M.Sc. (Occupational Medicine) RESEARCH PROJECT

THE SOURCES OF LEAD POLLUTION AND ITS EFFECTS ON CHILDREN LIVING IN THE MINING COMMUNITY OF KABWE, ZAMBIA

by

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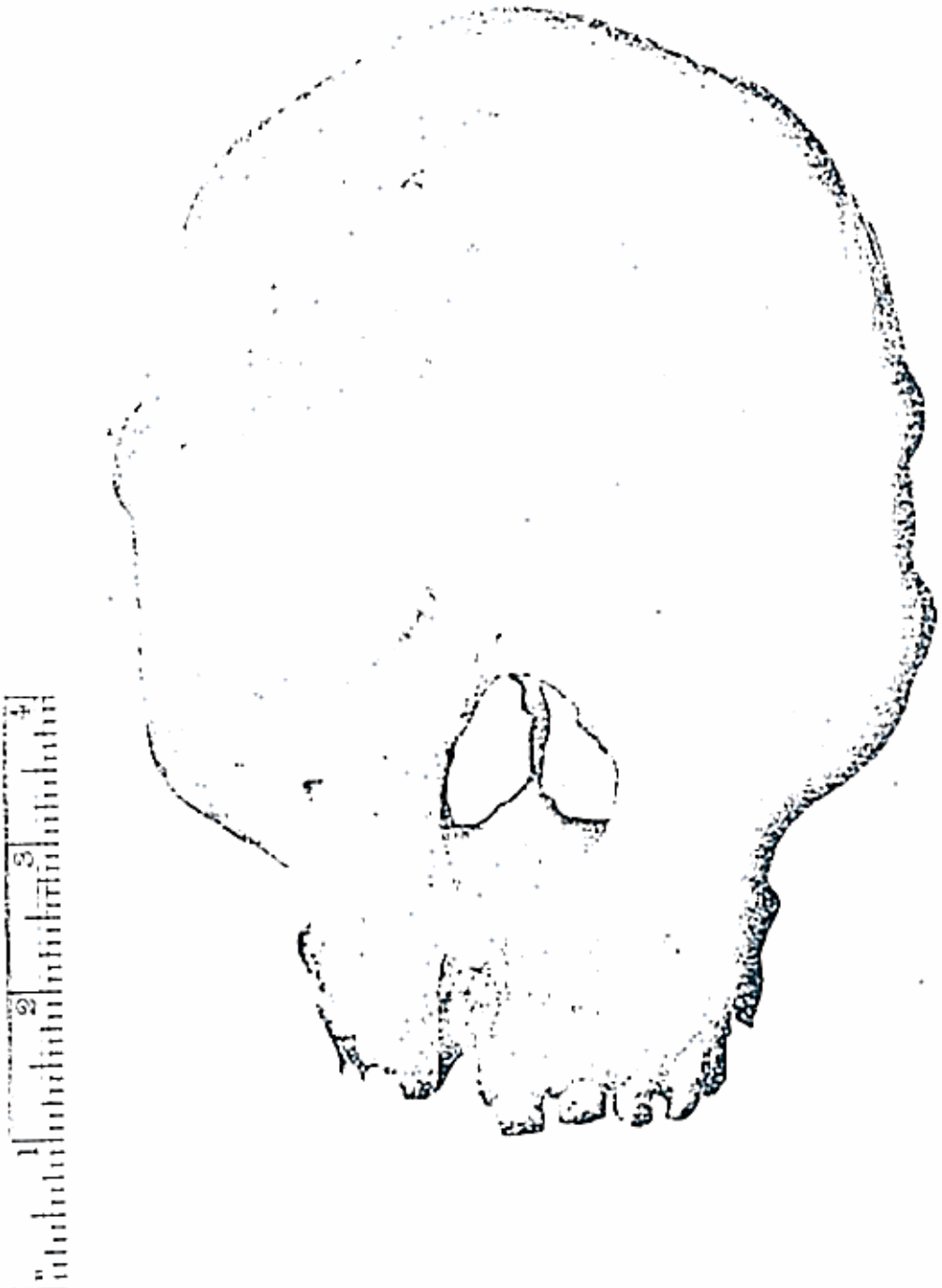

HOMO RHODESIENSIS

(By courtesy of the British Museum, Natural History)

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PREFACE

The demand for lead increased considerably during the industrial revolution, and again during the 20th century with the introduction of new uses for the metal, particularly as an additive to petrol. Despite this increasing usage the incidence of lead poisoning has fallen remarkably since the turn of the century and to-day is an uncommon event in Britain. This has been achieved through research, legislation and improved working conditions. Consequently, attention is now focused on biochemical abnormalities which are precursors of the disease, and it has been possible to examine communities adjacent to industrial plants which might exhibit such biochemical aberrations as a result of industrial pollution.

Zambia passing through its own industrial revolution has benefitted

from the research and experience of developed nations and thereby has achieved comparable monitoring and control of workers health. Similarly, interest has been directed to peripheral industrial communities which attract families from the country hitherto unused to urban society, creating problems of housing, education, especially in the squatter areas, and compounding the difficulties of health control, reminiscent of England in the early 19th century.

This dissertation is concerned with an investigation of four communities in the lead industrial town of Kabwe, Zambia. It appraises the extent, severity and consequences of lead pollution and makes recommendations for its control and treatment.

PURPOSE OF THE ENQUIRY

Recent world wide concern with the hazards of lead pollution and the death of some eight Kabwe children following convulsions and coma directed attention to the probable risks facing the Kabwe communities. It was therefore decided by the writer to initiate a series of investigations to be recorded and summarised individually to cover the following subjects:

1. A survey of the sources of lead pollution in the Kabwe area.
2. Blood lead concentration in children in each of Kabwe's main community districts.
3. The relationship of infants' cord blood lead concentration to that of its mothers.
4. The effects of 'inherited' blood lead concentration on the infant's birth weight and red blood cell values.
5. A radiological survey of lead in bones of young children.

6. The management and treatment of lead encephalopathy

7. Prevention of lead intoxication.

The work covered the period from January 1971 to September 1974.

A BRIEF HISTORY OF ENVIRONMENTAL LEAD INTOXICATION

Lead is widespread throughout the earth's crust in an average concentration of 16 parts per million, but in areas where mining occurs the surface concentration may be several thousand times greater. It is most commonly present as cuboid crystals of the sulphide contained within the ore, galena.

It has a melting point of only 327 °C, such a temperature is easily obtained in a domestic hearth, and owing to its abundance and low melting point, little ingenuity would be required to smelt the ore. Not surprisingly therefore, lead has been in the service of man since ancient times.

Natural lead compounds are variably soluble and can be taken up by plants, and thereafter ingested and absorbed by animals and man. Lead may enter the atmosphere naturally through

volcanic activity and weathering of mineral deposits, and recycled to earth, after being dissolved in rain and snow.

PRE HISTORY

In 1921 parts of a skeleton of Homo Rhodesiensis was discovered in a cave during mining excavations at Broken Hill Lead Mine, Zambia. The skeleton comprised almost a complete skull, portions of limb bones and pelvis; all were encrusted with lead and zinc ore. They were believed to be about 40,000 years old, although a more recent estimate is 100,000 years (LE GROS CLARKE 1970).

Homo Rhodesiensis used stone implements and was familiar with fire, but had not reached the sophistication necessary for purposeful smelting of lead ore. A spectroscopic analysis of the skeleton has revealed a high lead content (OAKLEY 1930) which leads one to speculate on the probable effects of lead

on the health of primitive man who lived in the environs of Broken Hill Mine.

ANTIQUITY

Lead was used in the ancient civilizations of India and China. Later, when most of Europe was still in the bronze age, the use of lead in the developed civilization of Asia Minor, spread to Egypt and eventually to Greece and Rome.

In Asia Minor the Hittites mined galena primarily to obtain silver; the contained lead sulphide was converted to the oxide and removed by a process of cuppellation (DERRY 1960). The Egyptians used lead for making statuettes, dishes, and fishing net weights, and galena as a cosmetic.

Inevitably workers were affected by the fumes generated in the early industrial processes involved, but it was Hippocrates in

460 BC who was the first to record toxic manifestations due to lead, when he described abdominal colic occurring in a miner (HUNTER 1962). Later, Nikander, a Greek poet of the 2nd century BC vividly expressed in verse the clinical features of colic, pallor, constipation paralysis and occular disturbances resulting from plumbism (MAJOR 1939).

The Romans mined lead in various parts of Europe including Spain, Britain, Germany and it assumed a very important role in their technology. They used it in a decorative capacity but much more significantly for the Roman aristocracy they also used it to make water pipes and to line bronze cooking pots and cups; whilst the poor used earthenware.

Wine merchants added to their wines a syrup made from

unfermented grape juice boiled down in lead lined vessels. Some recipes included the addition of lead to sweeten food prepared in bronze pots which otherwise had a bitter taste (TANNAHILL 1968).

According to Suetonius the decline of the Roman empire was the result of apathy and gluttony. Gilfillen has developed the hypothesis that the decline was due to lead poisoning amongst the aristocacy which was instrumental in the decay of Roman culture and eventually of the empire. What Seutonius described as apathy might well have been the effects of lead intoxication.

The first known description of an epidemic of clinical lead poisoning was given by Paul of Aegina 629-690. It began in Roman territory and caused colic, epilepsy and death, and

Von den gifftigen Bösen Dämpffen
vñ Reüchen/ der Metall/ als Silber/ Queck-
silber Bley vnd anders/ so die Edlen hand-
werck des Goldtschmidens/ vnd ander arbai-
ter in des feür sich gebrauchen müssen/ Wie sie
sich da mit halten vnd die gift vertreibē sollē.

V. Engelshofen. 1593. 777 6

Concerning poisons and the evil vapours of smoke and of metals, such as silver, quicksilver, lead, and others which the worthy trade of the goldsmith and other workers are compelled to use. How they should conduct themselves concerning these matters and how to dispel the poison.

ELLENBOG'S MANUSCRIPT TITLE PAGE

(By courtesy of the Wellcome Institute of Historical Medicine)

gives some credence to Gilfillen's theory.

MIDDLE AGES

Ulrich Ellenbog wrote the first ever work on industrial hygiene in 1473 but which was not published till 1524. "When ye master and men work silver with lead ..... guard yourselves as far as ye may from the vapour and smoke, for it is poisonous to you ... do it in the open air and not in a closed room ..... turn away therefrom and bind up the mouth ..... lead is a cold poison for it maketh heaviness and tightness of the chest, burdeneth the limbs and offtimes lameth them.....' Translated from 15th Century Swabian by C. Barnard 1932.

In 1724 Huxlams observed an epidemic of abdominal colic occurring in Devonshire which he correctly associated with cider drinking. It was George Baker, however, forty three years later

who astutely noted that cider drinkers in Hereford, Gloucester and Worcester were not similarly affected. He went on to prove experimentally that the Devonshire colic was due to the presence of lead in the cider which came from the use of lead lined cider presses. When the lead lining was removed, after much controversy, the epidemic cleared up. (MAJOR 1939).

A few years earlier Tronckin had shown that a similar epidemic called colic Pictonum in Amsterdam was due to the presence of lead in drinking water which had been collected from rain running off leaden roofs.

Meanwhile in 1700, Ramazzini published his remarkable treatise on the'Diseases of Workers' which directed some attention to ill health resulting from occupations.

In hisbook he described the effects of lead poisoning in potters,

'In almost all cities there are workers (potters) who habitually incur serious maladies from the deadly fumes of roasted and calcined lead (used) for glazing their pots, ... their whole body takes in the lead poison that has been melted, hence they are soon attacked by grievous maladies. First their hands become palsied, then they become paralytic, splenetic, lethargic, cachetic, toothless, so that one rarely sees a potter whose face is not cadaverous, and the colour of lead (WRIGHT 1940).

INDUSTRIAL REVOLUTION

The Industrial Revolution in England beginning in the second half of the 18th century caused a migration of people from the country to the towns in search of work in the factories. Many cases of lead poisoning occurred as a result of ignorance of hazardous industrial processes.

The appalling conditions in which people laboured began to stir the conscience of a nation ripening for social reforms. It was in this setting that Charles Turner Thackrah, a Leeds physician produced in 1832 the first British work on occupational diseases, 'The Effects of Arts, Trades, and Professions and of civic states and Habits of Living on Health and Longevity'.

Of lead mines he wrote, "Miners rarely work more than six hours a day, yet seldom attain the age of 40 ..... Smelting is considered a most fatal occupation. The appearance of the men is haggard in the extreme'. Of the manufacturers of white lead he wrote, "Some complain of headaches, drowsiness, sickness, vomiting, griping obstinate constipation .... Persons commence the manufacture about the age of 20; many soon leave from broken health; those who endure the employ do

not remain, on average, longer than the age of 45, and during one-third of these 25 years, the men are laid up in bed, or decrepid from colic or palsy'.

It was significant that Thackrah revealed in the title of his work an awareness of a deep, social implication resulting from industrial hazards. There followed the Factory Act of 1833, with the appointment of the first Factory Inspector and the limiting of child labour to 9 years of age and over. The growth of public health was beginning, but public health and industrial medicine developed and were to remain separate disciplines.

Greenhow, 1859, an epidemiologist and lecturer in public health at St. Thomas's, was amongst the first to compare mortality rates in lead and non-lead mining communities and concluded that a higher mortality in the former community was

associated with lead mining. Under his influence factory inspectors could enforce employers to attempt to control dust by the use of fans (NEWHOUSE & SCHILLING 1973). A further step followed in 1864 when legislation appeared which forbade women and children to take meals in the dipping rooms in potteries. Manufacturers were familiar with leadless glazes but were reluctant to introduce them in case the industry suffered. Eventually, legislation was necessary to prohibit lead glazes but poisoning from their sources was not eliminated until 1950 (SCHILLING & HALL 1973).

TWENTIETH CENTURY

Sir Thomas Legge was appointed the first Medical Inspector of Factories. Through him, the Home Secretary, Mr. Asquith, intoruced in 1895 the Factory & Workshop Act in which lead poisoning became a notifiable disease and which did much to

reduce its incidence.

Legge propounded his famous aphorisms; his 3rd stated, 'Practically all industrial lead poisoning is due to the inhalation of dust and fumes and if you stop their inhalation you will stop the poisoning' (HUNTER 1962). This important aphorism might well be applied not only within a lead industry but also to the surrounding general community, where persons may be at risk owing to industrial effluent.

An important crusader against the adverse conditions and effects of the lead industry was Alice Hamilton whose investigations resulted in improved working conditions and better surveillance in America. She became Assistant Professor of Industrial Medicine in the University of Harvard in 1919.

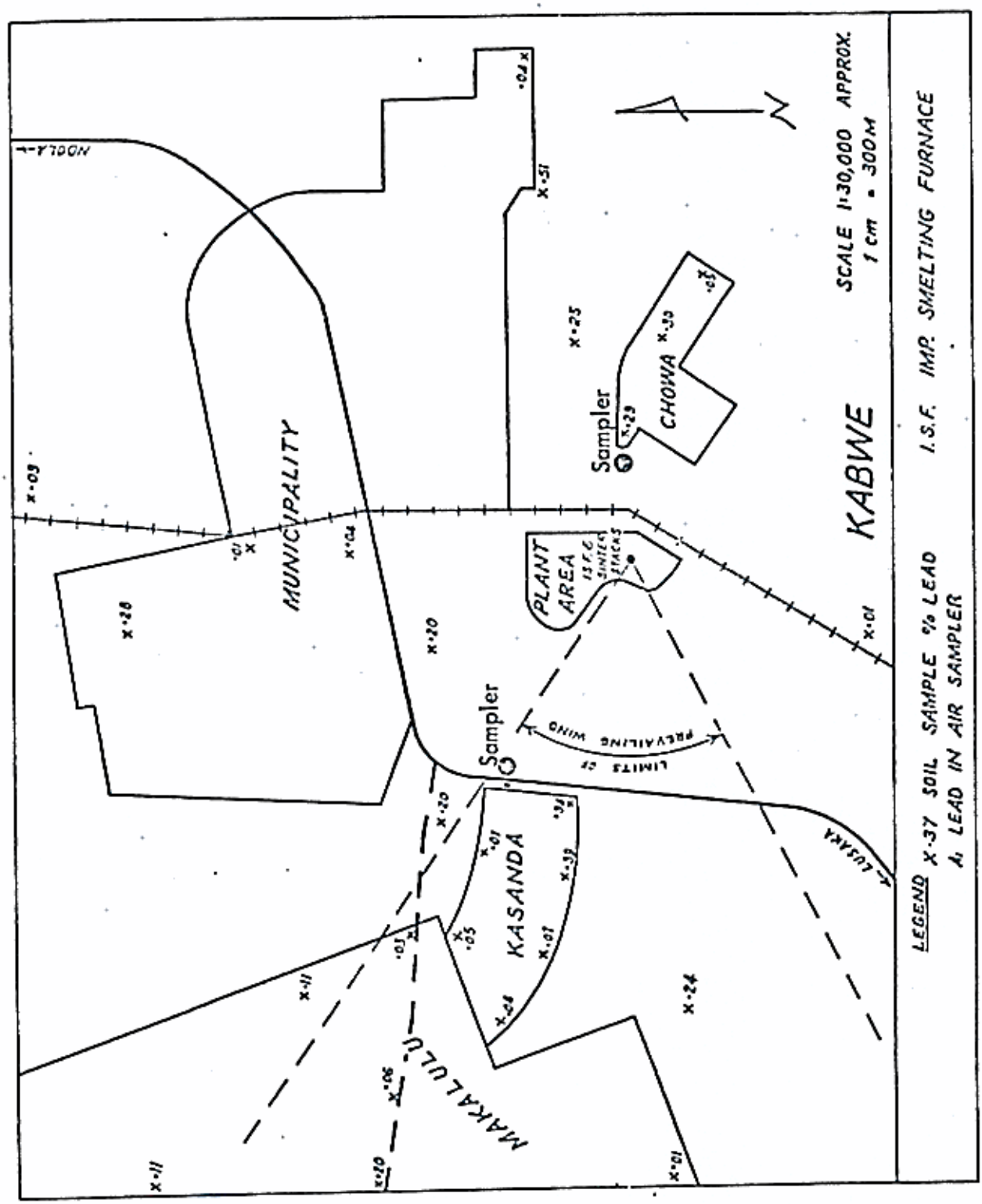
From time to time, moonshine whiskey has caused lead

human capacity for physiological adaptation, and emphasized the need for caution. He did not, however, share Patterson's pessimistic view.

In the past decade the demand for lead has increased at a rate of 3½% per annum. Despite some replacement by less noxious substances it continues to be used in a metallic form for pipes, cables, batteries, sheet roofing, solder, printing metal, and in compound form in petrol, plastics, paints, pigments and glass (STUBBS 1972).

To accommodate the increasing demand, new mines and smelters have developed in many parts of the world, creating localised increases in atmospheric lead with risk to workers as well as neighbouring communities.

FIGURE 1


KABWE
I.S.F. IMP. SMELTING FURNACE
SCALE 1:30,000 APPROX.
1 cm = 300 M
N
MUNICIPALITY
CHOWA
KASANDA
MAKALULU
PLANT AREA
I.S.F. SINTER STACKS
Sampler
Sampler
LIMITS OF PREVAILING WIND
LUSAKA
LEGEND X·37 SOIL SAMPLE % LEAD
A LEAD IN AIR SAMPLER

THE SOURCES OF LEAD POLLUTION IN KABWE

INTRODUCTION

Kabwe, a town in Central Zambia situated on a plateau 1,180 metres above sea level, has a population of about 64,000 residents in four community areas surrounding the Broken Hill Lead Mine (Fig.1). Mining began at Broken Hill about 70 years ago. Since then there has been an increasing annual output of zinc and lead, and smaller quantities of Cadmium and Silver. The main lead-ores are Galena, (Pb.S) and Cerrusite (Pb CO3).

