107604  609 000 31    000000 T01001001      0 0    0 1
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300791 1562  475  182  227 10 15675 A    2303 6611 36  2200 T 3 2 103398  651 333 41 F11010010 S11001011      0 0    0 0
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                                                                                  41 021110110    000000      0 0    0 0
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300797 1727  589  109  233 00  2154 A    2131 6611 41   408 T 4 2 104734  629 000 61    000000    000000   BR 1 0 HL 1 0
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                                                                                  61    000000    000000      0 0 BLL1 0

333406 1722  562  336  436 03  6426 A    4613 6611 18   177 T 3 2 124682  427 001 61    000000    000000      0 0    0 0
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331038	1575	721	250	265	20	12053	A	4333	6611	25	214	T
331043	1538	535	215	263	00	10697	A	4411	6611	21	334	T
331066	1526	569	238	245	00	4833	A	2936	6611	31	344	T

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301068 1702 730 349 440 02 1155 A 4707 6611 18

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301092 1714 784 328 399 10 2057 A 4701 6611 19

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301118	1651	704	353	410	00	15064	A		5636	6611	7
301122	1576	752	334	262	00	13473	A		2636	6611	43
301142	1753	618	346	407	00	380	A		2903	6611	31
301143	1660	713	283	375	00	6589	A		3603	6611	19
301144	1646	819	311	279	00	13110	A		4311	6611	26

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301199	1575	553	219	256	10	12414	A		4203	6611	24
301229	1689	666	225	286	10	7790	A		4336	6611	32
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331259	1702	760	132	251	20	14340	A	3609	6611	50
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301469	1631	574	278	353	0C	12708	A	3134	6611	45	183	T	0	2	108754	586	001	61	000000	000000		0		0	B	1	0	
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301472	1570	553	140	250	10	9568	A	3205	6611	34	849	T	5	2	105159	633	002	31	000000	T01001001		0		0		0	0	
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301499	1664	789	316	372	00	13601	A	2811	6611	33	494	T	4	2	111817	556	000	61	000000	000000		0		0		0	0	
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                          32  000000 T10011011      0  0    0 0
                          31  000000    000000      0  0    0 0
                          32  000000    000000      0  0    0 0

487 T 3 2 105726  517 030 61 P01010010 T01001001      0  0    0 0
                          61  000000 S11011011      0  0    0 0
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                          61 P01211001 T10011001    0  0    0 0
                          61  000000    000000      0  0    0 0
                          61 P10100000 T11001001    0  0    0 0

468 T 4 2 106373  621 033 61 P11010010 S11001001      0 H 0 B  0 0
                          61  000000 S11111111      0  0 BR   1 0
                          61  000000 T12111111      0 H 0 BB 1 0
                          61  000000 T22111111      0 H 0 BR 1 0
                          61  000000 S11111110      0 P 0 BL 1 0  1
                          61 P22110110 S11001000    0 L 0 LL 1 0

 57 T 4 2 125131  424 031 61  000000    000000      0  0    0 1  1
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                          62  000000    000000      0  0    0 2
                          61  000000    000000      0  0    0 1  1
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                          61 P11010000    000000    0  0    0 0  1

 75 T 4 2 127276  431 033 61  000000    000000   B  0  0    0 0
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359 T 3 2 124394  431 001 61  000000    000000      0  0    0 0
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1491 T 4 2 111176  562 001 61  000000    000000      0  0    0 0
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301545	1714	633	274	343	00	2950	A		3534	6611	31
301558	1595	541	183	214	22	15080	A		1210	6611	43
301569	1753	703	408	468	12	242	A		5036	6611	15
301575	1714	434	293	365	02	7013	A		4836	6611	18
301591	1464	692	243	424	32	3907	A		4536	6611	23
301613	1497	633	241	322	10	14232	A		7636	6611	33

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301641	1702	767	182	252	20	15472	A		2302	6611	26	49 T
301643	1575	834	219	276	20	10335	A		4305	6611	22	184 T
301647	1626	689	254	327	00	12743	A		2813	6611	18	383 T
301649	1697	666	314	395	00	12100	A		2331	6611	43	364 T
301655	1613	786	265	105	12	4090	A		1405	6611	32	290 T
301657	1626	576	177	246	10	7361	A		3636	6611	30	353 T
301658	1638	545	220	302	00	7356	A		2612	6611	31	406 T

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2 2 131415  448 000 32   000000 T22011011    1  0    0 0
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331664	1471	729	235	306	00	15055	A		1936	6611	41	278 T
331733	1621	743	306	364	00	2738	A		4208	6611	23	224
331726	1664	739	271	310	10	600	A		4834	6611	17	305
331736	1626	753	248	303	00	14321	A		2991	6611	27	324
331755	1554	517	336	360	00	4693	A		4839	6611	15	58
331759	1681	826	236	511	00	1151	A		3727	6611	29	16
331416	1405	599	169	235	10	13222	A		2834	6611	34	725

301819	1668	558	204	242	00	3936	A	2201	6611	40
301822	1562	636	242	324	02	15647	A	4238	6611	34
301835	1638	577	184	276	20	9539	A	2130	6611	35
301833	1526	551	239	306	20	12206	A	2335	6611	32
301844	1638	762	193	272	20	10795	A	2635	6611	30
301845	1714	644	332	366	00	6491	A	4309	6611	15
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331869	1651	713	302	401	10	8677	A		4234	6611	23	56
331890	1722	745	266	438	00	13626	A		2639	6611	40	374
331899	1645	619	287	367	00	12260	A		3737	6611	29	159
331902	1748	956	335	441	00	6485	A		4837	6611	16	42
331935	1751	737	319	439	00	6745	A		4012	6611	16	263
331946	1681	733	274	358	10	8329	A		3830	6611	17	19
331951	1714	781	430	455	10	14659	A		4338	6611	18	37

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                31  P3111111        000000          0  0    0    0 1
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A 2 104601  [illegible] 012 31      000000  T01001001       0  0    0    0 0
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332026	1638	733	238	280	10	4059	A	4335	6011	29	316	
332049	1656	653	305	302	10	2441	A	4534	6011	21	48	
402050	1707	503	202	254	00	4205	A	2307	6011	43	2347	
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302130	1575	719	218	262	30	2864	A	3630	6611	33
302156	1732	749	324	342	00	2826	A	4304	6611	23
302159	1651	713	197	286	00	13754	A	4009	6611	26
302163	1638	580	335	304	30	5150	A	3512	6611	33
302179	1676	735	284	325	10	4796	A	4604	6611	23

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430091	1714	628	208	399	00	67	A		4911	6611	17
430109	1714	1323	251	396	13	12348	A		4911	6611	17
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000309	1765	1051	344	432	10	2774	A	3303	6611	22	76	T
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(SI)

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900398	1485	728	335	393	11	4330	A		4509	6611 23
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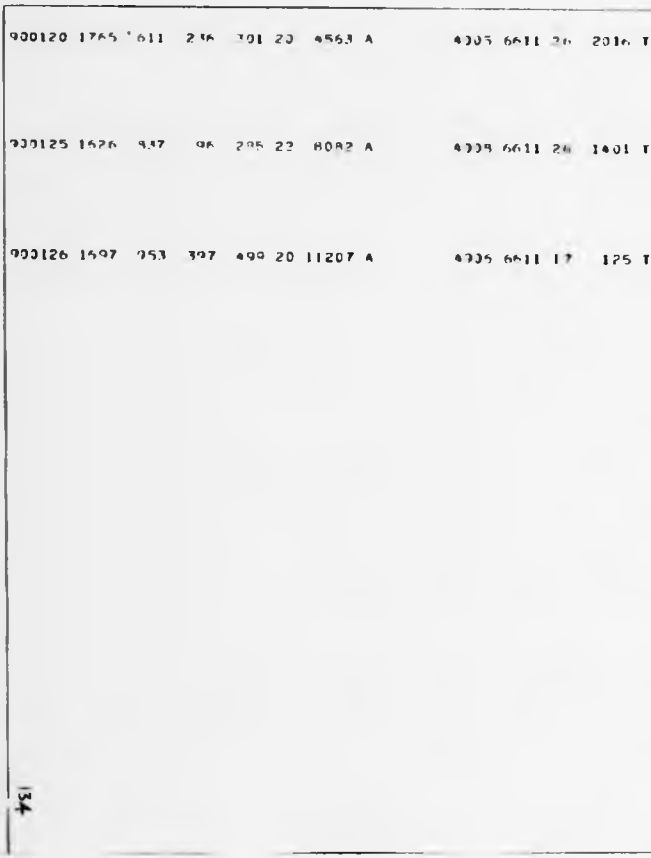
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930125 1526 937 96 295 22 8082 A 4335 6611 26 1401 T
930126 1597 953 397 499 20 11207 A 4335 6611 17 125 T
134

COHORT "A"

"Less-than-normals": Dead

pp. 136 - 151

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332336	1456	672	196	315	10	10235	D	72	144		4339	6611	26
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233373	1783	633	255	456	00	13966	D	76	151		3339	6611	36
333247	1626	653	392	379	00	6940	D	73	151		3209	6611	32
433359	1732	937	285	407	00	8120	D	73	151		4335	6611	27

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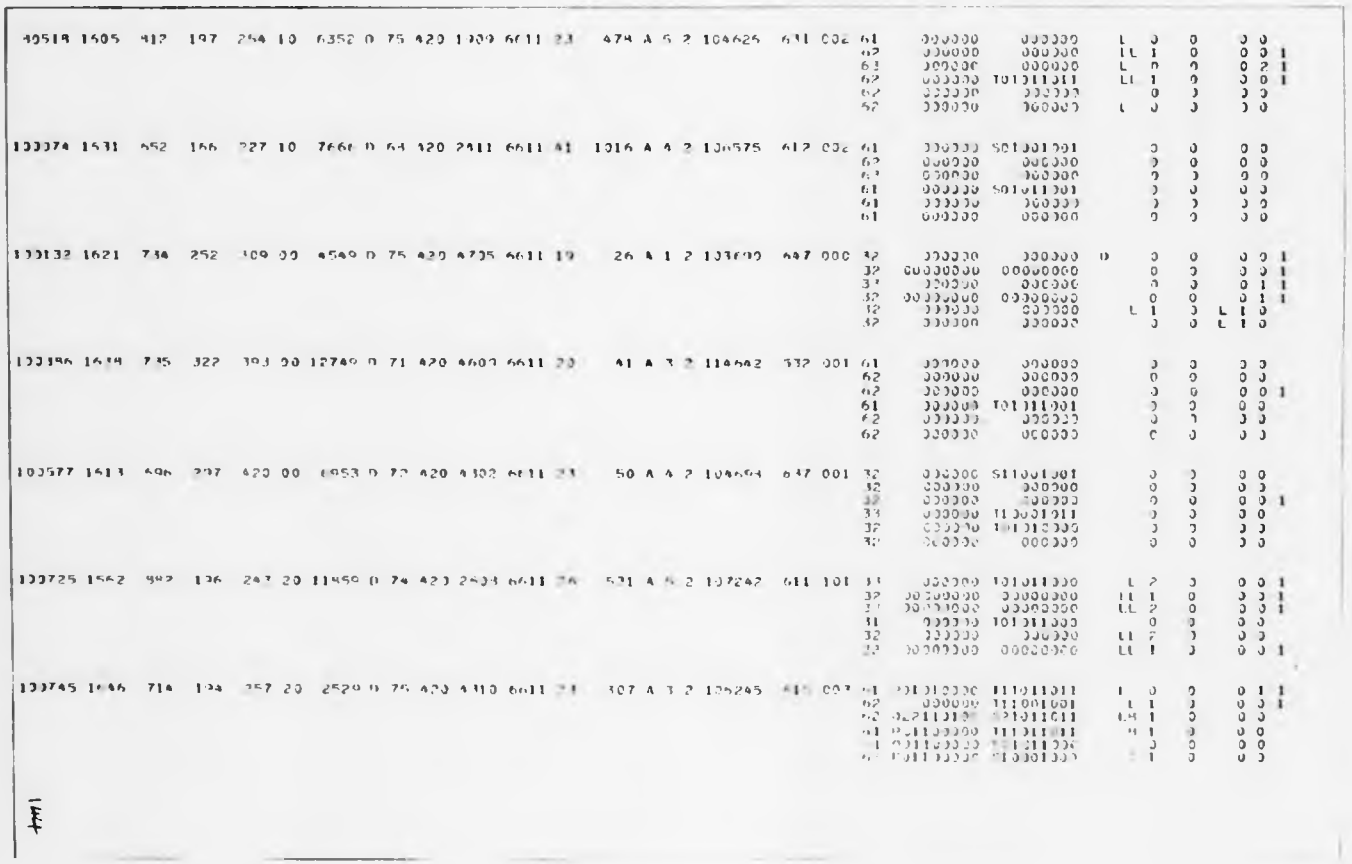

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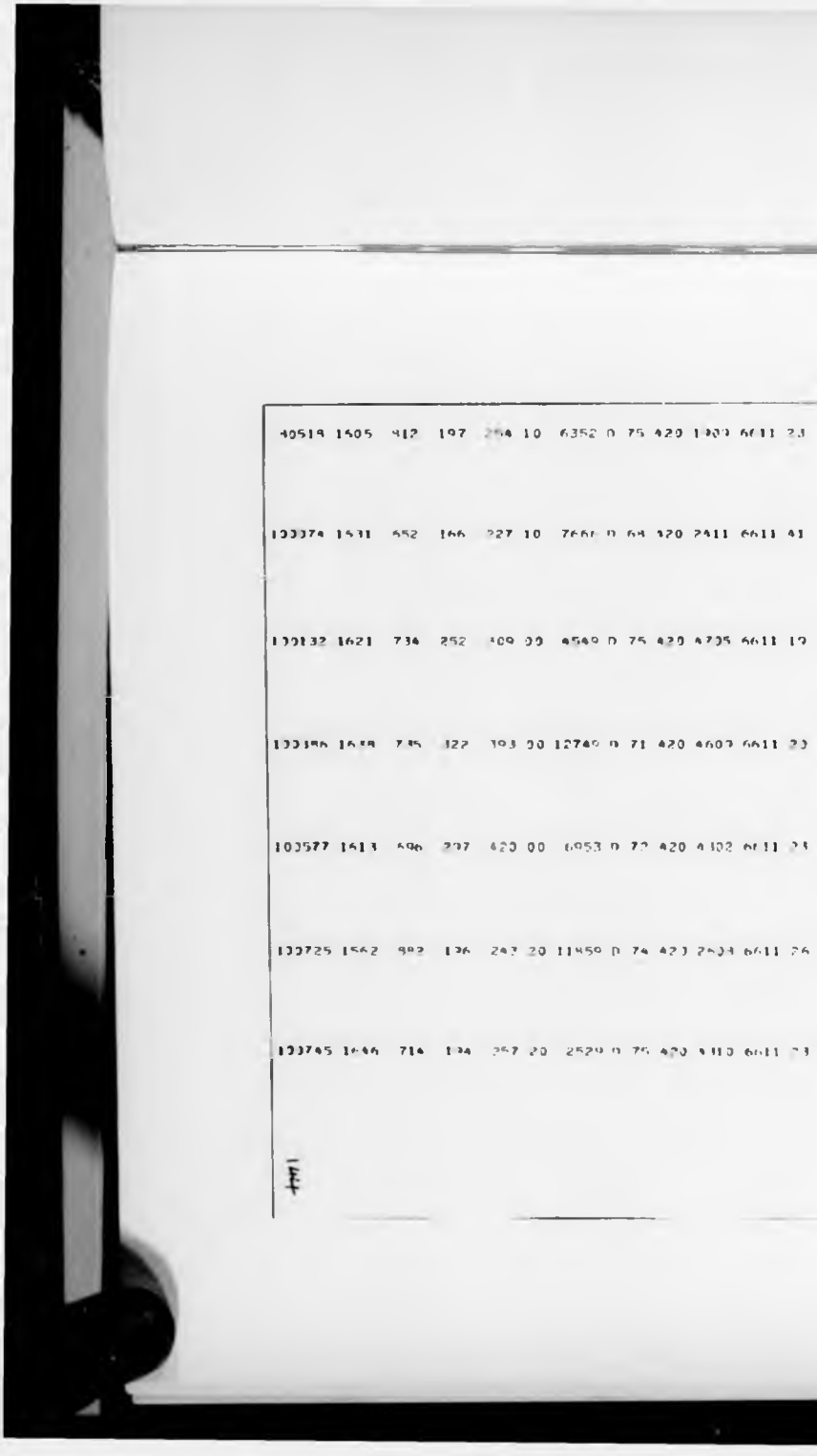

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330391	1413	749	155	262	22	10759	D	68	420	2513	6611	38	
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330662	1580	551	285	381	00	2072	D	71	423	3610	6611	35	
331939	1570	703	192	250	33	8414	D	69	423	2726	6611	39	

433081	1664	624	292	404	10	7747	0	70	420	6611	6611	27
433302	1587	671	204	256	00	9342	0	69	420	1311	6611	47
433332	1664	755	236	266	01	7578	0	70	420	2530	6611	40
533124	1697	586	302	366	10	10973	0	73	420	7330	6611	33
533224	1438	772	180	367	00	12947	0	75	420	6511	6611	1
533268	1758	863	314	366	10	846	0	72	420	7530	6611	31
533345	1656	533	131	232	00	5147	0	69	333	6511	6611	1

146

6301E7	1A00	628	1D4	77A	10	0092	D	75	420	4905	6A11	14
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730324	1A1A	742	1D7	611	02	11947	D	75	420	5A19	6A11	13
730540	1A05	740	215	103	11	12561	D	73	420	5911	6A11	13
730178	1A51	6A0	234	7H1	21	10206	D	7E	432	1H04	6A11	4A
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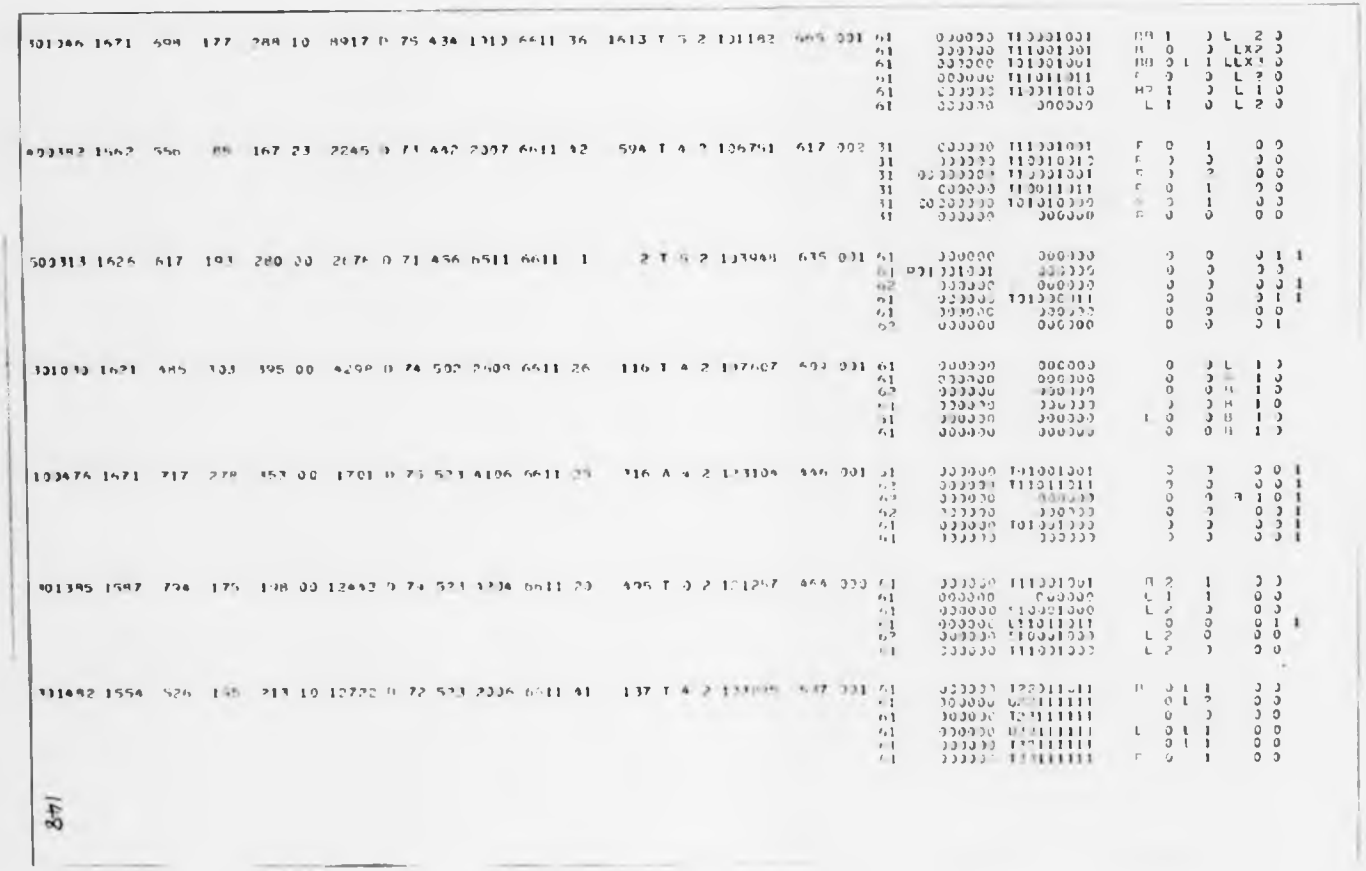
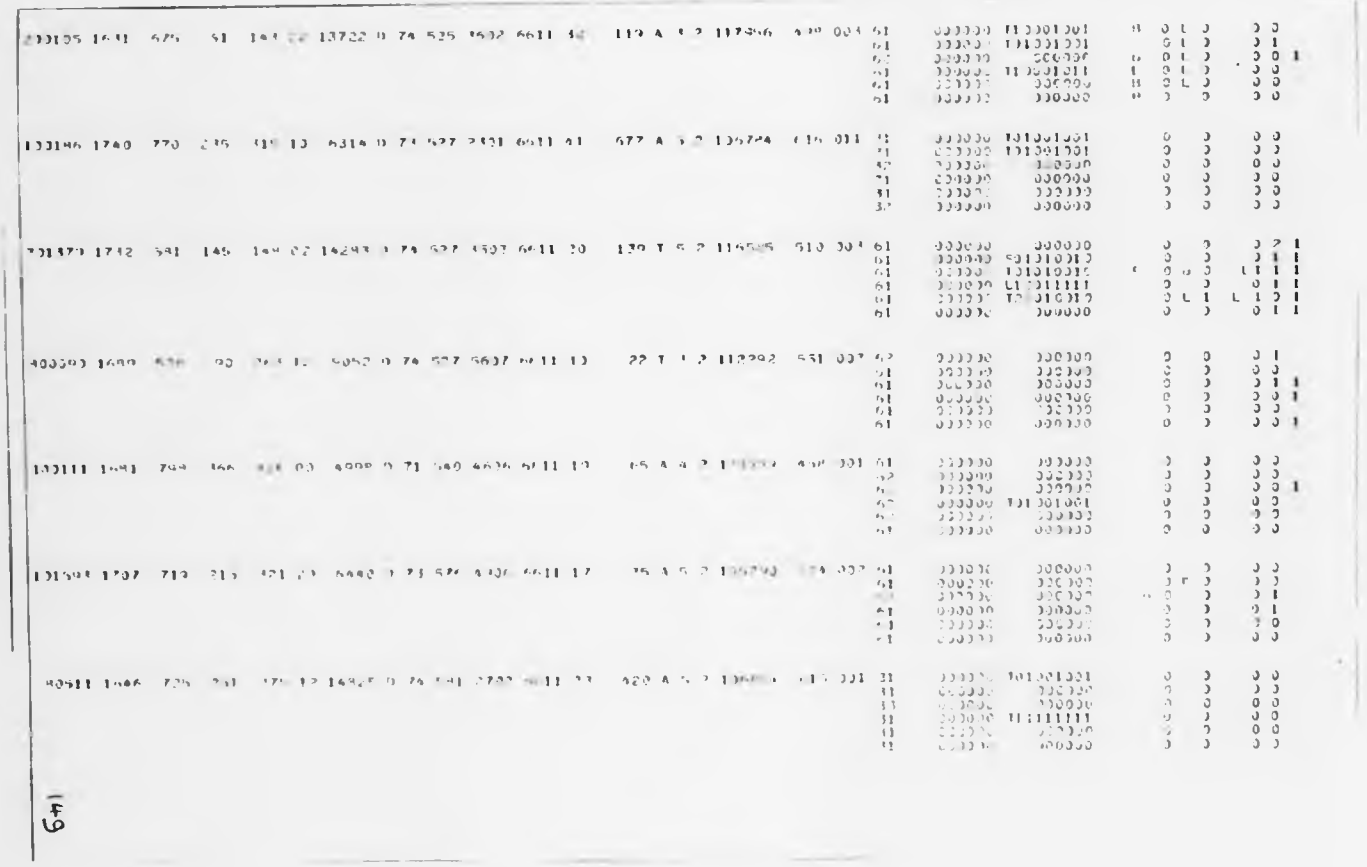
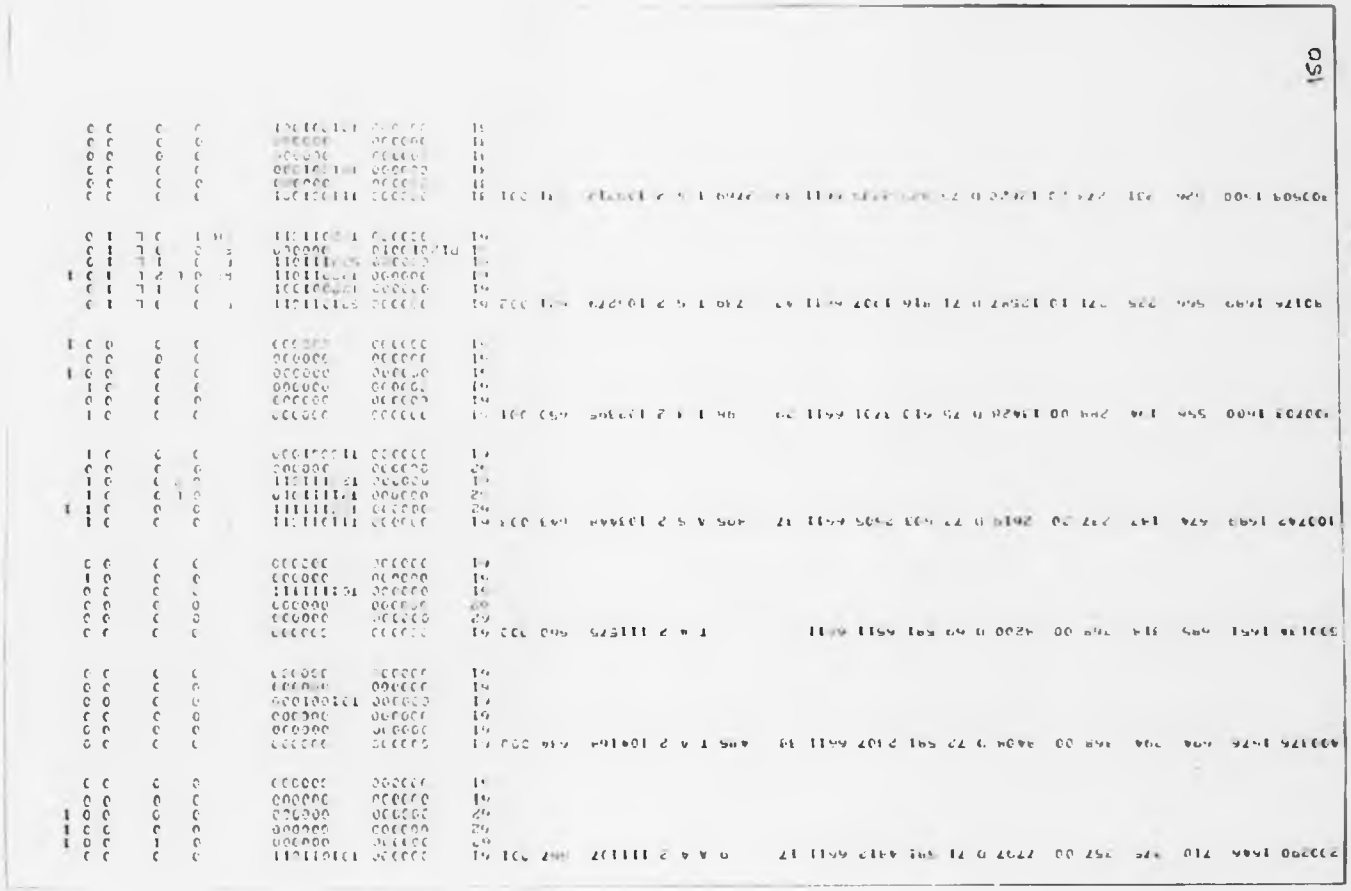






RECORD LAY-OUT: COHORT "B"

#	Length	Item, including units and "format"
1	6	Identity number
2-4	6	Date of birth (day/month/year)
5	5	Film reading sequence number
6	1	Status (Alive, Dead or Lost to view)
7	2	Year of death, or loss (blank if alive)
8	3	Cause of death * (blank if alive or lost to view)
9	4	Date of start of first job (year/month)
10	4	Date of end of last recorded job (year/month)
11	2	Net service (years: truncated) (I2)
12	5	Accumulated dust exposure (mpcf.y: truncated) (I5)
13	1	Mining area (Asbestos or Thetford Mines)
14	1	Reader (as recoded, see p. 180)
15	1	"Normal" or "Less-than-normal"
16	1	Smoking code (see p. 180)
17	43	X-ray readings (see next page)

* First character R or S for accidents at asbestos work

LAY-OUT OF X-RAY READINGS: COHORT "B"

#	Length	Print cols.	Item
1	1	59	Film quality (+; +/-; +/--)
2	9		Small rounded opacities
		61-63	type; profusion
		64-69	zones (RU; RM; RL; LU; LM; LL)
3	9		Small irregular opacities
		71-73	type; profusion
		74-79	zones (RU; RM; RL; LU; LM; LL)
4	2		Large opacities
		81-82	well defined; poorly defined
5	1	84	Costophrenic angle(s)
6	3		Pleural thickening
		85-87	diffuse; plaques; grade
7	1	89	Irregular diaphragm
8	1	91	Irregular cardiac border
9	4		Pleural calcification
		93-96	diaphragm; wall; other; grade
10	1	98	"Additional symbols" (count)
11	1	100	"Comments" (1 = Yes)

COHORT "B"