THE KABWE COMMUNITIES

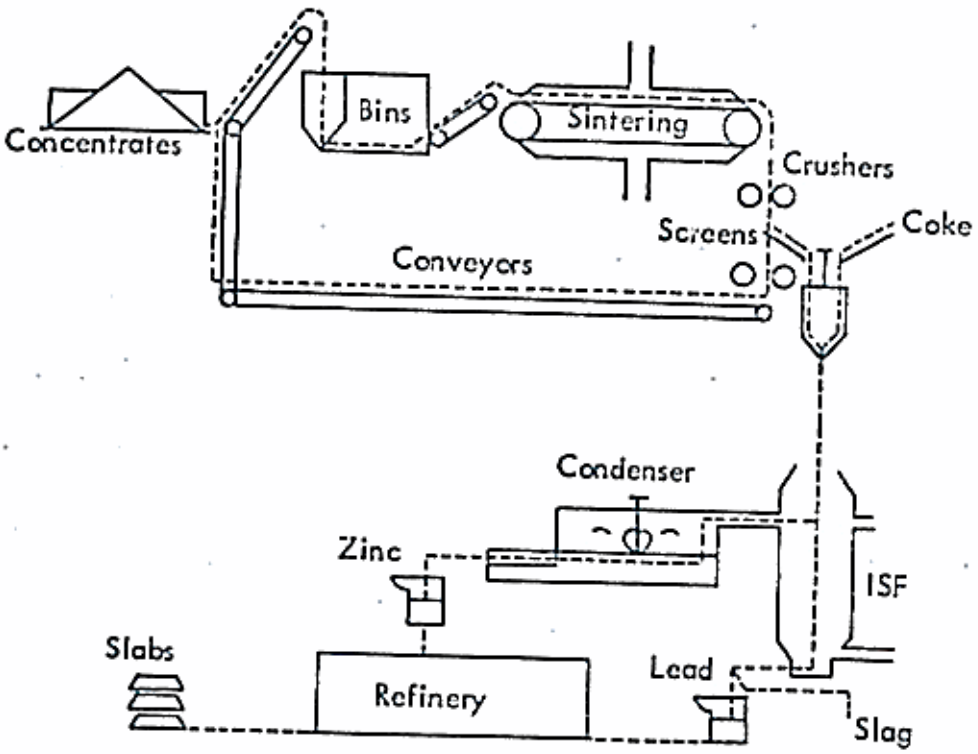
1. Kasanda: is the main residential area for African mine employees and their families. Up to 1973, its

population was 11,000 and was subsequently reduced, between January and June 1973, to 8,000 following a rehousing scheme. It covers an area of 650,000 square metres, the centre being 2,200 metres from the smelter furnace stack and is swept by the prevailing wind from the direction of the smelter. (Fig. 1).

2. Chowa: a smaller residential area, situated to the east of the mine is not subject to significant wind-borne lead. It has a population of 3,000 mine employees and their families, removed from Kasanda to better housing between January and June 1973.

3. Makalulu: a large squatter area of 3,000 population, adjacent and to the west of Kasanda.

FIGURE 1[2]


Concentrates
Bins
Sintering
Crushers
Screens
Coke
Conveyors
Condenser
Zinc
ISF
Slabs
Refinery
Lead
Slag

STEPS IN EXTRACTING LEAD AND ZINC FROM THE ORE

Concentrator

1. The main ore galena, (zinc and lead sulphide) is concentrated to remove waste matter. (Fig. 1[2]).

2. It is then loaded into bins and conveyed to the sinter.

Sinter

3. In the sinter the sulphides are roasted to oxides

$$ZnS + O_2 \rightarrow ZnO + SO_2$$

$$PbS + O_2 \rightarrow PbO + SO_2$$

and then pass on to the crusher, then through screens which recycle unsuitable material through the sinter.

4. The crusher discharges into the smelter furnace

Imperial Smelter Furnace

5. The temperature of which is maintained between the

boiling points of lead and zinc. Zinc having a lower boiling point becomes gaseous and is carried off to the condenser, whilst the lead remains molten and can be topped off at the base of the furnace.

$$ZnO + CO \rightarrow Zn + CO_2 \uparrow$$

$$PbO + CO \rightarrow Pb + CO_2 \uparrow$$

Condenser

6. The gases are cooled in the condenser and a molten pool of metal is formed from the surface of which zinc is topped off.

(ROACH 1974)

SOURCES OF LEAD POLLUTION

The sources of lead pollution in Kabwe are as follows:

1. The effluent from the stack of the mine Smelter Refinery and waste ore products dumped to the west of the mine Smelter.
2. Petrol exhaust fumes from vehicles travelling through the area.
3. Ground lead.
4. Contamination of mine workers clothing and body surface.
5. Vegetation grown within the lead area.
6. Drinking water.

ATMOSPHERIC LEAD

The prevailing wind blows from the mine westerly to Kasanda, across open country almost unimpeded by trees or buildings.

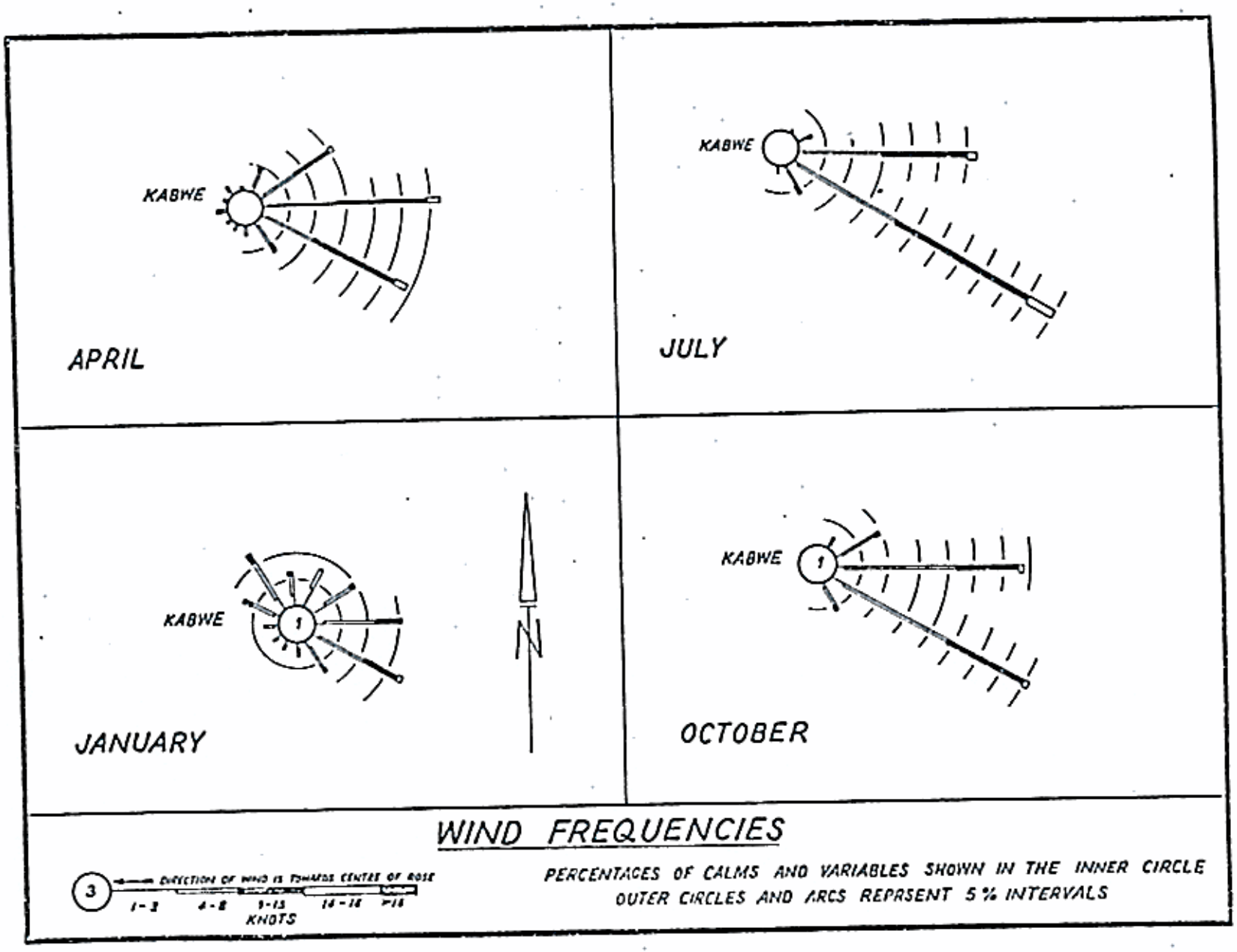
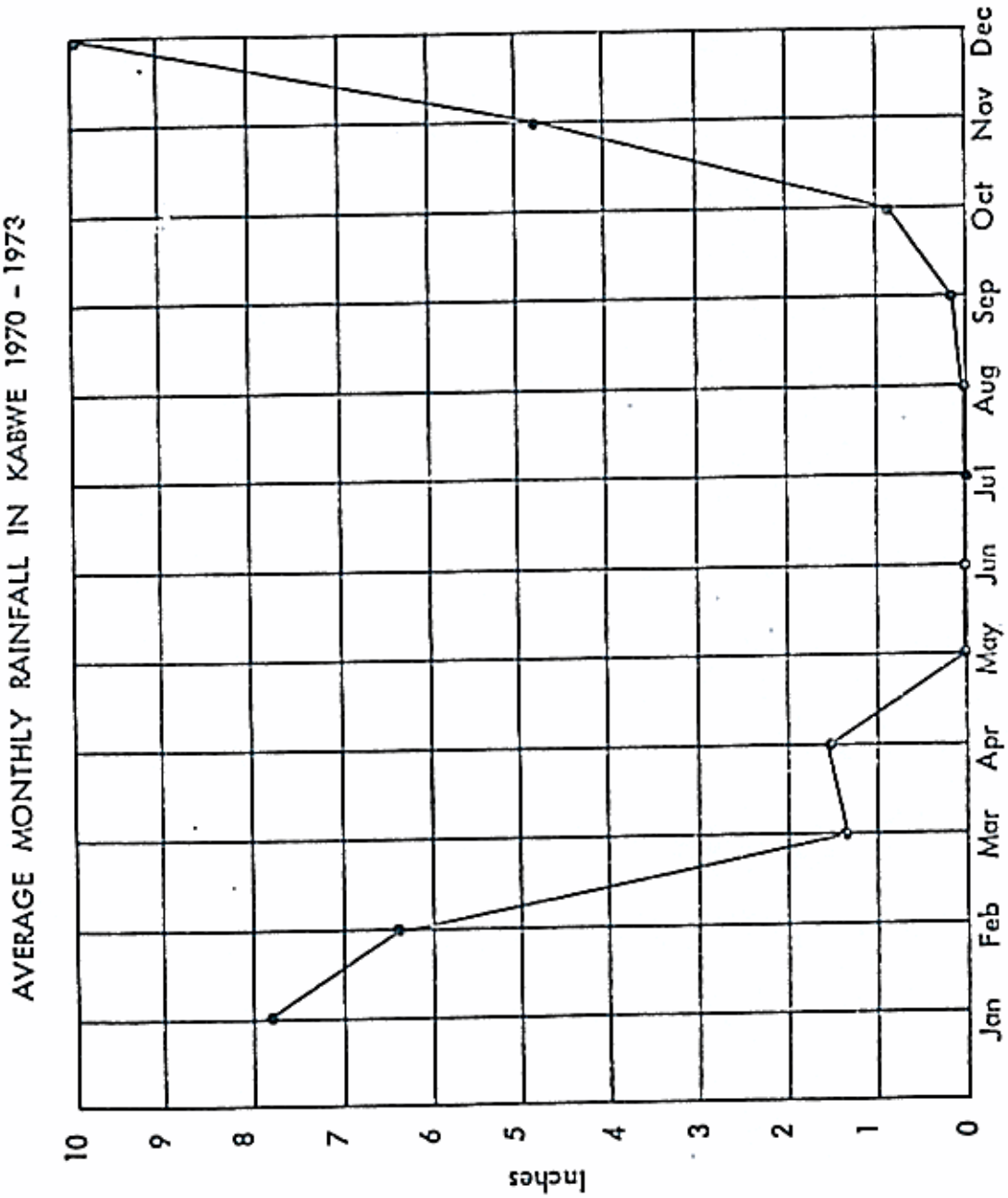

KABWE
APRIL
KABWE
JULY
KABWE
JANUARY
N
KABWE
OCTOBER
WIND FREQUENCIES
3
DIRECTION OF WIND IS TOWARDS CENTER OF ROSE
1-3
4-8
9-13
14-16
>16
KNOTS
PERCENTAGES OF CALMS AND VARIABLES SHOWN IN THE INNER CIRCLE
OUTER CIRCLES AND ARCS REPRSENT 5 % INTERVALS

FIGURE 2

FIGURE 2^2


AVERAGE MONTHLY RAINFALL IN KABWE 1970 - 1973
inches
0
1
2
3
4
5
6
7
8
9
10
Jan
Feb
Mar
Apr
May
Jun
Jul
Aug
Sep
Oct
Nov
Dec

MONTHLY MEAN WIND SPEEDS FOR 1967 - '72 KABWE STATION 663

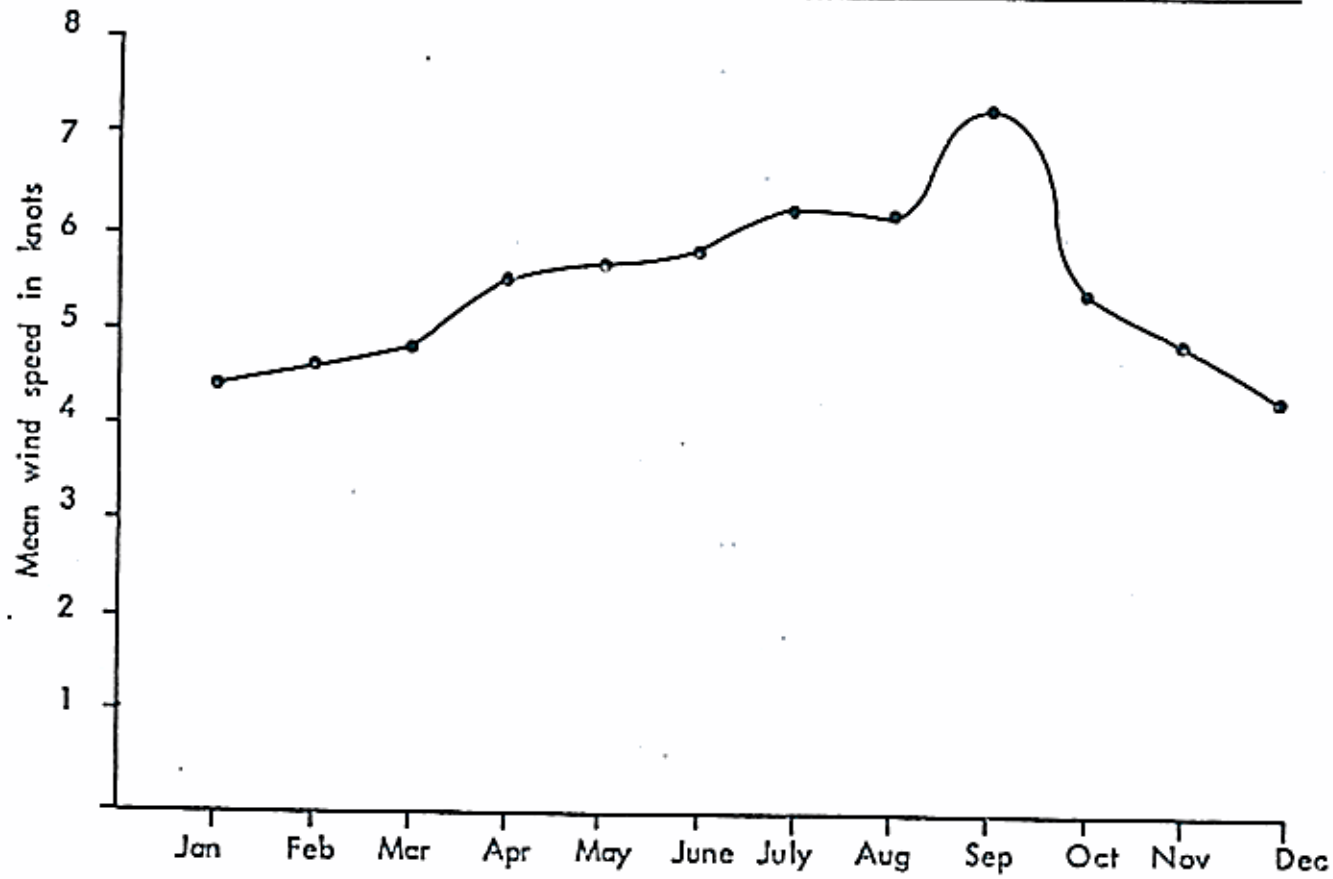


FIGURE 23

During the rainy season, however, from November to March the winds are variable in direction. (Fig. 2[1]). This wind takes up lead particles from the effluent of the Imperial Smelting Furnace and Sinter Plant stacks creating a looping or fumigating plume. The wind also picks up particles from the waste ore deposited on the ground on the lee side of the mine forming ground level dust clouds which sweep towards Kasanda.

Lead in air was monitored from the top of a two storey building on the eastern border of Kasanda (See Fig. 1'A') 1,500 metres from the plant. (Method: See Appendix 1). A second sampler was positioned in May 1974 in Chowa which then enabled a comparison of lead in air concentrations between the two mine townships. Chowa's was well within the normal limits. (See Table 1).

TABLE 1

RESULTS OF LEAD IN AIR SAMPLING

		KASANDA SAMPLER A[1]	CHOWA SAMPLER A[2]
April	1973	.0143 mg/m^3	
May	1973	.0088	
June	1973	.0094	
July	1973	.0087	
August	1973	.0071	
September	1973	.0029	
October	1973	.0090	
November	1973	.0106	
December	1973	.0079	
January	1974	.0020	
February	1974	.005	
March	1974	.013	
April	1974	.014	
May	1974	.018	.002
June	1974	.010	.0015
July	1974	.014	.001
Average Monthly		.00968mg/m^3 .00235	.0015
USA Environmental Protection Agency - standard		.002 3 mths. .005 30 day	

The Kasanda average monthly figure is above the U.S.A. standard of .005 mg/m^3 for a 30 day average period. The low figure for September reflected a partial shut down of the Imperial Smelter Furnace, and the lower figures of December, January and February reflected not only some days of shut down but also wind change as occurs at this time of the year during the rainy season. (Fig. 2) Obviously the Kasanda community is subject to an abnormally high atmospheric lead concentration. Owing to fall out and dispersal of the lead particles the concentration expectedly lessens with increasing distance from the source, i.e. pollution decay, so that Makalulu area is less affected, this is reflected in the lower blood lead concentrations of children living in Makalulu compared with those of Kasanda. The lead in atmosphere figures also reflected the

contribution of motor exhaust, as the monitoring was done close to the main road running between Kasanda and the Mine plant. Petrol contains tetra methyl or tetra-ethyl lead, added as an anti-knock agent, also 1,2, dihaloethanes, e.g. ethylene dibromide and ethylene di-chloride, which react with the decomposition product lead oxide, to form lead halide. Thus motor exhaust contains organic as well as inorganic lead. The former occurs when petrol combustion is incomplete. Organic lead is absorbed via inhalation and also, being fat soluble, via the skin.

Tetra-alkyl leads have a lower atmospheric threshold limit value (.075 ug/m^3) than has inorganic leads, and in the liver are converted to toxic tri-alkyl leads which have a predilection for the central nervous system.

Inorganic lead in car exhaust increases with engine speed, (especially during accelerations approaching 60 m.p.h.) and traffic flow density. The average hourly flow rate of motor vehicles was 140 between the hours of 7.30 a.m. and 4 p.m. Monday to Saturday. The contribution from such a small traffic flow is relatively insignificant. Consider Fleet Street's mean figure of .0063 mg/m^3 for a seven month period, April to October 1972 for 'saturated' traffic flow during the hours of 9 a.m. to 5 p.m (LAWTHER 1972).

LEAD IN SOIL

The percentage of lead in soil and its distribution is indicated in Fig. 1. Soil samples of Kasanda and Chowa were collected by the writer and the samples assayed by Mr. R. HOARE, M.Sc. head of the Broken Hill Assay Lab., other Kabwe samples were

similarly assayed but were collected under direction of Mr. E.KING and Prof. LANE. Lead is widespread over Kabwe, but it is more concentrated in Kasanda, Chowa and Makalulu areas. Some of this lead can be expected to be of natural occurrence, but much is lead oxide fall out originating from the smelter stack. It is thought that lead oxide reacts with atmospheric sulphur dioxide to produce lead sulphate which is soluble. Samples taken showed variable water solubility of up to 3% and an average solubility of 60% in 0.1 N Hydrochloric Acid (LANE 1970). All samples taken at depths of 10 cm showed very much reduced concentrations, with similar solubility properties. (Tables 2 and 3).