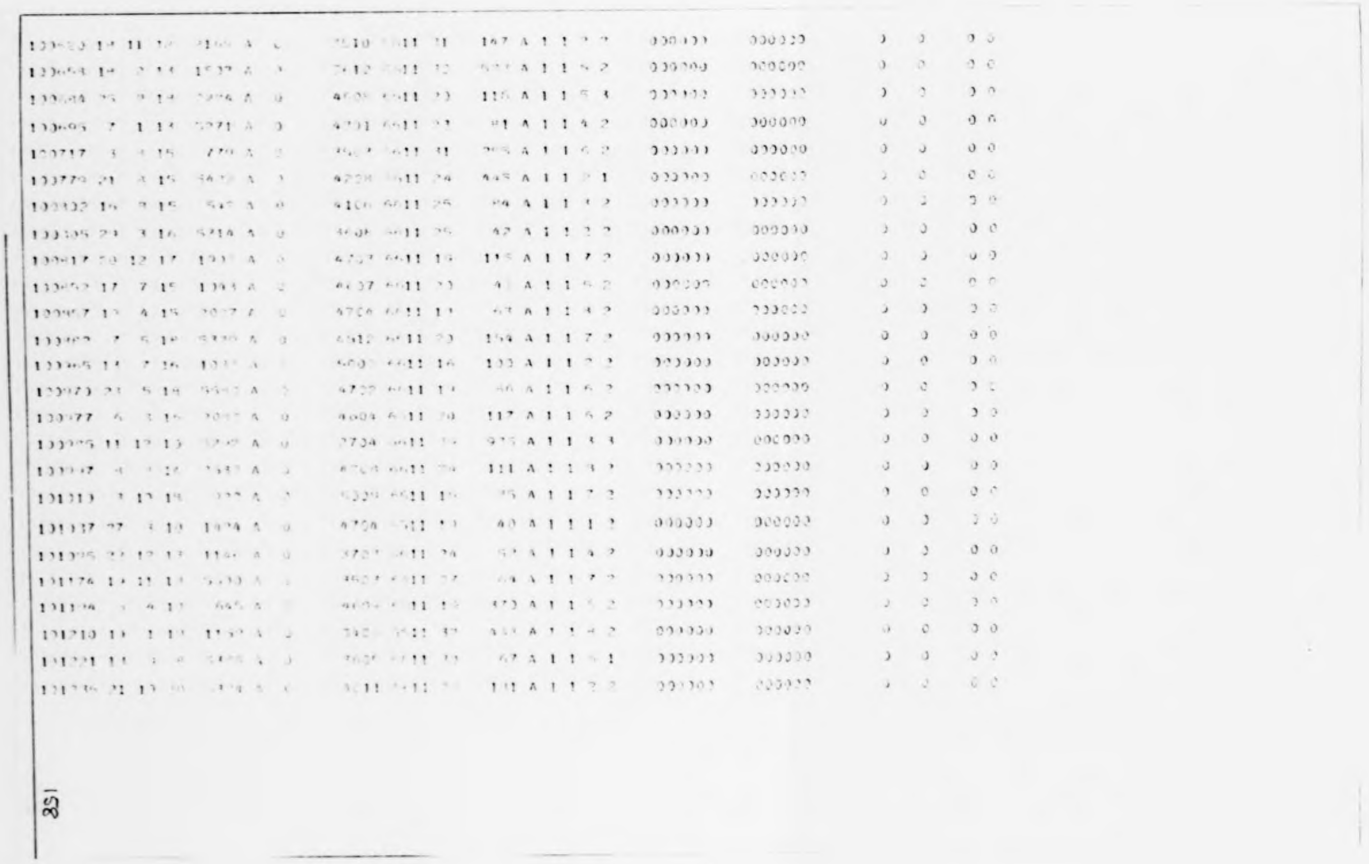
Reader 1

"Normals"

pp. 155 - 177

"Less-than-normals"

pp. 178 - 184



251

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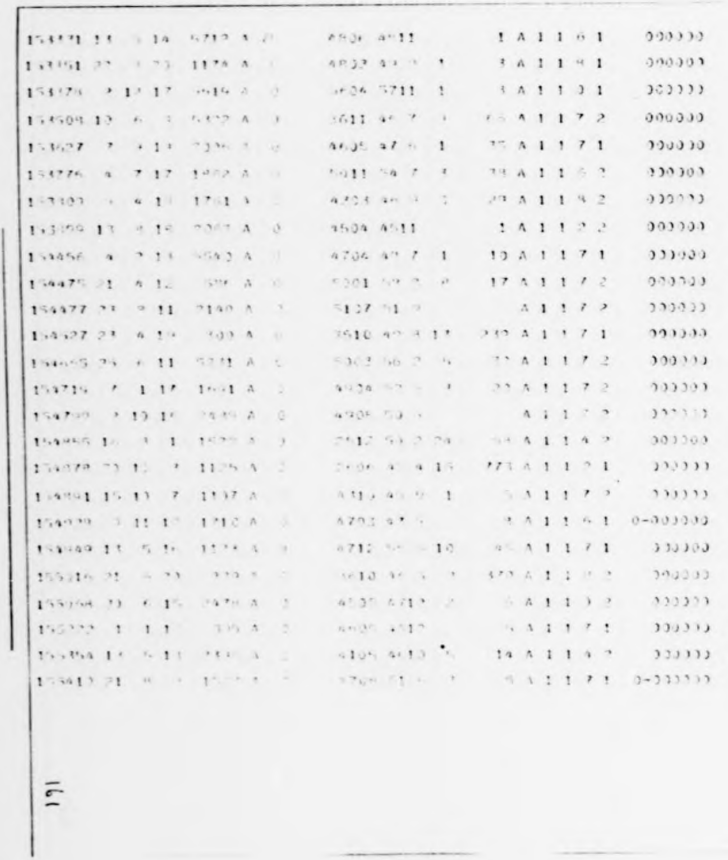

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155531	30	3	15	719	A	3	4229	51	11	11	26	A	1	1	3	1	000000	000000	0	0	0	0	
155540	15	1	19	1473	A	0	5105	51	6			A	1	1	9	1	000000	000000	0	0	0	0	
155779	21	5	6	1221	A	0	3606	47	5	11	542	A	1	1	7	2	000000	000000	0	0	0	0	
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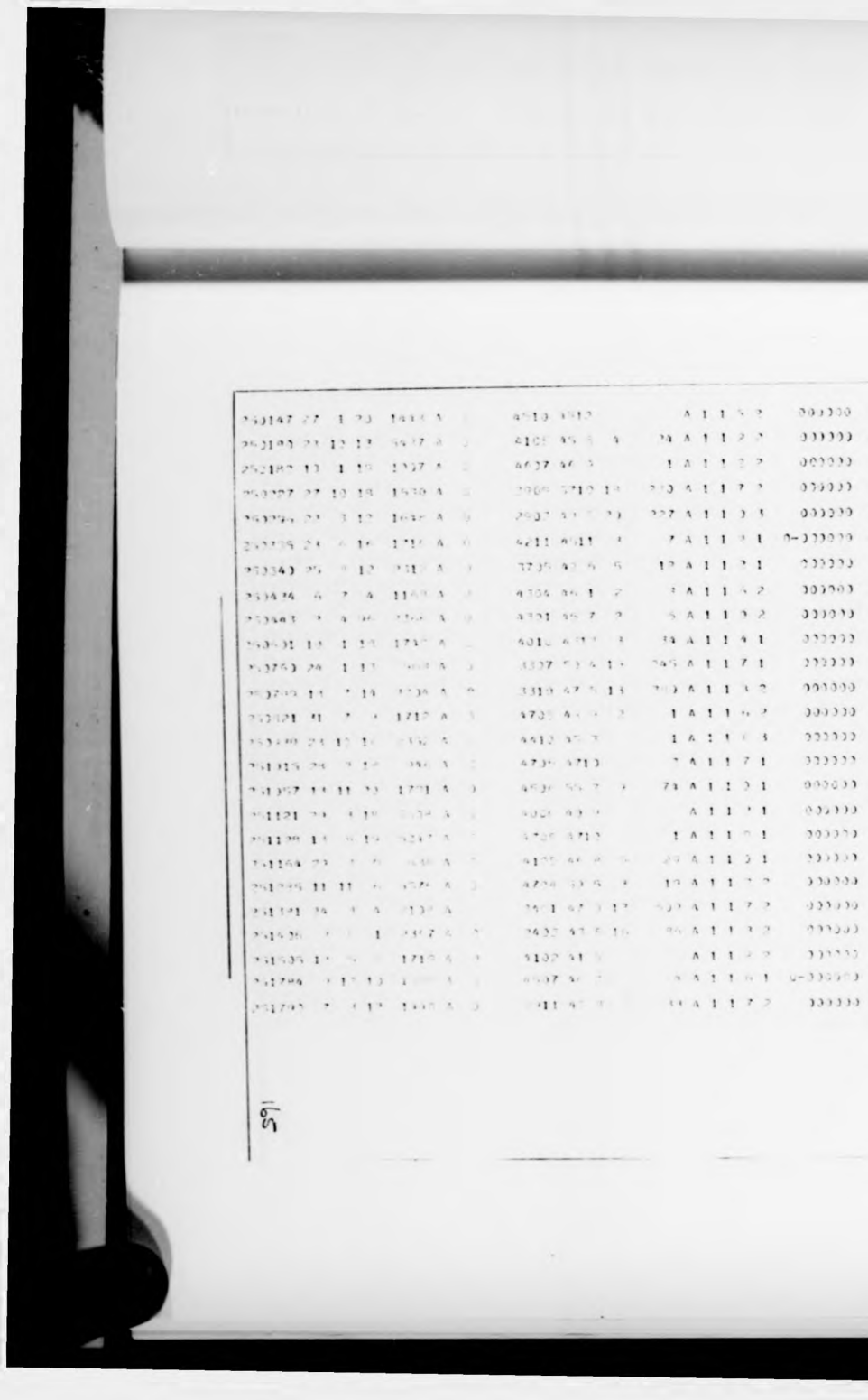

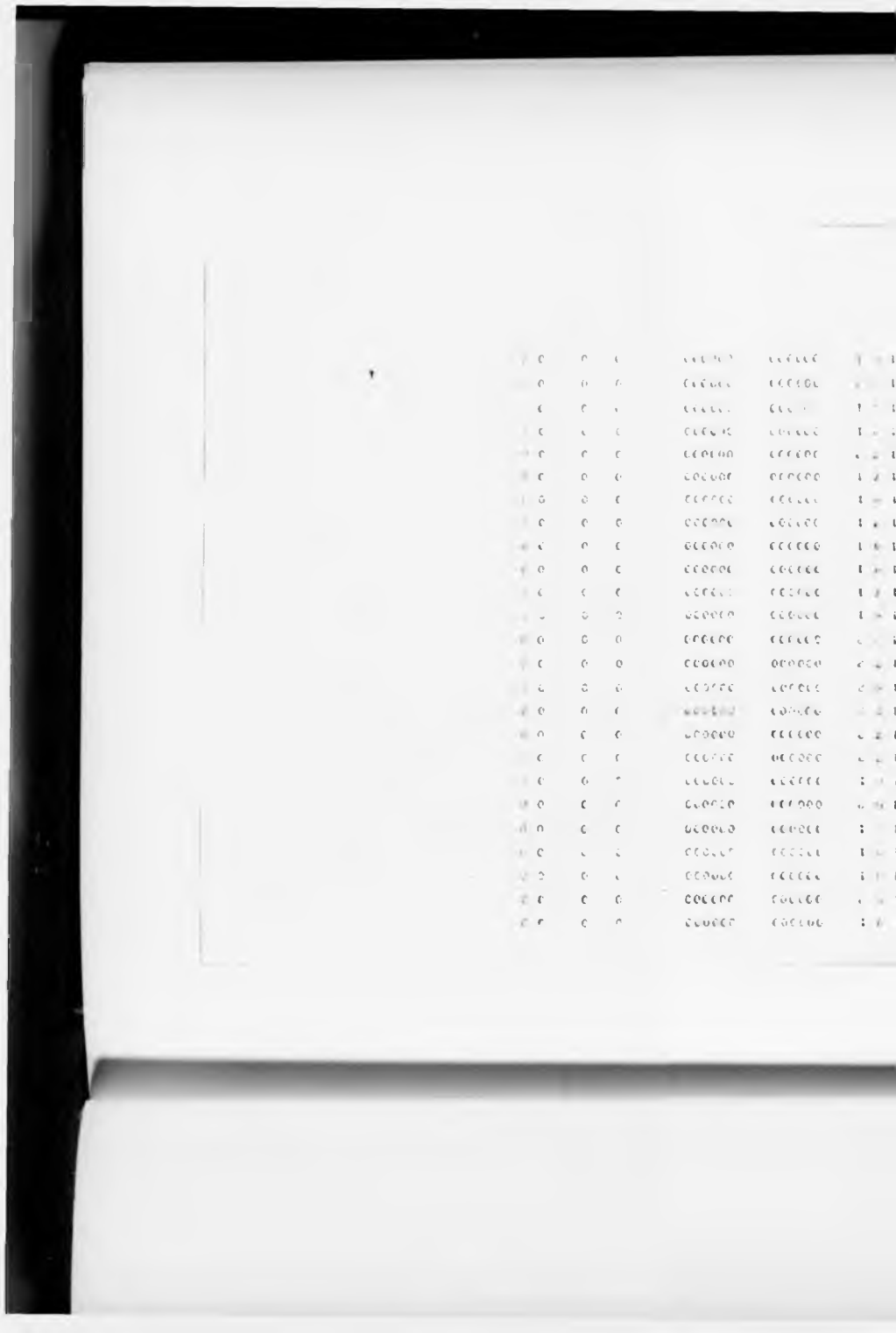

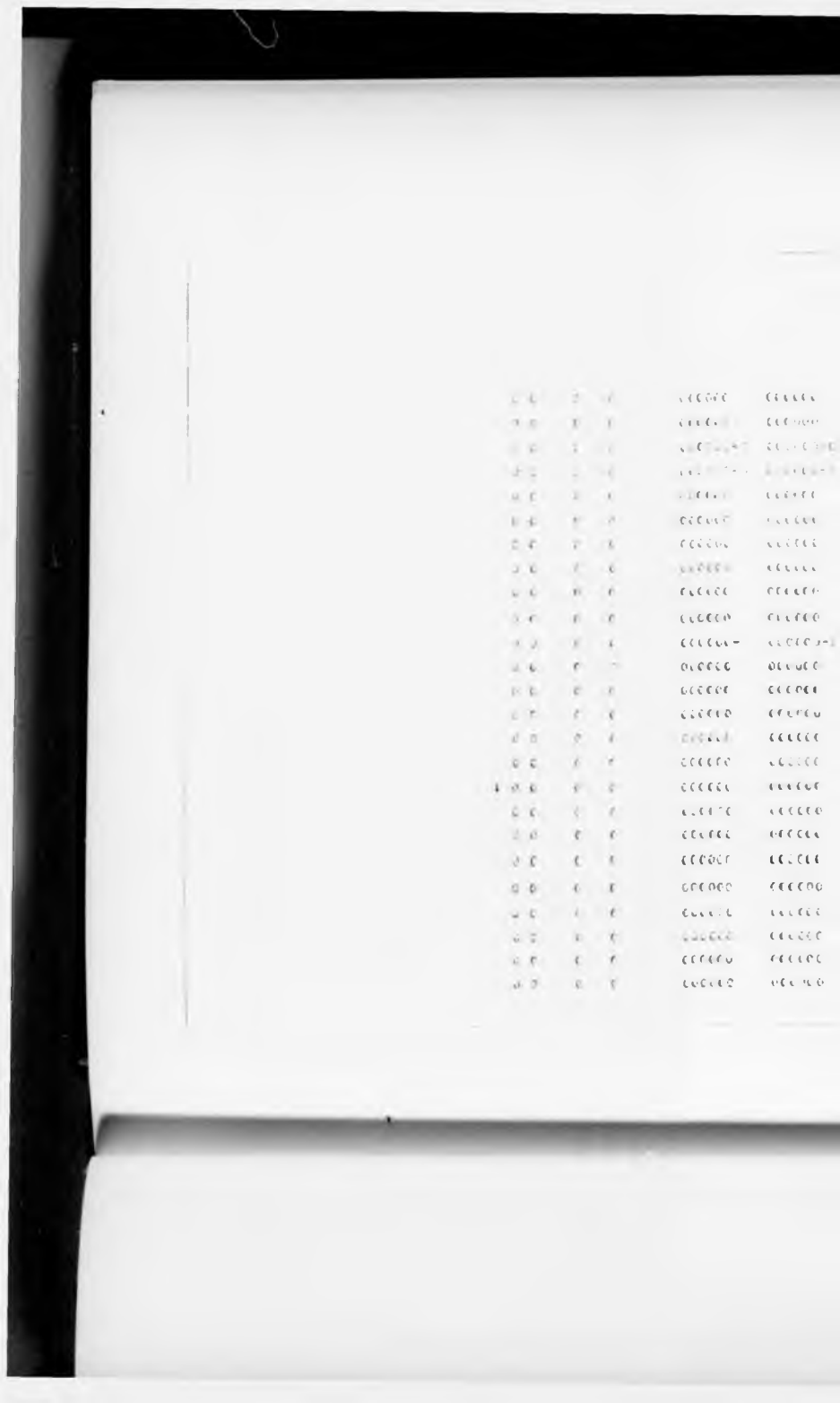

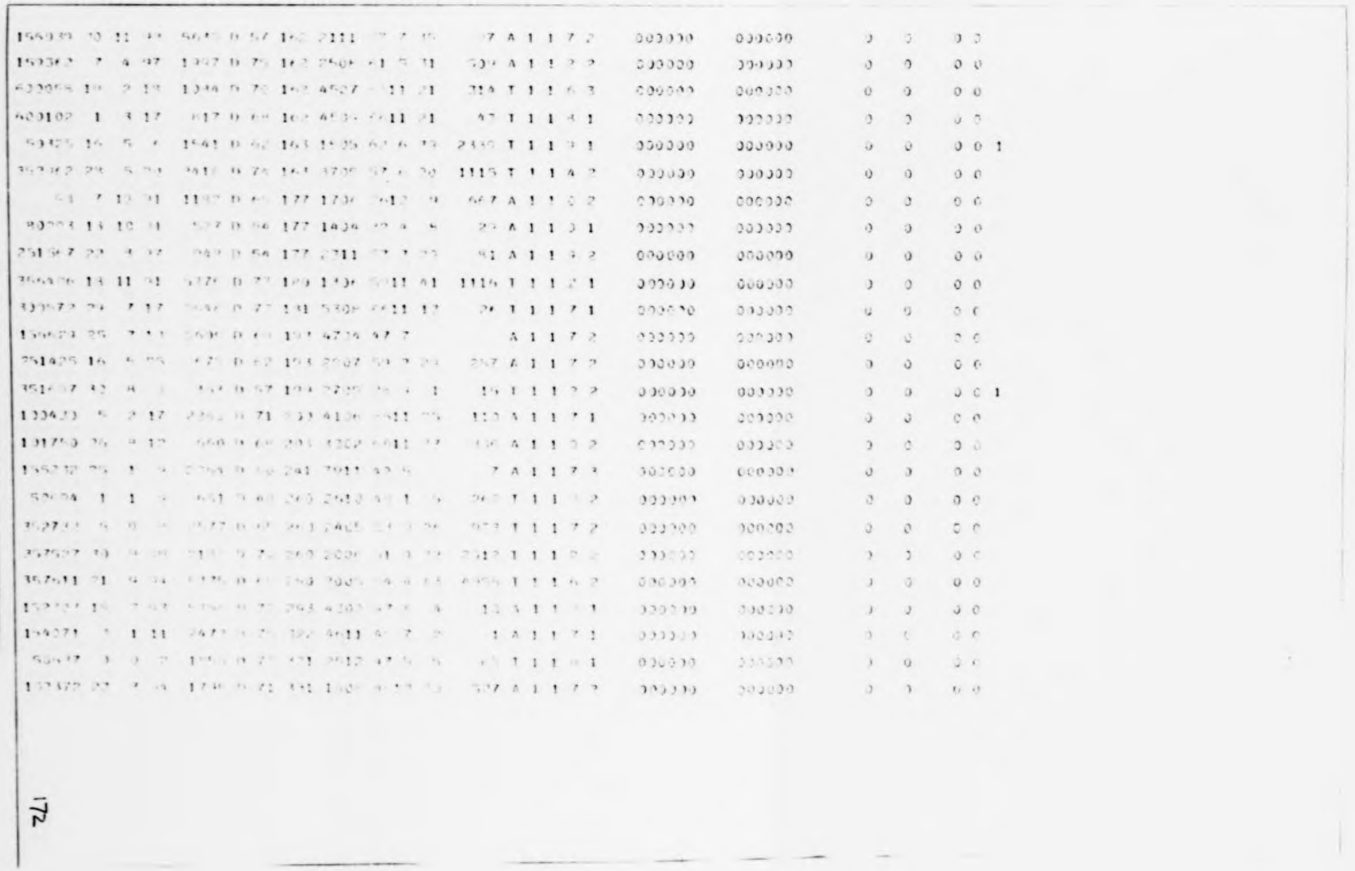

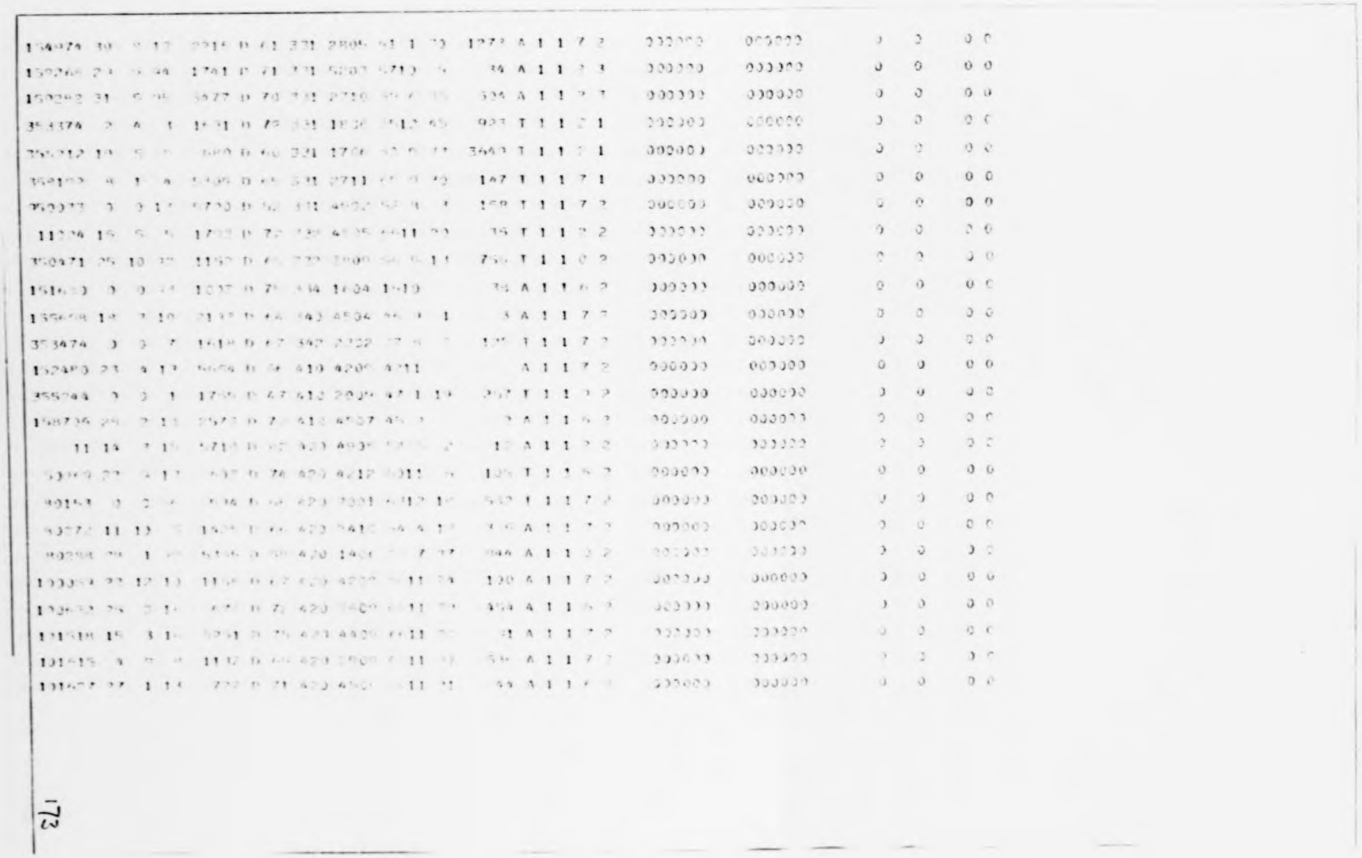

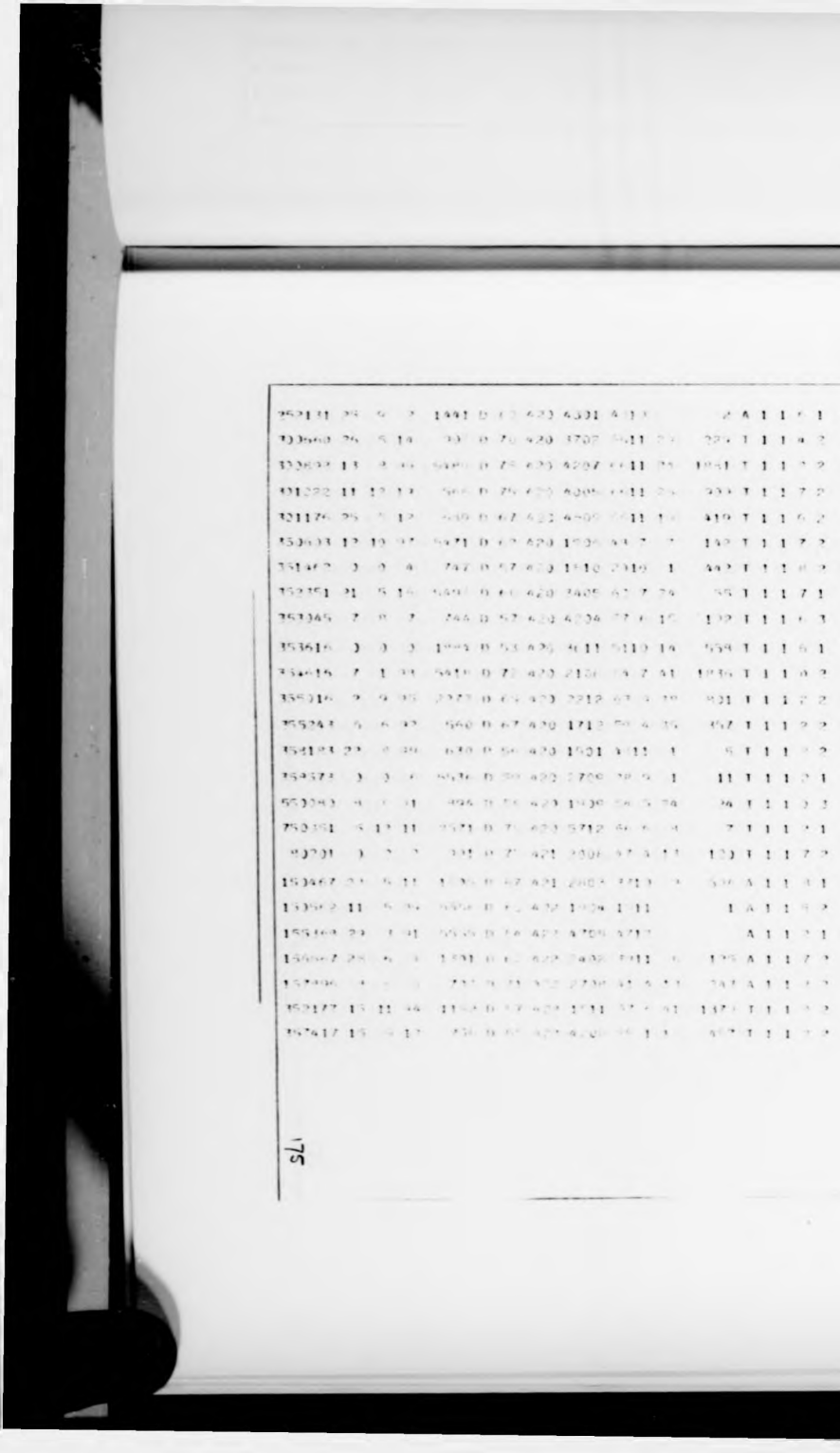

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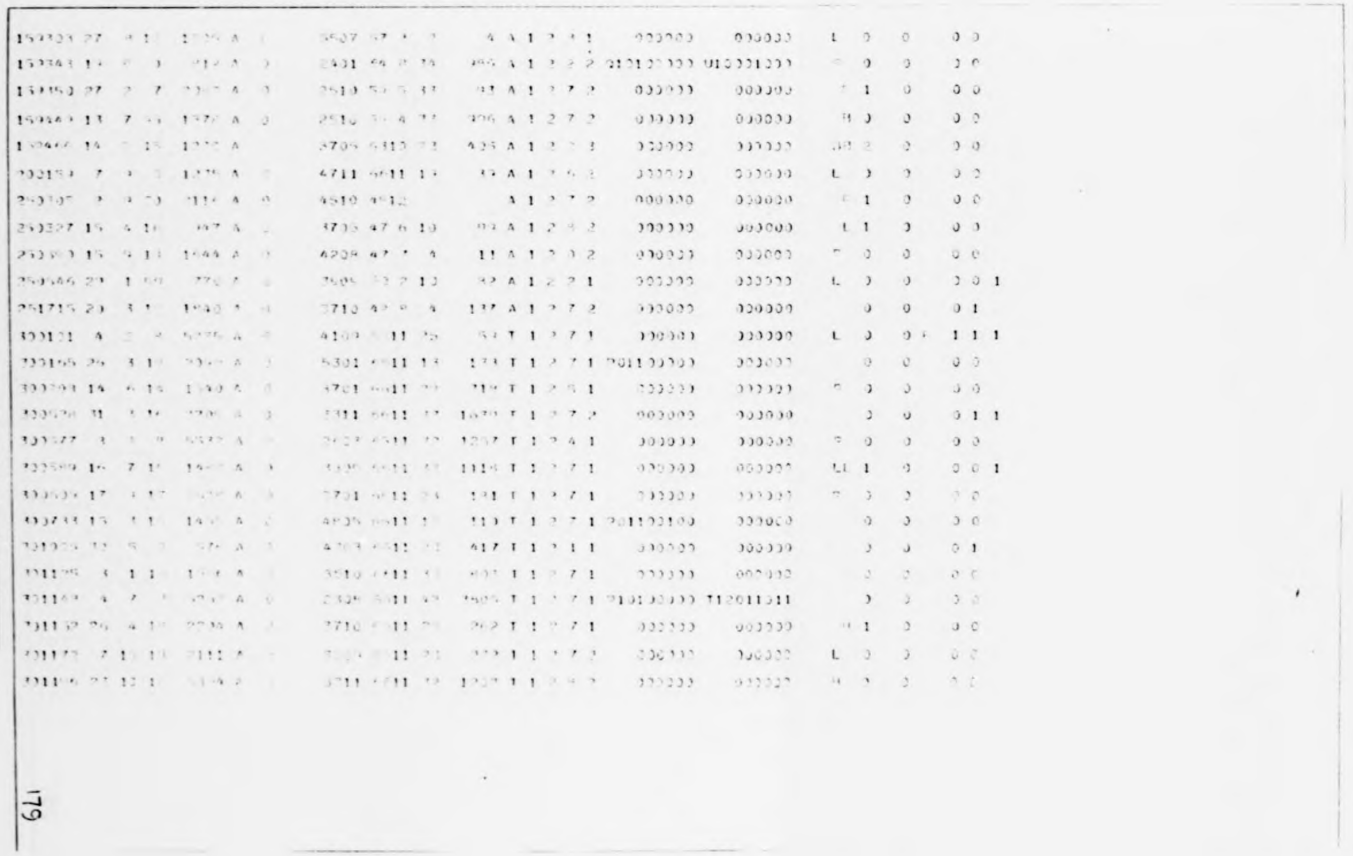
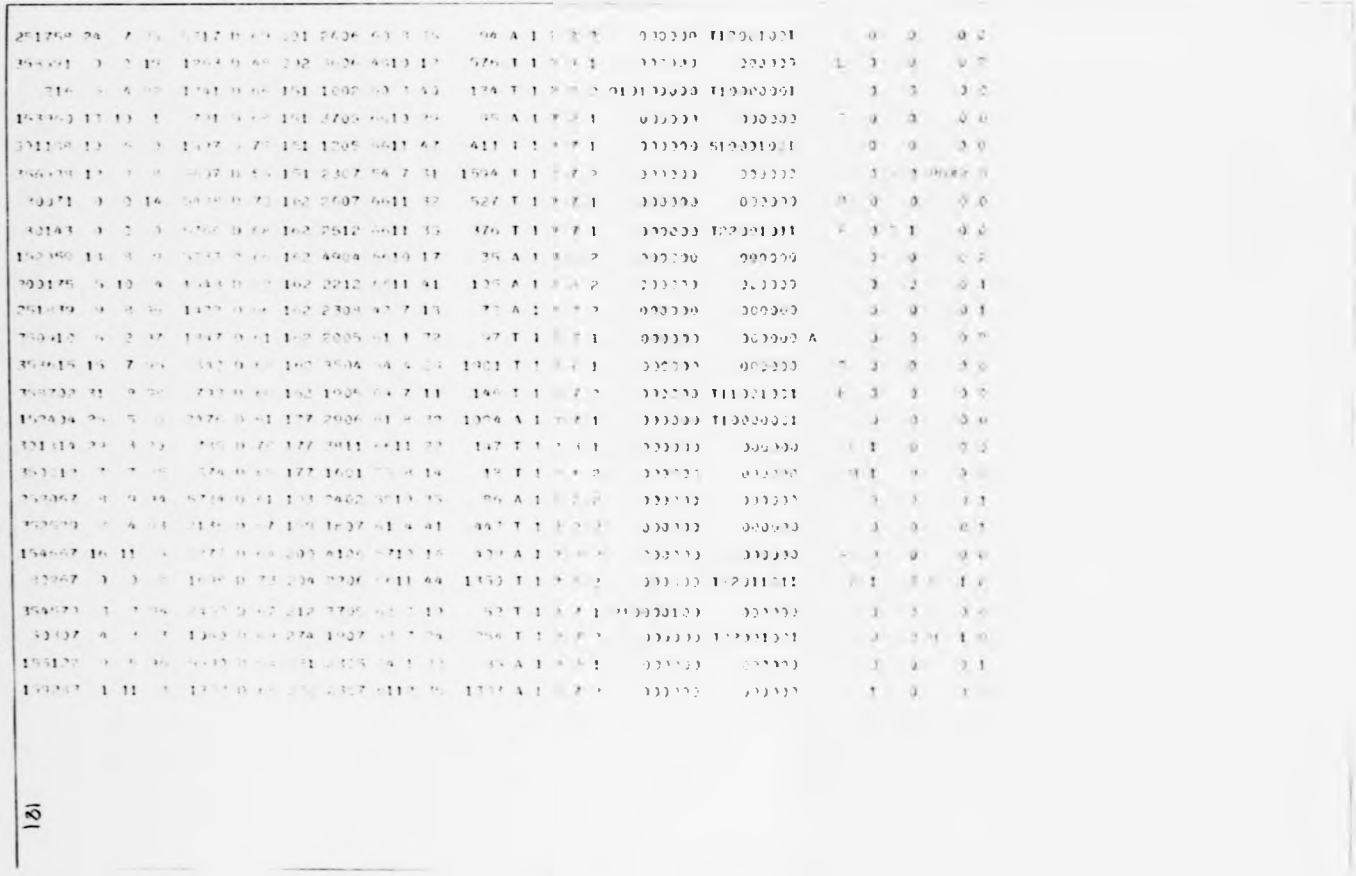
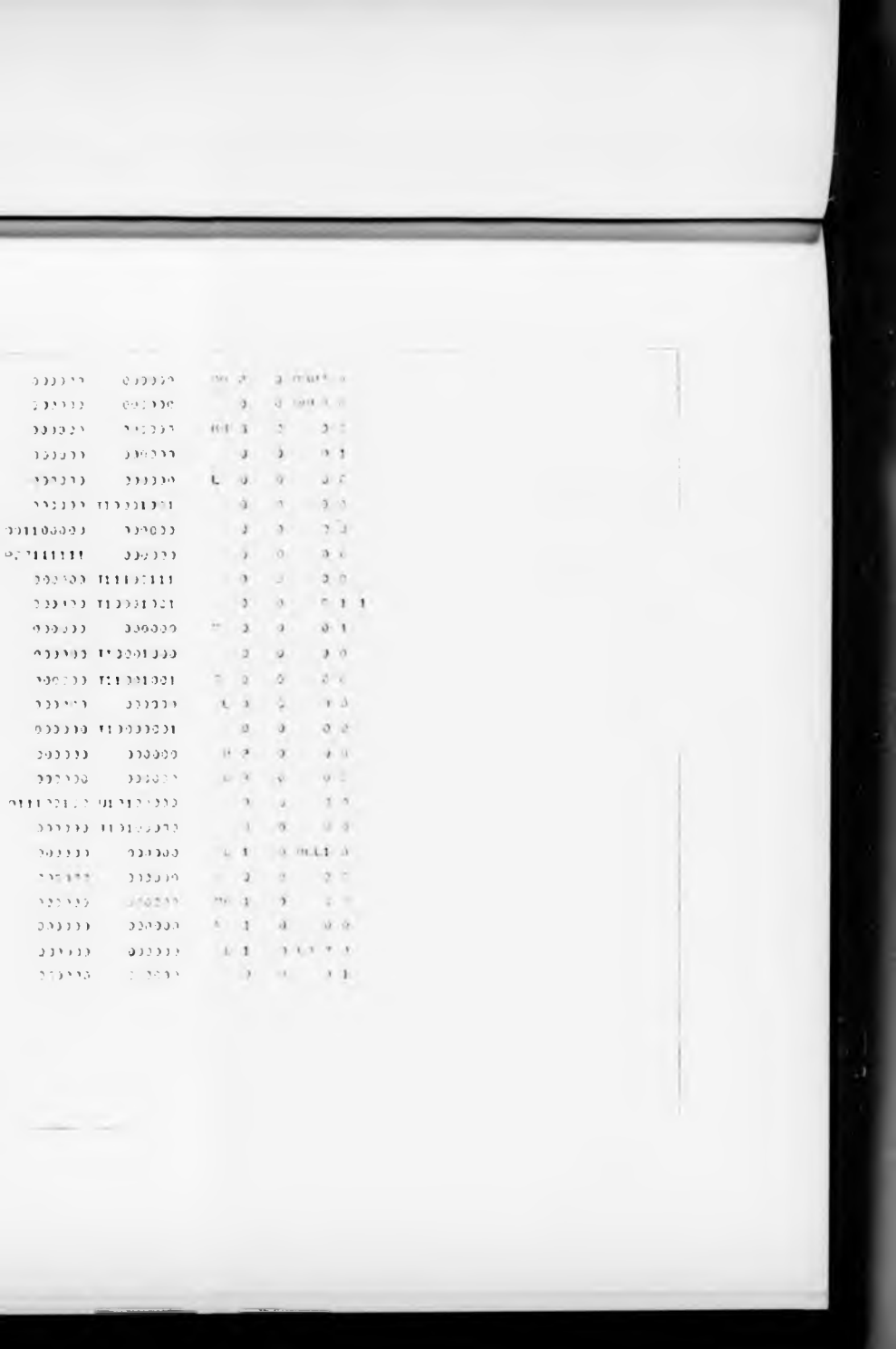






COHORT "B"

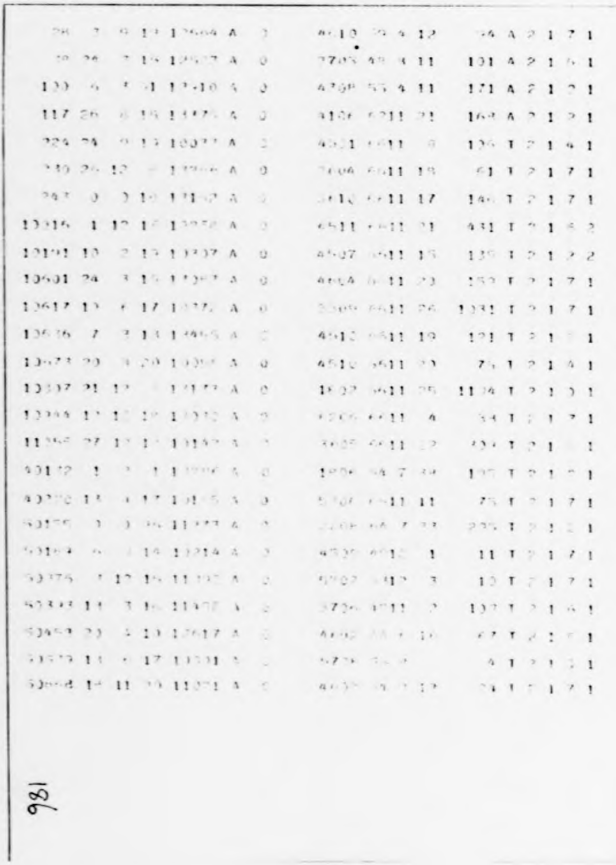
Reader 2

"Normals"

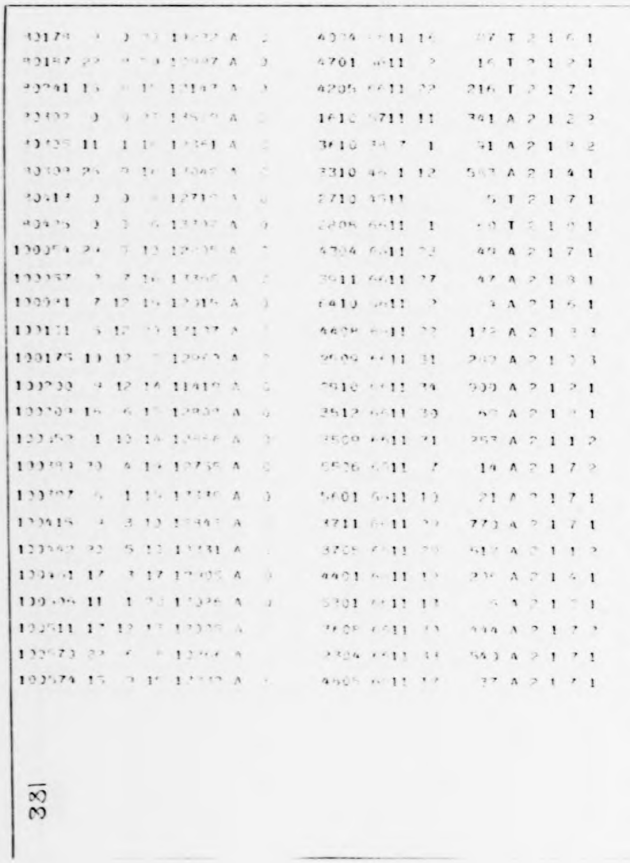
pp. 186 - 208

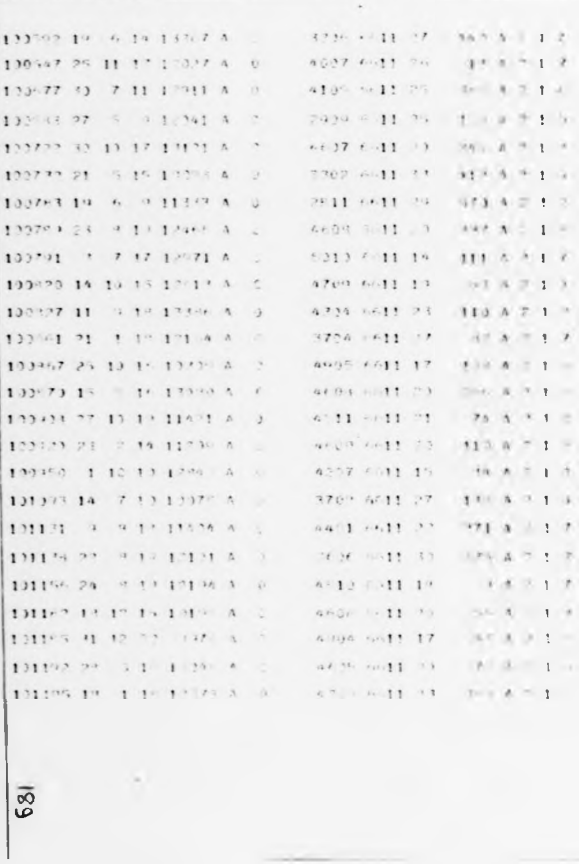
"Less-than-normals"

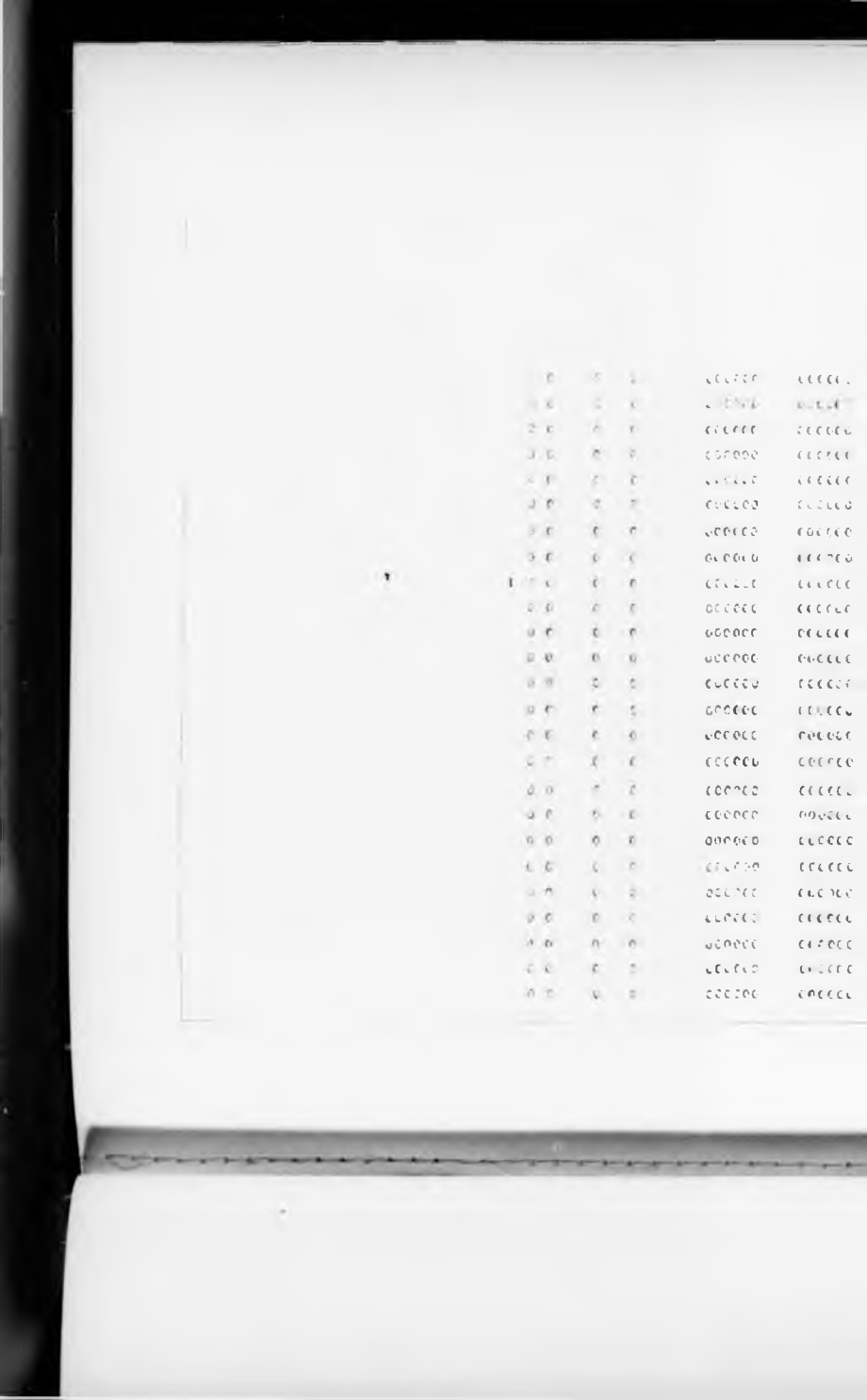
pp. 209 - 216

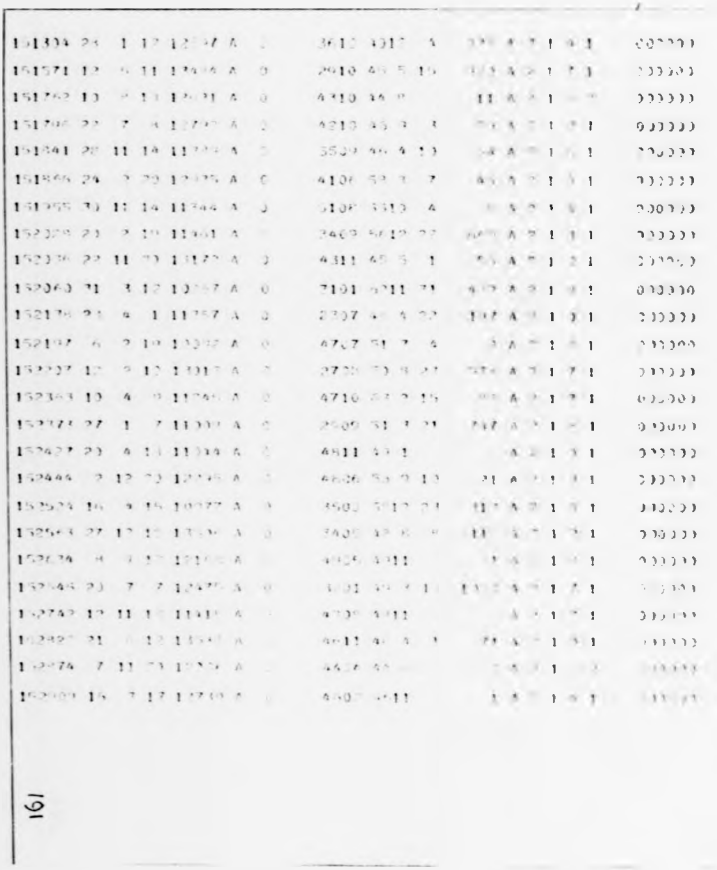


981









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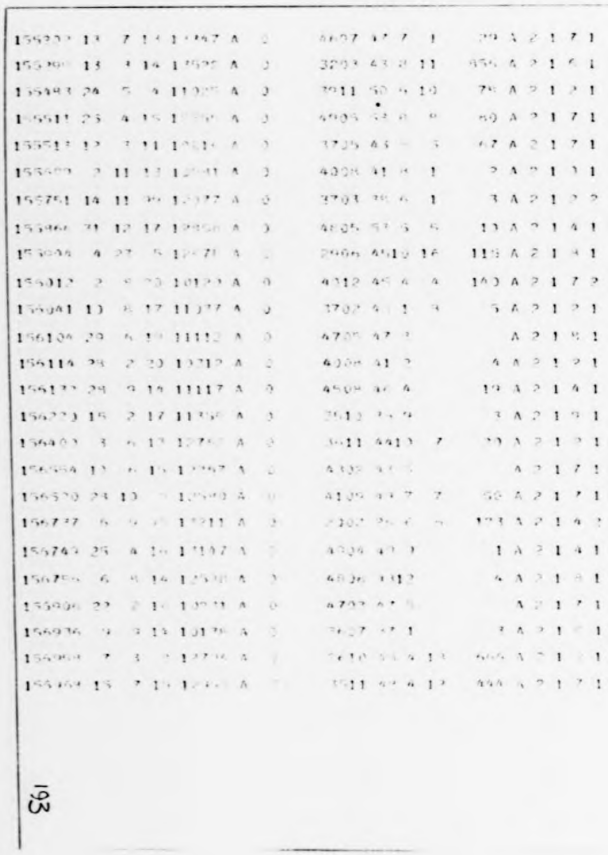

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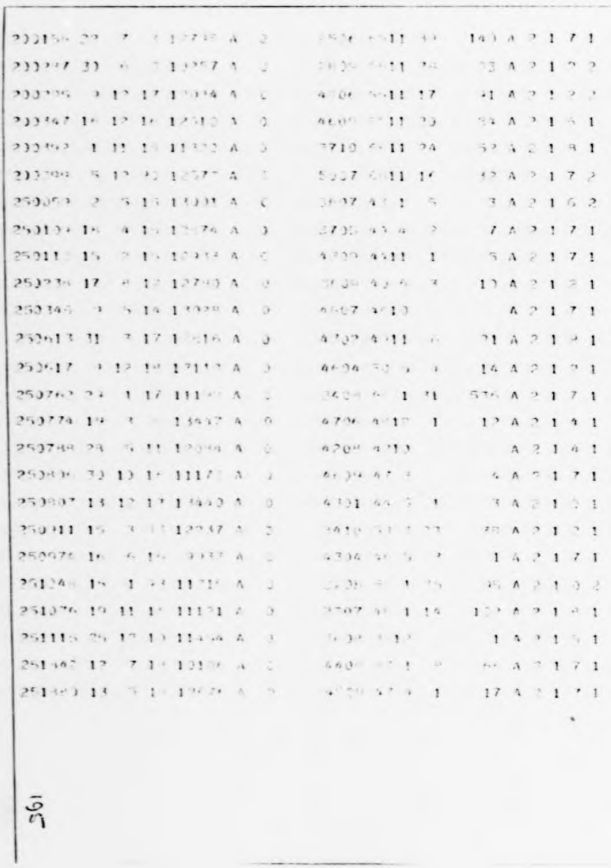

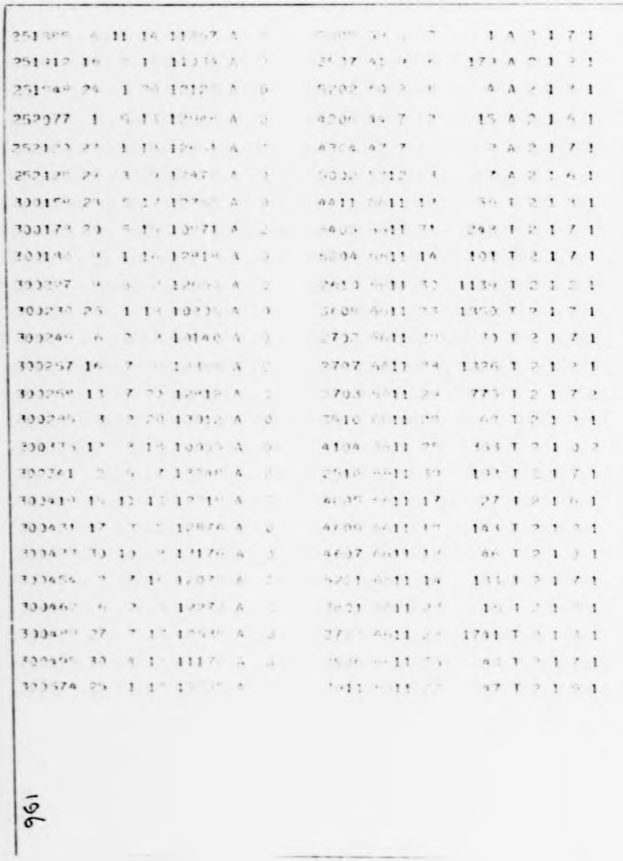

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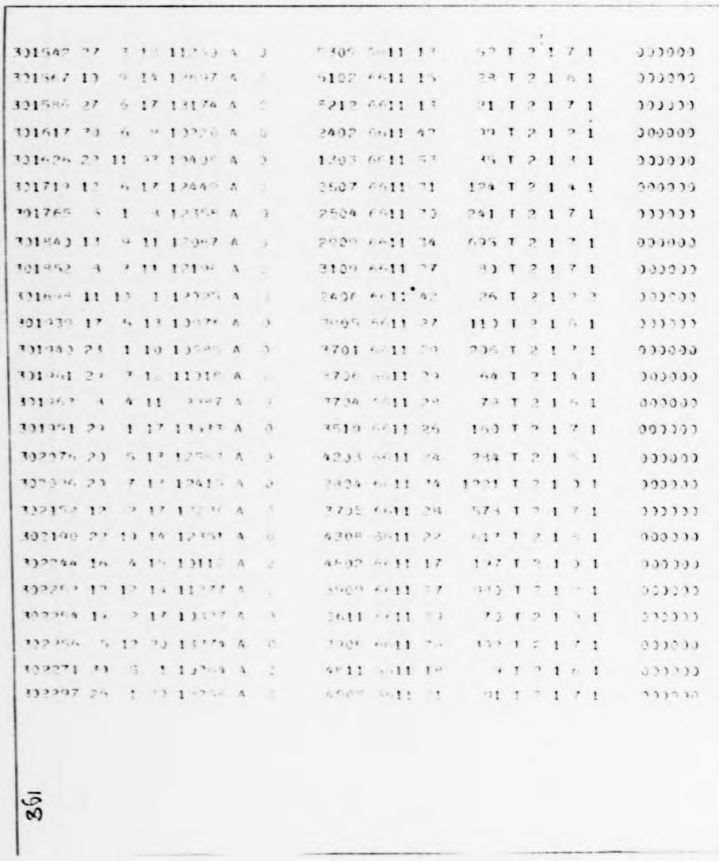

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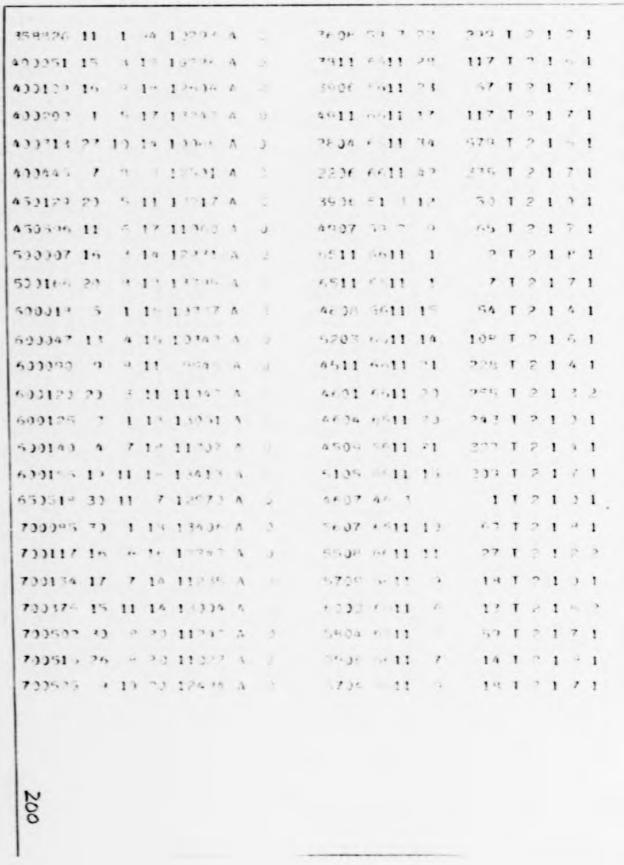

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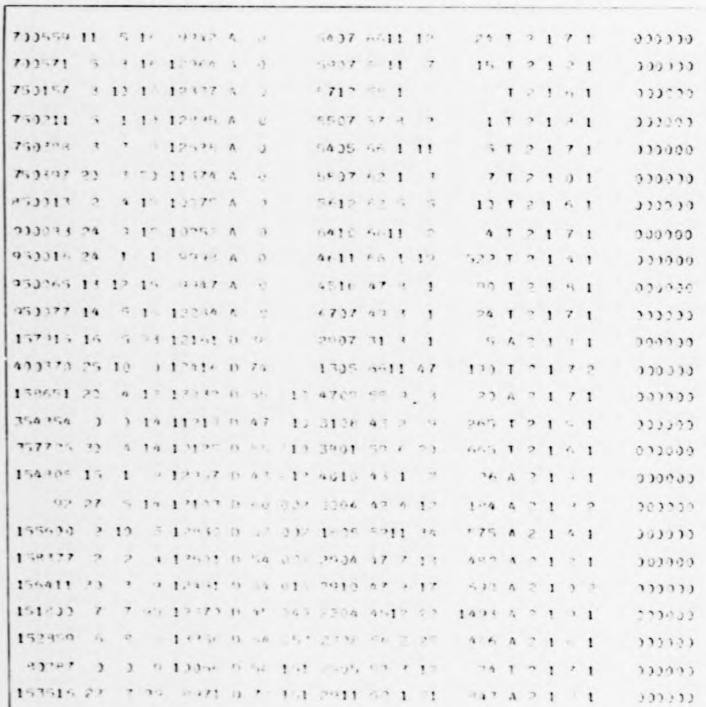

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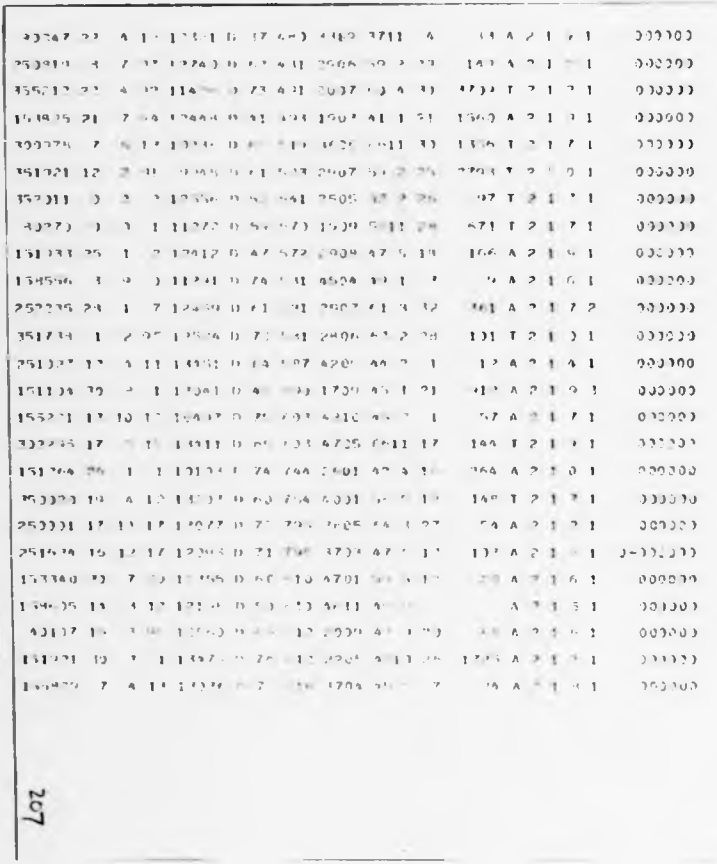

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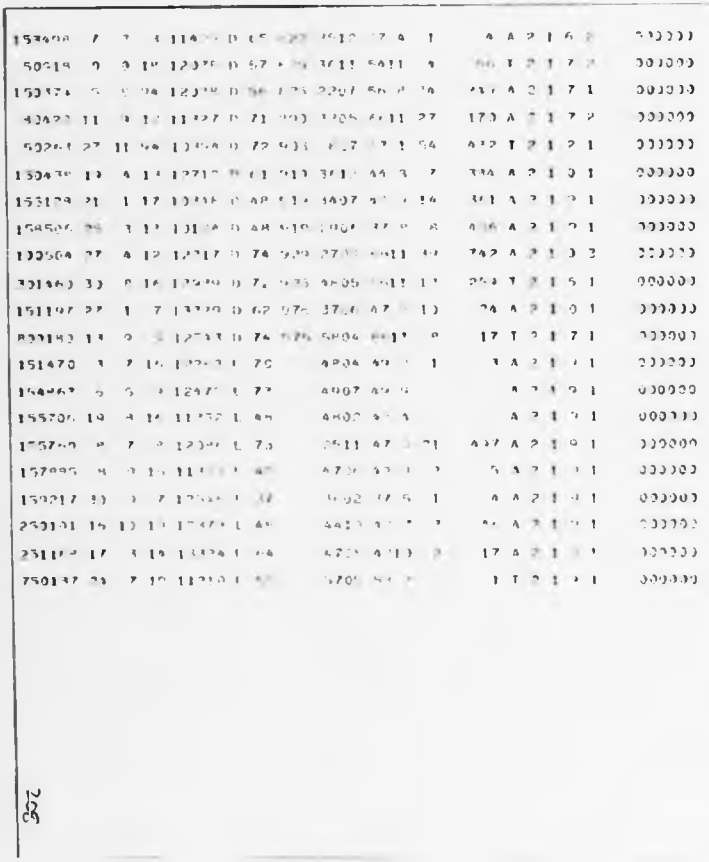