PATERNAL OCCUPATION

No relationship was shown between the occupation of the

TABLE 2

SOLUBILITY OF SOME LEAD COMPOUNDS

COMPOUND		SOLUBILITY mg/100 mls water at 20°C
Lead dioxide	PbO_2	Insoluble
" carbonate	$PbCO_3$	1.1
" monoxide	PbO	17.0
" sulphate	PbSO4	42.5
" sulphide	PbS	124.4
" di-iodide	PbI_2	630.0
" mono-iodide	PbI	1000.0
" bromide	$PbBr_2$	8441.0
" chloride	$PbCl_2$	9900.0
" nitrate	$Pb(NO_3)_2$	376500.0

Waldron, H.A. & Stöfen, D.

TABLE 3

LEAD CONTENT IN SOIL

Location	Lead Content	Author
India	.04 - .06 ppm	Bagchi et al 1940
Canada	.5 - 1000 "	Warren & Delavault 1960
East Africa	.6 "	Chamberlain 1960
Sarawac	4.8 "	Kehoe et al 1933
New Jersey Loan	13.9 - 95.7 "	Prince 1957
Soil near lead ore vein	20.0 10,000 "	Huff 1952
Near site of coal burning	534.0 12,340 "	Manley 1937
Near lead-zinc smelter	778.0 40,175 "	Pakhotina 1958

Adapted from Sub-clinical Lead Poisoning by Waldron, H.A. & Stöfen, D.

Derbyshire Soil	500 - 10,000 ppm	Barltrop et al 1974
Soil around Broken Hill, Zambia	100 - 9,400 ppm	

father and children's blood lead, in fact the fathers of affected children were found to be employed in a variety of occupations, including those jobs with no direct access to lead. As mine workers live in either Kasanda or Chowa, the contribution made by lead contaminated clothing to these communities would be much the same. There are adequate shower and laundry facilities provided on the mine but it seems that these facilities are not fully utilised.

Vegetation Lead: The amount of lead in vegetation varies with the plant species and also depends on the quantity of available soluble lead in the soil, it being greater by up to 10 times in plant samples taken close to highways (CHOW, 1973).

The amount of soluble lead available to plant roots is comparatively small, and even less is transmitted up the

stem. (JONES 1972). Root crops, therefore, generally contain relatively more lead than leaf or fruit crops. It is possible, however, for plants to take up lead from the atmosphere through their aerial parts and this route may account for the high lead content found in maize cobs examined by REILLY. Of any lead in vegetation which is ingested, only about 5% is actually absorbed.

REILLY and REILLY (1972) in a survey showed that vegetation in the Kabwe area had a higher lead content compared with similar plant species in other areas and that Kabwe maize grain contained 28 ppm. The British Food Standard Committee sets different levels for different foods, but with a general upper limit of 5 ppm (EGAN 1972). Thus, it would seem that the value of lead in maize is high. This may be of some

significance for a community such as Kabwe's whose staple diet is maize; if, however, locally grown maize made a significant contribution to raised blood lead one would expect that the inhabitants of Chowa, where the ground lead is also high, would be similarly affected by lead as Kasanda inhabitants. No doubt lead in maize contributes to raised blood leads but as maize is reaped for only 2-3 months in the year, and none is stored by the mine small holders, less than a quarter of the maize consumed is lead affected as further requirements are supplied by traders who buy from distant non lead affected farms.

Lead in Water: domestic water comes from boreholes in the Makalulu area, and is pumped to Kasanda, Municipality and Mine plant areas. Water samples collected from the five

operative boreholes as well as from domestic taps in the supplies areas showed a lead concentration of between 10 and 20 ug/litre. As the recommended upper limit set by the World Health Organisation is 50 ug/litre the supply was well within the safety limit. Fortunately, lead piping is not used for conducting water to any of the communities, and as all Kabwe water is hard the problem of lead solubility, resulting from the association of soft water in lead pipes does not arise.

SUMMARY

The main sources of lead pollution are considered. The greatest contribution is from atmosphere which derives its lead from the Smelter and Sinter Furnaces as well as from

the ground. Some of this lead is inhaled, and some, following fall out, ingested. Other sources such as contamination of workers person and clothing, vegetation, motor exhaust and water are of secondary importance to the Kabwe problem.

BLOOD LEAD CONCENTRATION IN CHILDREN

INTRODUCTION

Lead absorption first occurs in utero, and at the time of birth, the infant has a blood lead concentration only a few microgrammes below that of its mother's. Subsequent to establishing an independent existence, lead may be ingested with a body absorption of about 10%, and also inhaled with a degree of absorption which has been variously estimated from 10 - 50% (BARLTROP 1972). The object of this investigation was to determine the pattern of blood lead concentration in African children from early infancy to 16 years of age in each of the four residential community areas of Kabwe. Two of these areas, Kasanda and Makalulu, are subject to atmospheric lead pollution emanating from the neighbouring Broken Hill

Lead Mine, (Fig. 1) which adds considerably to the blood lead concentration of the inhabitants. Ground lead concentrations are somewhat similar in all four areas but increased quantities are to be found in parts of Kasanda. The survey was conducted over a complete year from January 1973 to January 1974.

Blood samples were taken from children, in each year of age up to 16 years, on their first attendance at the clinics.

Method of Blood Lead Determination

Blood lead concentration was determined using an atomic absorption spectrophotometer, following extraction with 0.5 cc ammonium pyrrolidine dithiocarbamate and 5 cc n-butyl acetate. During each batch of lead estimations the spectro-

BLOOD LEAD CONCENTRATIONS / YEAR OF AGE AS AT 31-1-'74

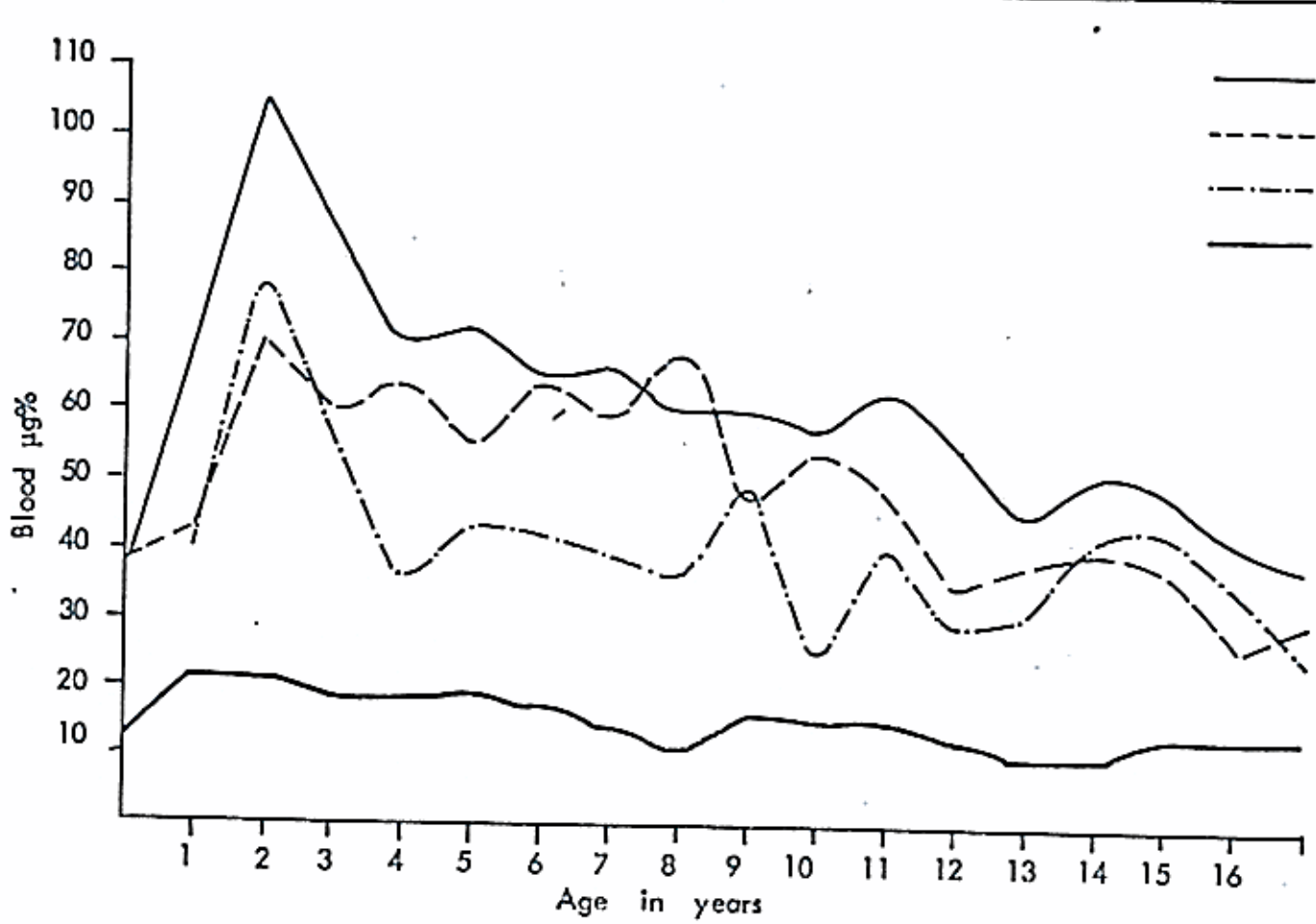
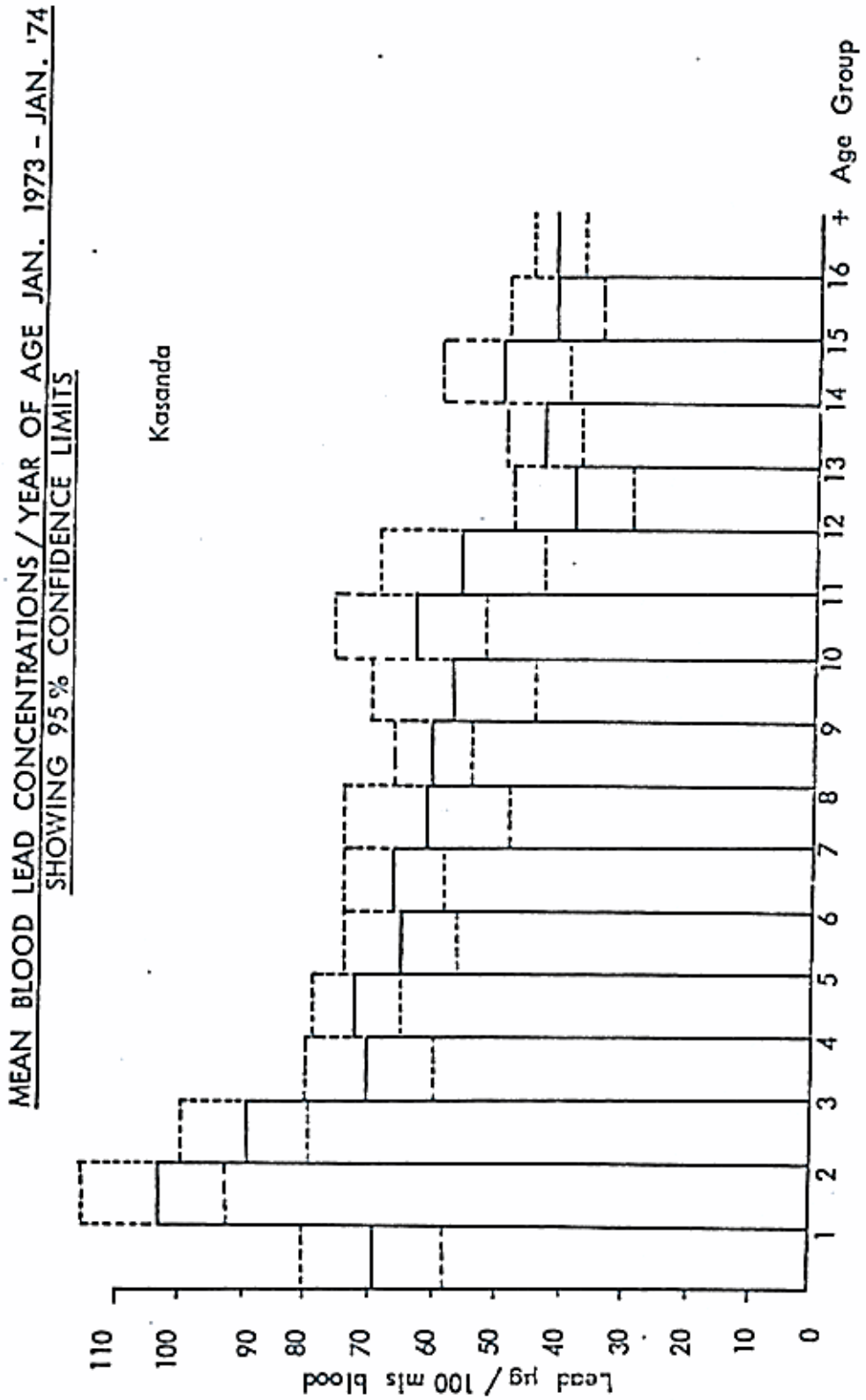


FIGURE 3.1

FIGURE 3^2


MEAN BLOOD LEAD CONCENTRATIONS / YEAR OF AGE JAN. 1973 - JAN. '74
SHOWING 95% CONFIDENCE LIMITS
Kasanda
Lead µg / 100 mls blood
0
10
20
30
40
50
60
70
80
90
100
110
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
+
Age Group

photometer was checked against a known standard blood sample. (Appendix 2). The average result of two readings on a 5 c.c. venous sample of blood, collected in a Lithium heparin tube was recorded. The accepted normal upper limit of blood lead for urban populations is 40 ug/100 mls. Values between 40-60 suggest abnormal exposure and over 60 ug evidence of increased lead burden (CHISOLM, 1970). A comparison of mean blood lead concentrations in each of the four groups, and for each year of age is shown. (Fig 3^{1}).

KASANDA (Fig. 3^{2}) was the most severely affected group mean blood lead levels rapidly rising from a mean level at birth of 37 ug to 103 ug at two years of age; a sharp fall to 71 ug by 4 years, and thereafter a gradual decline to adult levels of about 40 ug/100 mls. Thus the evidence indicated an increased

MEAN BLOOD LEAD CONCENTRATIONS / YEAR OF AGE JAN 1973 - JAN '74
SHOWING 95% CONFIDENCE LIMITS

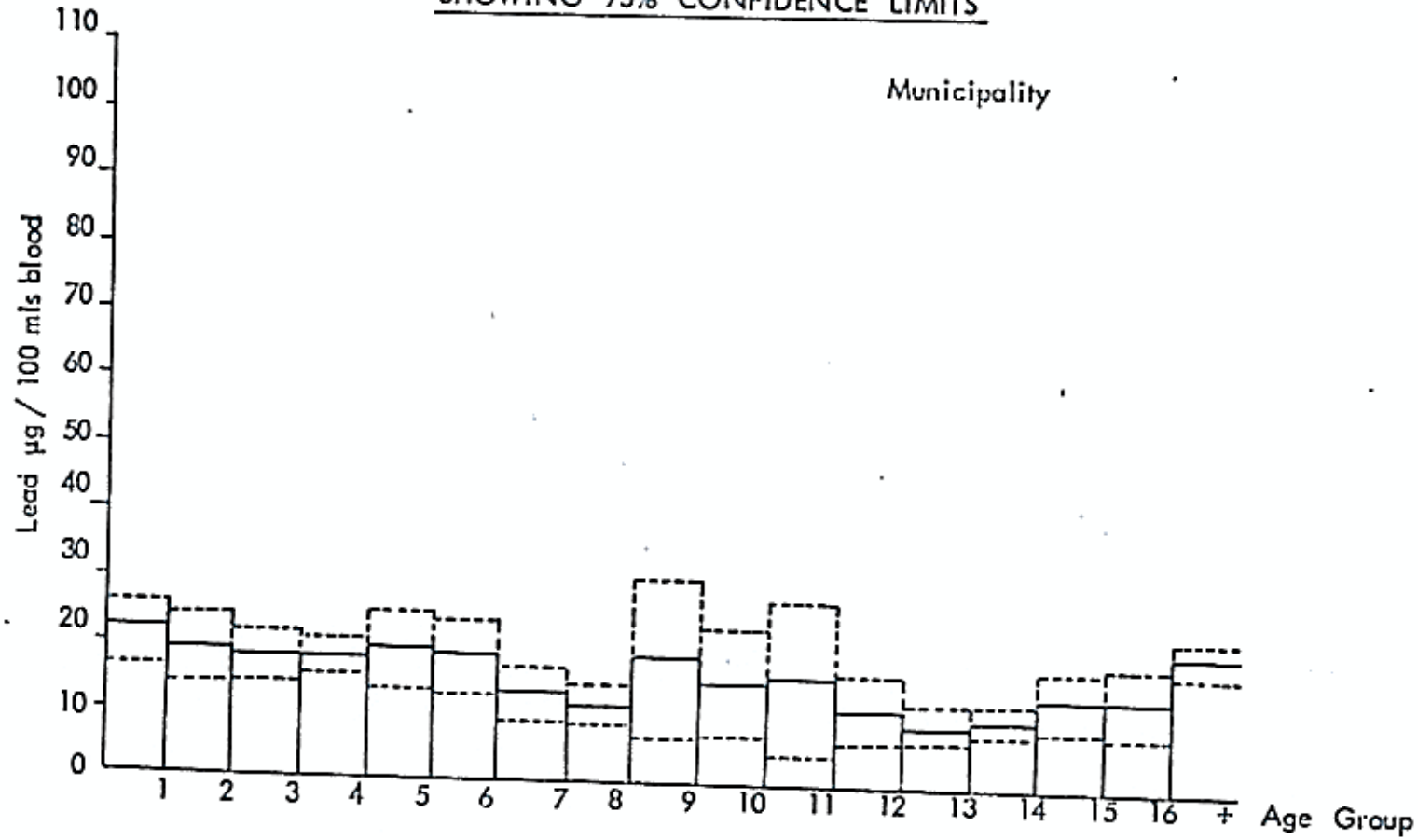


FIGURE 3.3

lead burden affecting all people in this group, again two year olds being most severely affected. In fact of the 91 children between 1 and 2 years attending Kasanda clinic for minor ailments, 89% of them had blood leads over 60 ug/ 100 mls. Commonly, lead levels over 100 ug seemed not to affect the children, but 11 from this area were admitted to hospital with lead encephalopathy during 1973.

MUNICIPALITY (Fig 3[3]) represents the normal range of blood lead levels to be expected in a community 'unaffected by lead pollution'. Ranging from a mean 12 ug at birth, rising to 22 ug in the first two years and thereafter dropping gradually to about 14 ug in late childhood. These infants' cord blood levels of 12 ug/100 mls relate closely to values obtained by various investigators of urban and suburban communities.

MEAN BLOOD LEAD CONCENTRATIONS / YEARS OF AGE JAN. 1973 - '74
SHOWING 95% CONFIDENCE LIMITS

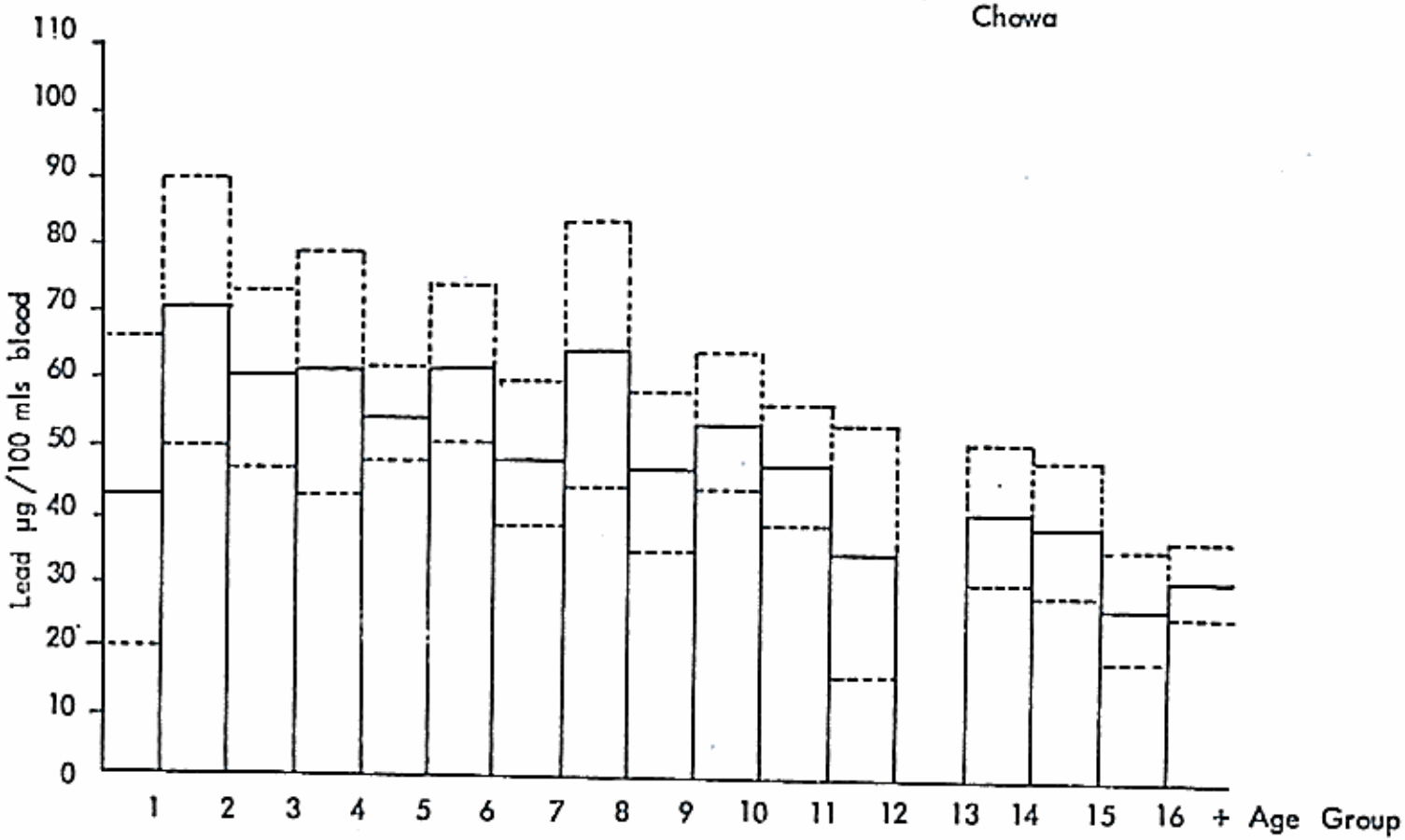


FIGURE 3.4

(BARLTROP 1968, HAAS 1972, HARRIS 1973, KUBASIC 1972, RAJEGOWDA 1972, SCANLON 1971).

CHOWA (Fig 3[4]) the 3,000 persons of this group were resident at one time in Kasanda. The graph illustrates how lead levels fall when persons with a high lead are removed to a cleaner area, reflecting an average period of approximately 6 months residence in the new township. That this fall was attributable to reduced lead inhalation was a reasonable assumption because Chowa being situated on the windward side of the Mine, is thereby subject to considerably less atmospheric lead; and because the contribution from pica in Chowa and Kasanda persons is of the same order, as the infants are of a common background and the ground leads have similar concentrations. It remains to be seen if lead levels in

MEAN BLOOD LEAD CONCENTRATIONS / YEAR OF AGE JAN. 1973 - JAN '74
SHOWING 95% CONFIDENCE LIMITS.

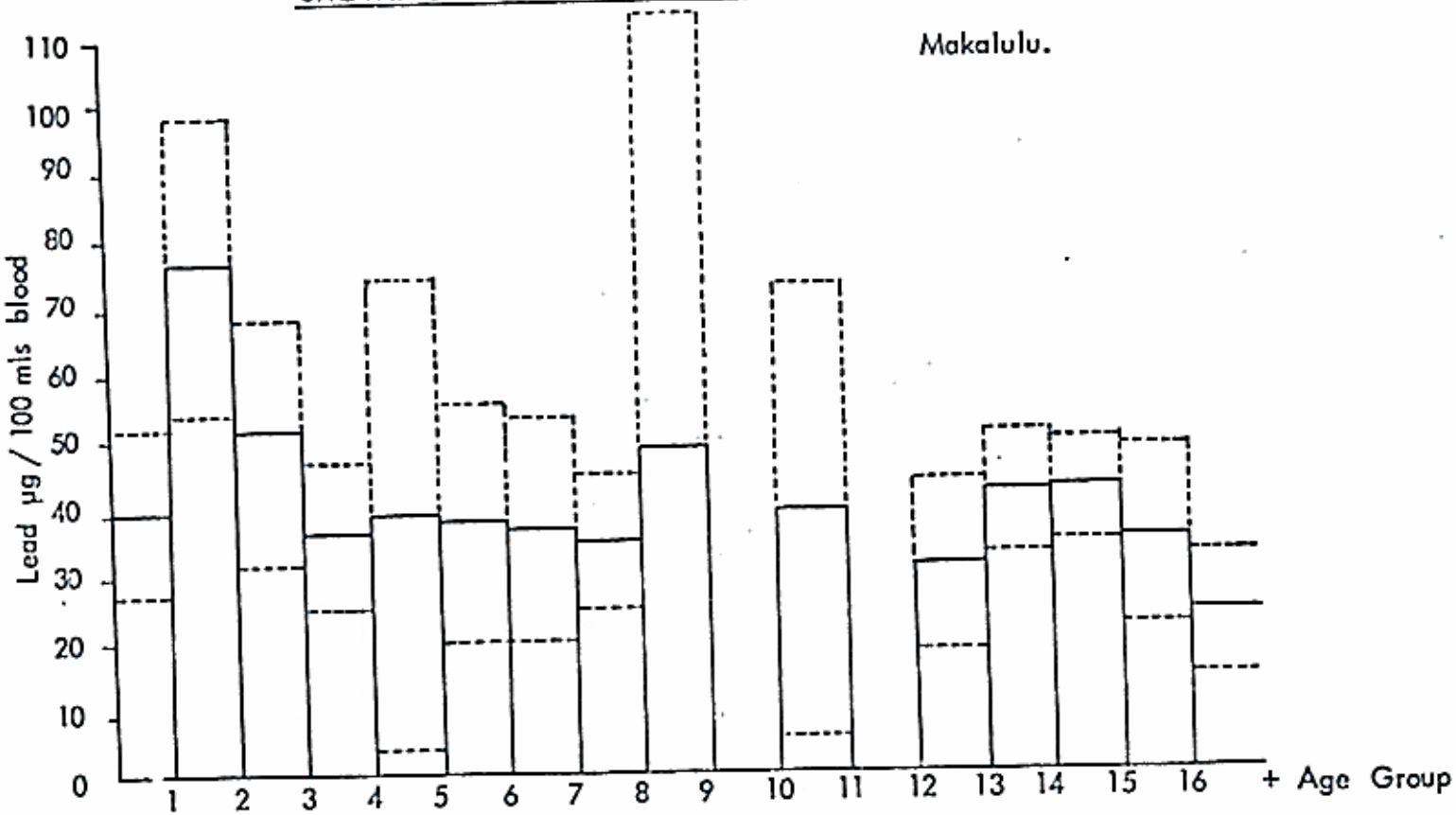


FIGURE 35

this group will continue to fall during the coming year.

MAKALULU (Fig. 3[5]) showed a high peak of 83 ug at 2 years, a rapid fall to 36 ug by 4 years and mildly undulating levels between 20 - 50 in older children.

Makalulu is a very extensive area but most of the population resides close to Kasanda.

Table 4 compares Kasanda and Chowa blood lead concentrations of children at all ages. Their mean values are significantly different as shown by Student's 't' test. The most notable reductions occur in the infant age groups most susceptible to lead encephalopathy and thus the transfer of families from Kasanda to Chowa appears to be justified.

The fact that they moved into an area of equivalent ground

lead concentration does not jeopardise them any more than the Municipality persons who have normal blood lead concentration.

Barltrop & Strehlow (1974) investigating children in the Derbyshire area where ground leads are comparable to those of Kabwe (500 - 10,000 ppm) found that the children's blood leads were not raised to a level considered to be biologically significant.

CONCLUSIONS

All four communities showed rising blood lead concentrations from birth, reaching a peak in infants between the ages of 1 - 3 years and thus emphasising the increased susceptibility of toddlers to lead absorption. An infant's inherited lead load forms a significant base on which further lead loads may

TABLE 4

COMPARISON OF KASANDA & CHOWA MEAN BLOOD LEAD CONCENTRATIONS

Mean blood leads ug/100 mls

Age - years	Degrees of freedom	KASANDA	CHOWA	Mean difference	t	p
0-1	34	70	43	27	3.15	<.01
1-	108	104	70	34	4.96	<.001
2-	68	90	60	30	3.0	<.01
3-	31	71	61	10	2.48	<.02
4-7	100	67	58	9	2.67	<.01
8-11	51	59	51	8	3.29	<.01
12-15	64	43	34	9	2.35	<.02
16+on	80	42	30	12	3.27	<.01

Blood lead distributions did not vary sufficiently from the normal to preclude the use of Student's "t" which is robust and valid for marked departures from normality.

be built during its first three years of life. In Kasanda infants it has been shown that the inherited lead load was unusually high at 37 ug/100 mls. It is also shown that children from the 4th year of life have reduced blood lead concentrations in each successive age groups even though they remain in the high lead environment.

These high blood leads in toddlers reflect in part their reduced ability to deposit lead in their bones (BARRY, 1973) but growth after the second year provides a larger reservoir in terms of bone capacity for lead deposition with subsequent reduction in blood concentration.

It being customary for breast feeding to continue into the second year and very often into the third in these infants,

it would be interesting to measure the amount of lead transmitted via the breast milk of mothers whose blood lead levels are high; and also to determine the role of a predominantly milk diet in aiding lead absorption in these early years, particularly as factors which encourage calcium deposition also encourage lead deposition in bone. (LLOYD-DAVIES, 1973).

The graph emphasises the danger of assuming that normal range blood concentrations in older children and adults also reflect normal levels in infants. Indeed, toddlers can be expected to have abnormally high levels when older children and adults have levels approaching the upper limit of accepted normal. Safe levels in toddlers can be expected, however, when mothers and older children possess levels

below 20 ug/100 mls. A survey within families confirmed that a child's lead level was within normal range when its mother's was below 20 ug/100 mls. and abnormally high when its mother's was above 30 ug/100 mls.

SUMMARY

Blood lead concentrations for persons in each year of age up to 16 years is shown, for residents of four communities within the proximity of Broken Hill Lead Mine, Kabwe. The graph indicates that children between the ages of 1 - 3 years have higher blood lead concentrations than other children subject to a similar lead environment. It also shows the degree of fall in blood lead concentration which follows when persons are removed from an area with a high atmospheric lead content to a cleaner one.

HIGH LEAD CONCENTRATION IN CORD BLOOD

INTRODUCTION

Lead is known to cross the placenta from mother to foetus. This was first demonstrated by Baumar; 1933, in experiments on the rat. Later, Earltrop, 1968, showed that in the human such lead transfer began about the 12 - 14 week of gestation. Lead transfer continues thereafter throughout foetal life and at the time of delivery a good correlation between the lead concentration in the mother's blood and infant's cord blood is obtained (EARLTROP, 1968, HARRIS, 1972, HAAS, 1972).

Investigations have shown that for communities unexposed to abnormal lead environments, the mean blood lead concentration in mothers lies between 13.9 ug and 16.89 ug/100 mls.

and in full term infants the cord blood level lies between 10.8 ug and 14.98 ug/100 mls. (Table 5.)

OBJECT

This investigation was undertaken to determine if a good correlation of lead concentration between the mother's blood and infant's cord blood was maintained in the presence of abnormally raised lead levels. This involved a community living within a 3,000 metre radius of Broken Hill Lead Mine, Kabwe, Zambia, which was subject to a high atmospheric lead concentration. Conducted over a complete year to obviate gross seasonal differences, 125 mothers and their newborn infants were examined. These representing 1 in 5 deliveries recorded at the clinic of the lead affected community.

TABLE 5

ug LEAD/100 mls BLOOD

	Number examined	Mothers' Mean Value	Infants' Cord Mean Value	Cord Range
Scanlon 1971	13 Urban		22.1± 8.7	10 - 39
	15 Sub Urban		18.3± 4.03	13 - 26
Barltrop 1968 U.K.	29	13.9	10.8	4 - 24
Harris 1972 U.S.A.	24	13.2 ± 4.2	12.3 ± 3.3	10 - 20
Haas 1972 Germany	294	16.89 ± 8.7	14.90 ± 8.57	2 - 47
This Report) Control Group	33	14.72 ± 7.5	11.78 ± 5.6	3 - 29
) Mine Group	125	41.2 ± 14.39	37.00 ± 15.3	10 - 84

Values ± one standard deviation

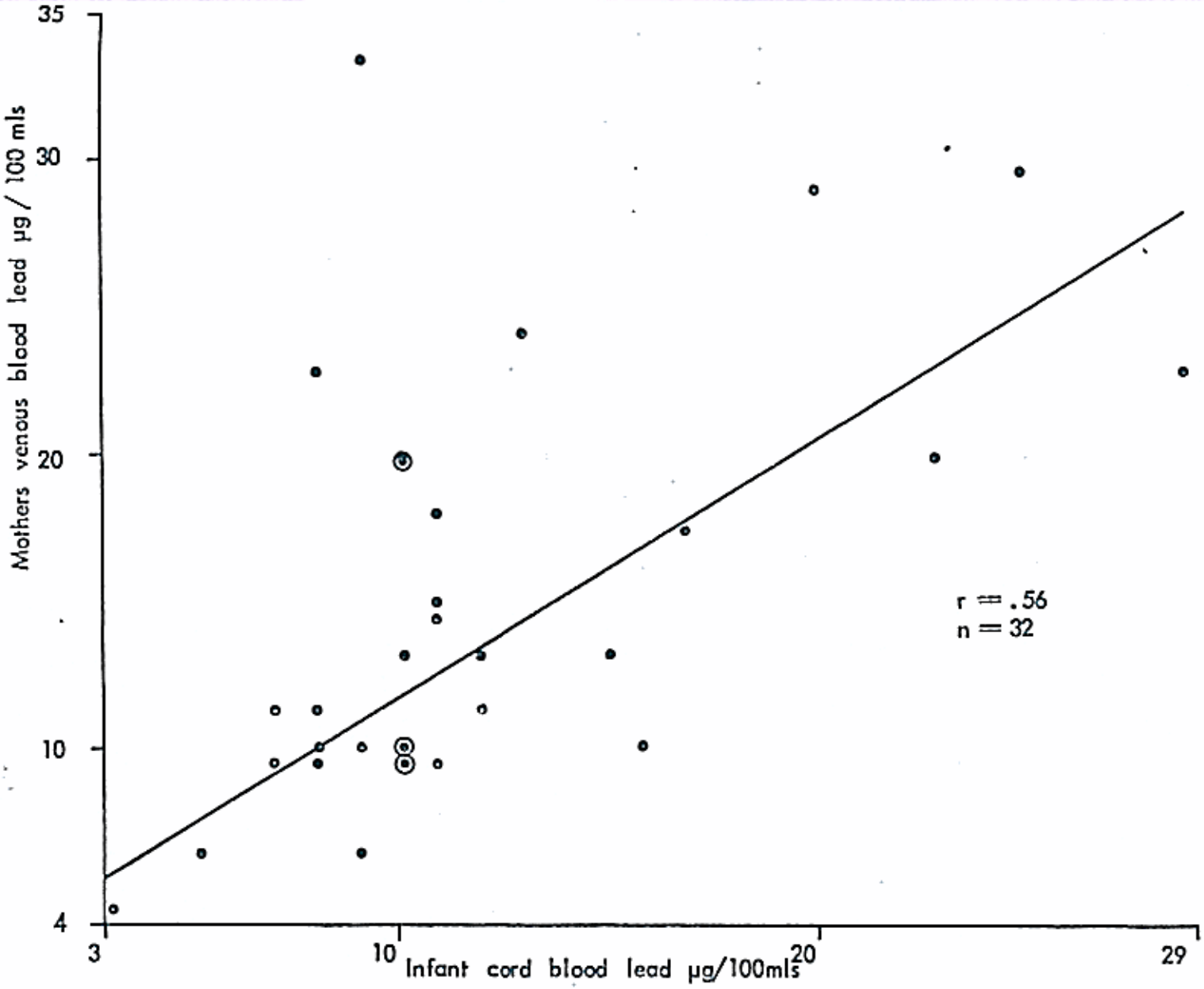

32 MOTHERS WITH INFANTS. CONTROL GROUP.
Mothers venous blood lead µg / 100 mls
35
30
20
10
4
3
10
20
29
Infant cord blood lead µg/100mls
r = .56
n = 32

FIGURE 4.1

Over the same period, a control group of 33 mothers and their infants were examined, these being resident in a different district of Kabwe "unaffected by atmospheric lead pollution."

METHOD

Blood samples, each of 5 c.c. were collected in a lithium heparin tube from an antecubital vein of the mother at delivery and from the umbilical cord of the infant. The contained lead was determined using an atomic absorption spectrophotometer following extraction with ammonium pyrrolidine dithiocarbamate and n-butyl acetate. During each batch of lead estimations the spectrophotometer was checked against a known standard blood sample.