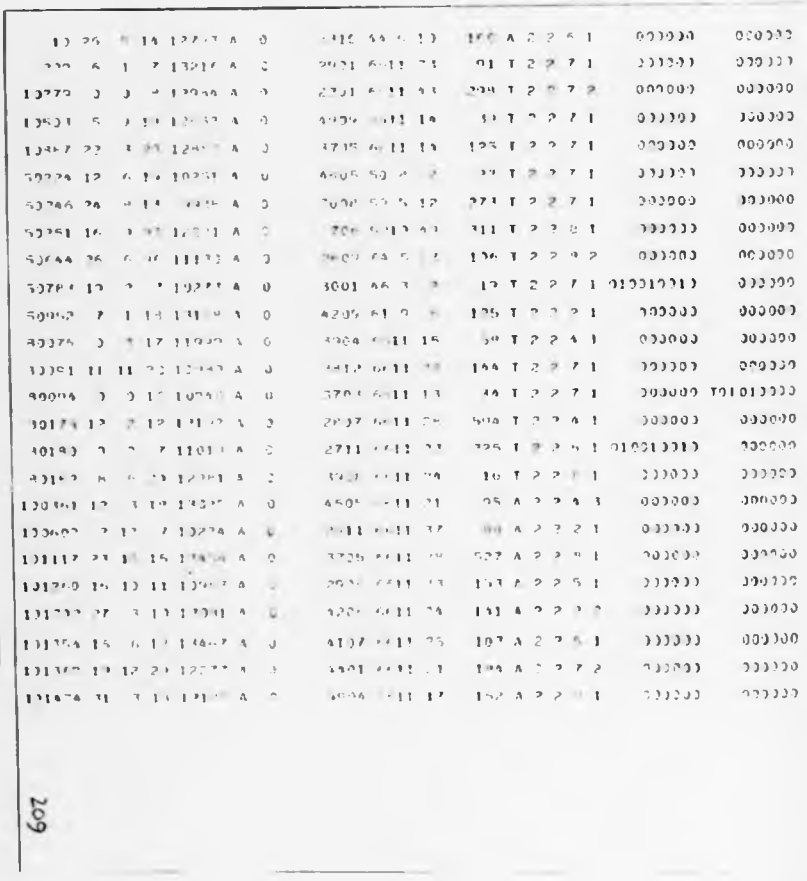

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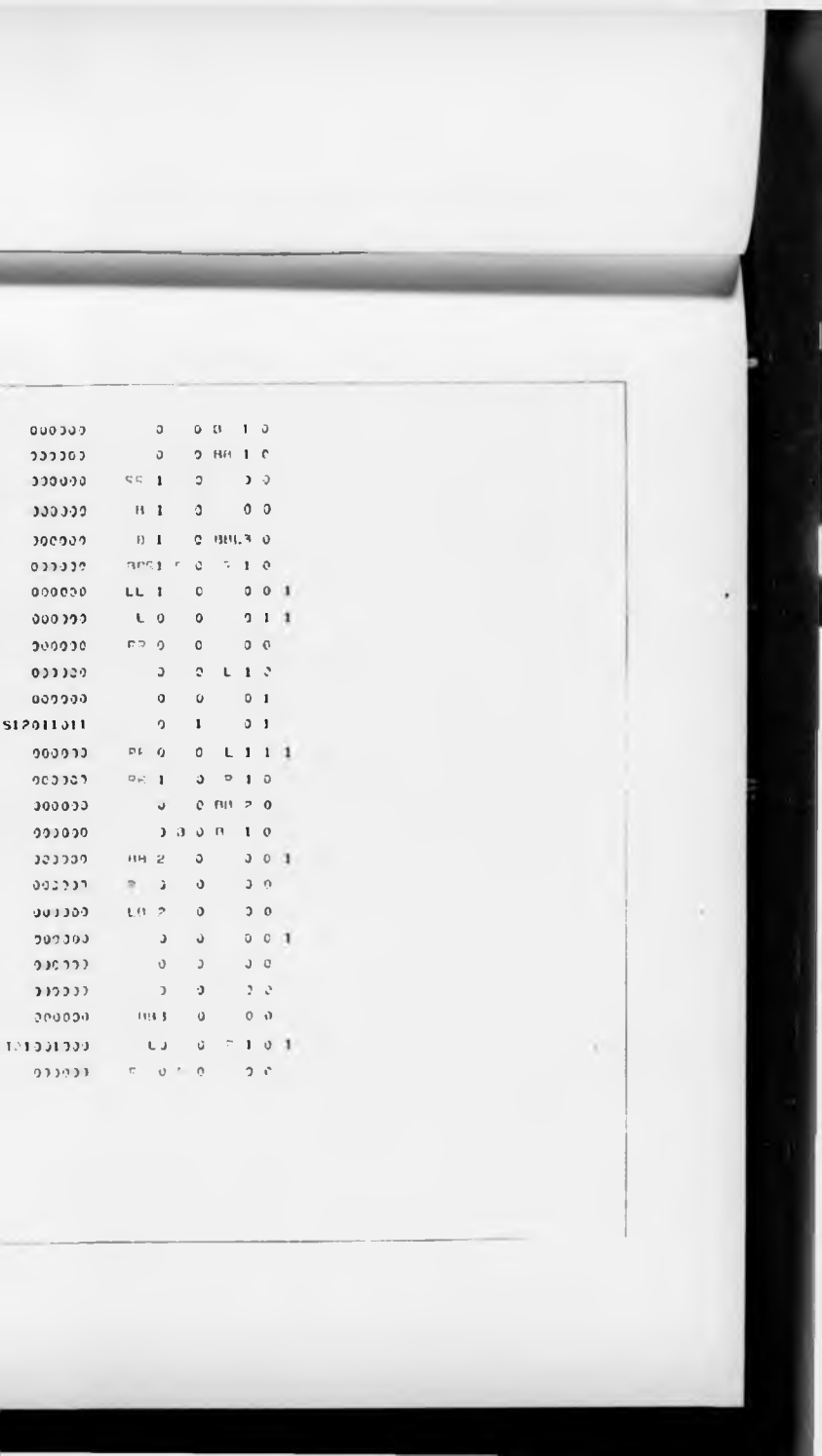

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302116	21	12	10	10371	A	0	3701	6611	23	242	T	2	2	7	1	001010000
302113	29	7	10	12444	A	0	2205	6611	38	1590	T	2	2	7	1	001010010
302114	24	2	16	13234	A	0	4206	6611	24	[illegible]	T	2	2	3	2	001011000
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357588	24	11	36	12645	A	0		1710	64	7	34		277	T	
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433379	13	9	23	10063	A	0		3802	4611	27			209	T	
433556	15	5	33	12281	A	0		2229	4611	44			29	T	
433396	13	0	16	13115	A	0		2805	4611	10			634	T	
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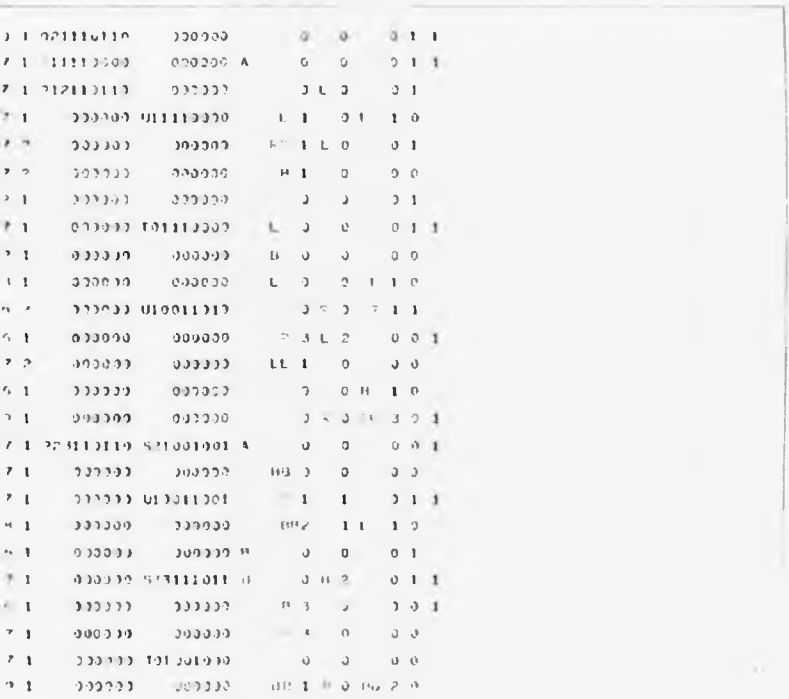

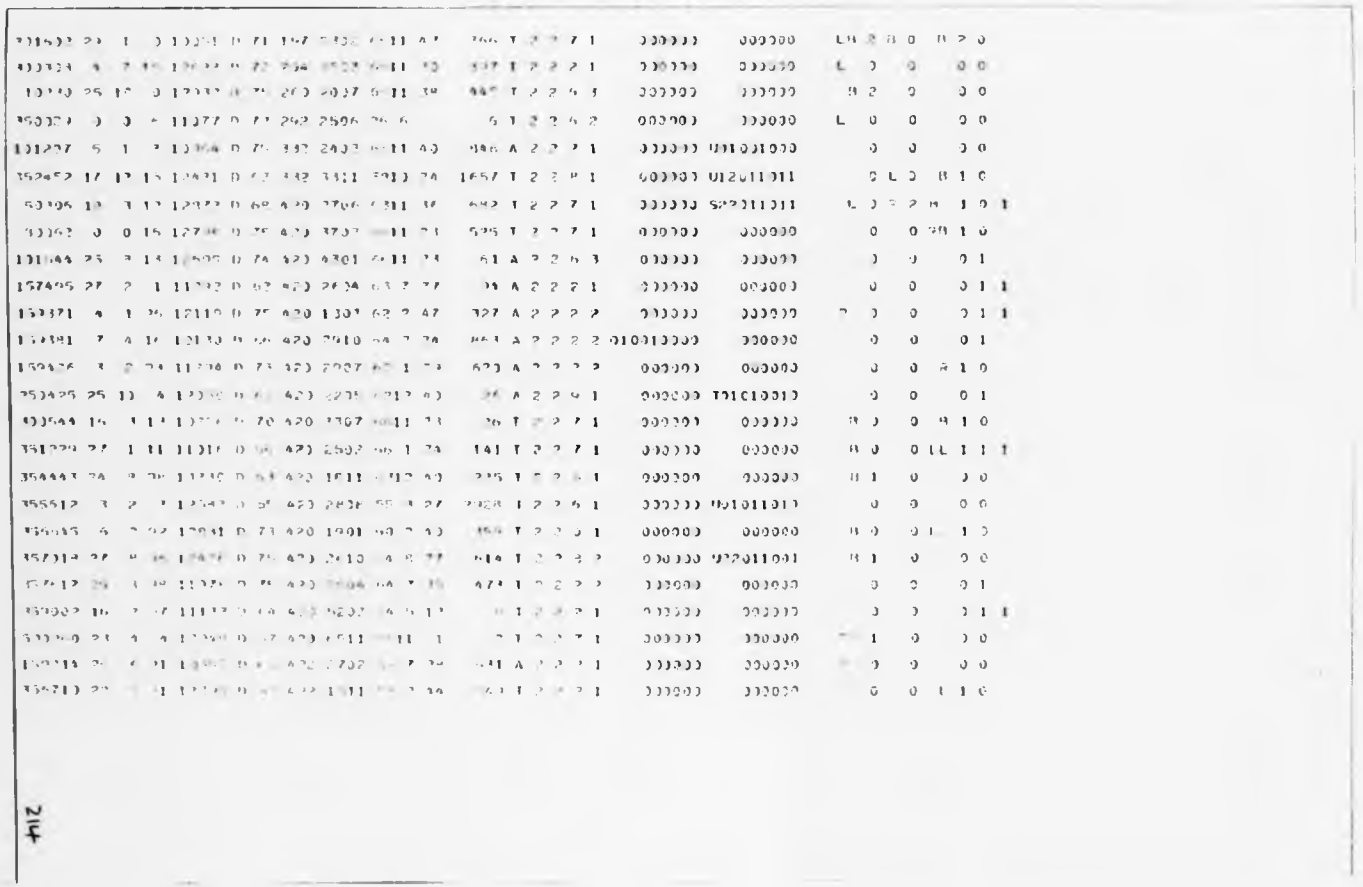

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356189	1	4	0	11139	D	56	434	1510	5511	40		797	T	2	2	3	1
352533	23	2	3	13416	D	69	442	2002	61	7	43	692	T	2	2	7	1
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354014	1	12	95	10026	D	58	581	1206	56	5	36	5148	T	2	2	8	2
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COHORT "B"

Reader 3

"Normals"

pp. 218 - 236

"Less-than-normals"

pp. 237 - 248

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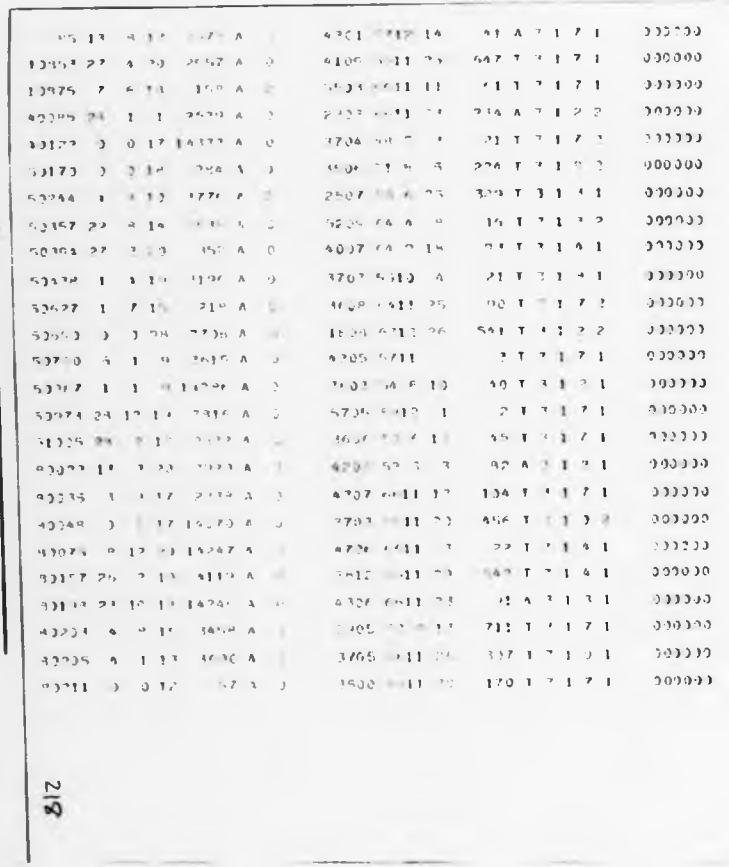

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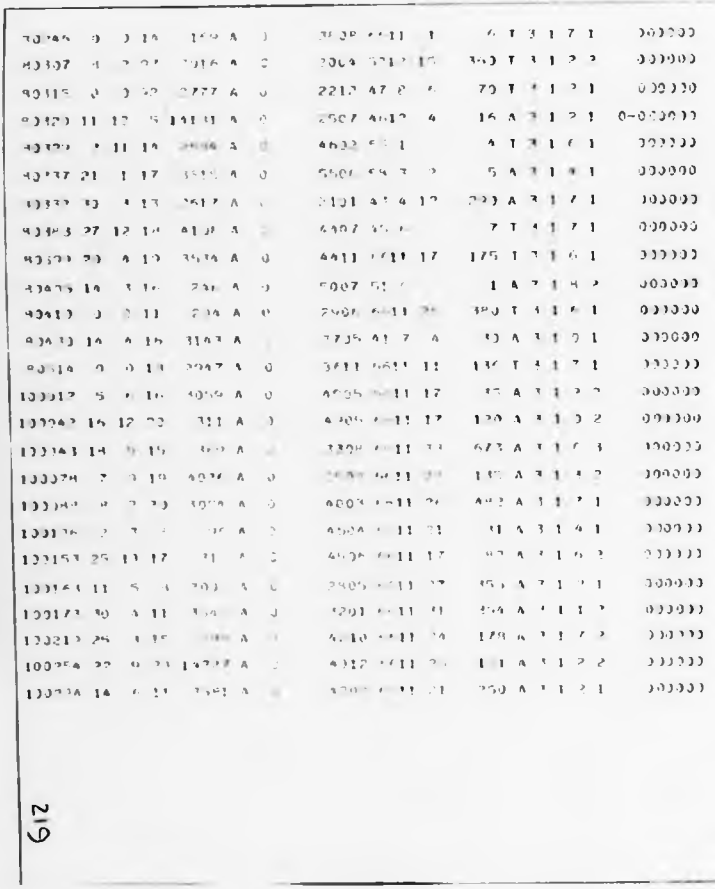

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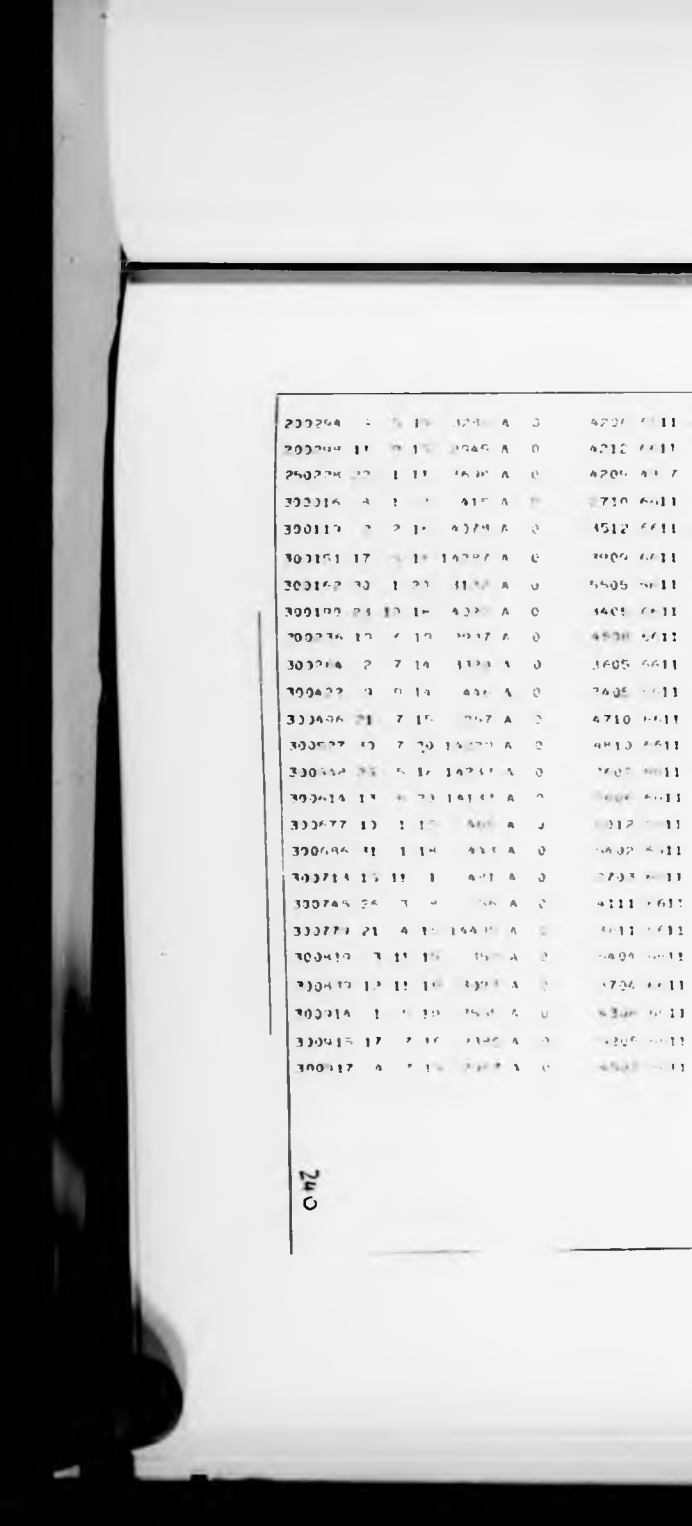

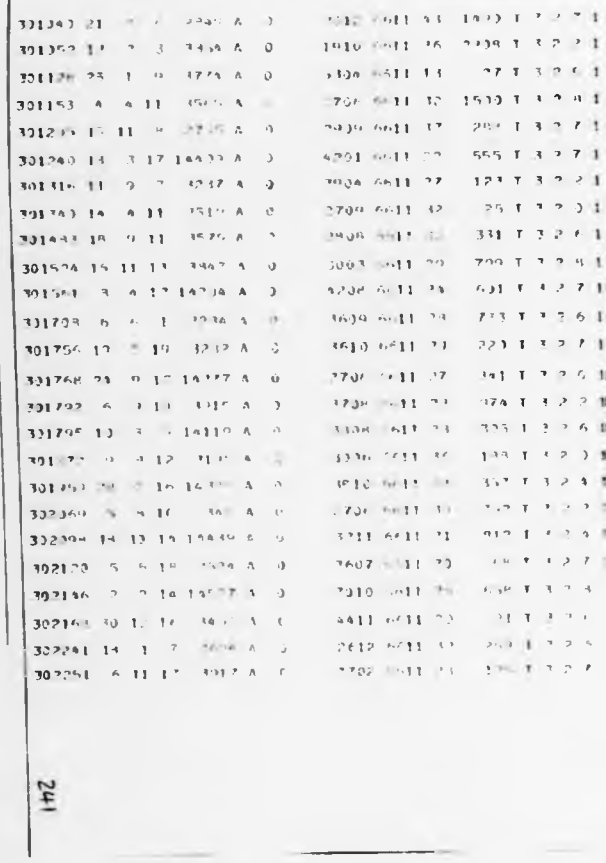

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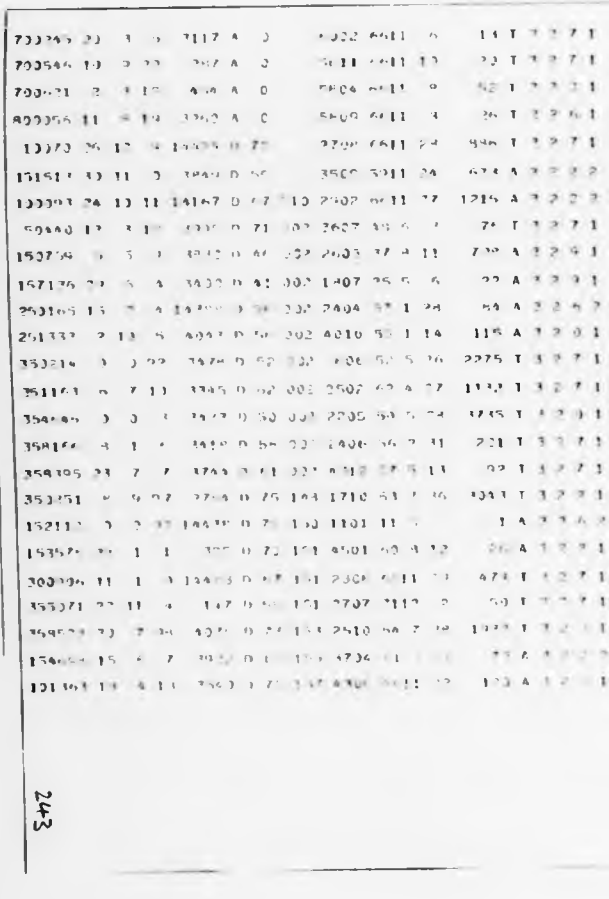

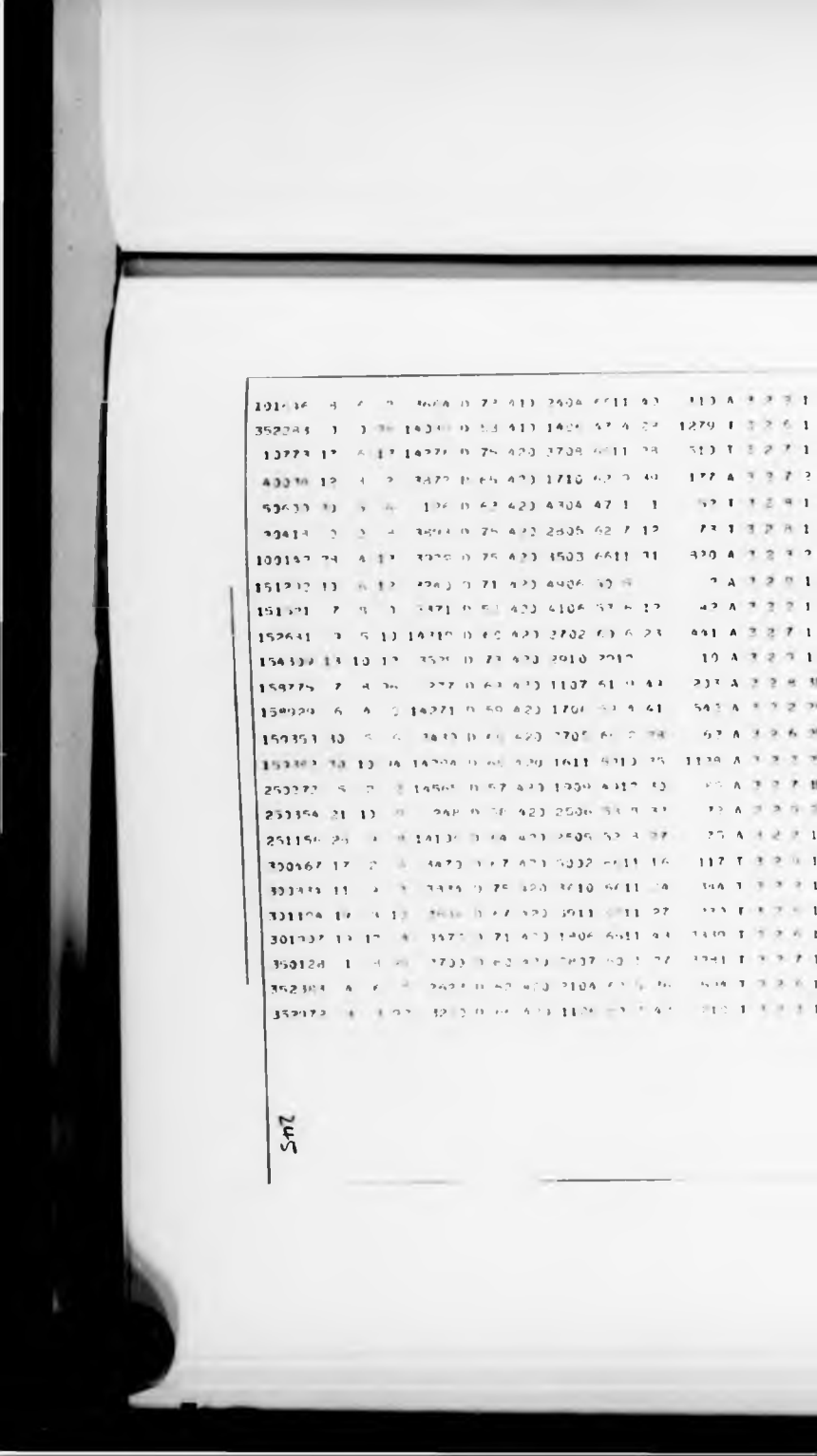

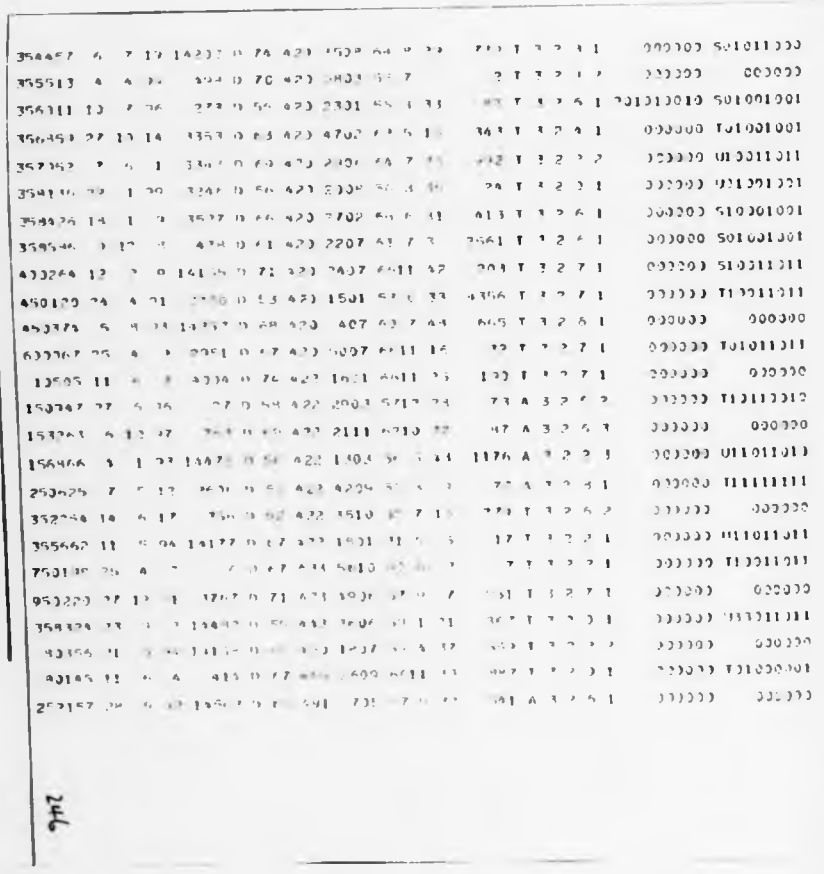

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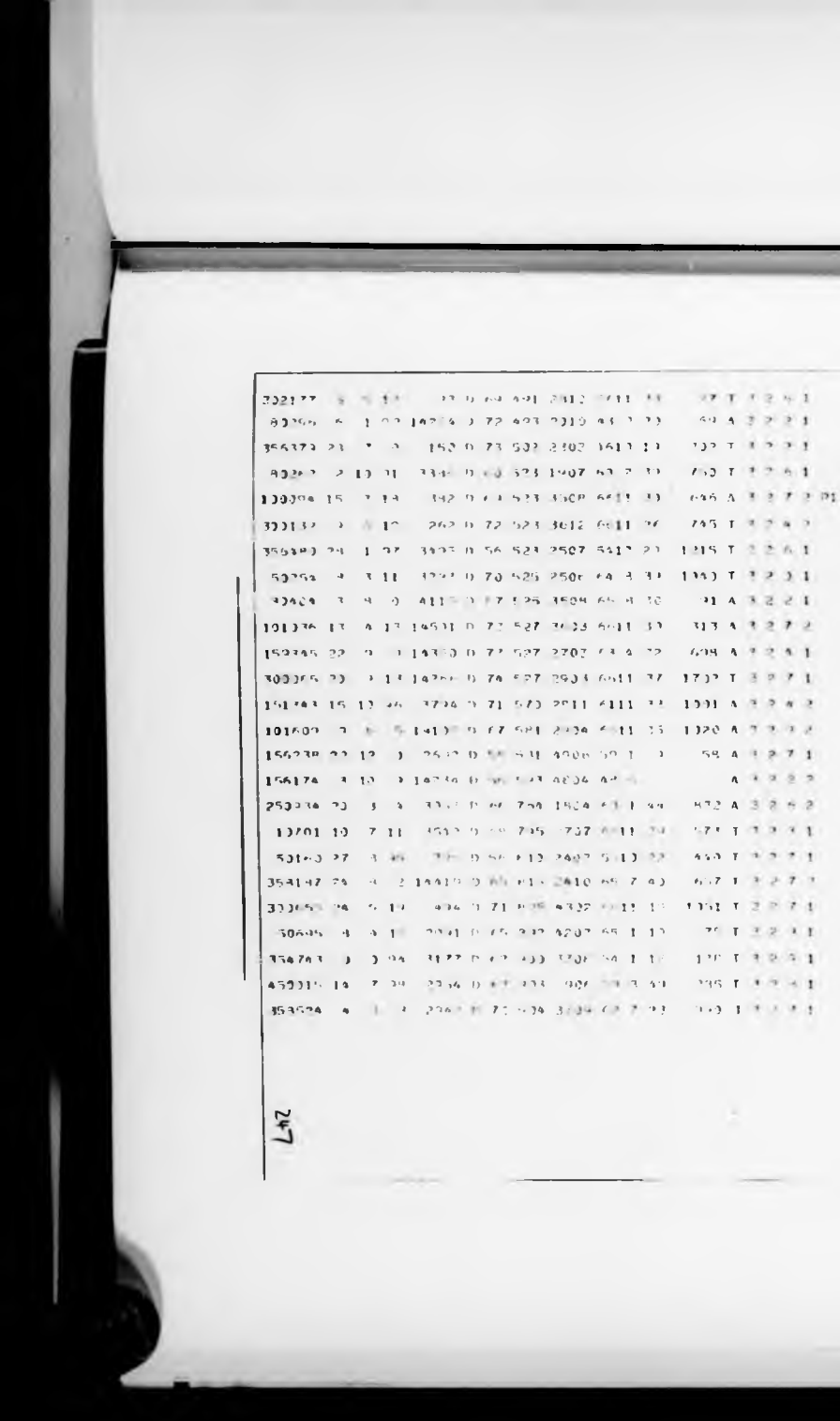

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COHORT "B"

Reader 4

"Normals"

pp. 250 - 268

"Less-than-normals"

pp. 269 - 281

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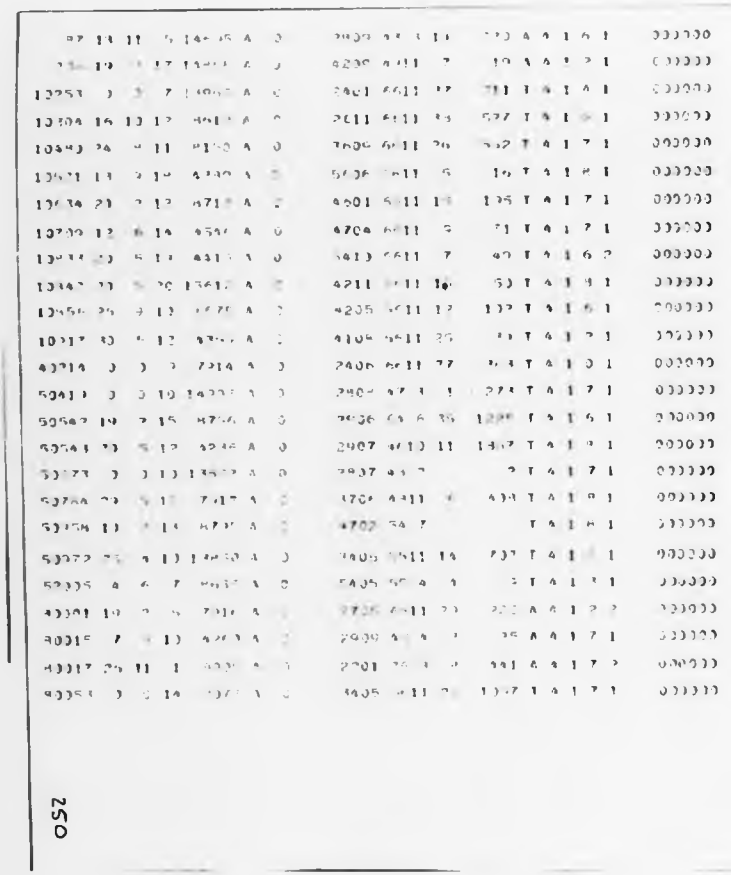

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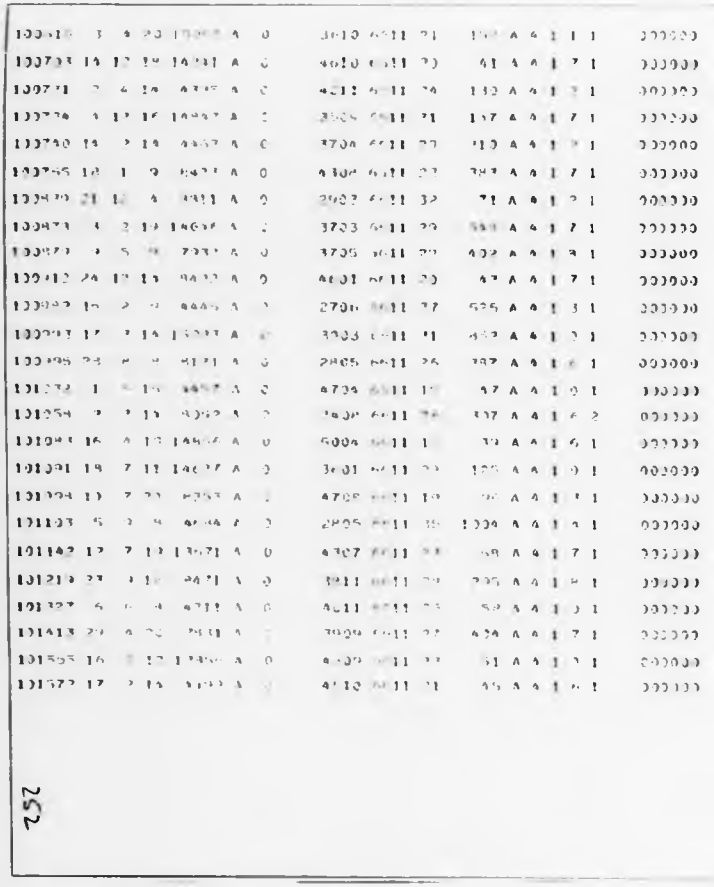

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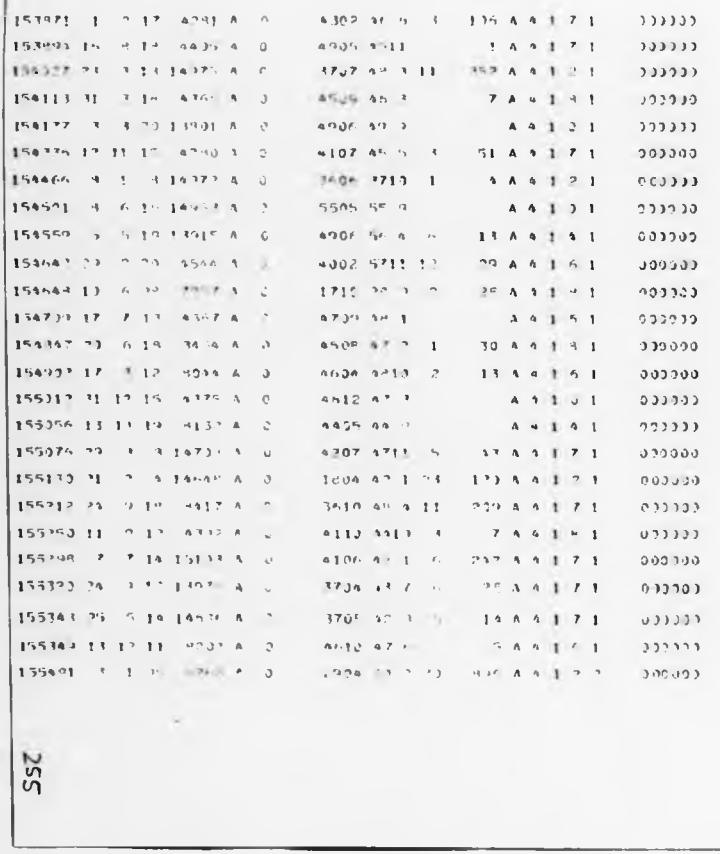

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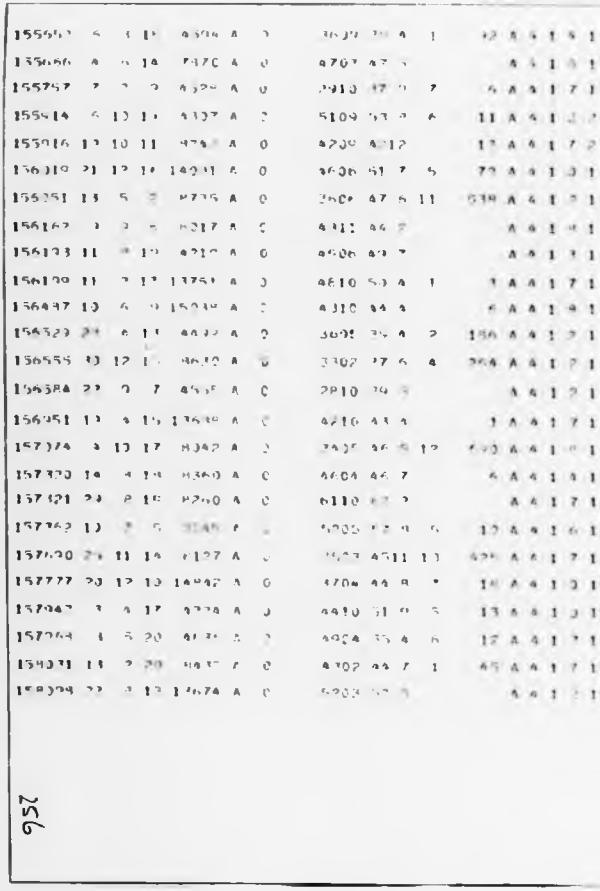

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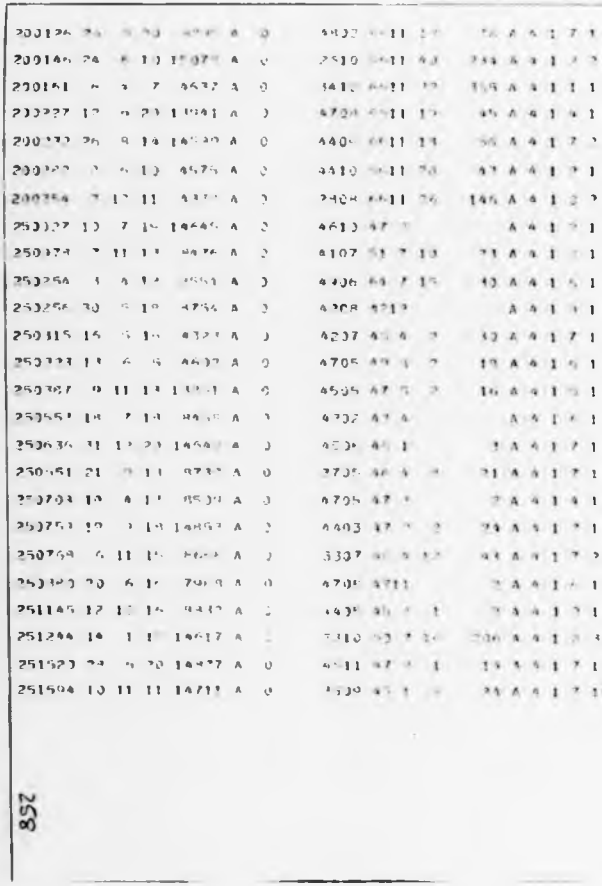

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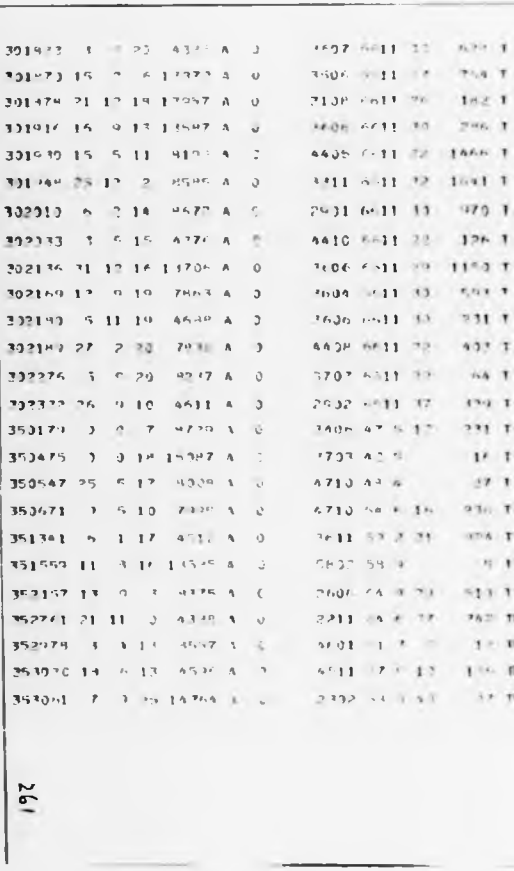

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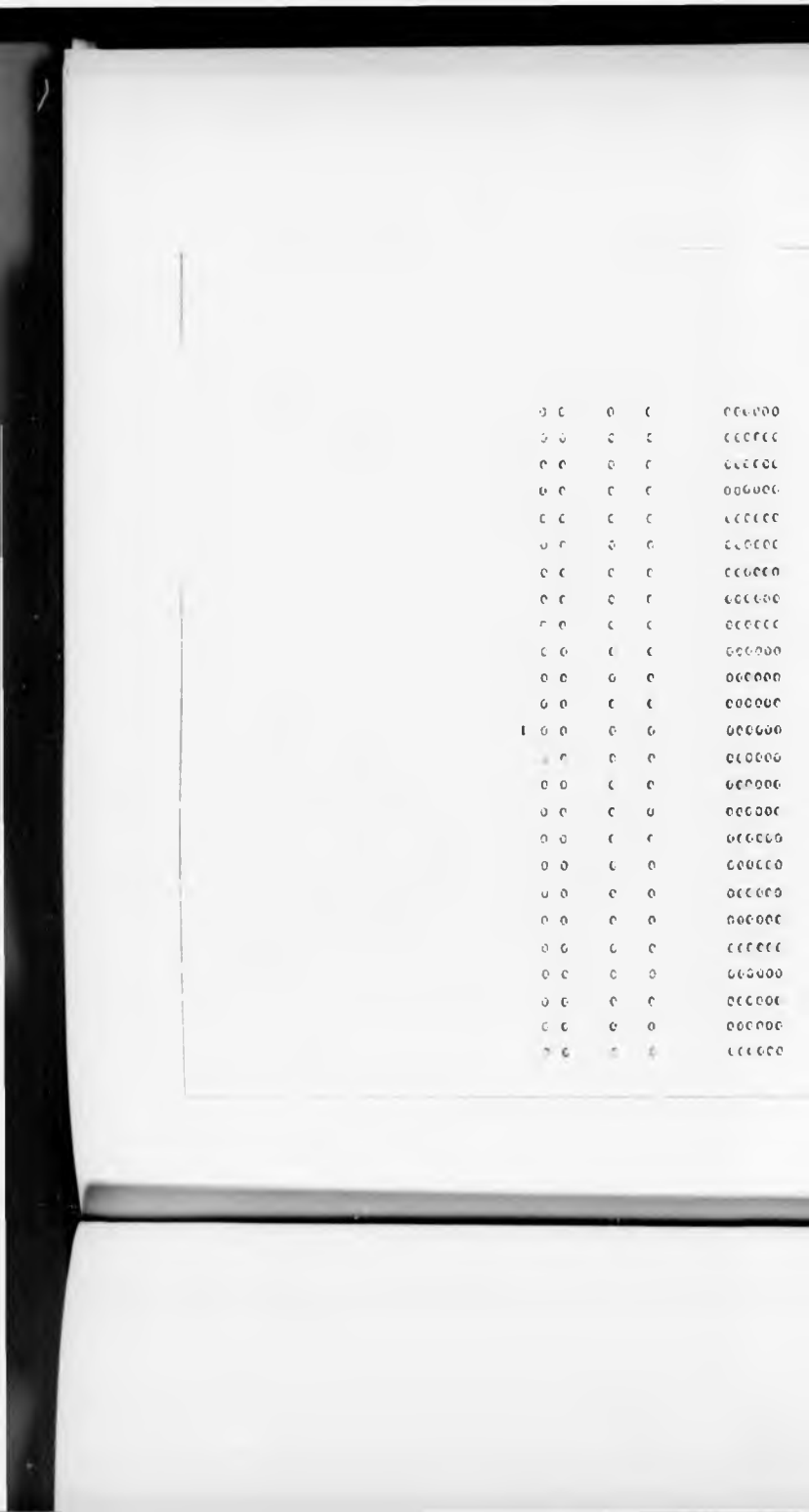

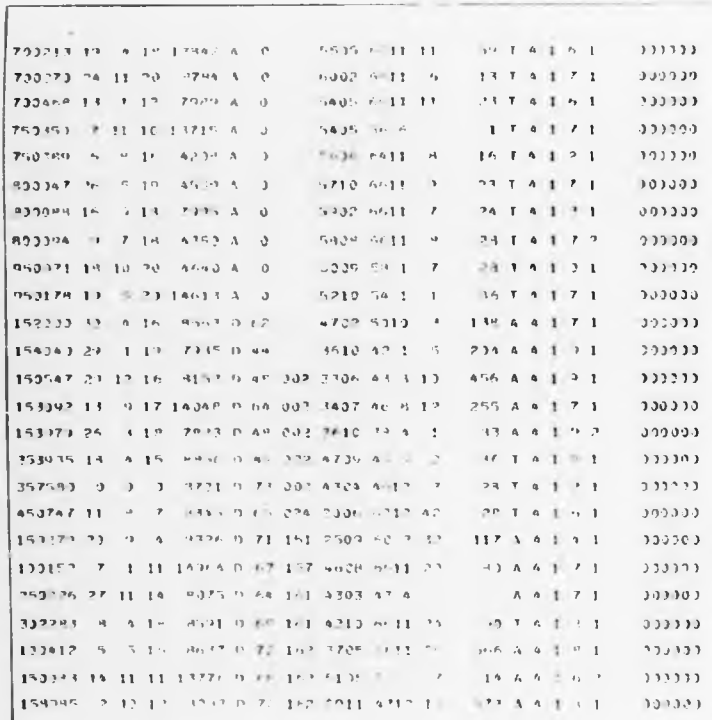

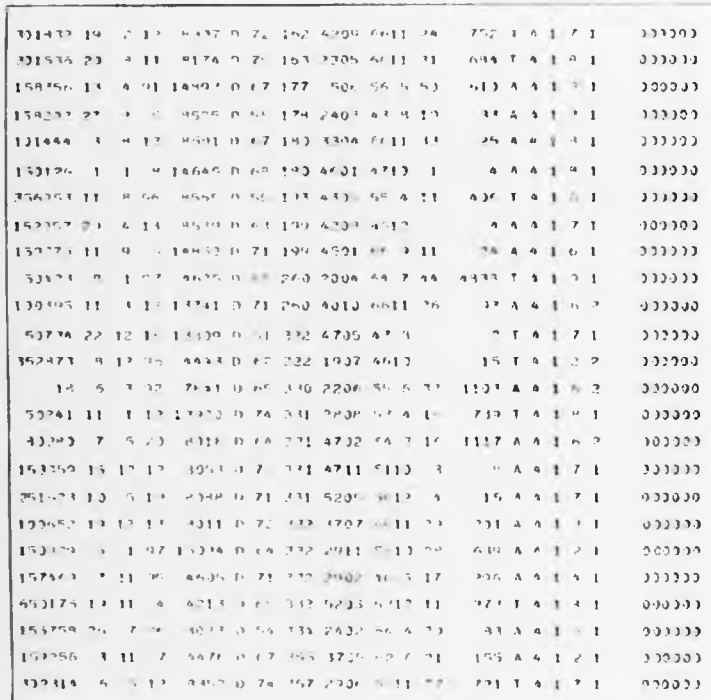

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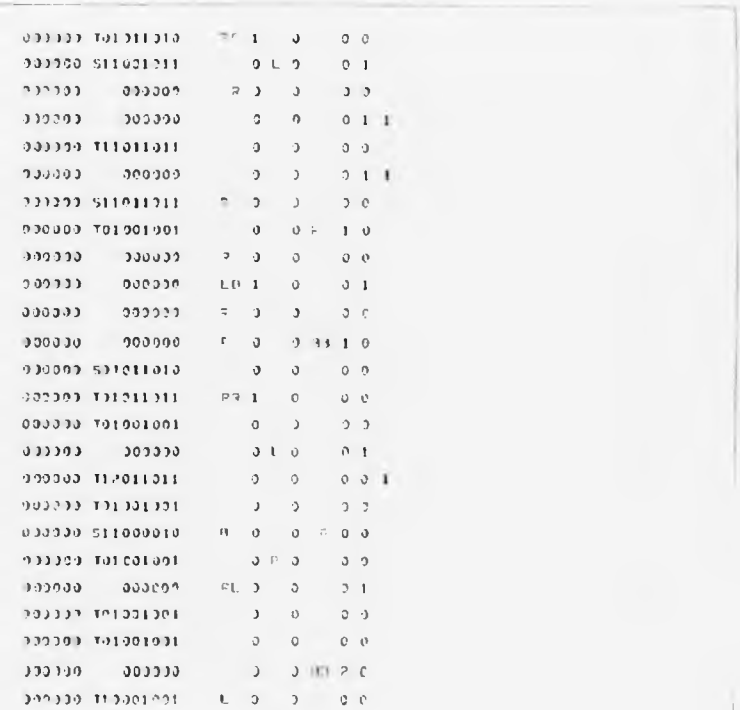

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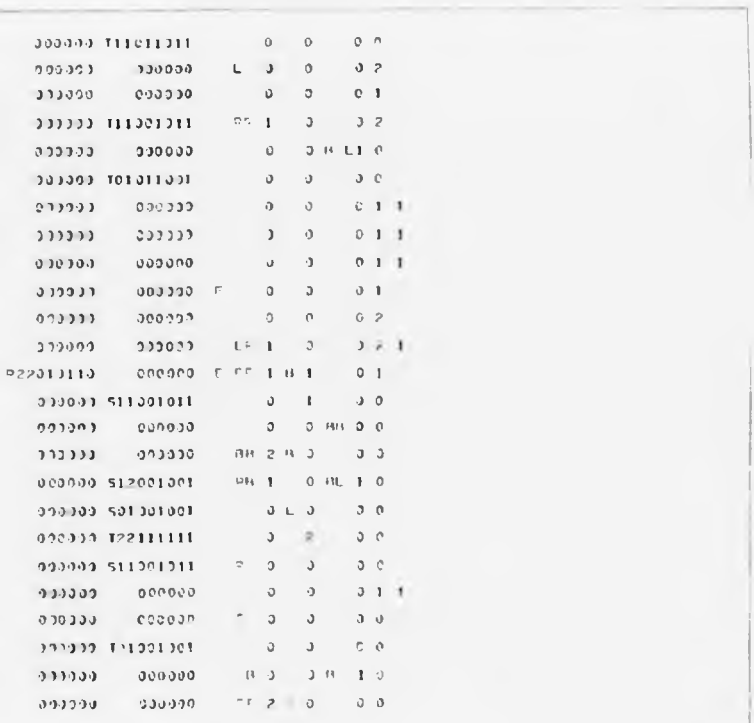

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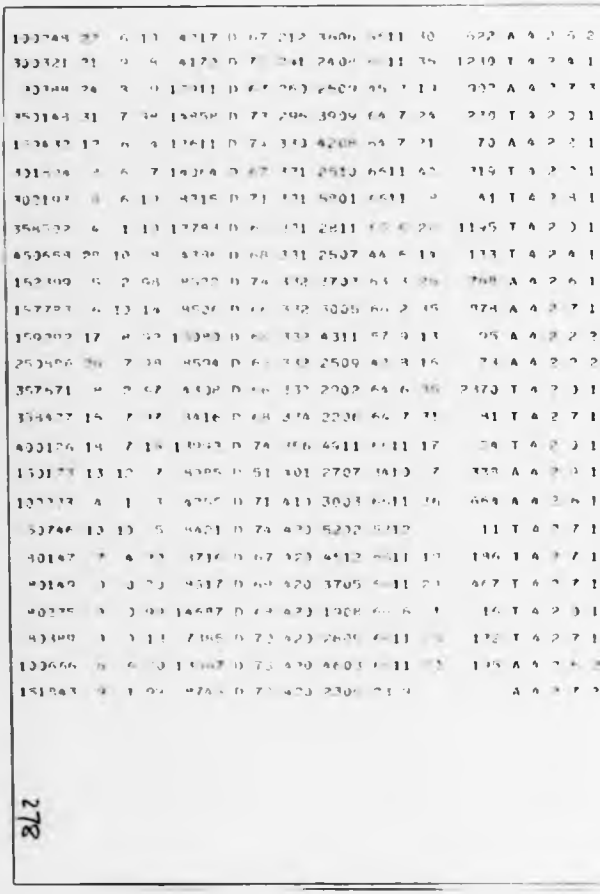

272

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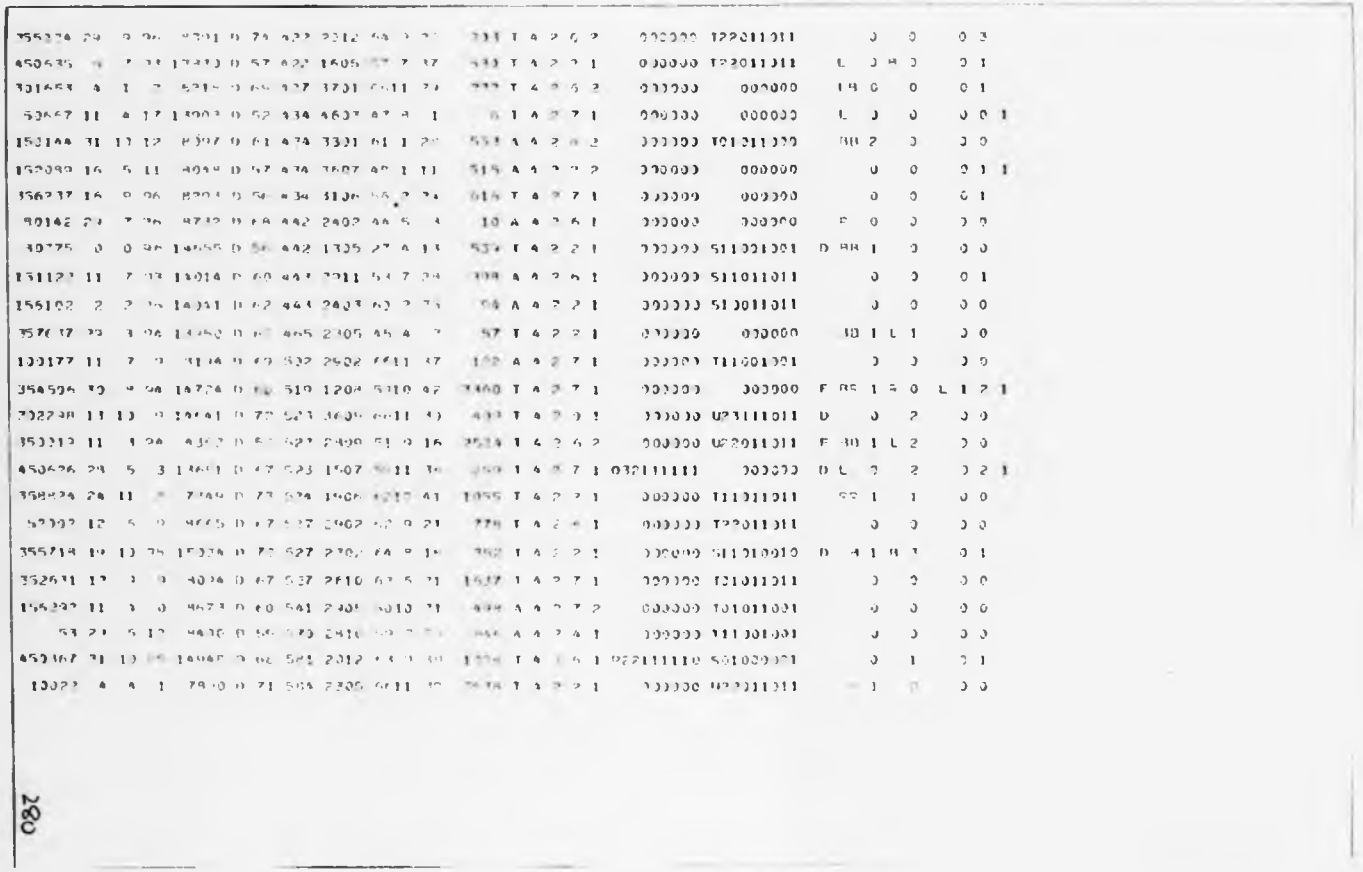

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COHORT "B"

Reader 5

"Normals"

pp. 283 - 303

"Less-than-normals"

pp. 304 - 314

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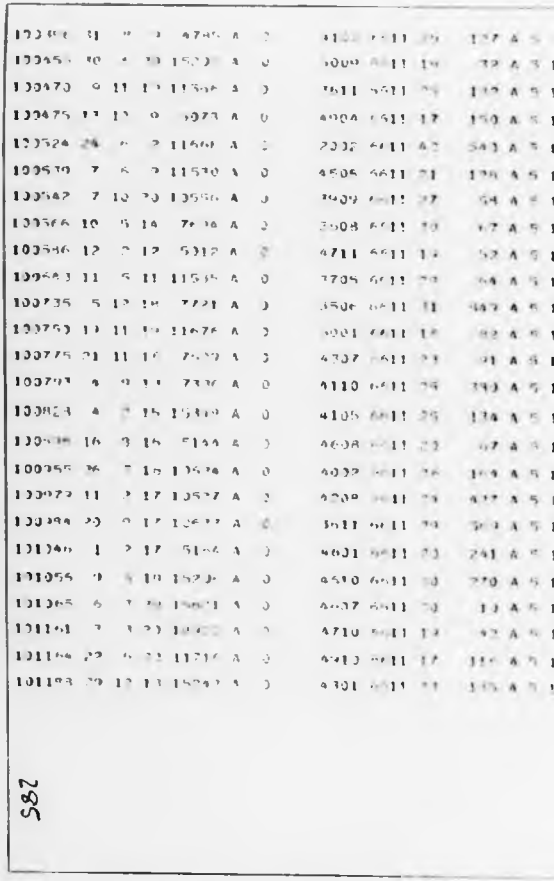

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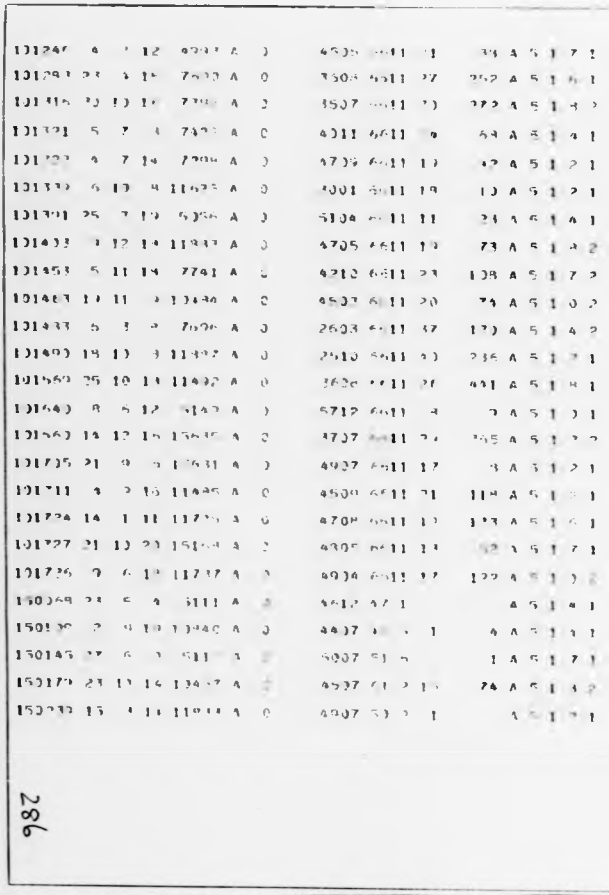

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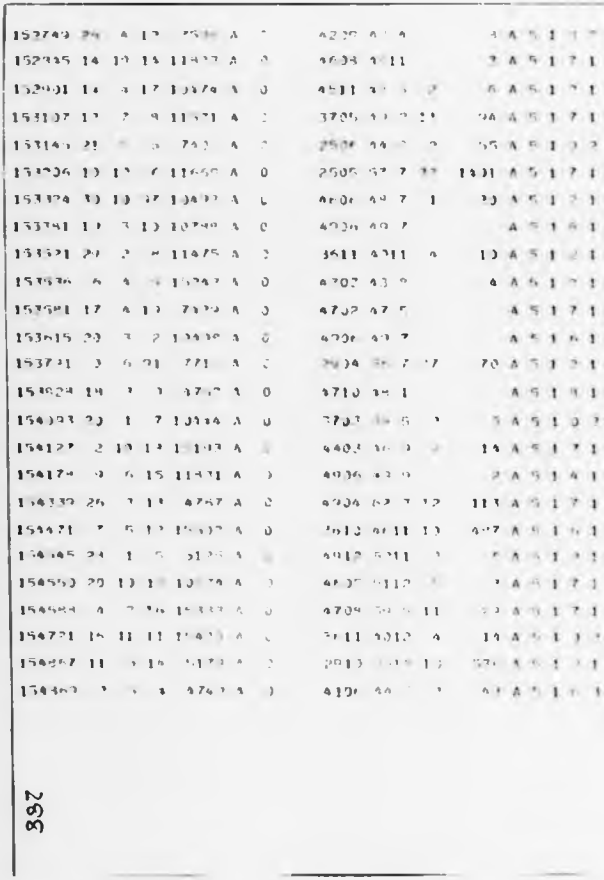