RESULTS

The control group, despite a fairly wide scatter, Fig. 4[1]

showed a good significant correlation ($p = <.001$) between lead concentration in the blood of mothers' and infants' cords. The blood leads ranged from 3-29 ug with mean levels of 14.72 ug/100 mls and 11.78 ug/100 mls for mothers and infants respectively. These figures are much the same as those obtained by other investigators of urban and suburban communities. (Table 5) Scanlon, (1971) examing 13 infants born of urban mothers and 15 infants born of suburban mothers showed no relatively significant relationship between umbilical cord blood lead levels and residency: the lead in air concentrations over the respective residencies, being 2 ug/m^3 for the urban and 1 ug/m^3 for the suburban. The 125 Kasanda mothers and infants resident within the proximity of the lead mine where there was an atmospheric lead load of 9.6 ug/m^3 showed

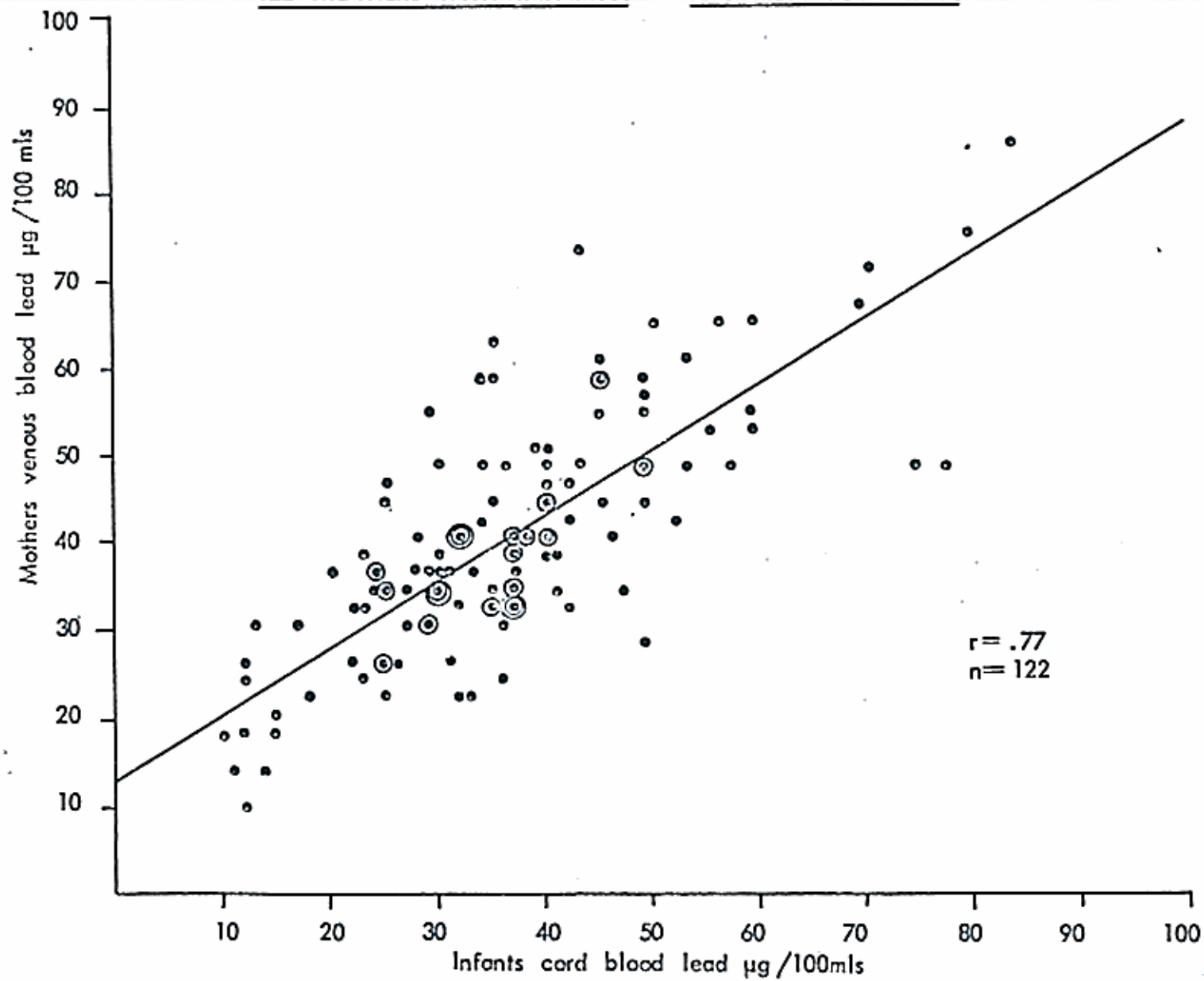

122 MOTHERS WITH INFANTS. KASANDA TOWNSHIP.
Mothers venous blood lead µg /100 mls
100
90
80
70
60
50
40
30
20
10
r = .77
n = 122
10
20
30
40
50
60
70
80
90
100
Infants cord blood lead µg /100mls

FIGURE 42

higher blood leads than has previously been reported for such pairs, (Table 5), these ranged from 10 - 86 ug with mean values of 41.2 ug/100 mls and 37 ug/100 mls for mothers and newborns respectively.

There was also a very significant correlation between these values ($p = <.001$). Fig. 4^2. This investigation confirms that even at raised levels the infants blood lead concentration at birth is still directly proportional to that of its mother. Of lead in blood, 95% is carried by the red cell, the remainder in the plasma. Plasma lead is in equilibrium with lead in neighbouring soft tissue (BARRY 1972), the concentration of which varies with the nature of the tissue and is related to the lead supply from the external environment. There is an equilibrium between lead in the foetus and its maternal

areas is reported to be increasing annually (CHOW 1973).

SUMMARY

Following delivery, blood was taken from 125 mothers, and from their infants umbilical cords for estimation of lead concentration. They were resident in Kasanda, a township within a radius of 3,000 metres of Broken Hill Lead Mine where the atmospheric lead concentration per monthly average was9.6ug/m^3. Their mean blood lead levels were high being 41.2 ug and 37 ug/100 mls for mothers and infants respectively, with a good correlation (r = .77). Thus, it is apparent that an infant's blood lead at birth follows closely that of its mother's even at high levels, a factor of significance for a community subject to a lead polluted environment and of particular relevance should

EFFECTS OF HIGH LEAD CONCENTRATION ON THE NEWBORN

INTRODUCTION

Lead is transferred from the maternal to the foetal blood via the placenta, from about the 12th - 14th week of gestation (BARLTROP 1968) establishing a good correlation of 'normal' lead concentration in mother and baby's blood by the time of birth (BARLTROP 1968, HARRIS 1972 and HAAS, 1972).

In the present investigations the lead concentrations in the mother's blood and infant's cord blood were above normal, maintaining the significant correlation between the two, and thereby the infants were exposed to an unusually raised lead concentration for most of their foetal lives. This fact initiated

an enquiry into the possible adverse effects of such a raised lead on the foetus, and was restricted to an examination of the birth weight and red cell values of the newborn. Kabwe, Zambia, presented two distinct communities for a comparative study, one with an abnormally high blood lead resident in the Mine Township, Kabwe, and the other with a normal blood lead concentration, resident outside the Mine area.

METHOD

One hundred and twenty five cord bloods of infants with a mean lead of 37.0 (their mothers 41.2) were compared with thirty three cord bloods of infants with a mean lead of 11.70 ug/100 mls (their mothers 14.72 ug/100 mls). A 5

c.c. blood sample was collected from each infant's cord in a lithium heparin tube and examined at Kabwe Mine Laboratory for haemoglobin, (Hb), red blood cell count (RBC) using a Coulter counter, and packed cell volume (PCV) by the micro-hematocrit method using a Hawsley centrifuge. The infants were weighed immediately following delivery.

RESULTS

The results obtained from the two groups are given, in Table 6, in addition to Professor Ezeilo's figures from 75, Lusaka, Urban infants.

These latter figures are the only ones so far published for Zambian cord bloods. They, and the Kabwe control group

show very similar values for the red cells and, therefore, form an important base line for comparison with the mine group. The lead level of the Lusaka infants was not known, but assumed to be within the range of normal for urban and suburban communities. This range of 10.8 - 22.1 ug/ 100 mls has been established by various investigators:- (BARLTROP 1968, HARRIS 1972, HAAS 1972, REJEGOWDA 1972, SCANLON 1971)- and the Kabwe control group fell within this range. The mine mothers living closer to the ante-natal clinic attended earlier and more frequently for ante-natal care which probably contributed to the better haemoglobin concentration found amongst the mine mothers.

BIRTH WEIGHT

Lead is said to cause abortion in women who worked in the

potteries in the days before protective measures were in operation (HUNTER). Amongst the women resident in the mine township lead did not appear to be a contributory factor to abortion, possibly because their mean blood lead level of 41.2 ug/100 mls was not high enough. In this investigation there was no significant difference in birth weights between infants of the mine group compared with the controls, their mean values were 3.22 and 3.10 Kg respectively. (Table 6.).

Five of the mine infants weighed between 2.04 and 2.5 kgs. and three from the control group weighed between 2.18 and 2.5 kgs.

Rejegowda 1972, examined 100 infants in New York with a

TABLE 6

TOWN	KABWE				LUSAKA
	MINE		CONTROL		
Number examined	125		33		75
	Mothers	Infants	Mothers	Infants	Infants
Blood Lead u/100 mls	41.2 ± 14.39	37 ± 15.3	14.7 ± 7.5	11.78 ± 5.6	
Hb g/100 mls	13.57 ± 1.4	15.2 ± 1.8	12.4 ± 1.89	15.7 ± 1.7	15.6 ± 1.9
P.C.V.	41.6 ± 3.48	47.9 ± 5.59	40.25 ± 4.54	49.78 ± 5.16	49.3 ± 6.2
M.C.H.C. %	32.5 ± 1.9	31.7 ± 1.7	30.8 ± 2.2	31.5 ± 1.6	31.6 ± 2.7
Birth Weight Kg.		3.22 ± 0.46		3.10 ± 0.49	

Values ± One S.D.

mean cord blood lead of 20 ug/100 mls, their mean birth weight was 3.17 kgs. not significantly different from the Zambian newborn infants; 14 of them weighed less than 2.5 kgs. The birth weights of these Zambian infants compare very favourably with European infants. This has not always been so, for a 2.5 kg. birth weight amongst Zambian infants was once the exception rather than the rule. These improved birth weights are due to the better nutrition of the mothers,probably a consequence of a higher family income and improved education during the past few years.

RED CELL VALUES

Despite the better haemoglobin levels in the mine mothers,

FIGURE 5[1]

HISTOGRAM OF CORD - BLOOD HAEMOGLOBIN

CONTROL GROUP

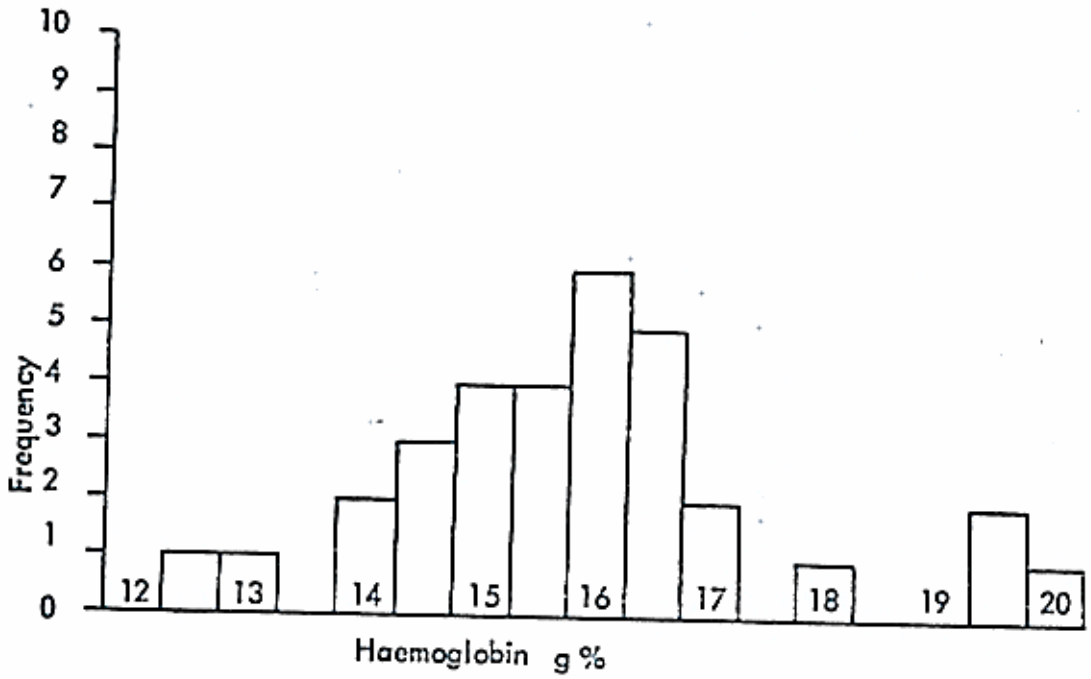
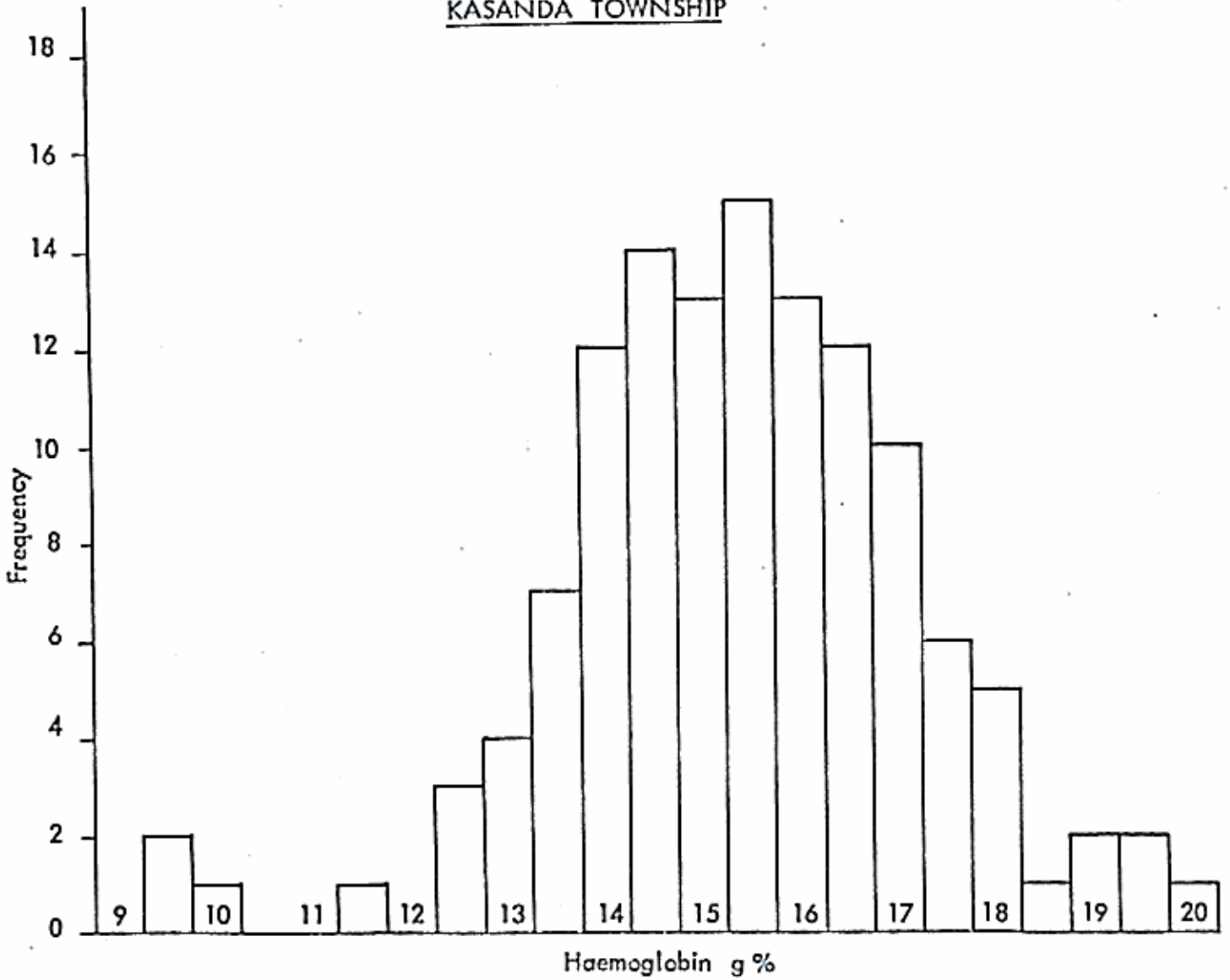


FIGURE 5[2]

HISTOGRAM OF CORD - BLOOD HAEMOGLOBIN KASANDA TOWNSHIP



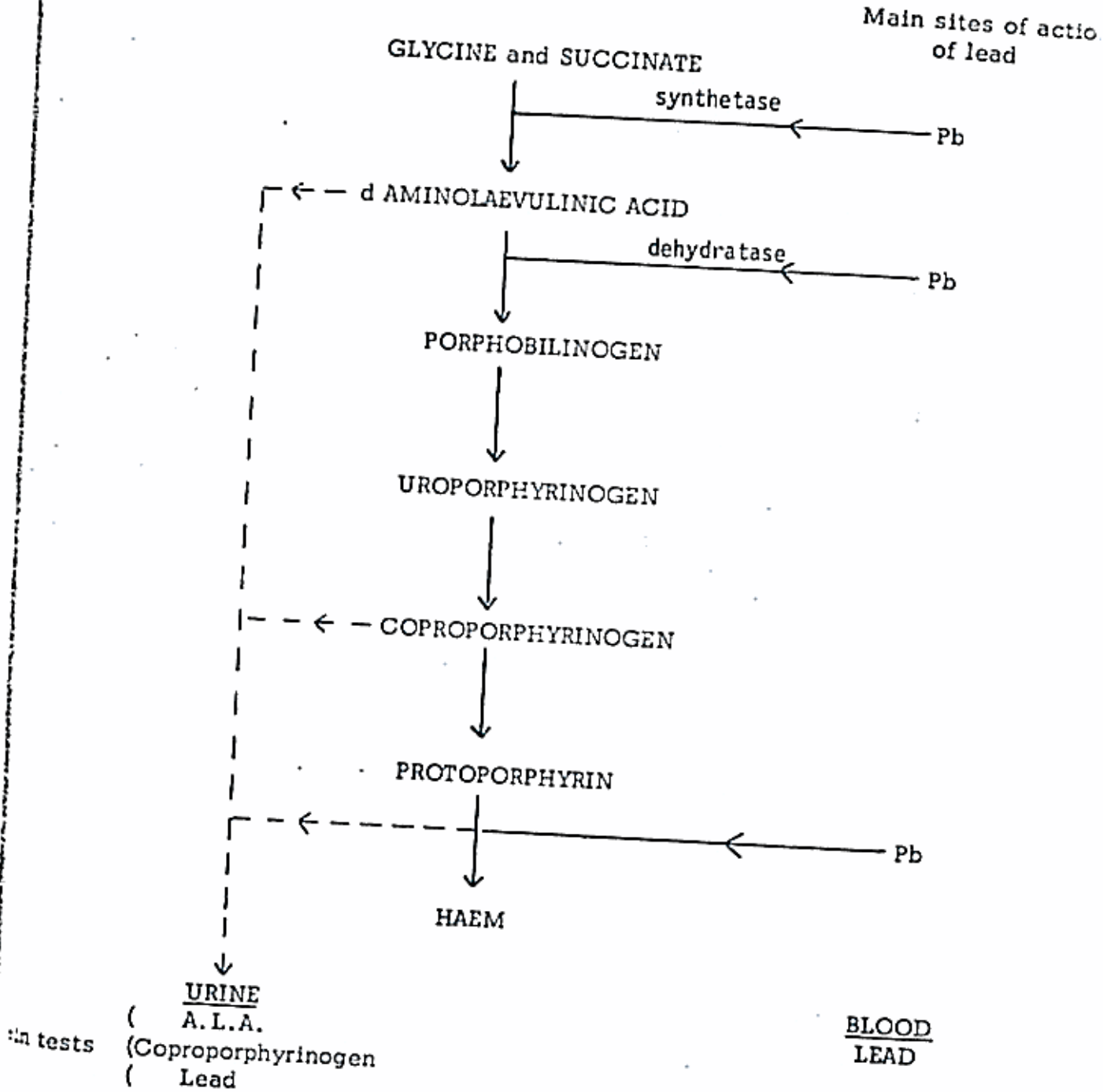
excess, depresses the action of various enzymes acting particularly on amino laevul inic acid (ALA). FIG. 6. In addition, it has been shown that lead reduces the life span of red cells. (DE BRUIN, 1971).

In adults, blood lead levels below 80 ug/100 mls have yet to be shown to affect haemoglobin levels (KING, 1971). In industry, a blood lead of 80 ug could be trusted as the earliest indication of the existence of widespread hazard (KEHOE, 1971). On the other hand it has been shown that blood lead levels of 40 ug/100 mls reduced the activity of alpha laevulinic acid dehydratase although the haemoglobin levels were unaffected (HERMBERG, NIKKANEN, 1970).(MILLER, 1970).

Betts, 1973, however, reported anaemia in nine children of

FIG. 6.

EFFECTS OF LEAD ON BLOOD

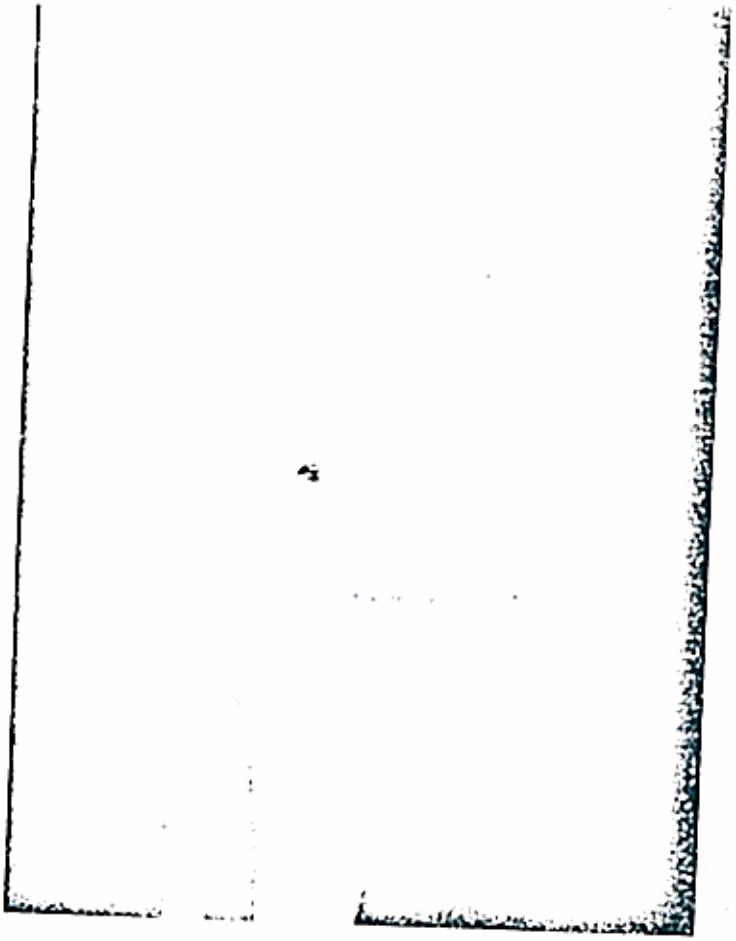


After SAYERS, M.H.P.
Medical aspects of lead absorption in industrial diseases.
LEAD DEVELOPMENT ASSOC. 1973

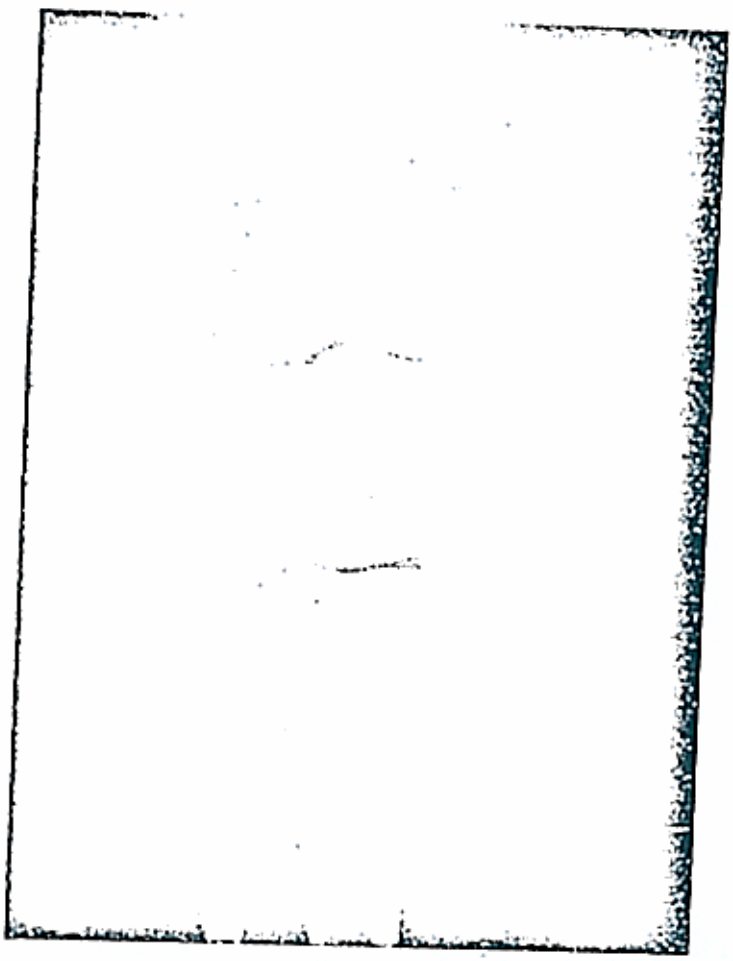
thirty eight examined with lead levels between 37 - 60 ug.

CONCLUSION

It would seem that infants can tolerate blood lead levels in utero of 37 ug/100 mls without adverse effects on their birth weights, haemoglobin concentrations, or packed cell volumes, as no significant differences were shown in these values compared with the controls. On the other hand, 20 (16%) mine infants were born with haemoglobins below 13.7 g/100, and might be considered anaemic. As there are so few published figures available for Zambian cord bloods it is possible that the values for haemoglobins obtained in the mine infants' are, in fact, within the normal range for this ethnic group.



Child Aged 2 Years
Blood lead 27 µg/100 cc



LEAD IN BONE AND BLOOD LEAD CONCENTRATION

OBJECT

This survey was carried out to determine if an X-ray of a long bone would assist in the diagnosis of children admitted to hospital with suspected lead encephalopathy. The X-ray being more readily obtainable than the blood lead concentration.

INTRODUCTION

In adults 90% of the body lead is stored in the skeleton. Once deposited in bone, lead is not easily released, except perhaps during conditions of fevers, acidosis, excess intake of alcohol and malignancy, consequently lead tends to increase in quantity and concentration with age. Young children, however, because of a relatively greater proportion of haemopoietic bone, are less able to store lead in this way.

This reduced capacity of lead storage in toddlers may account for their higher blood lead concentrations compared with older children and adults (BARRY 1972). Lead is selectively deposited as a triple phosphate at the metaphysis of long bones forming transverse bands which are opaque to X-rays. These bands are easily viewed in the very young, but after the age of five years the opacity merges with the increasing density of the surrounding bone and becomes more difficult to detect.

The lead band is related to duration and rate of lead exposure as well as velocity of growth. It is, however, not specific for lead as a similar appearance occurs commonly in normal healthy children and also with the deposition of bismuth

and of phosphorus. Furthermore, Park's stress lines especially when thick may also be confused with a lead line. In addition to the opacity of the lead line at the metaphysis, splaying of the ends of long bones occurs in chronic cases of lead intoxication (CAFFEY).

REVIEW OF LITERATURE

Betts et al, 1973 examined 52 children's knee X-rays and showed a correlation between the density and thickness of the lead line and the blood lead concentration.

The lead line was graded 'o' or '1' if considered to be a normal variant, '2' if lead poisoning was a possibility, and '3' if lead poisoning was considered certain. It was shown that, with one exception, grade 3 indicated blood

lead levels above 37 ug/100 mls, but grades 0, 1 and 2 failed to distinguish between blood lead levels ranging from normal to 60 ug/100 mls. LEONE (1968) examined 30 children and found no correlation between blood lead concentration and the lead line on X-ray.

PRESENT SURVEY

The present survey of 100 knee X-rays of children between the ages of 6 months and 6 years, and resident in Kabwe, Zambia, failed to produce a convincing correlation between the thickness or density of the lead line and blood lead concentration.

All the X-rays were taken by the same radiographer, using non-screen Kodirex film at constant focus film distance.

CORRELATION OF LEAD LINE IN BONE AND BLOOD LEAD CONCENTRATION

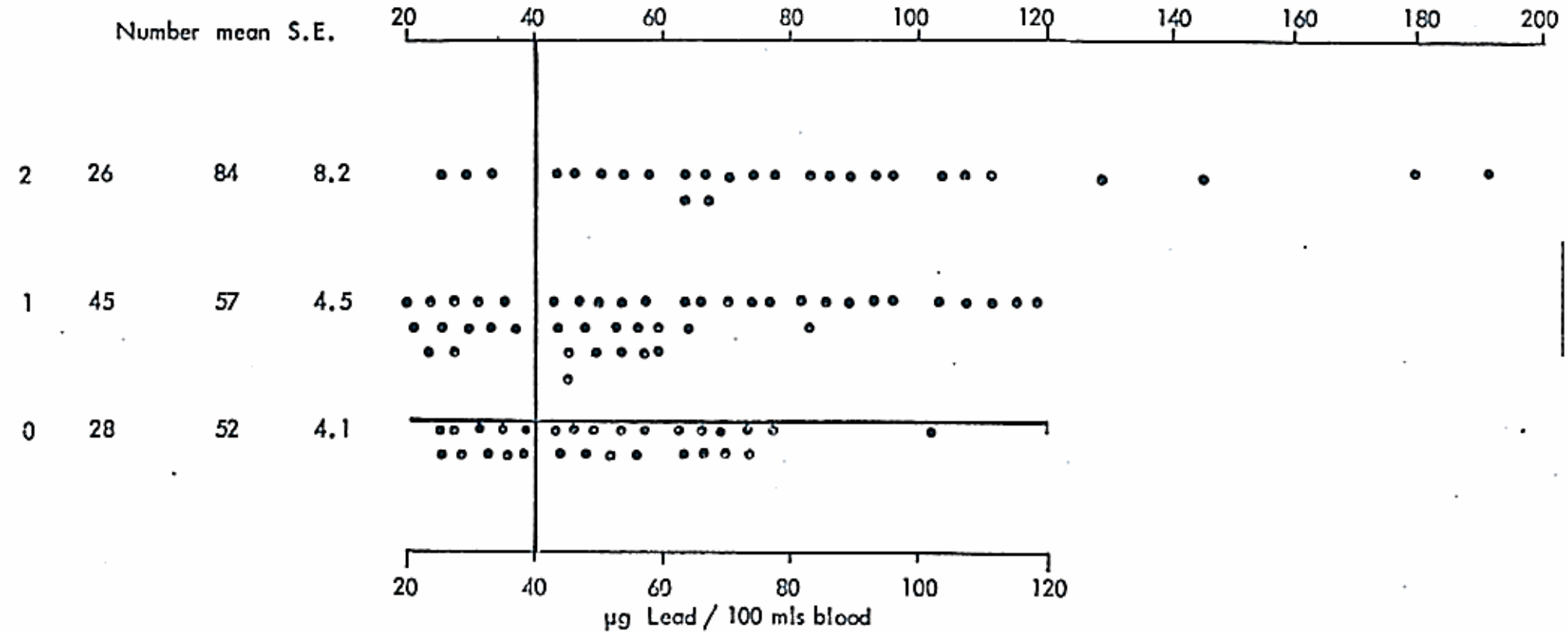


FIGURE 7

Lead line grade---0 = Less than 3mm.

1 = 3 - 4mm.

2 = More than 4mm

The thickness of the lead line at the distal metaphysis of the femur was graded '0' if considered to be within normal limits. (< 3 mm thickness) '1' if lead deposition was a doubtful possibility, and '2' if a high probability of lead deposition was anticipated, (> 4 mm thickness). The lead line was not always clearly demarcated which made grading difficult at times.

From the graph, (Fig. 7) it can be seen that grade '0' covered a range of blood lead concentrations from 24-105 ug/100 mls; although only one blood lead was over 78 ug, grade '0' could not be relied on to exclude lead intoxication. Grade '1' covered a range from 20-120 ug/100 mls, 33 out of 45 of the blood levels were above 40 ug/100 mls. This group included seven children who had never lived in

the lead area and whose blood lead levels were, expectedly, below 33 ug/100 mls. Four others also had normal blood lead concentrations but had formerly resided in an area with a high lead environment. This grade was similarly non specific. Grade 2 covered a wider range than the others; all with the exception of three, were above 40 ug/100 mls. This was the only grade in which a high probability of lead intoxication could be more confidently expected. Eighty two of the hundred children had spent most of their childhood in the high atmospheric lead environment of Kasanda. (Table 7[1]). There appeared to be no clear relationship between duration of exposure to lead and the radiological grade.

TABLE 7[1]

RADIOLOGICAL GRADE AND DURATION OF EXPOSURE TO LEAD OF 82 KASANDA CHILDREN

Radiological Grade	EXPOSURE		
	Under 2 yrs.	Over 2 yrs.	Over 4 yrs.
2	9	7	7
1	6	19	12
0	13	8	1

However, when the mean blood lead concentrations of the 82 Kasanda children are considered in relation to their age and radiological grade, then those under 2 years of age with a grading of 2 have highly significant lead levels, they are in fact in the lead encephalopathy range.

TABLE 7[2]

MEAN BLOOD LEAD CONCENTRATION

Radiological Grade	AGE		
	Under 2 yrs.	Over 2 yrs.	Over 4 yrs.
2	125.2 ± 7.2	65.77 ± 6.2	61.57 ± 7.6
1	64.28 ± 12.1	65.89 ± 8.1	59.7 ± 3.8
0	54.9 ± 13.7	71.33 ± 11.2	64.0

Mean blood lead $\pm$ 1 Standard Error

Factors mitigating against a good correlation are, first, that in acute cases of lead intoxication, although the blood lead is raised, there is insufficient time for lead to be deposited in sufficient quantities to be detected radiologically. This occurred in one case graded 'O' in which the blood lead was 105 ug/100 mls. Secondly, patients removed after long periods of residence in a high lead environment, have high blood leads which fall at a faster rate than lead can be eliminated from the bone. This occurred in two patients who were of grade 2, and whose blood lead levels were below 40 ug/100 mls. Thirdly, because in some children unexposed to an abnormal lead environment, an opacity may occur at the metaphysis which is similar in appearance to that of a lead line, 14 children, who had never lived

within a high lead environment and had blood levels of 40 ug/100 mls or less, 5 of these were of grade 'O', 8 of grade '1' and 1 of grade '2'. Finally, it has been shown that blood lead concentrations fall after the age of 2 years, even if the children remain exposed to a high lead environment (Fig.3[1]). If this fall in blood lead level is due to an increased deposition of lead in bone, the absence of correlation between blood lead concentration and lead grade is even further emphasized.

CONCLUSION

No satisfactory correlation between the appearance of the lead line radiologically and the blood lead concentration was apparent in this series. Such an X-ray is no more than a diagnostic aid in cases of chronic lead poisoning. However, a higher probability of lead intoxication may be

LEAD ENCEPHALOPATHY TREATED WITH ORAL PENICILLAMINE BP

INTRODUCTION

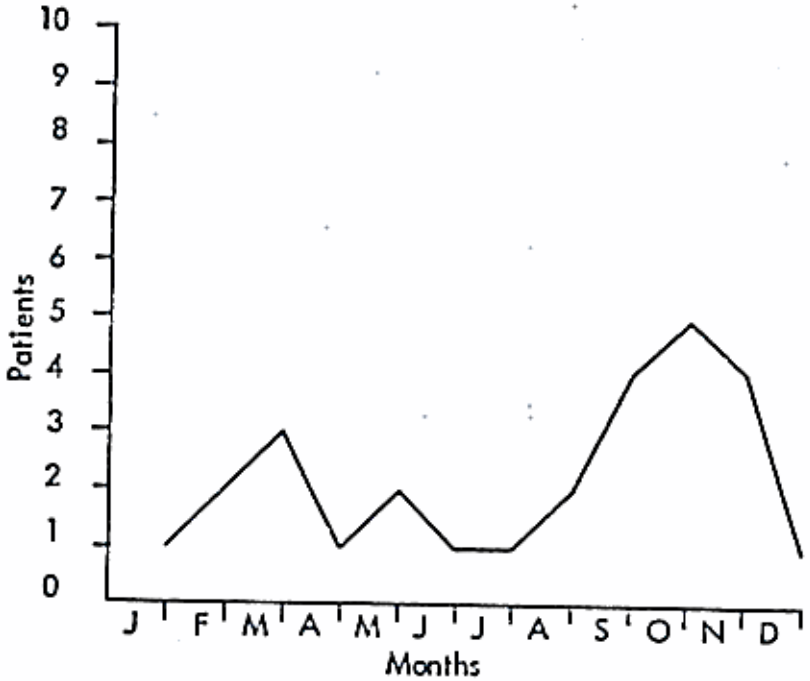
Over a period of $2\frac{1}{2}$ years, 27 children were admitted to Kabwe General Hospital, Zambia, suffering from lead encephalopathy. They were between the ages of 10 - 30 months with one exception, aged 4 years. All were residents of Kasanda, a Mine Township within a radius of 3,000 metres of Broken Hill Lead Mine. Kasanda's population was 11,000 up to 1973, when it was subsequently reduced following a rehousing scheme to 8,000.

ANNUAL, SEASONAL INCIDENCE

During the period unver review, only one case was admitted during 1971, 14 in 1972, 11 in 1973 and in 1974 up to the

FIGURE 8

LEAD ENCEPHALOPATHY PATIENTS
ADMITTED TO HOSPITAL



time of writing in July only 2. Most cases were admitted during the months from September to November which is the end of a dry and dusty season. Fig. 8 and Fig. 2^3, 2^2.

PRESENTATION

The ratio of males to females affected was 12 : 15. All presented with generalised convulsions, twenty one were noted to be in coma. (Table 8.) Four presented with hydrocephalus (Cases 3, 4, 11, 13). The first case seen was paralysed in both legs for 3 weeks following recovery from coma. An external rectus palsy occurred in seven of the infants, (Cases 4, 11, 12, 16, 17, 18, 27). Malnutrition was common to all, but four were severely malnourished, (Cases 13, 15, 25, 26;) the mean weight of the infants was

8.5 kg. All were aneamic, their mean haemoglobin being 9.6 g%.

DIAGNOSIS

In order that there should be no delay in initiating life saving treatment, all children presenting with convulsions were considered to be suffering from lead encephalophathy and treated for such until proved otherwise. However, the final criteria for diagnosis depended upon the triad of cerebral dysfunction, i.e. convulsion and/or coma; a blood lead above 90 ug/100 mls; and an increase in cerebro-spinal-fluid protein content, as well as pressure, with little or no change in the other elements.

DIFFERENTIAL DIAGNOSIS

Other causes of convulsions and/or coma needed to be excluded, particularly malaria, meningitis, sickle cell disease, severe anaemia, upper respiratory tract infections

and hypoglyaemia.

TREATMENT

Convulsions were controlled by intramuscular paraldehyde 1½ c.c./5 kg. body weight, and following a complete physical examination, a lumbar puncture was performed, and blood taken for haemoglobin, white cell count, lead concentration, sickling of red cells, and a blood slide examined for malaria parasites. Each child's abdomen was X-rayed to exclude ingested lead, also a knee to detect the presence of an opaque lead line at the metaphysis; these being simple and convenient diagnostic aids in suspect cases of lead poisoning.

Through a naso-gastric tube, penicillamine tablets, crushed and mixed with a little water was introduced into the stomach, in a single initial daily dose of 60 mg/Kg body weight and subsequently single daily doses of 30 mg/KG.

It was considered vital that this treatment was started without waiting for confirmation of the blood lead concentration, and could be discontinued if the case proved not to be one of encephalopathy. An appropriate antibiotic was given to combat any accompanying respiratory or alimentary infection. Fluid electrolyte balance and anaemia were corrected as necessary.

CULTURAL DIFFICULTIES

The local population invariably sought traditional treatment for convulsions, bringing their infant to hospital only after this had failed. The response to oral penicillamine was remarkably good, most infants admitted in coma regained consciousness within a few days of treatment. Mothers would then agitate for their infants to be discharged. As by this time the blood lead was still very high, discharge to

their polluted home environment could not be risked. Consequently, the course of penicillamine was prolonged to 12 - 14 days and mothers seeing that treatment was continuing desisted in their agitation for discharge. Even so, several absconded with their infants against medical advice. For the few who co-operated a second course of treatment continued after a few days rest, for a further week. The opportunity to continue treatment to this stage or beyond was seldom permitted, but in 10 cases following a second course of treatment the blood levels were below the critical level of 90 ug/100 ug. Unfortunately, follow up of cases after discharge from hospital was the exception, as mothers were still very reluctant to bring infants for check-up.

MORTALITY

Of the 27 cases there were 4 death,(14.8%). In only two of these was death solely attributed to lead encephalopathy, and in these there was a possibility that treatment was not started promptly on admission. Another infant had a complicating severe broncho-pneumonia (Case 17), and the fourth was an extremely malnourished, and neglected twin, with a history of convulsions for five months prior to admission.(Case 13.)

CASE HISTORIES Fig. 9.