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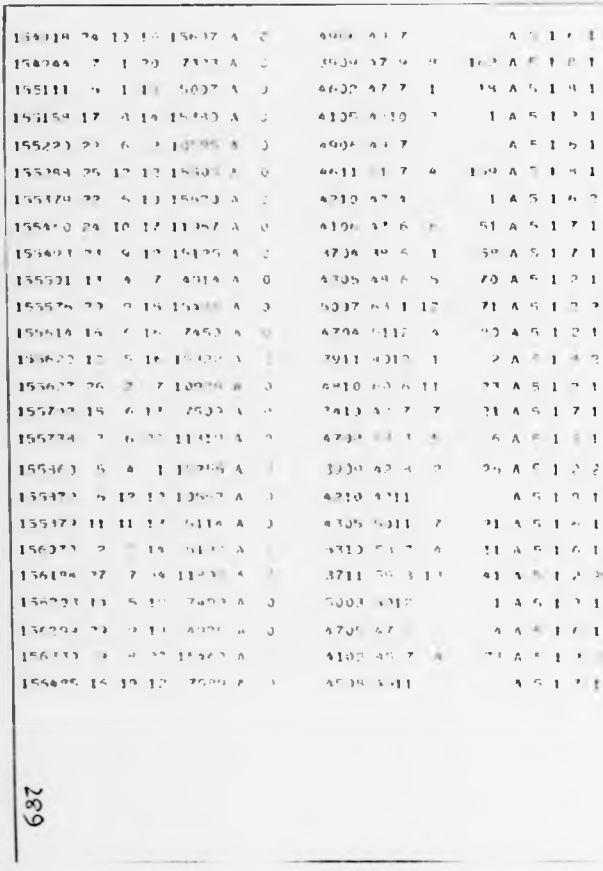

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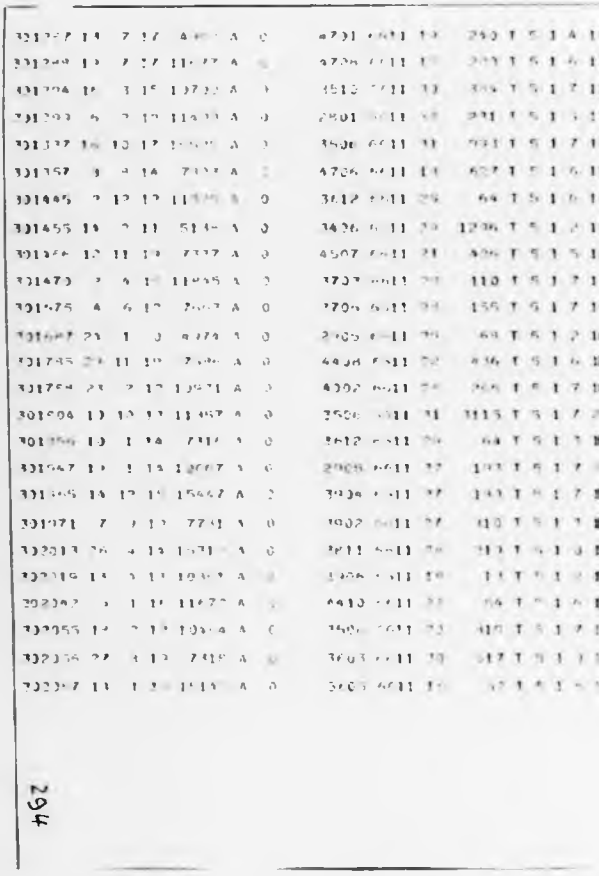

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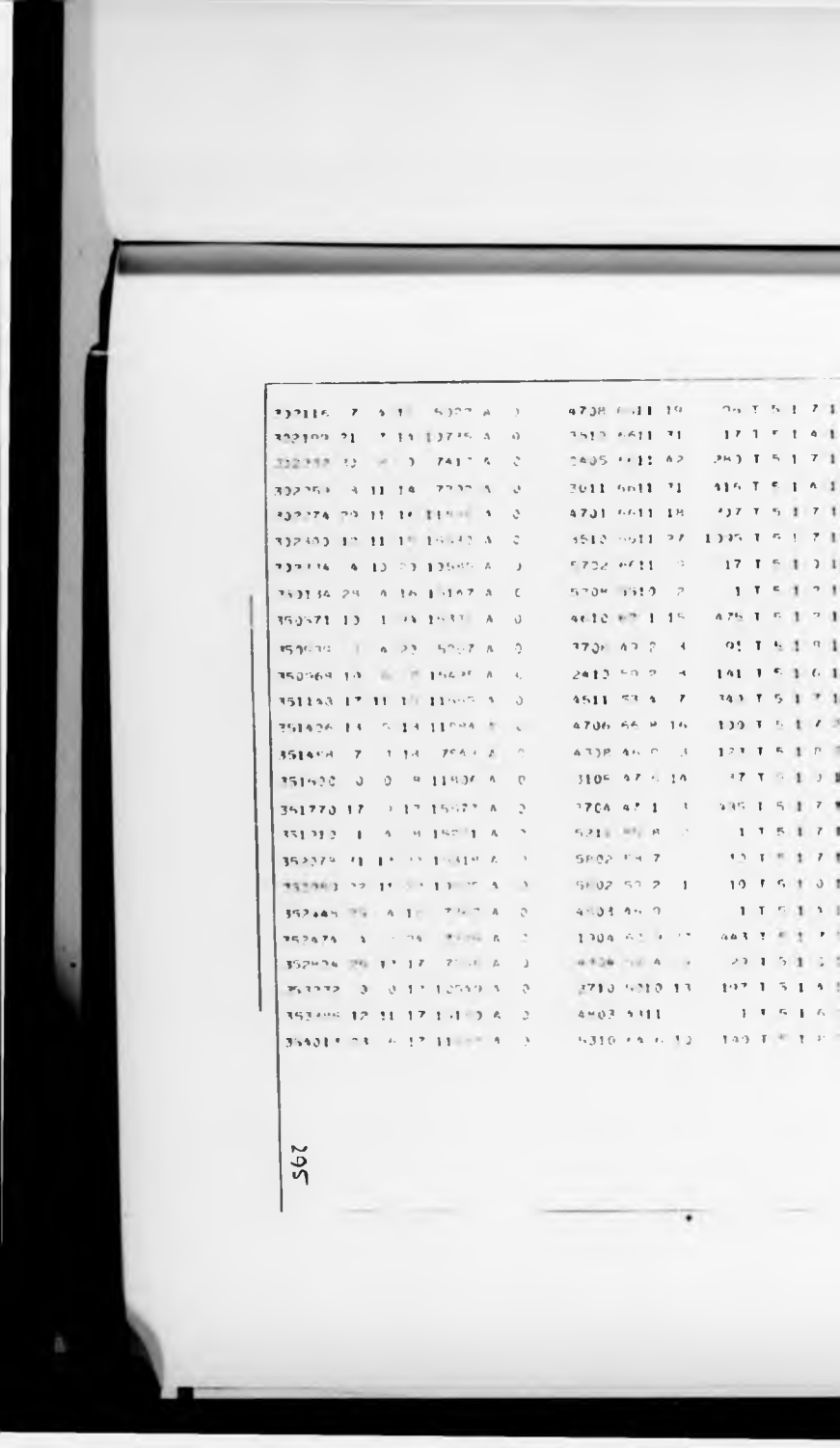

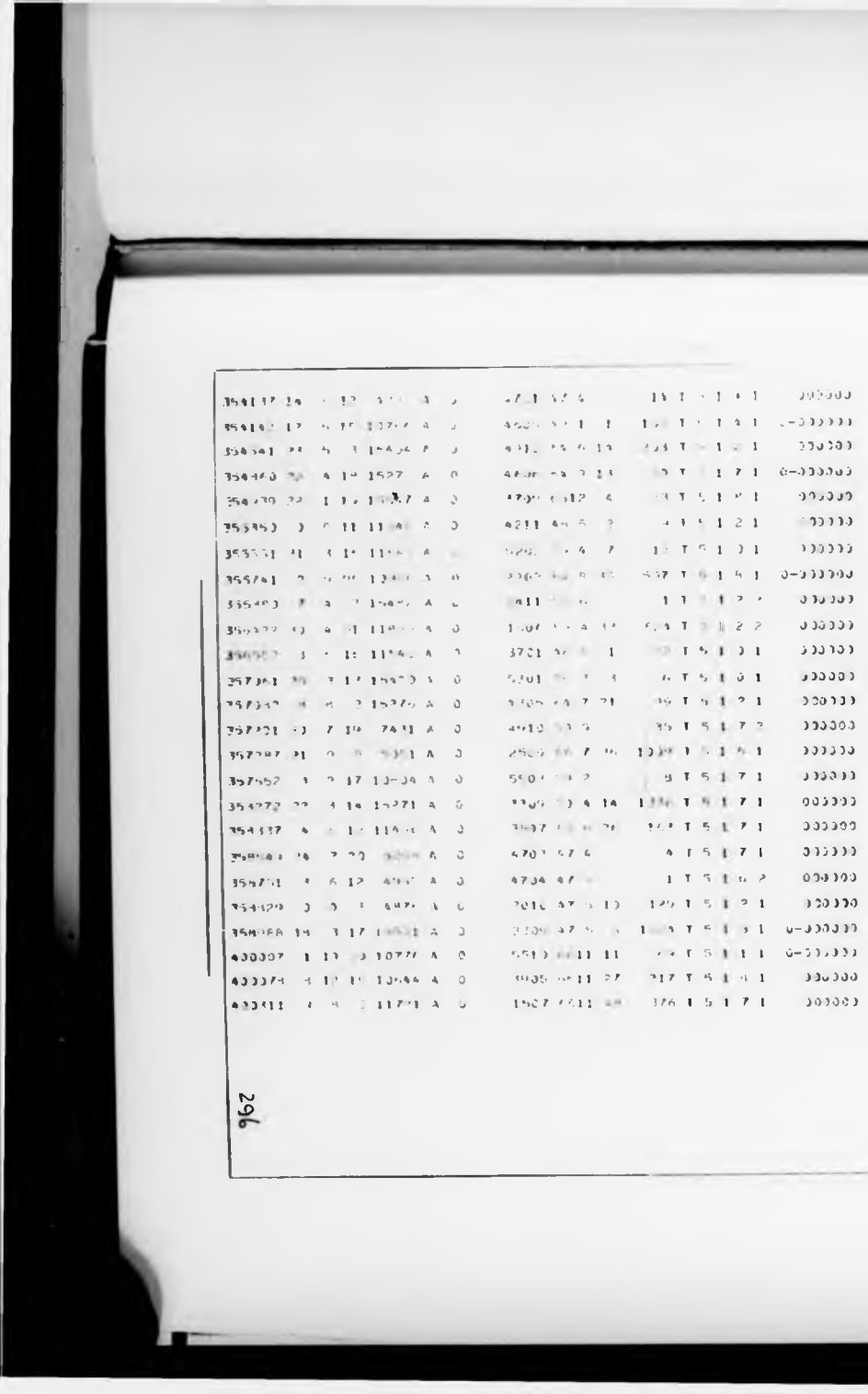

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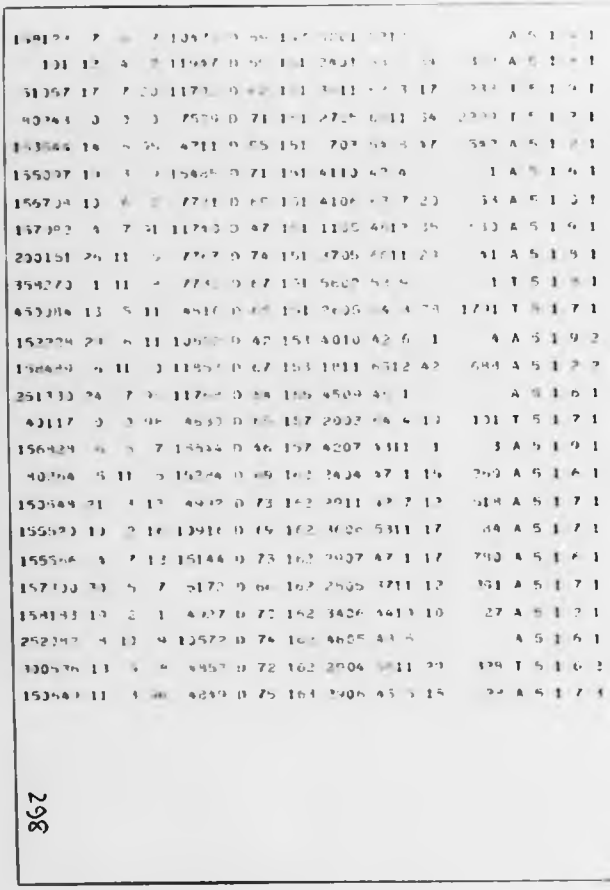

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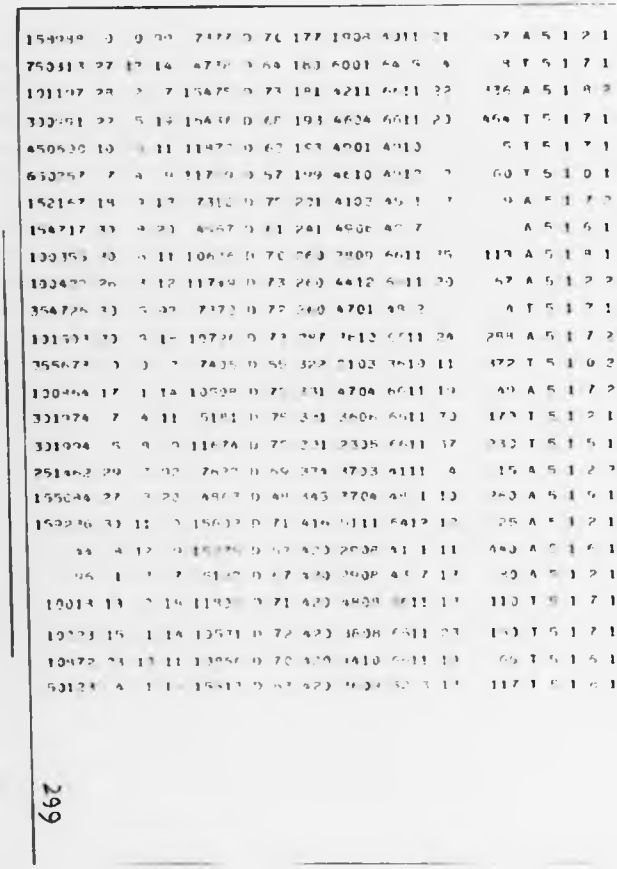

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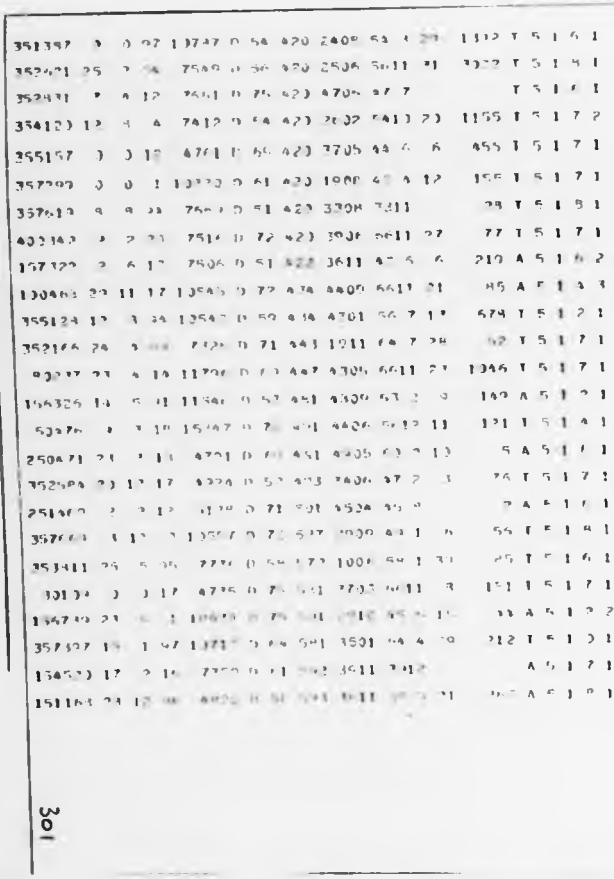

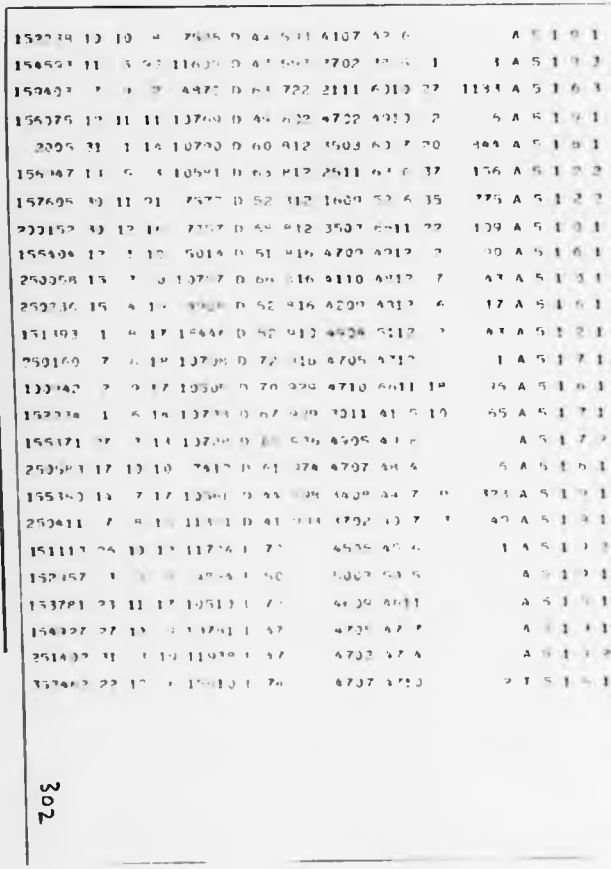

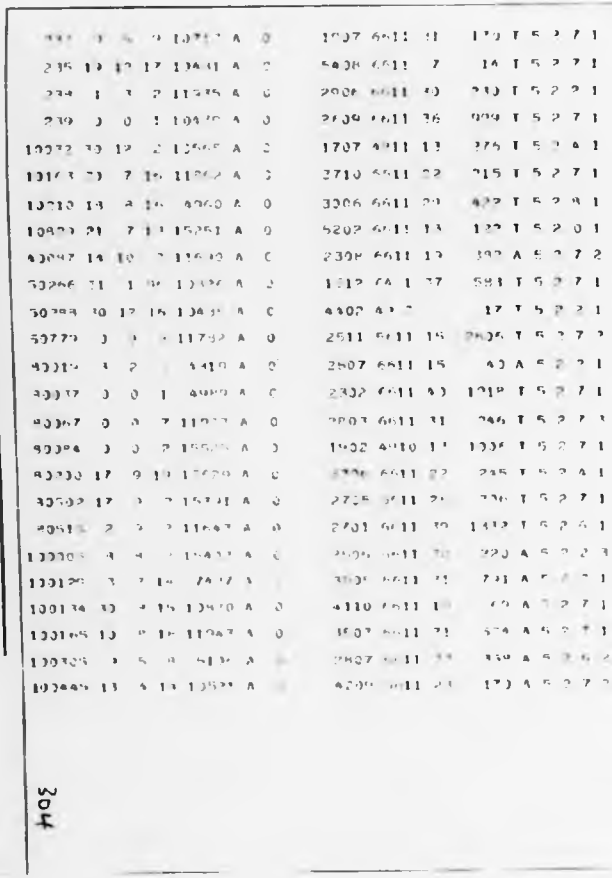

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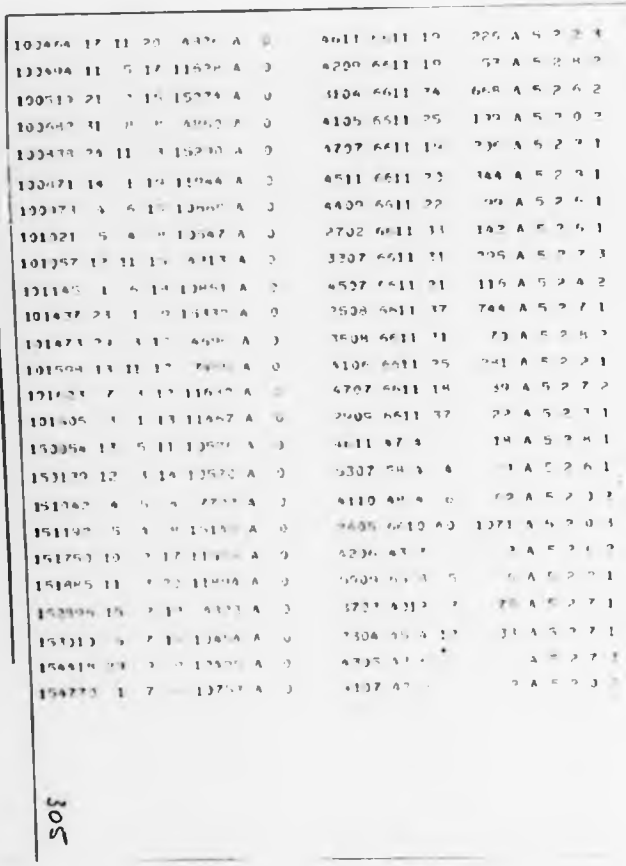

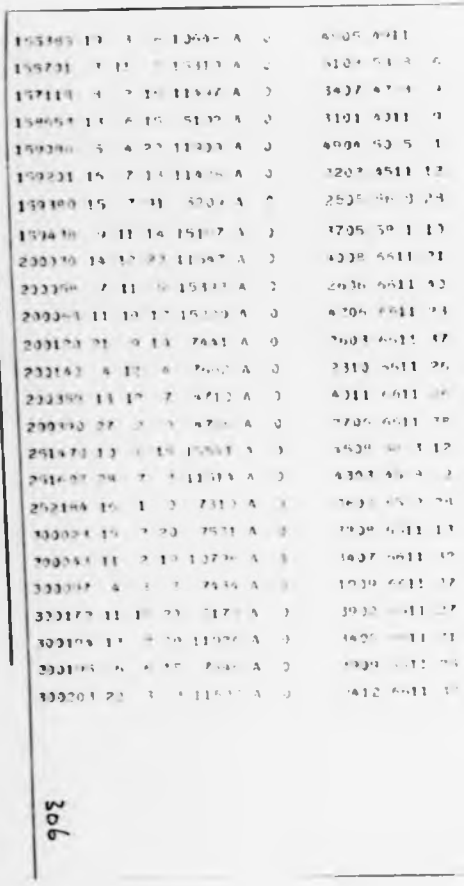

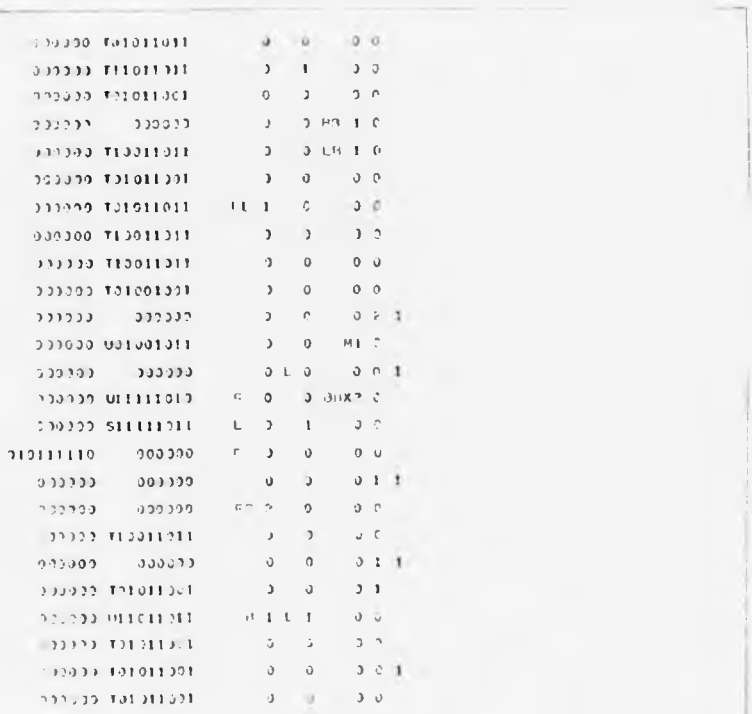

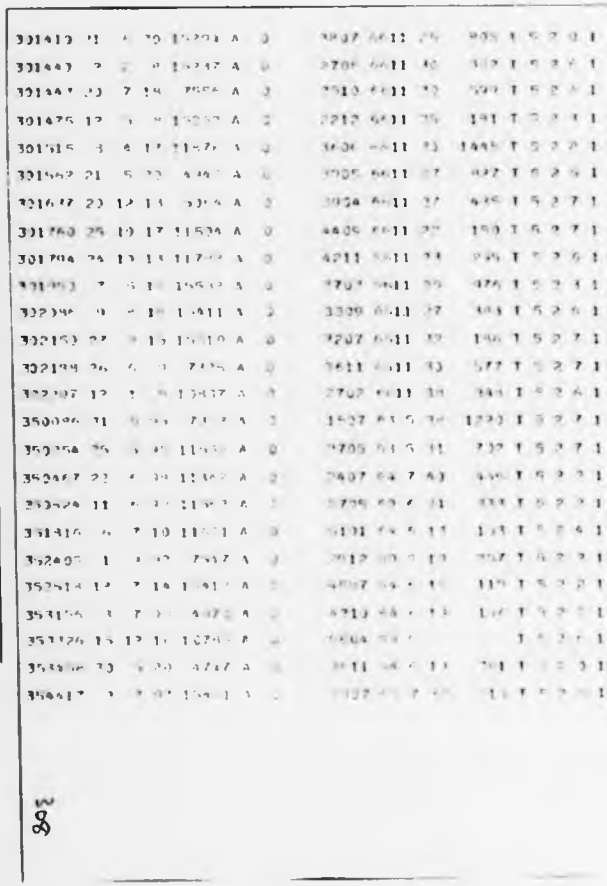

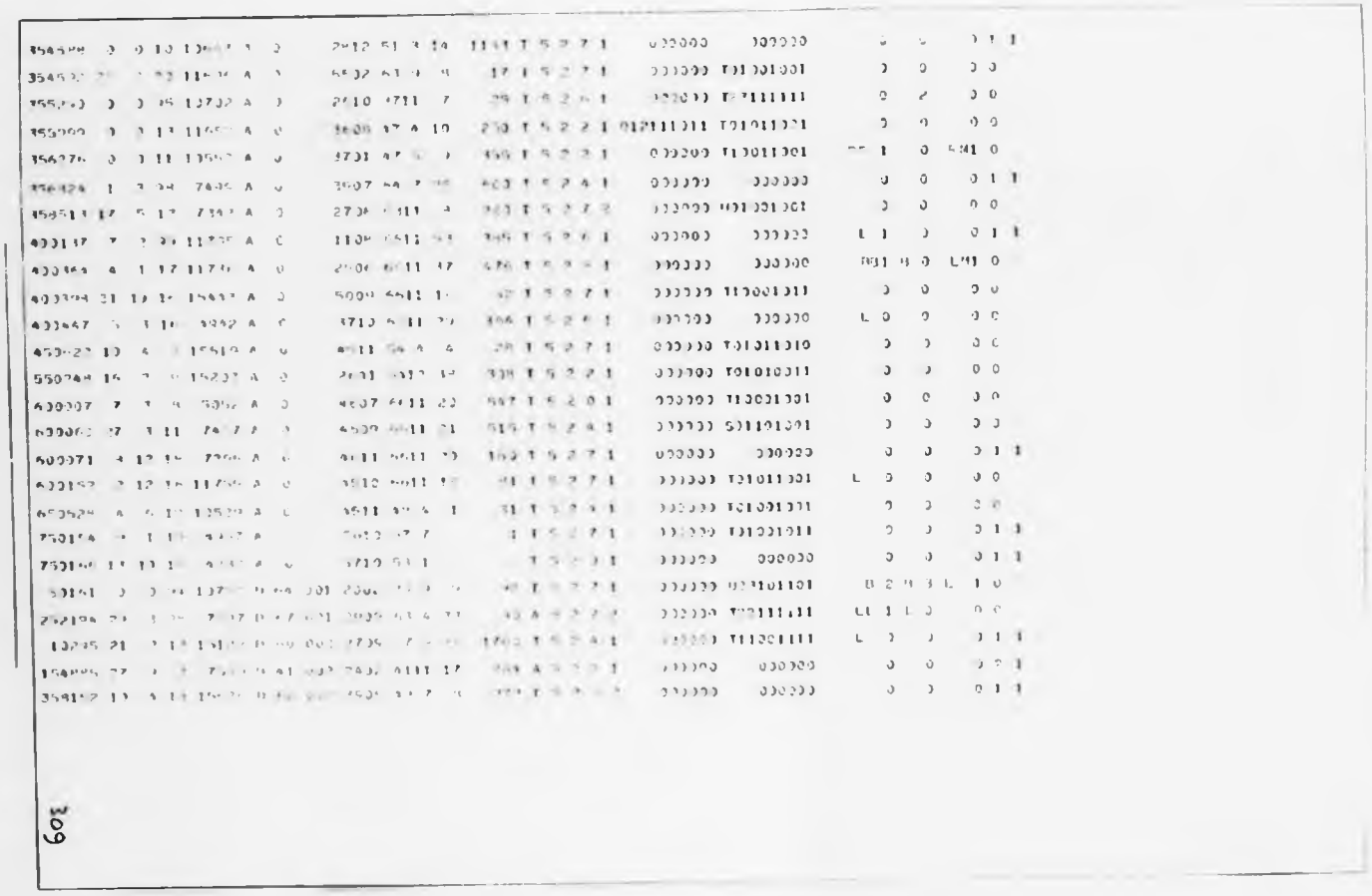

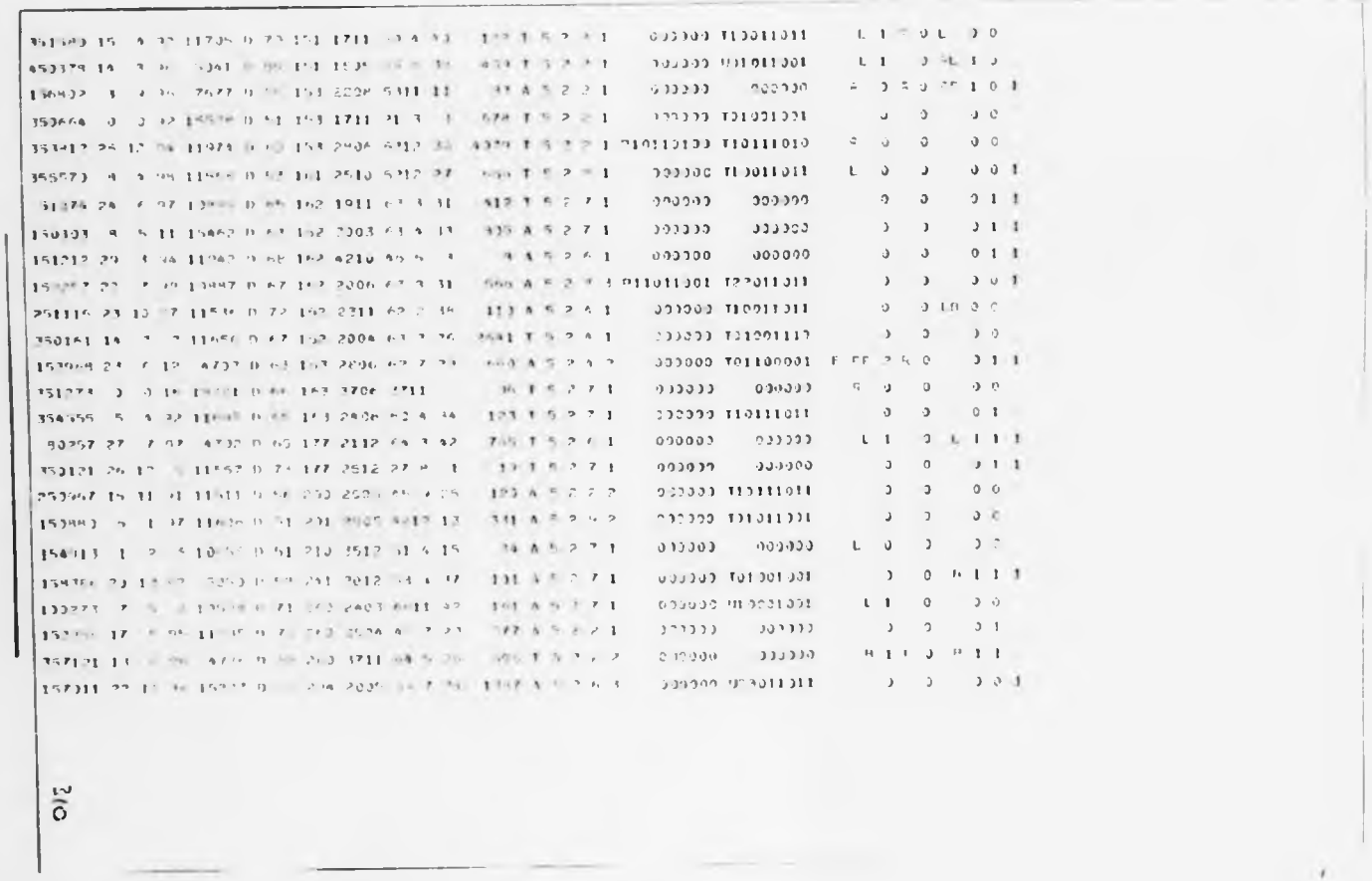

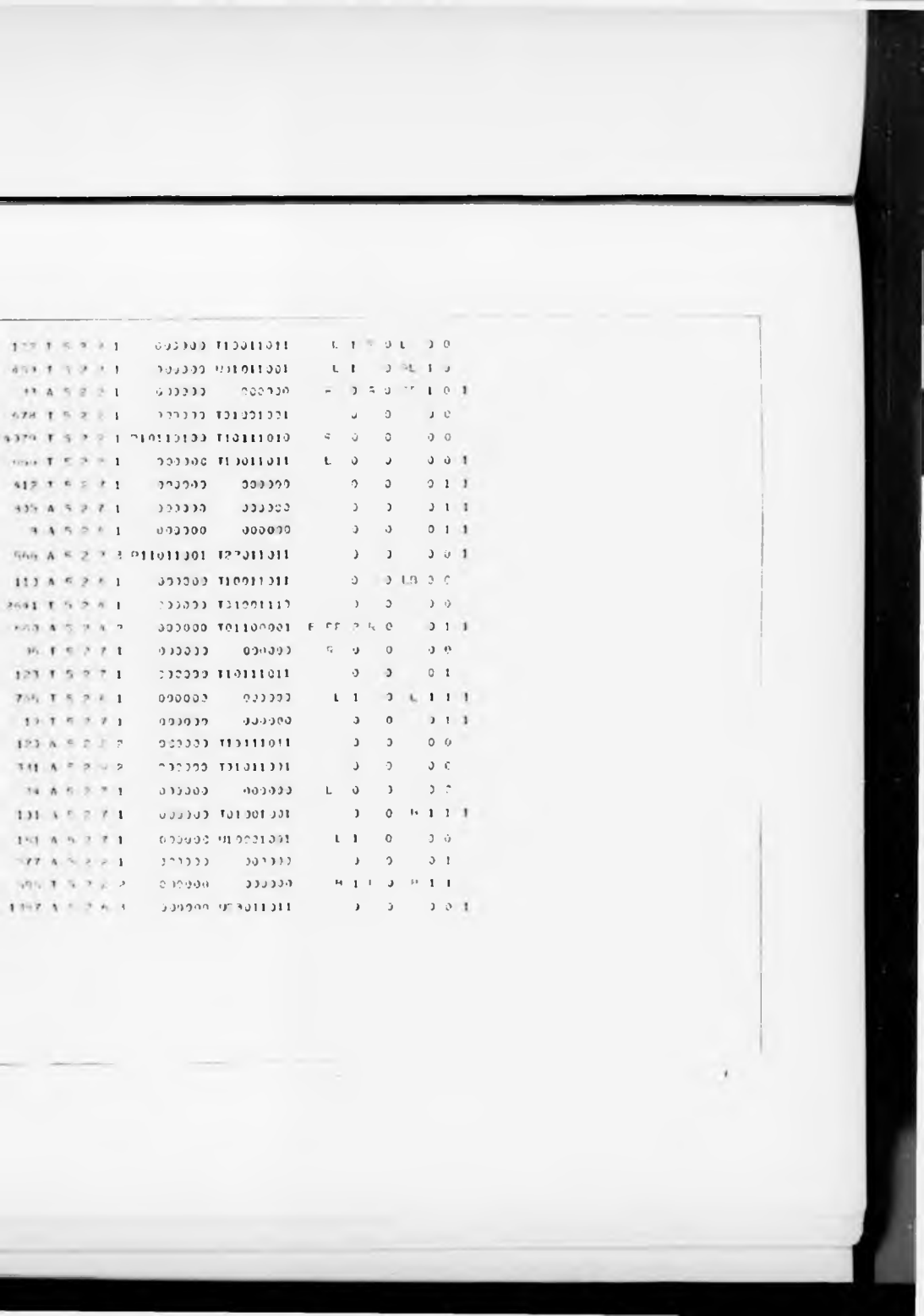

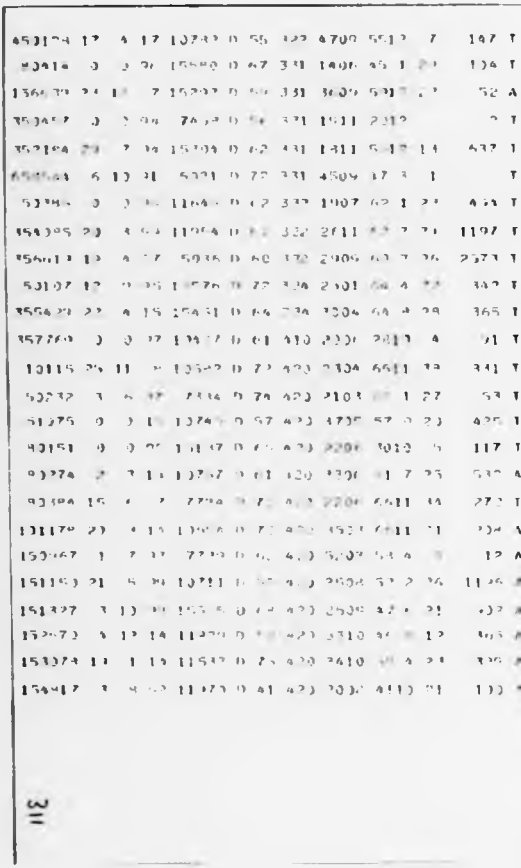

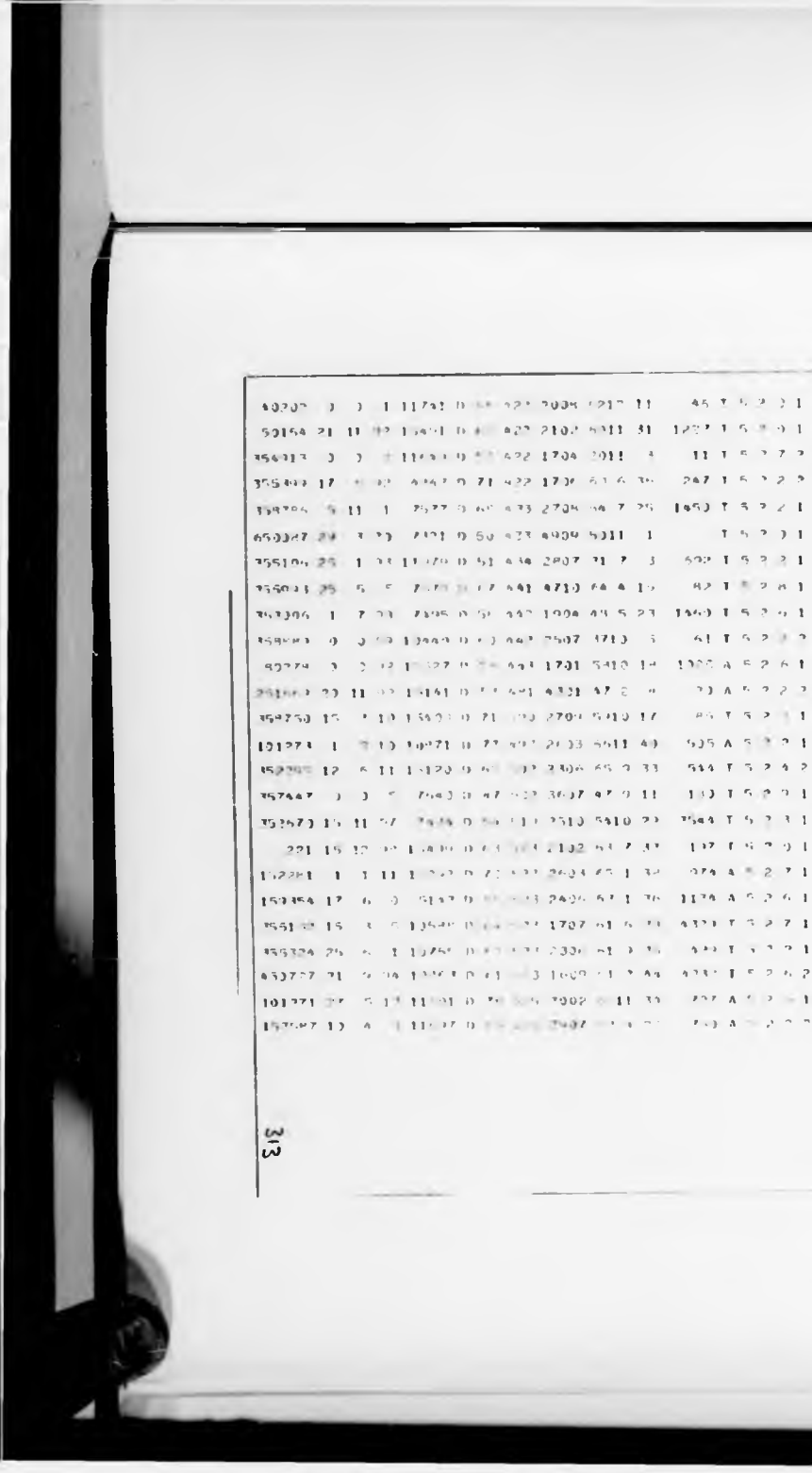

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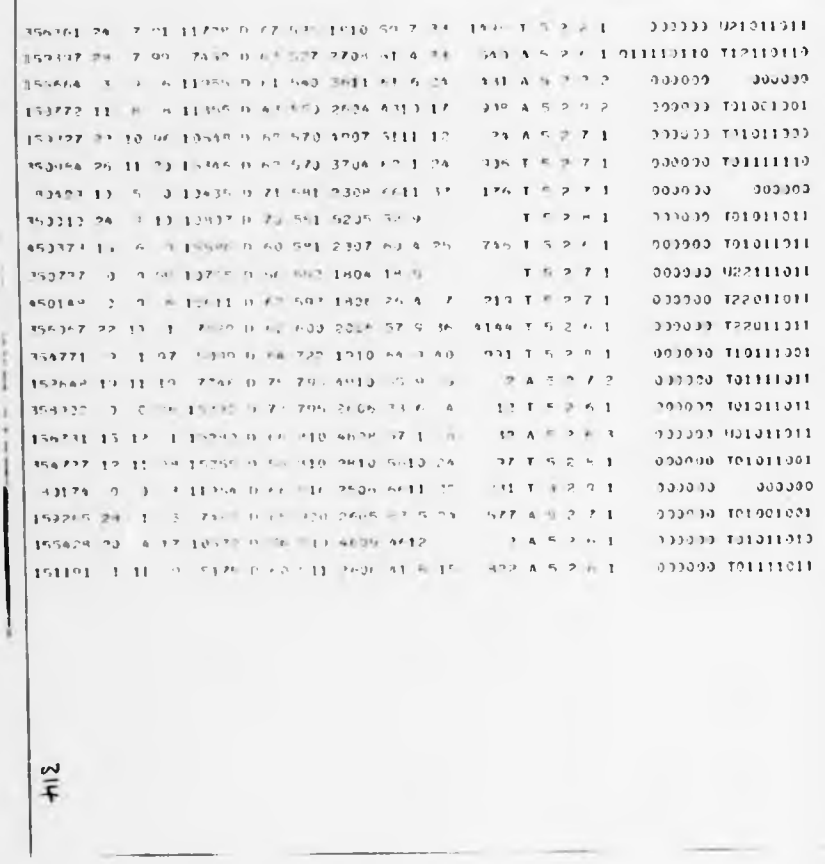

COHORT "B"

Reader 6

"Normals"

pp. 316 - 336

"Less-than-normals"

pp. 337 - 348

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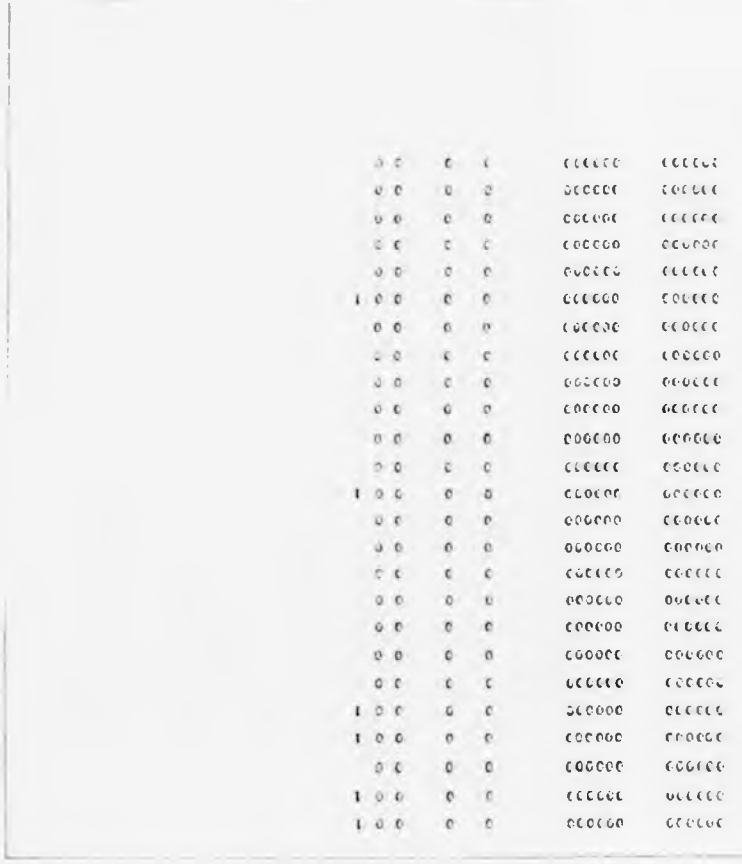

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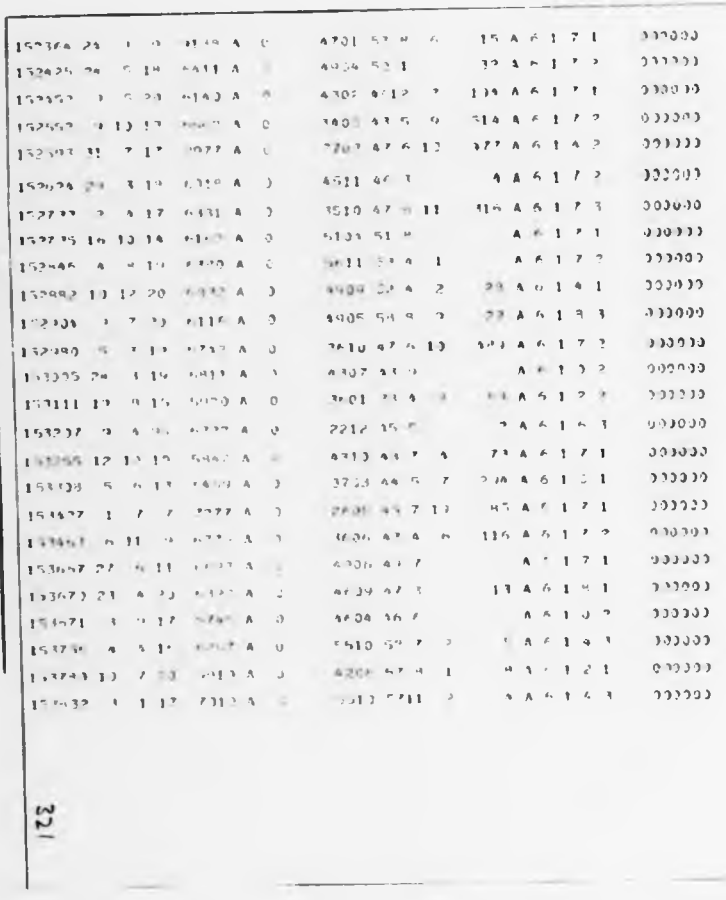

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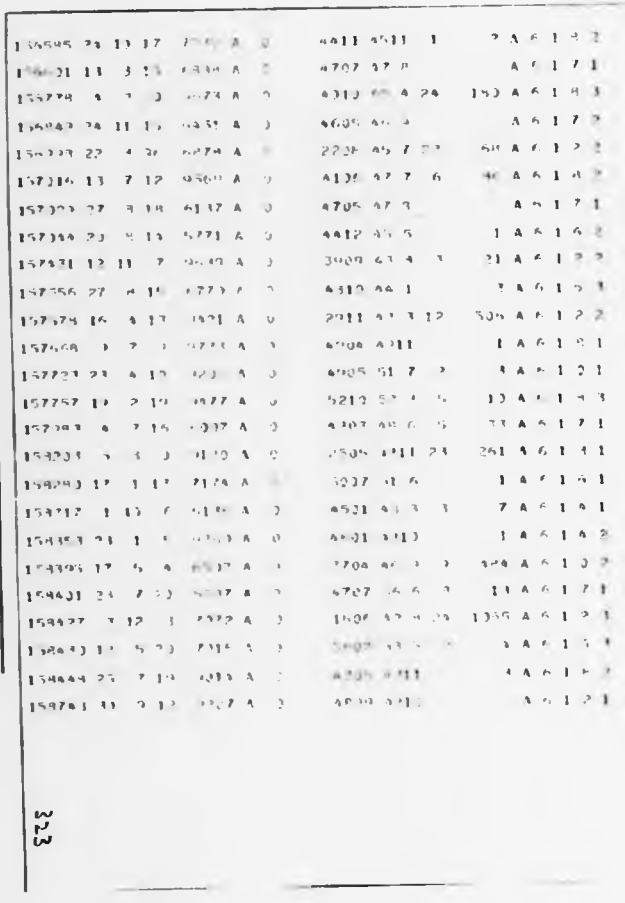

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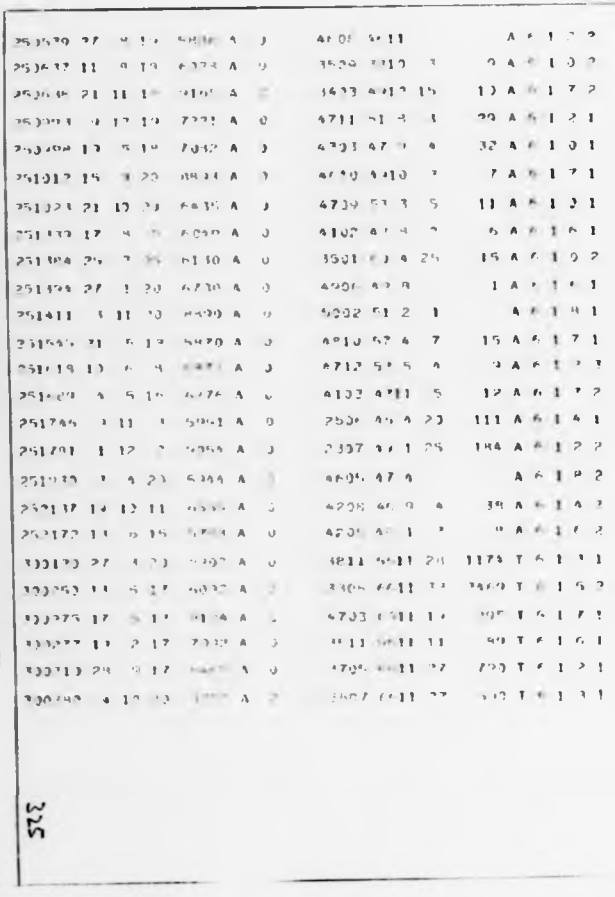

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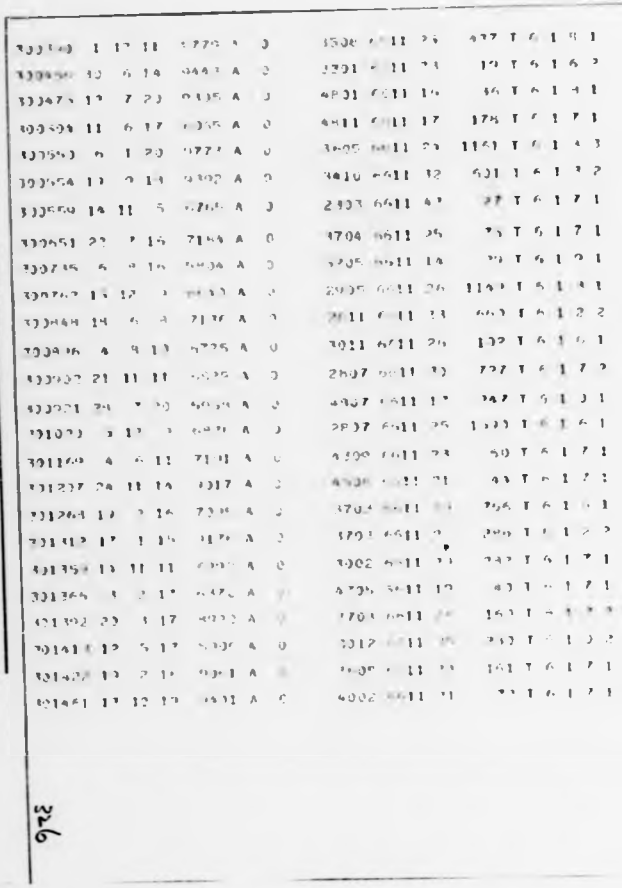

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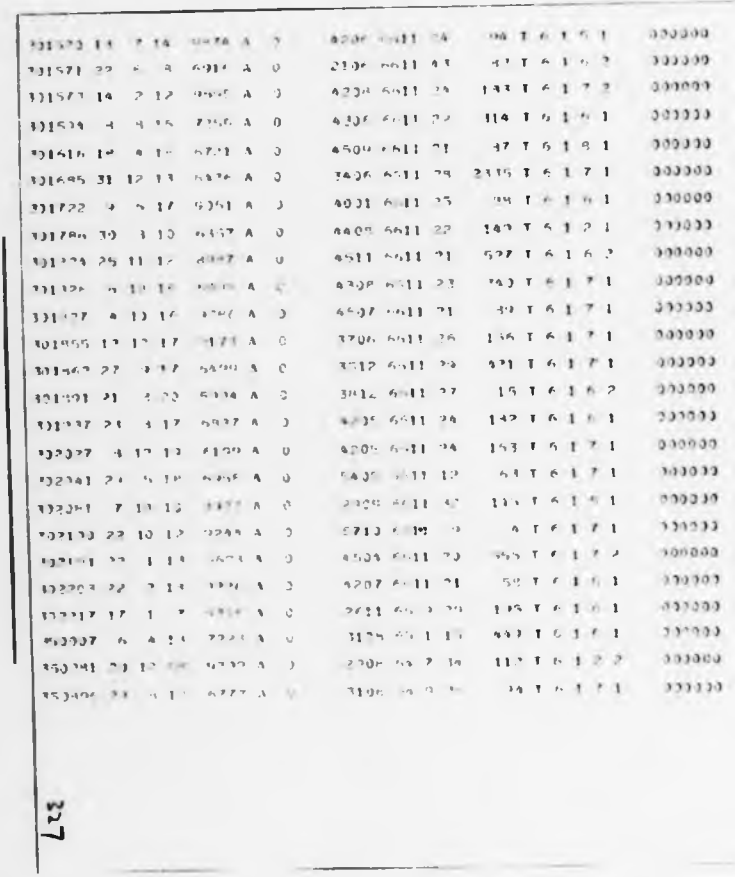

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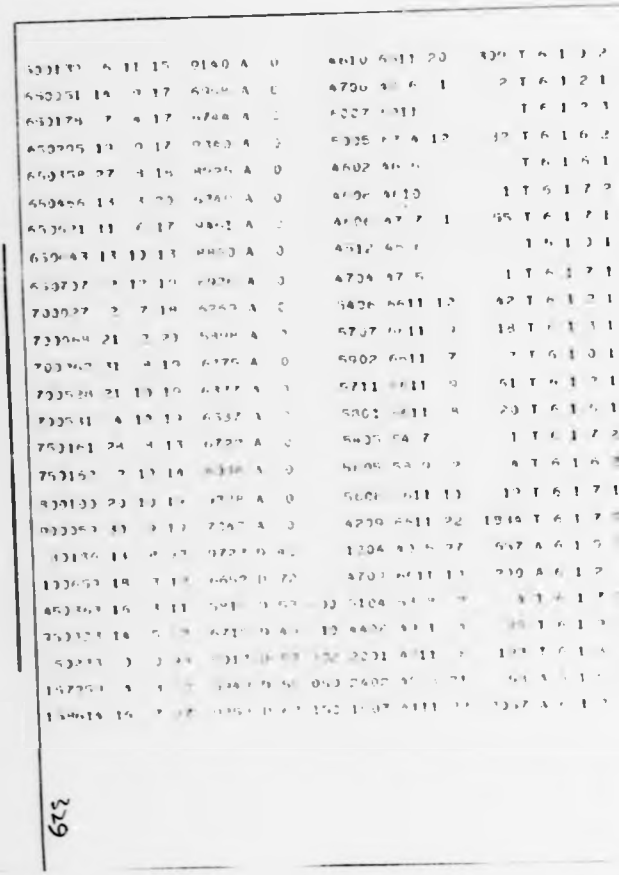

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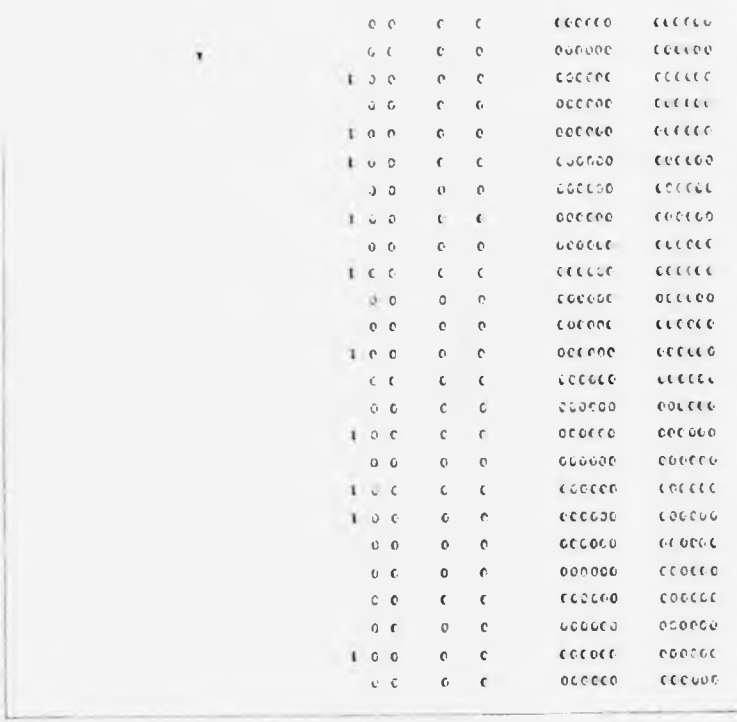

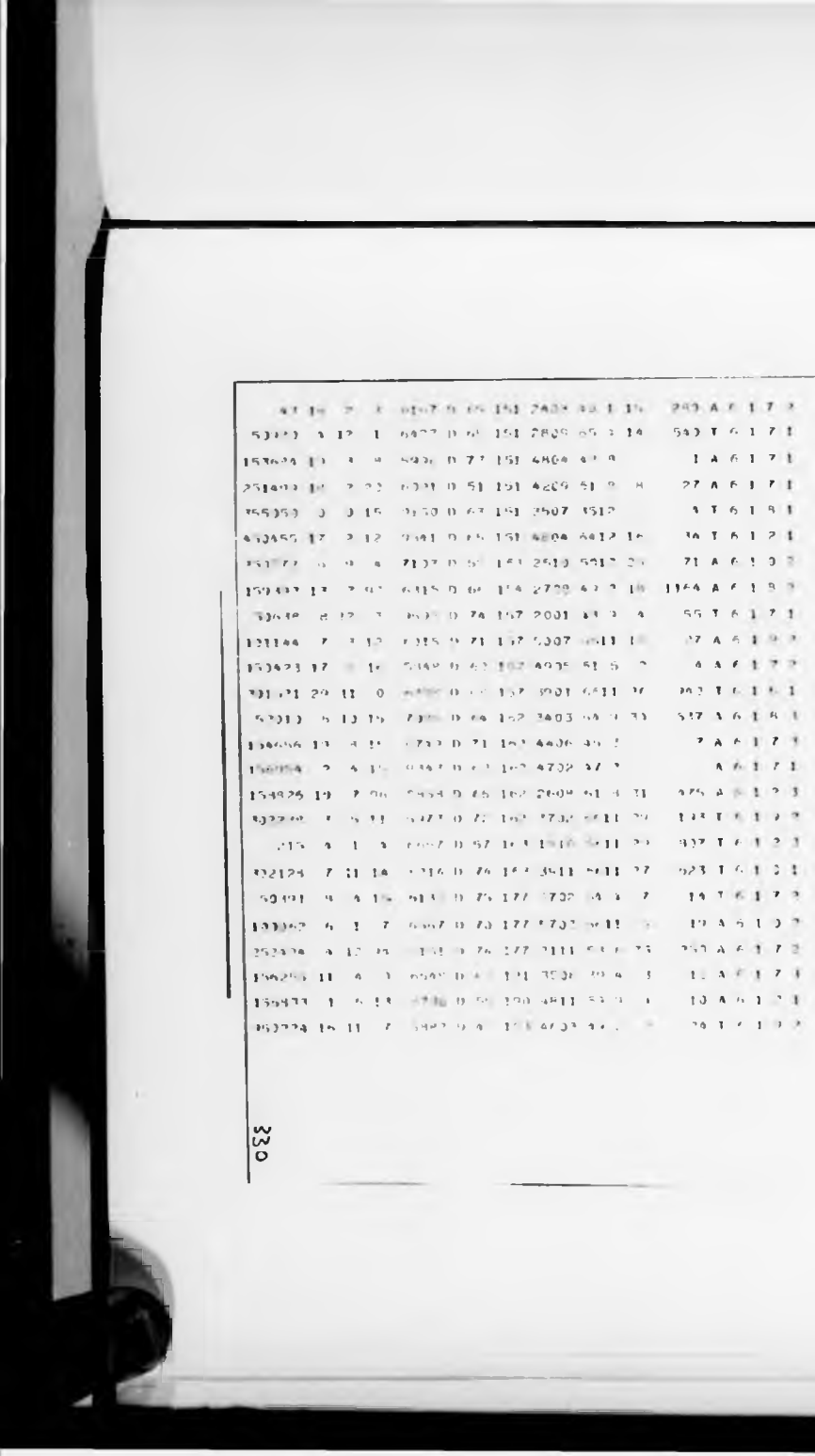

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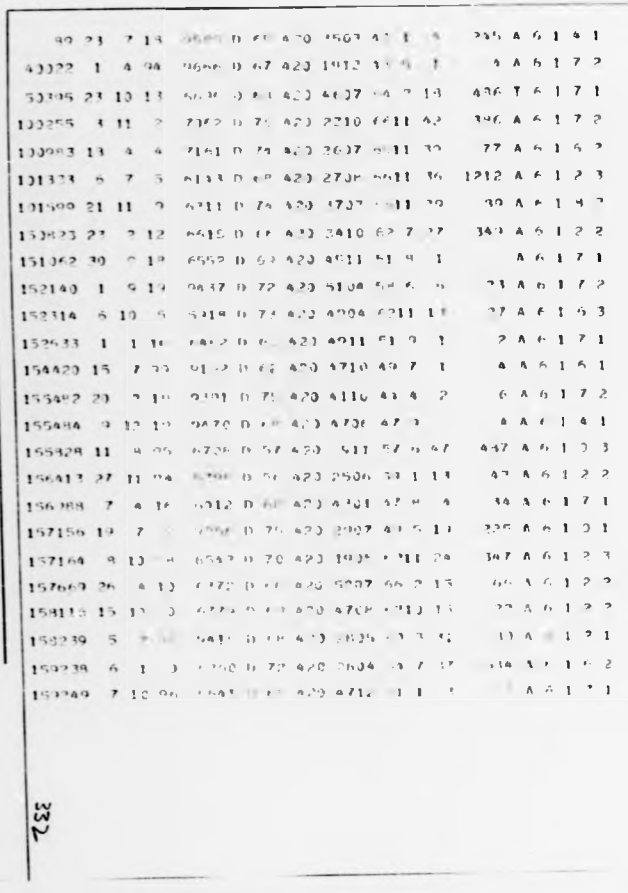