Case 1: Male infant, aged 1 year 2 months. Admitted December 1971, with sudden onset of convulsions, followed by coma O.E. malnourished infant, small for age, weight 6.8 Kg, comatose with occasional generalised convulsions. Temp. 99. Convulsions were controlled by I.M. paraldehyde

CASE No. 1. MALE AGED 1 YEAR 2 MONTHS. ADMITTED DEC. 1971

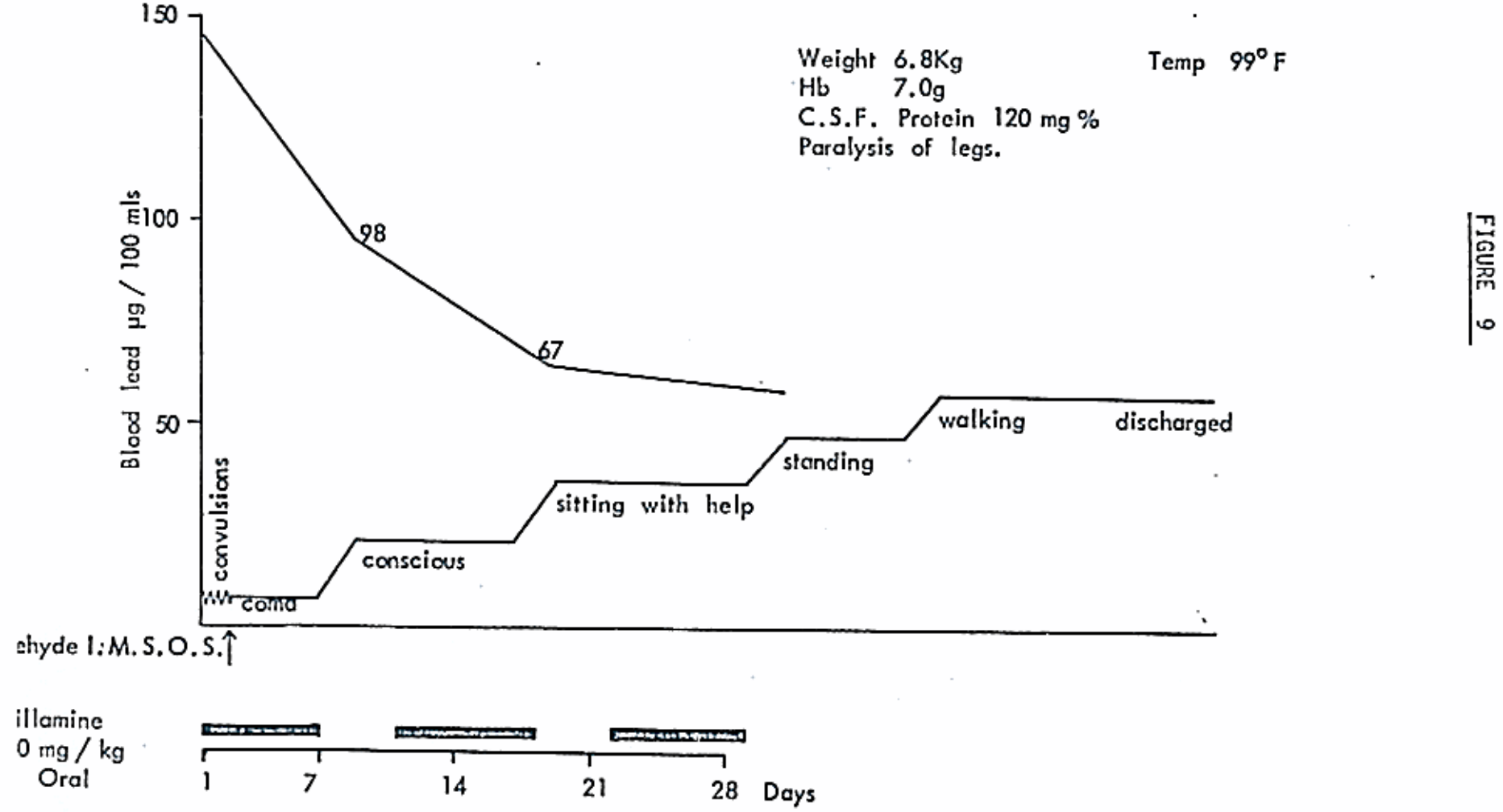


FIGURE 9

2 c.c. Following a complete physical examination, lumbar puncture was performed and blood taken for Hb, malaria parasites, blood lead estimation and sickling. Suspecting lead encephalopathy, penicillamine was started in a dose of 30 mg/Kg body weight daily, given via intra-gastric tube while awaiting laboratory results.

After two days the infant appeared to be responding and was fully conscious by the sixth day. Its legs however, remained flaccid for a further three weeks although daily improvement was observed. Following the first 7 day course of penicillamine the blood lead dropped from the original 145 ug/100 mls to 98 ug. After a few days rest a second course began which after a week resulted in a fall of blood lead to 67 ug. This was the unit's first case of lead encephalopathy treated with

penicillamine alone. Subsequent cases were treated with a 1st day loading dose of 60 mg/Kg body weight as opposed to the daily dose of 30 mg/Kg body weight and continued for 7-10 days according to response but later the initial course was prolonged to 10-14 days according to response and blood lead level.

The policy of interrupted courses of treatment was adopted to obviate any likely depressant action of penicillamine on the bone marrow. The large initial loading dose and subsequent daily doses up to 14 days dependent upon the response of the infant appeared to be without any complication.

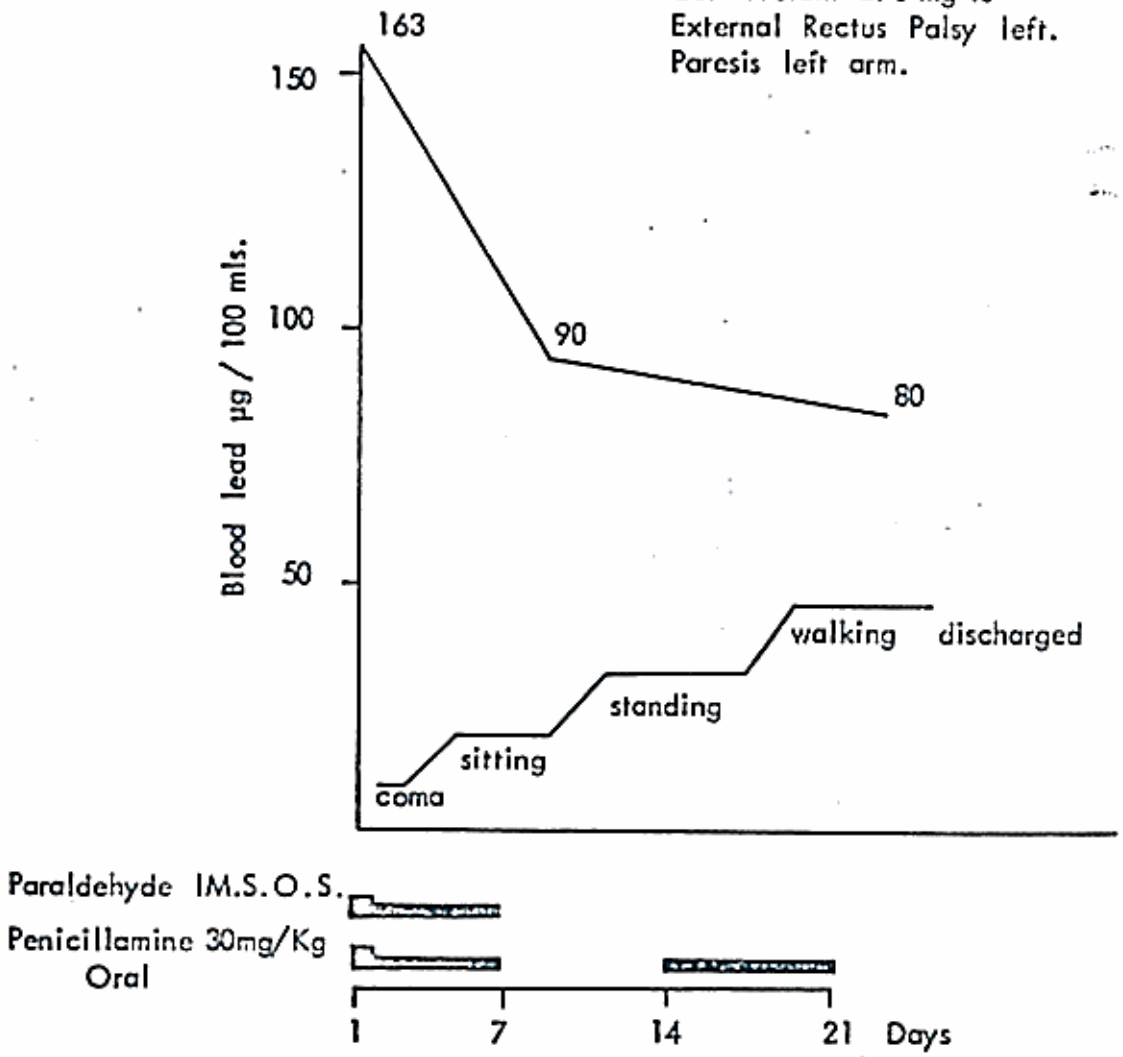
CASE No. 12 Fig.10.: Male infant, aged 1 year 6 months. History of sudden onset of coma with convulsions

FIGURE 10

Case No 12. ADMITTED SEPT. 1972

MALE AGE 1 YEAR 6 MONTHS.

Weight 9.07 Kg
Hb 11.1
CSF Protein 276 mg %
External Rectus Palsy left.
Paresis left arm.

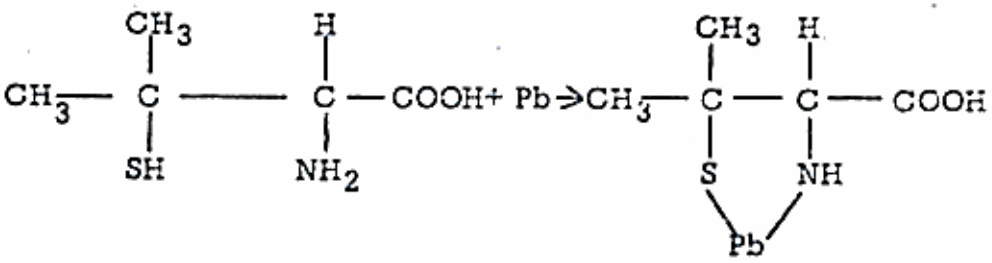


O.E. Deep coma with spasms of generalised convulsions and left external rectus palsy. Convulsions controlled with paraldehyde and patient examined and investigated routinely for suspected lead encephalopathy. Treatment began with penicillamine in an initial dose of 60 mg/Kg body weight and continued at a daily dose of 30 mg/Kg. The infant responded in a manner which had become expected, being conscious by the third day and standing by the end of a week. The external rectus muscle was by then normal but a paresis of the left arm persisted for another week.

The blood lead originally 276 ug/100 mls dropped to 90 ug after the first 7 days course and was 80 ug following the second.

PENICILLAMINE

Penicillamine is a very effective chelating agent. Its action being dependant upon the presence of a-SH group in its molecule. Having combined with lead, the new compound is excreted in the urine.



It was first introduced by Walshe in 1956 for the treatment of Wilson's disease and has been used in lead poisoning since 1957. (Boulding & Baker 1955).

Harris(1958) compared intravenous calcium disodium versenate with oral penicillamine in two cases of lead

poisoning, and concluded that lead elimination was slightly greater with versenate than with penicillamine, but that the ease of administration and lowered toxicity outweighed the 'trifling superiority' of versenate.

Chalmers and Whitehead (1964) found oral penicillamine reliable in eliminating lead, and that its toxicity was slight, but owing to its nephrotoxic effects recommended intermittent therapy. Selander et al (1966) found no therapeutic advantages in giving penicillamine intermittently. They also compared versenate with penicillamine and found the latter a good alternative, but considered that versenate should possibly be preferred in severe cases.

Simpson et al (1964) treated a case of chronic encephalopathy

using penicillamine initially for three days, then followed this with versenate and found that the output of lead was greater with penicillamine.

TOXICITY OF PENICILLAMINE

Toxic effects of penicillamine have been reported. Skin sensitivity is said to occur in 30% of cases (Schienburg 1967). Renal damage was first reported by Goldberg (1963), the earliest sign being protein uria. It was thought that the toxic effect was on the tubules, but kidney biopsy in three cases showed focal glomerulitis (Rosenburg & Hayzlett, 1967). Granulocytic leucopenia occurs often in the early weeks of treatment, but improves spontaneously when treatment is stopped. Agranulocytosis has been reported on several occasions, and there has been one death recorded.

(Carlos et al 1964, Conway and Walker, 1967, Selander & Cramer 1965, Golding, 1968).

COMPARISONS OF TREATMENTS USING

a. Edathamil calcium disodium (EDTA) alone.

b. EDTA, combined with 2, 3 - dimercapto-1-propanal (B.A.L.).

c. Penicillamine alone.

Established methods of treatment involve either EDTA alone or EDTA combined with B.A.L.

Whitfield et al 1972 reviewed 23 adult cases, 8 of which they saw personally, treated with EDTA alone and noted a delayed response due to cerebral oedema. There were three deaths. Mortality 13%.

Chisholm (1968) noted that in children treated with EDTA

alone, further cerebral impairment as well as risk of anuria occurred during the first 48-72 hours. He recommends a combination of BAL and EDTA. His work, plus that of Coffin, describes 46 children with acute encephalopathy, 26 of whom had severe disease, similarly treated, with only one death. On the other hand, Greengard and associates report that 5 of 11 children with encephalopathy who were treated with the BAL, EDTA combination died. This striking difference requires explanation. Dosage and mode of administration of BAL and EDTA were similar in the 3 series (CHISHOLM 1968). Mortality 10.3%.

This fair comment of Chisolm's is interesting, but it is very likely that different responses to similar treatments reflect differences in severity of the encephalopathy. Previous experience in Kabwe using I.V. EDTA and BAL resulted in all 8 children dying but at the time there was general inexperience

SUMMARY

Oral penicillamine was used in the treatment of 27 cases of lead encephalopathy in children between the ages of 10 months and 4 years. Their response was prompt; most cases regaining consciousness within a few days of treatment.

There were no toxic manifestations apparent and there were only four deaths; in only two of these, however, was death attributed solely to lead encephalopathy. This mode of treatment was found particularly effective and suited conditions prevailing locally, where skilled personnel were not always available to administer intravenous therapy.

CASE NO.	SEX	AGE yr.	AGE mths.	WEIGHT KG.	CONVULSIONS	COMA	OTHER PRESENTING CLINICAL FEATURES	ON ADMISSION Hb g%	ON ADMISSION CSF Protein mg%	ON ADMISSION Pb/ug 100 mls Blood	Penicillamine days of Treatment @ 30 mg/Kg Body weight	Blood Pb After-Treatment	Progress	Blood Pb following 2nd course Penicillamine	
1	M	1	2	6.8	+	++		7	120	145	7	98	Comatose 6 days, Paralysed in legs 4 weeks	67	Fully recovered.
2	F	1	2	6.8	+	+		8.3	250	245	10	75	Quick recovery	74	Fully recovered.
3	M	1	0	7.26	+	++	Hydrocephalus; skull circumference 20".	9.2	98	156	7	80	Comatose 3 days		Fully recovered.
4	M	1	0	8.16	+	++	Hydrocephalus and widely separated sutures. Ex-rectus palsy.	8.4	100	200	8	147	Comatose 3 days, Sat up 10th day.		Fully recovered
5	M	1	6	8.16	+	+		10.6	158	205	8	146	Prompt recovery	87	Fully recovered
6	M	2	6	11.3	+	+	Upper respiratory infection.	8.4	160	154	10	120	Comatose 2 days, recovered quickly	87	Absconded
7	F	1	7	8.62	+	+		9.5	136	125	9	114	Comatose 3 days, recovered quickly	84	Fully recovered
8	M	1	6	9.5	+	+		8.2	80	170	10	90	Comatose 2 days.	94	Fully recovered
9	F	1	0	8.16	+	+		8.6	140	140	10	76	Comatose 2 days.	71	Fully recovered
10	F	1	6	8.31	+	+	Bronchitis	12.5	132	385	7	195	Slow progress. Athetoid movmts.	120	Absconded
11	F	0	11	7.26	+	+	Distended ant; font: Ex rectus palsy.	10.1	62	321	10	205	Responded quickly	105	Absconded
12	M	1	6	9.07	+	++	Ex rectus palsy	11.1	276	163	7	90	Coma 2 days. Walking 12th day.	80	Fully recovered
13	F	1	4	6.8	+	++	Hydrocephalus. Severe malnutrition.	10.5	212	405	-	-	Died within 24hrs.	Lead encephalopathy	
14	F	1	0	8.16	+	++		9.2	168	230	-	-	Died within 23hrs.	Lead encephalopathy	
15	M	1	10	10.0	+	+	Marasmus	8.9	204	210	7	115	Coma 2 days. Sitting 3rd day		Absconded
16	F	1	0	5.9	+	+	Ex rectus palsy	10.4	120	161	14	98	Very slow progress, Athetoid movmts.		Fully recovered
17	M	1	6	9.07	+	+	Bronco-pneumonia; Ex rectus palsy.	9	140	256	-	-	Died within 24hrs. Lead encephalopathy	Pneumonia	
18	M	1	3	8.25	+	+	Ex rectus palsy	8.8	65	166	14	101	Very slow progress Sitting up 18th day	73	Absconded
19	F	2	6	11.3	+	+	Bronchitis.	11.6	48	127	7	85	Recovered quickly	-	Fully recovered
20	M	4	0	13.6	+	+		13.8	84	90	10	70	Coma 2 days, weak for 11 days	-	Fully recovered
21	F	1	4	6.8	+	++	Broncho-pneumonia.	8.5	236	235	14	96	Coma 5 days. Good progress	-	Absconded
22	M	1	0	6.8	+	+		9.5	74	134	10	123	Recovered quickly	-	Absconded
23	F	1	7	9.07	+	++		9.6	94	203	-	-	Died within 24hrs.	Lead encephalopathy	
24	F	0	10	6.8	+	+		9.6	134	131	10	80	Recovered quickly	Fully recovered	
25	F	1	6	9.07	+	+	Severe malnutrition.	7.9	160	160	-	-	Comatose 3 days	-	Absconded
26	F	1	11	11.0	+	+	Severe malnutrition, Microcephaly.	8.0	100	240	14	110	Recovered quickly	-	Absconded
27	F	1	3	7.77	+	+	Ex rectus palsy. Rt. facial palsy	9.5	120	230	8	90	Coma 3 days; Right facial palsy	55	Fully recovered

PREVENTION OF LEAD POLLUTION AND INTOXICATION

ENVIRONMENTAL FACTORS

The prevailing wind reaches speeds up to 18 knots throughout the year and carries lead particles from the effluent of the mine and the ground in its vicinity to the Kasanda-Makalulu area. The emission from plants needs to match stack height in order to reduce community atmospheric pollution to a minimum. Complete enclosure and exhaust system are desirable but difficult to introduce in older furnaces. Much has already been done to reduce lead in the effluent from the sinter and smelter furnaces, but still there remains the problem of wind-borne ground lead

contribution the barriers need to be continuous and extend across the entire width of 'waste' land. A quicker but temporary method requires a reticulated water pipe system with sprinklers to keep the surface of the waste area moist and thereby reduce wind take-up of the soil. Such watering might encourage growth of grass which would bind the soil and prevent its further erosion. Watering should be started in May and continued until the rains which can be expected in November.

Because of the water shortage, in the dry season, sprinkling should begin after the heat of the day, when evaporation is least, or during the day if dust clouds are exceptionally severe. Traffic leaving the plant area should have a wheelwash to reduce take-out lead.

EXHAUST FROM MOTOR TRAFFIC

A further contribution to atmospheric lead comes from motor vehicle exhaust. This is not particularly significant just now, but may become so as the traffic flow through Kabwe continues to increase. Lead emission from this source varies directly with the speed of the engine (McCallum 1972). There is therefore a case for ensuring traffic is confined within safety speed limits through the town. Further, all roads should be tarred; this has mostly been done in order to reduce dust created by traffic within the townships.

PROTECTION OF FOOD

Zambians are outdoor people; in their gardens they cook,

children play and families gather. These cooking areas, if asphalted, would also help to reduce dust from being blown into cooking pots and homes, and preclude infants from direct contact with lead contaminated soil during their play.

Areas around shops should similarly be asphalted and all food on display adequately protected from lead fall out. This applies particularly to such foods as dried meat, fish, bread and fruit which require very little preparation before eating; it being shown that the degree of lead contamination of food is proportional to its exposure time (Marks, 1974).

Unlicensed vendors should be discouraged from selling food

in the townships simply because they will not conform to the required hygiene standards.

Maize grown in the Kasanda area has a high lead content. This crop forms part of the staple diet of local people but in order to reduce its lead content should be cultivated in areas where the ground lead and atmospheric lead is least - that is, in areas further west or north of Kasanda.