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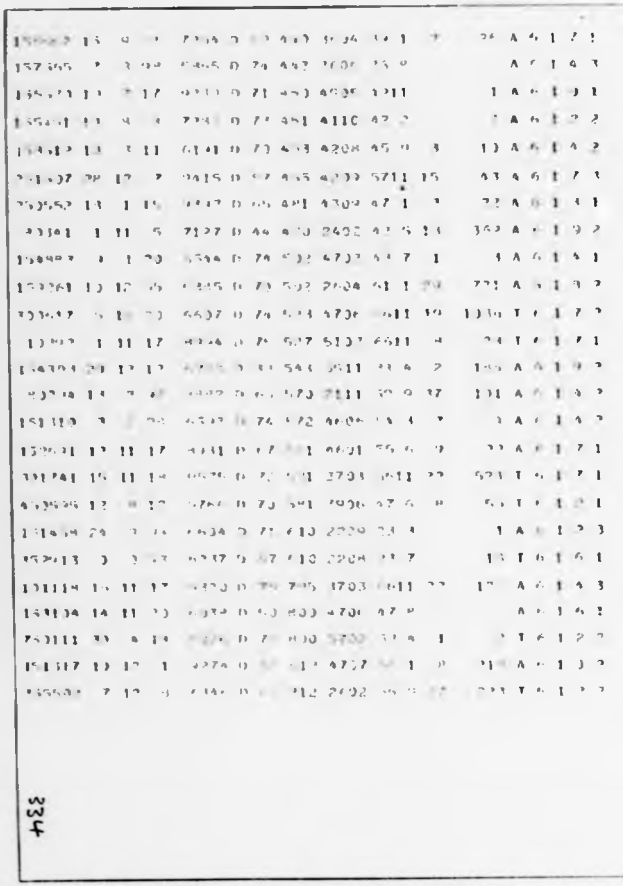

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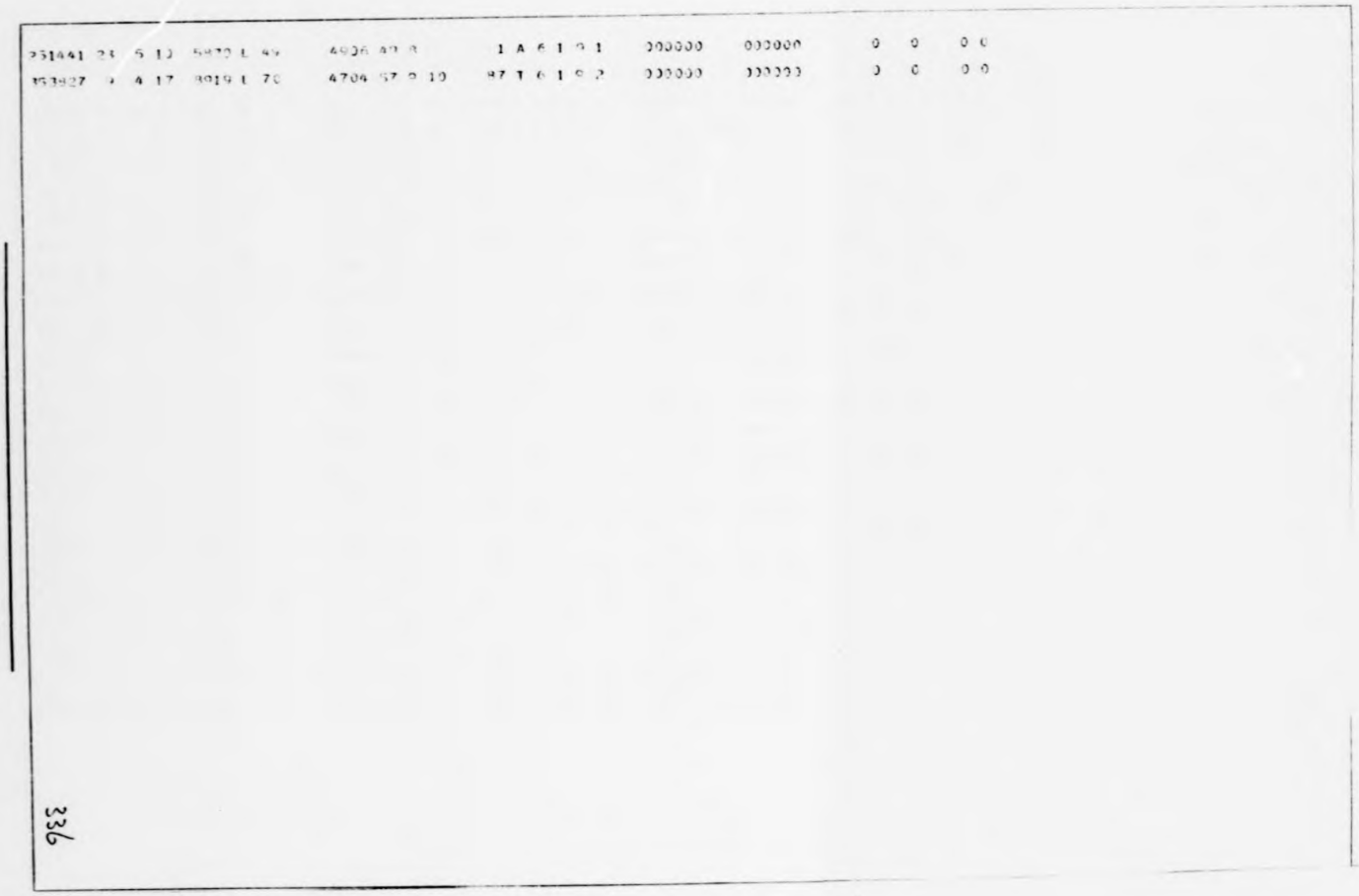
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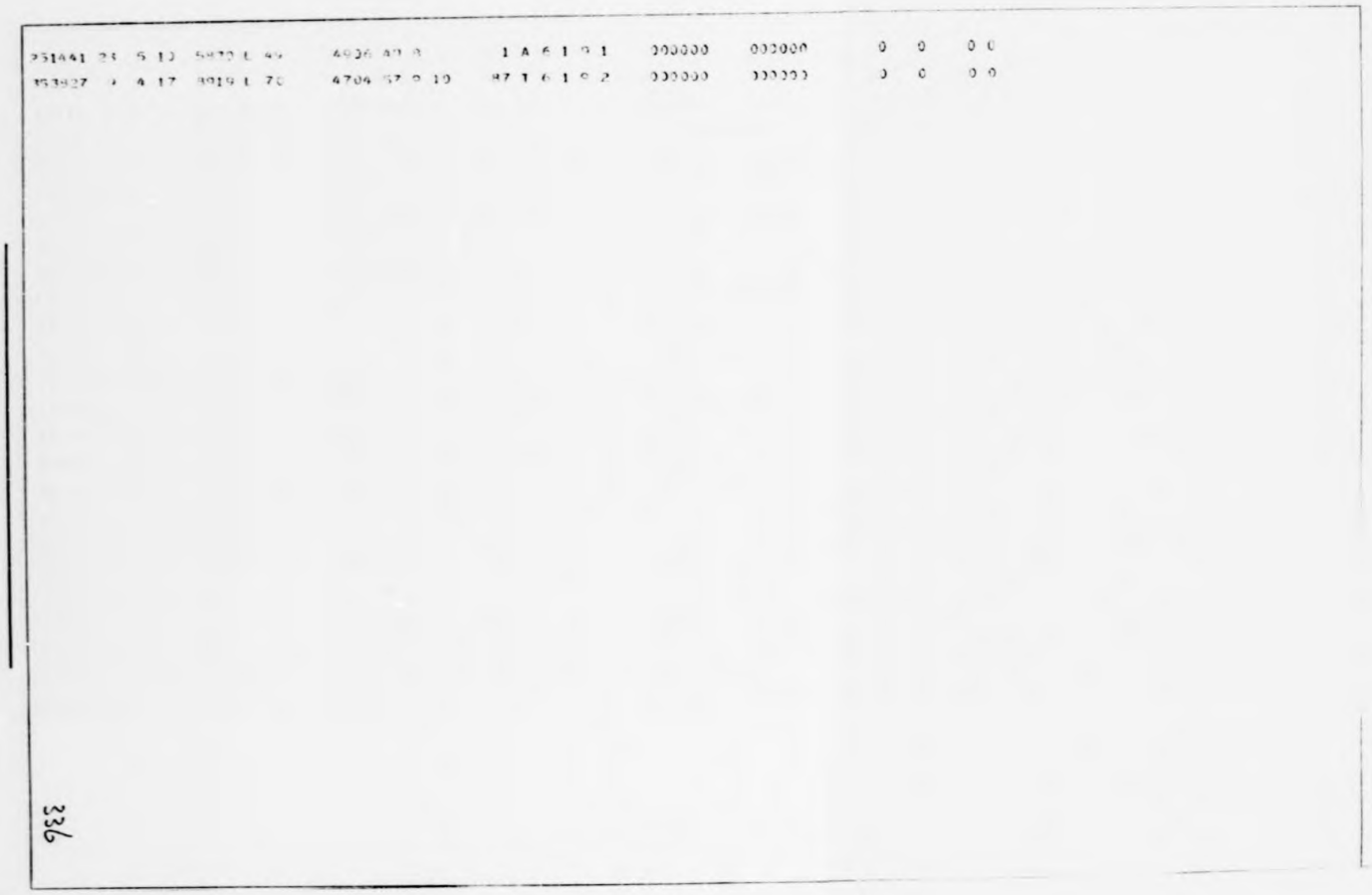
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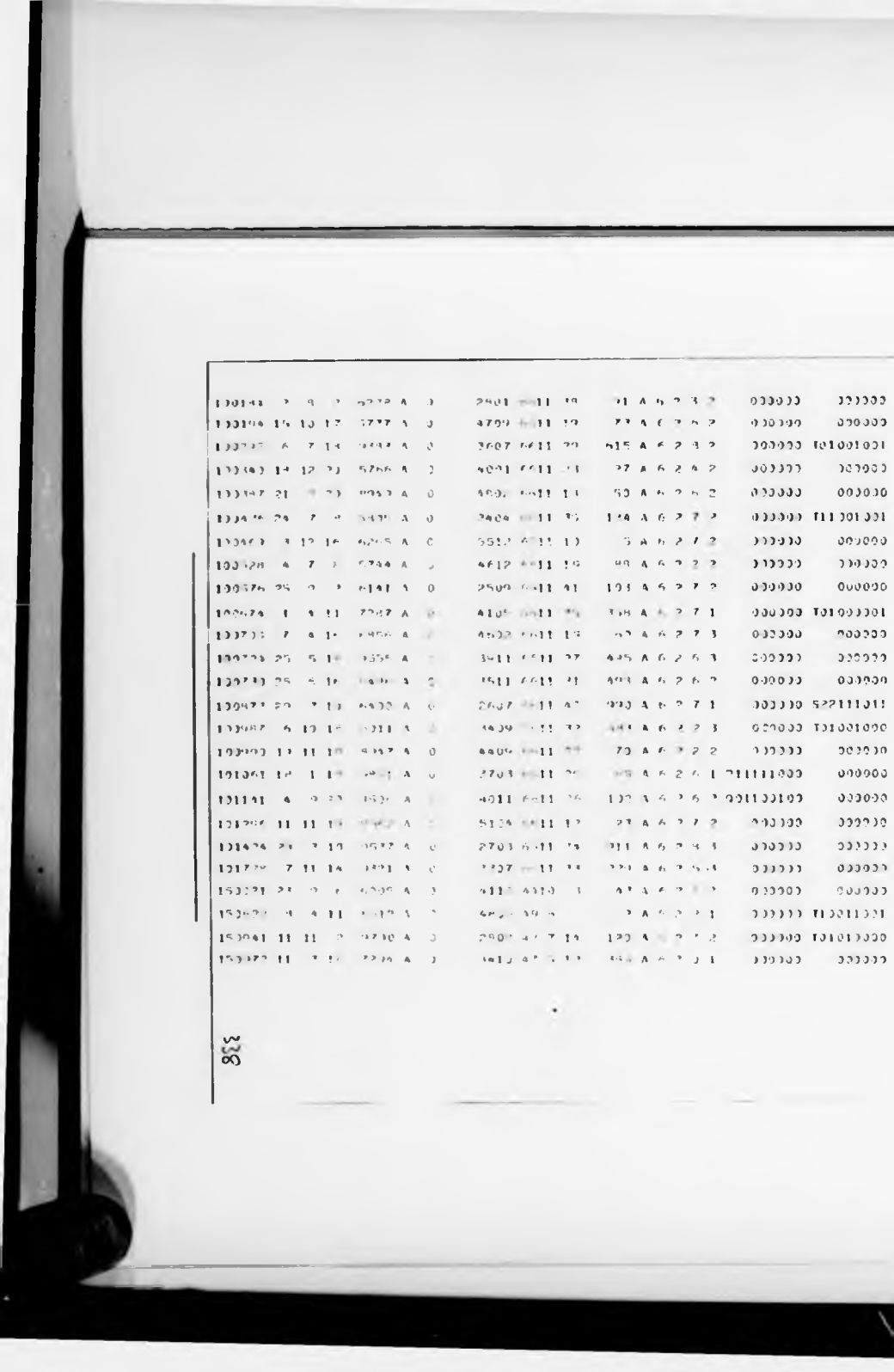

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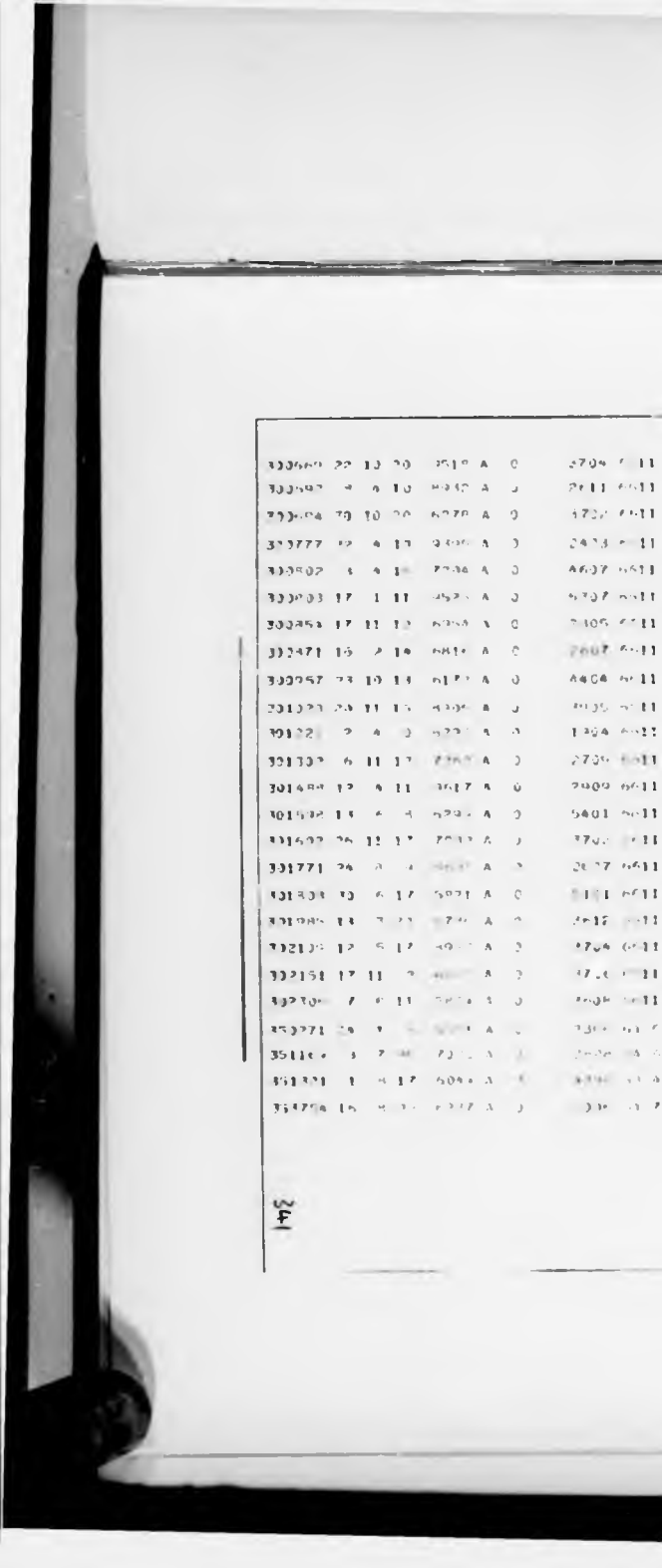

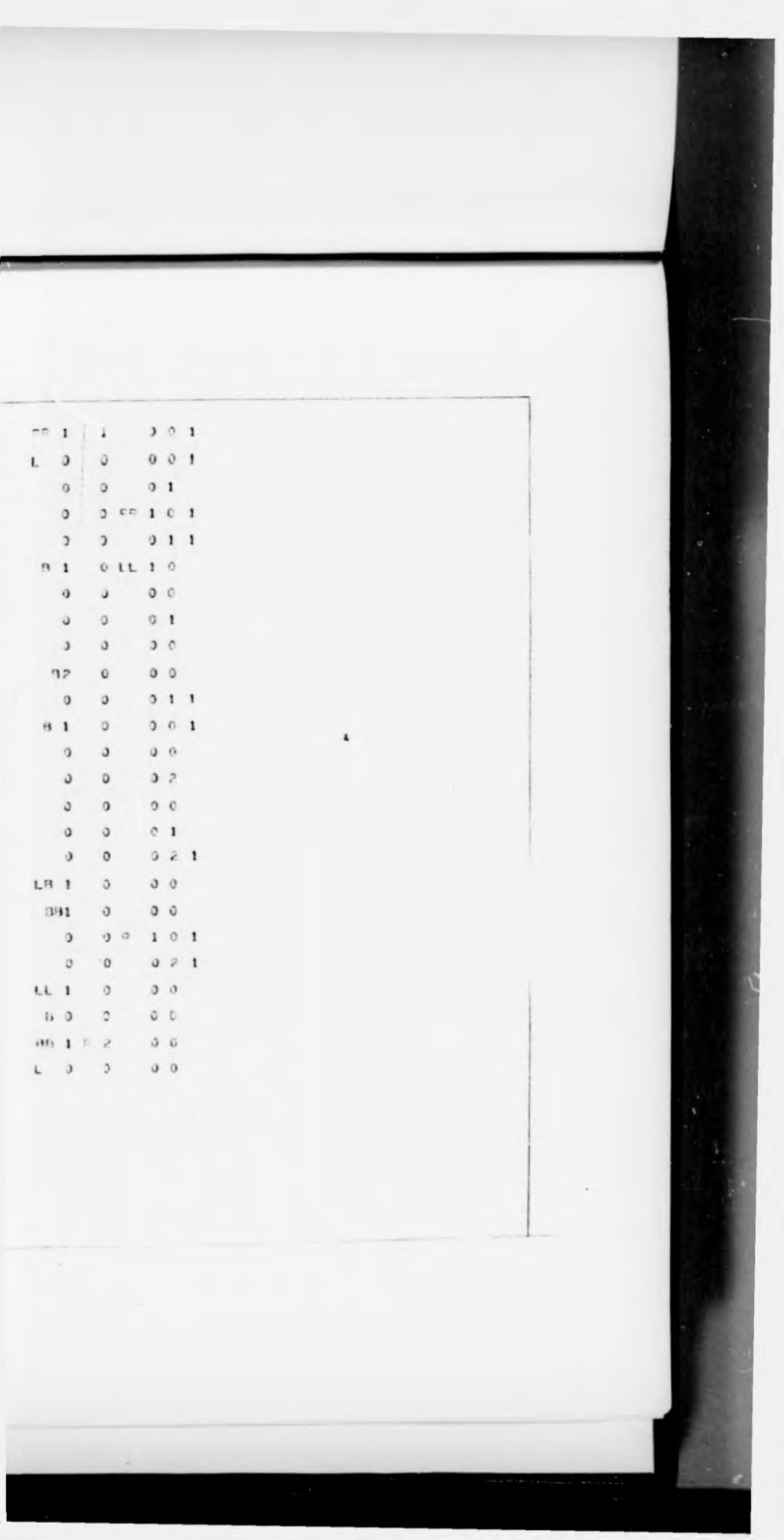

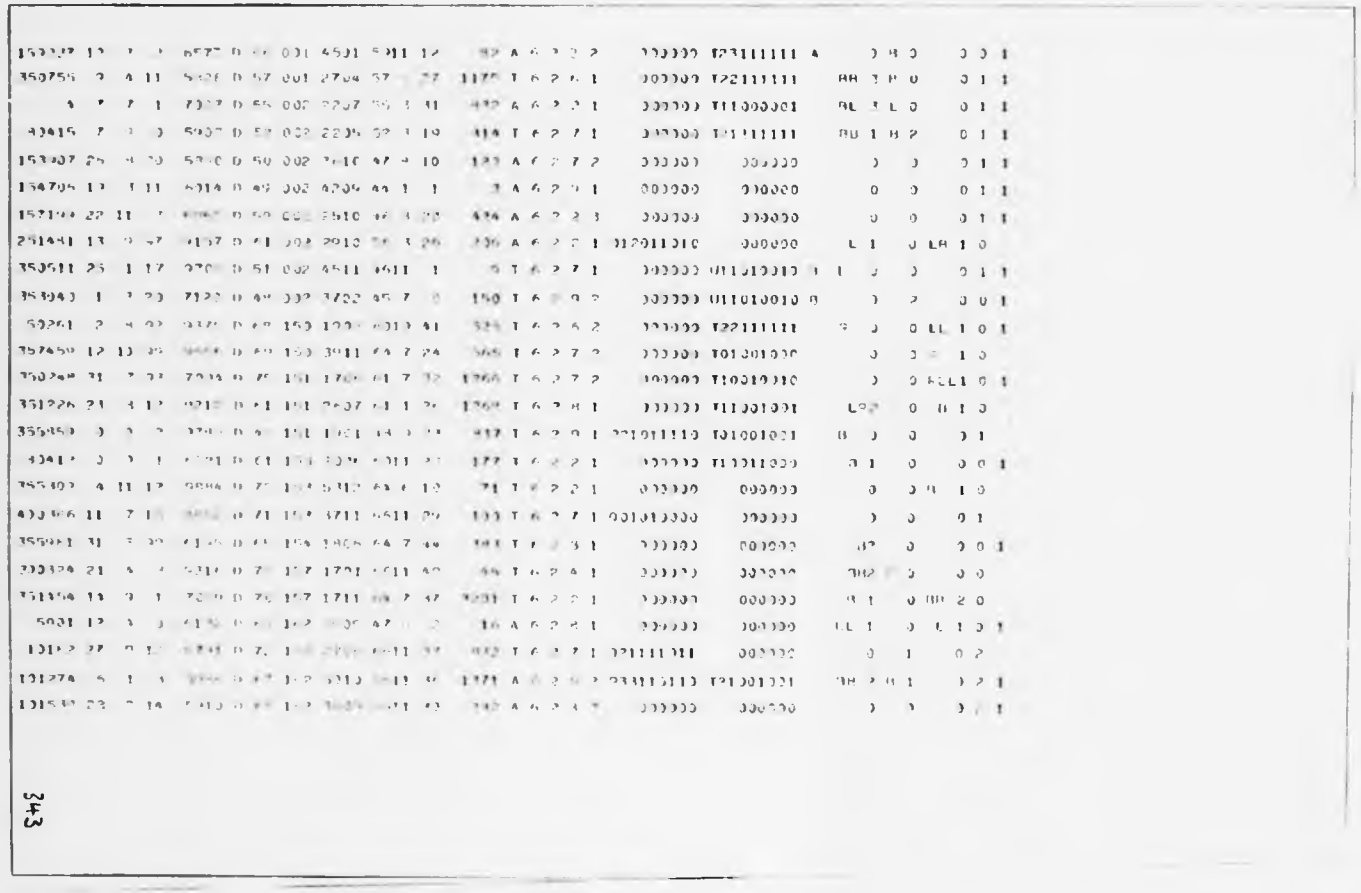

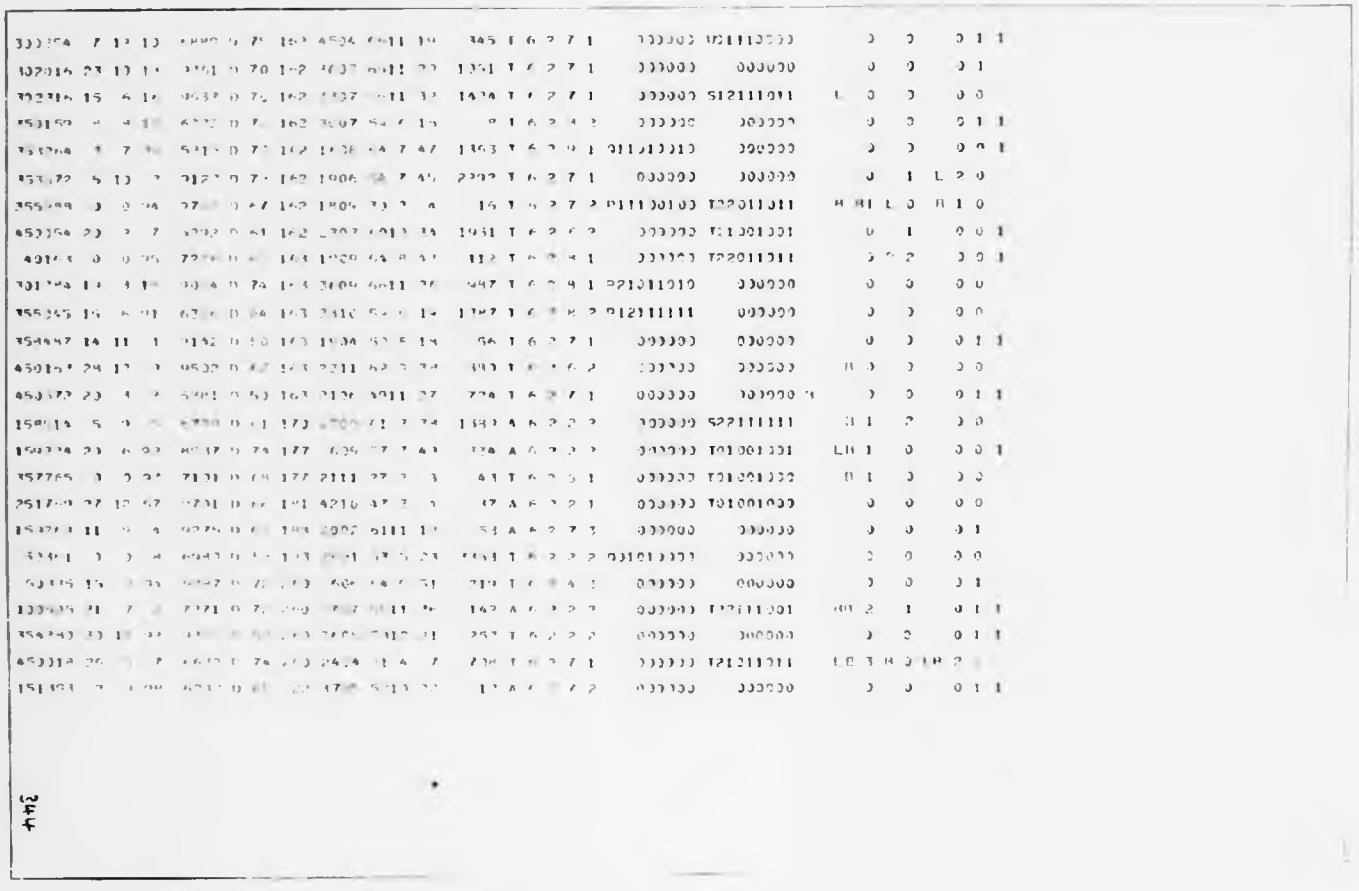

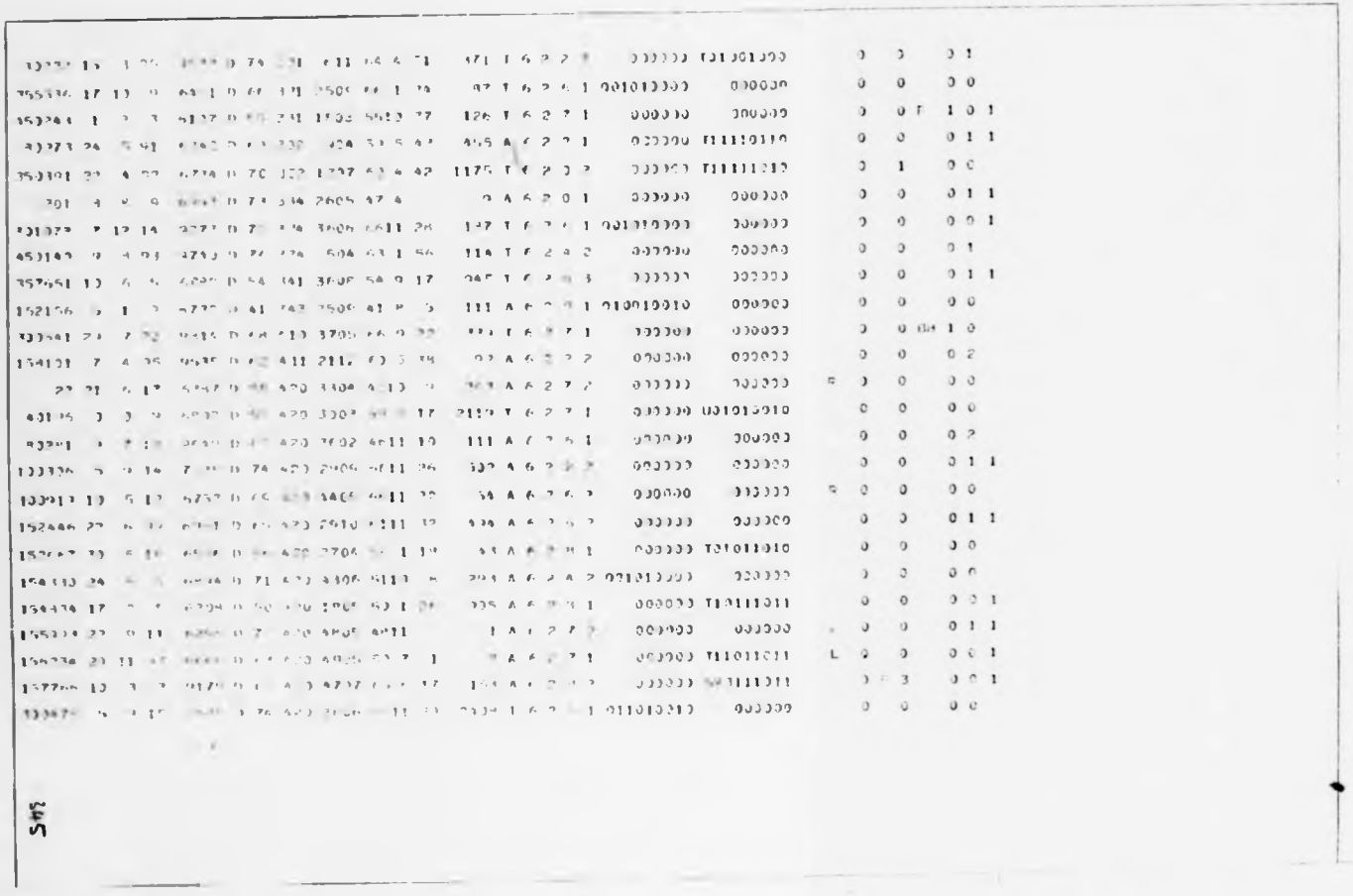

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