WATER CONTROL

Domestic water comes from bore holes and is pumped over lead polluted ground. The pipes are often illegally tapped and thereby the water may become contaminated. It is therefore necessary that the pipes are inspected routinely to exclude damage, and water samples taken periodically to

ensure it is not polluted.

CONTROL OF LEAD ON CLOTHES AND PERSON

Mine employees are liable to bring home lead particles on their clothes or person. They should therefore travel to work in their own clothes , change into disposable shirt and clean overalls on arrival, and store their own clothes in dust proof lockers, double lockers preferably, one for clean clothes, the other for dirty. At the end of their shift, they should change and shower completely, before returning home in their own clothes. Protective clothing is now being designed for workers in lead industries, providing caps, overalls buttoned up at the neck and changeable overshoes. In the heat of Kabwe, buttoned up clothes are unlikely to be tolerated. Head gear will need to remain the hard hat / in accordance

with safety regulations, and footwear will contine to be boots or steel capped shoes. Ideally these should be changed before leaving the plant area but little is to be gained from this if men need to walk over lead contaminated ground on their way home.

HOSPITALISATION

Patients with lead intoxication must be removed from the polluted environment before chelating agents are prescribed, otherwise they may sustain renal tubular damage.

The principle of removing employees with high lead from their work environment is an accepted one. Similarly, infants with high lead should be removed from their home

environment, especially as they may risk cerebral damage if lead treatment is not begun promptly. Mothers are reluctant to allow their children in to hospital because it is too far away from their homes and there is no other person to care for their other children left at home while they are visiting. To encourage a mother to allow her affected child to be hospitalized for treatment, nearby accommodation could be provided for her and her other children. This idea is not new to Africa and its revival in Kabwe would go far to promote better care for lead affected infants.

Three thousand persons have already been rehoused in good homes in CHOWA: to rehouse the remaining 8000 Kasanda inhabitants should not be necessary provided adequate

lead control measures continue to be enforced.

MONITORING

As part of the control programme it is important that monitoring of the atmosphere as well as biological monitoring continues. An important reflection of the blood lead state of the community is provided by blood sampling of mothers routinely at their first ante natal visit, as well as at delivery. The mother's blood lead being only a few microgrammes above that of her newborn infant's, it gives a measure of the inherited blood lead concentration and is also a useful clue to that existing in toddlers, as they invariably have normal leads when mothers are below 20 ug/100 mls and high leads when mothers lead levels approach 40 ug/100 mls.

EDUCATION

For any new health measure to be successful, it is necessary to obtain the co-operation of those directly involved. This often involves the child's aunt or uncle rather than its parent. Much can be done by a gradual system of education; here the community health nurse has an important role to play in the prevention of lead intoxication. She must obtain the confidence of local families so that she becomes a welcome visitor to the homes of the children with raised blood lead. Her special knowledge may help to determine factors contributing to intoxication. She should advise on personal hygiene, food preparation and storage and dust reduction. She may be able to help a mother who has to leave her home to visit a sick child in hospital, and arrange for children to attend the hospital for treatment when necessary. The co-operation of community services and unions through management is essential.

CONCLUSIONS

The purpose of this series of investigations was to enquire into the extent and severity of the lead problem in Kabwe. In so doing it has been possible not only to view the problem in perspective, but also to establish base lines for future epidemiological studies.

The body burden of lead depends upon the cumulative difference between the amounts of lead absorbed and excreted. Lead enters the body:

1. After crossing the placenta
2. Following ingestion
3. By inhalation

and is excreted via the urine and faeces, i.e.

$$B = (A - E)$$

B = Body burden
A = Amount absorbed
E = Amount excreted

An equilibrium is reached when excretion is equal to absorption. The time taken to reach half this equilibrium is the biological half life, which is of the order of several months.

This investigation shows that, of the four communities situated within a radius of approximately 3000 metres of the Kabwe mine smelter, only two, namely Kasanda and Makalulu were exposed to a high atmospheric lead environment. These two communities lay in the path of the prevailing wind blowing from the mine. This wind carried lead particles from the mine smelter stacks and from the ground in their vicinity.

Of the other two communities, the Municipality inhabitants

bore lead loads similar to the those found in most urban dwellers, whilst the Chowa lead loads were intermediate between Kasanda and Municipality having been removed from the former heavily exposed area to a cleaner one.

Placental Transfer of Lead

It was shown that a significant correlation between maternal blo and cord blood lead concentration was present even at very high lead concentrations.

The inherited mean lead load of the Kasanda newborn infants was 37 ug/100 mls - a level not before recorded in the literature. This level is close to the accepted 'normal for children'. It was therefore necessary to determine if this high level at birth affected infant birth

weight and their red cell values.

No significant differences, however, were noted in these parameters between the Kasanda and Municipality controls.

Haemoglobin Concentration of the Zambian Newborn

The mean cord blood haemoglobin concentration in both Kasanda and Municipality infants were not significantly different from those of Professor Egeilo's seventy Lusaka infants. Thus Zambian infants are born with a lower level of haemoglobin than Europeans, though this may not mean they are physiologically disadvantaged.

Blood Lead Levels in Children

Children exposed to a high atmospheric lead environment have high blood lead levels, this was most striking in

infants between the ages of 1 - 3 years. The Kasanda infants had a mean blood lead level of 103 ug/100 mls between the ages of 1 - 2 years, decreasing in older age groups. For any given atmospheric lead concentration blood lead level appears to be related to body surface area, or in other words to relative respiratory dose, : One litre of inhaled oxygen is needed in metabolism to produce approximately 5K calories. Calorie requirement at age 1 per hour is about 55Kcalories/m^2 body surface, at age 2 years 50Kcalories/m^2 body surface reducing in successive ages to about 30Kcalories/m^2 at age 60 years. Therefore, infants require more oxygen per body surface area than older children and thus will inspire a larger dose of any contained air pollutant, in this case, lead. That this factor should be

taken into account has also been suggested by Knelson, J.H. (1974). Thus, it is important to state a child's age in years or months when referring to its blood lead concentration.

Lead Encephalopathy

Although cases of lead encephalopathy can be anticipated at any month of the year, September and October was the period when most cases were admitted to hospital. This being the end of a very dry spell when the winds are strongest and the air hottest.

Twenty seven children ages 10 months to 4 years were admitted between 1971 and 1974, convulsing and in coma.

They were treated with penicillamine in an initial dose of 60 mg/kilogram body weight, followed by a daily dose of 30 mg/kilogram body weight; 23 responded remarkably well, 4 (15%) died. One of these was severely malnourished and another had severe broncho-pneumonia. The penicillamine treatment proved clinically satisfactory in its ease of administration and its effectiveness and not inferior to established regimes. Fewer and less severe toxic manifestations were experienced, than might have been expected with other regimes, it is therefore recommended in the treatment of lead encephalopathy in similar circumstances.

Future Investigations

It is planned to follow up the cohorts whose cord blood

lead have been determined, and all those children who have suffered from lead encephalopathy, to assess their future physical and mental development.

Although no evidence relating raised blood level and terotogenicity is so far apparent this matter should be further explored, certain authorities have expressed concern as to the possibility of tetragenic or mutagenic effects.

Lead ingestion is a traditional method of procuring abortions. The association should be more critically examined. In Kasanda, no association has been observed between high blood lead levels and subsequent abortion. Furthermore, no high lead levels were noted in some 15 blood estimations on those women who had recently aborted.

Evidence is accumulating that negroes in America with sickle cell disease are more prone to haemolysis if they have raised blood lead levels. Sickling occurs in about 1.5% of the children in the Kabwe area, the opportunity therefore to study the relationship between sickling in a community with high lead environment should be taken.

Glucose-b phosphate-dehydrogenase deficiency is reported to be common in negroes but its association with a raised lead has yet to be ascertained. This matter will need a pilot study to determine the incidence of the enzyme deficiency in the local population to see if it also should be further studied.

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APPENDIX I

LEAD IN AIR SAMPLING

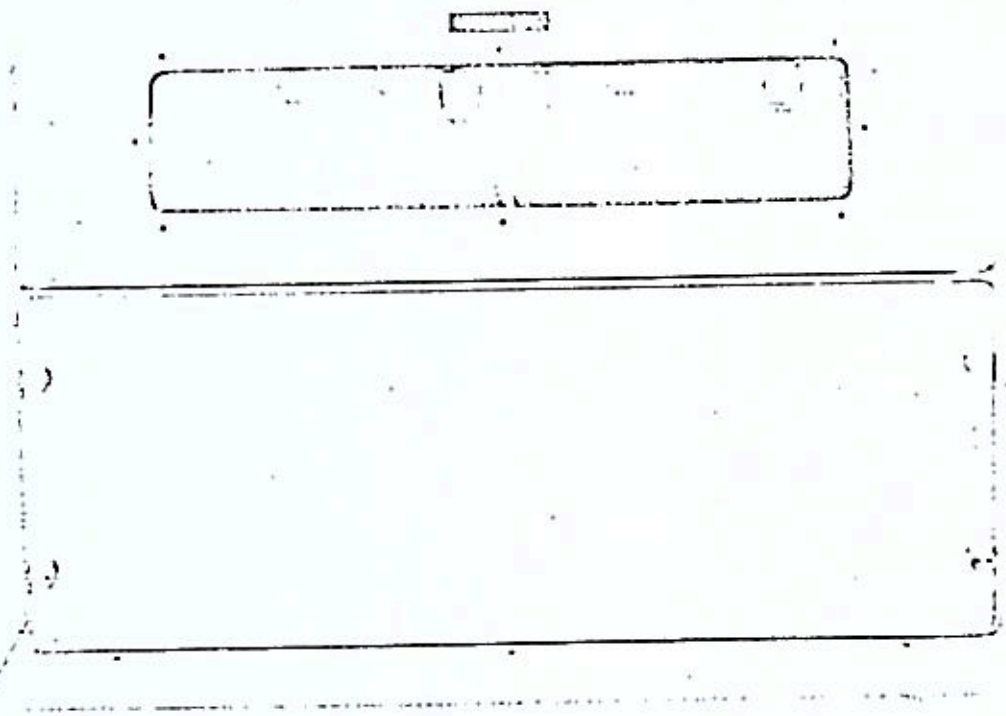
Atmosphere lead was measured by drawing samples of air through an 8 port valve, continuous, semi automatic sampler.

The air passing over a Whatman No. 2 filter paper for 24 hours in each successive port.

The filter paper with its filtered lead was digested with 15 mls of 10% nitric acid and boiled. The whole then filtered using Whatman 541 paper and the residue washed, the filtrate then boiled down to 10 mls cooled and diluted with 10cc water.

A reagent blank and standard solutions were prepared in a similar manner. Standards were prepared by the addition of aliquots of 10 ppm lead solution. Lead in the solutions was estimated by atomic absorption spectrophotometry.

SEMI-AUTOMATIC



AUTOMATIC OPERATION FOR 8 DAYS

MANUFACTURED TO D.S.I.R. SPECIFICATION

1in., 2in. or 4in. FILTERS

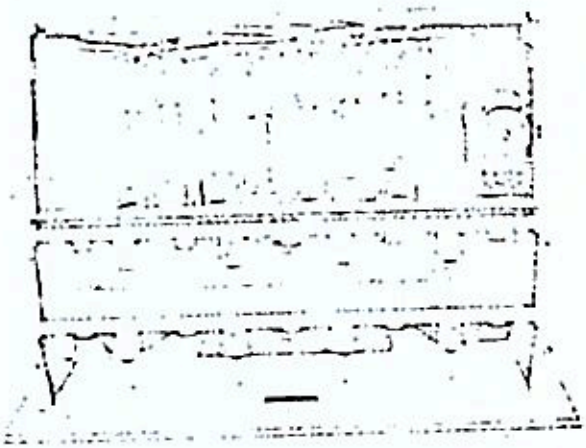
OPTIONAL TIMER FOR 8 READINGS IN 24 HOURS

SEMI-AUTOMATIC SMOKE AND SO_2 SAMPLER

Semi-Automatic Smoke and SO_2 Sampler, eliminates the need for daily replacement of smoke filter and hydrogen peroxide bubbler by automatically switching the air intake once every 24 hours to pass through a next adjacent filter and bubbler. A sequence of seven consecutive daily measurements can be made in this way, this being a particularly useful device for covering weekend periods. It can be used for any number of consecutive days without the necessity to seal off any unconnected ports.

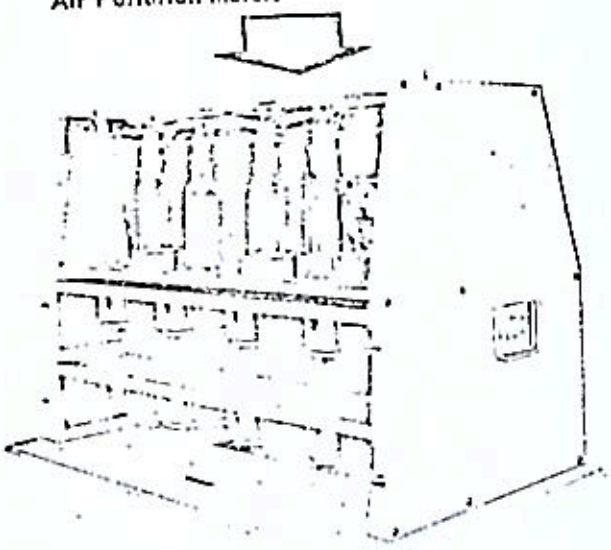
The 8 port valve, normally arranged to change filter and bubbler once in 24 hours can be extended by a simple switch operation to give a port change each 3 hour period i.e. 8 tests in 24 hours.

Alternative size filter clamp mounting is provided for 1in., 2in. and 4in. filters the equipment being fitted with 2in. clamps as standard.



The illustration shows the full accessibility of the filters and bubblers with the front cover removed.

Showing the position of the dial of the Air Pollution Meter.



Electrical Rating 200-250 Volt A.C. 50 c/s.

Overall Measurements

Length 30 inches

Width 18 inches

Height 20 inches

The complete instrument fitted with 2in. Filter Clamps and port change control once in 24 hours

Price: £110.0.0.

Extra for alternative port change control from 24 hours to 3 hour by simple switch

£15 - 0 - 0

The equipment is fully guaranteed and embodies quality materials and components throughout.

Technical literature available on request.

ENGINEERING DEVELOPMENT LTD

PRECISION TOOLS & INSTRUMENTS · ELECTRO MECHANICAL ENGINEERS

VERULAM ROAD · HITCHIN · HERTFORDSHIRE · ENGLAND

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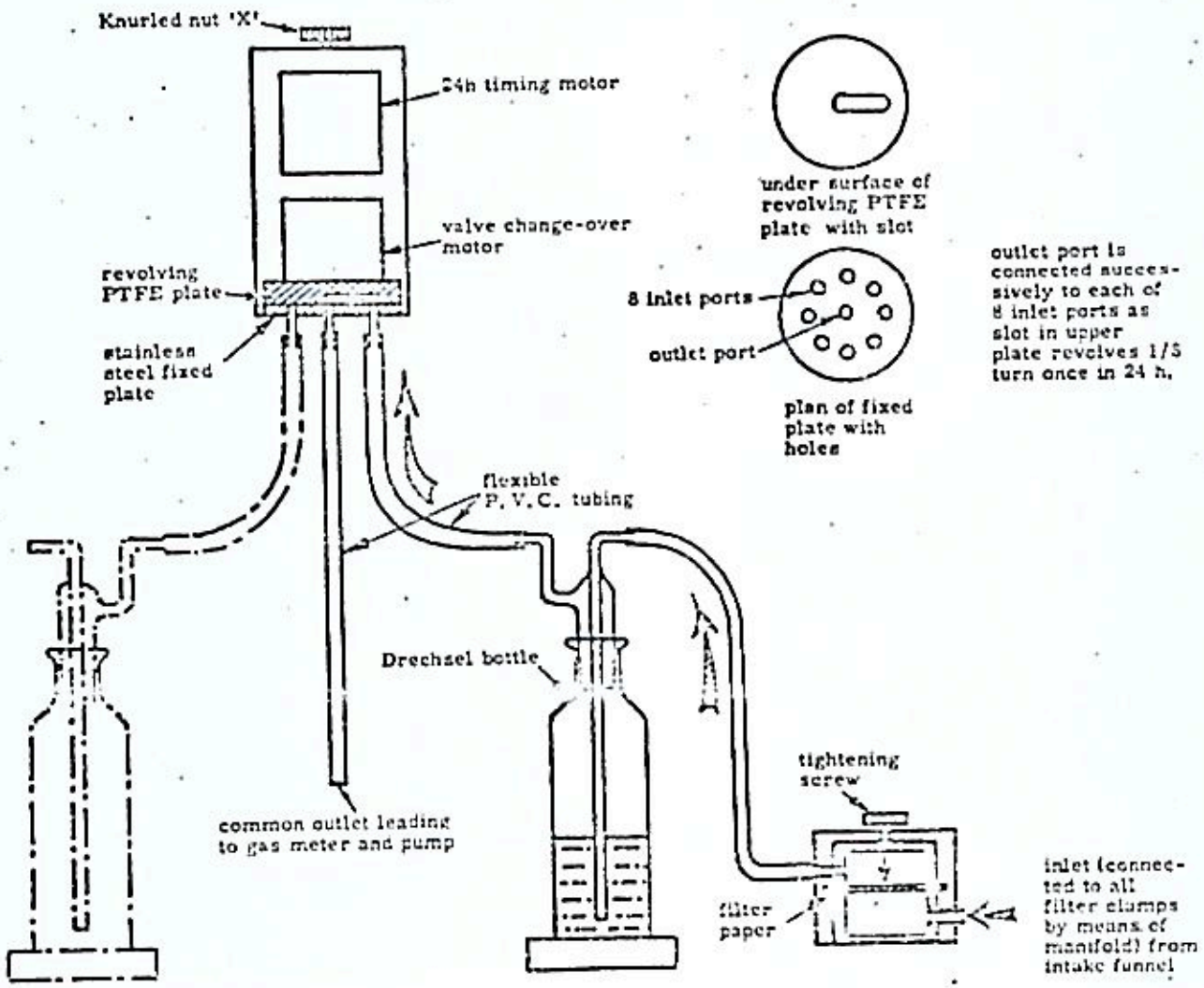
TELEPHONE: HITCHIN 3350

A SEMI-AUTOMATIC SMOKE AND SO_2 SAMPLER (8 PORT ROTARY VALVE)

General Description

This device eliminates the need for daily replacement of the smoke filter and hydrogen peroxide bubbler by automatically switching the air intake once every 24 hours so that it passes through a different filter and bubbler. A sequence of up to eight consecutive daily measurements can be made in this way without attention and the instrument should be particularly useful for offices working a 5-day week.



Only one pump and one meter are required; the pumping rate is sufficiently reliable for the daily volume of air to be determined by dividing the overall meter reading by the number of days the instrument is operated.

The device is extremely simple and should be less expensive than existing designs. It is expected to be commercially available by mid-1962 at a cost of £100 or less.

Setting-up Procedure

Note: It is important to keep the apparatus out of direct sunlight so that the hydrogen peroxide does not deteriorate before it is used.

Equipment

As in the case of other apparatus for daily sampling it is possible to buy the components separately or to buy complete self-contained instruments.

If, for example, the valve is being used for three days at the weekend the following equipment will be required in addition to the 8 port valve.

4 drechsel bottles (and at least one extra if the bottles are to be taken away for titration).

4 brass filter-clamps (size according to darkness of stain expected).

1 hard glass or stainless steel manifold (internal diameter of pipes should be $\frac{1}{4}$ inch).

The manifold should have one inlet, and one more outlet than the number of daily samples required. Fig. 2 shows a sketch of a manifold suitable for use for three days.

Fig. 2

Bends should have a radius of not less than 1 inch

Particular care should be taken to ensure that all joints in the manifold are air tight.

The complete selfcontained instrument will have 8 bubblers and clamps and will be mounted on a framework complete with manifold, meter and pump and enclosed in a box.

Procedure

It is first necessary to set the timing mechanism to operate the valve at the desired hour each day.

Allow $1\frac{1}{2}$ hours before the first sampling period is required to commence. Loosen the knurled nut (X in Fig. 1) and move the dial until the arrow is half way

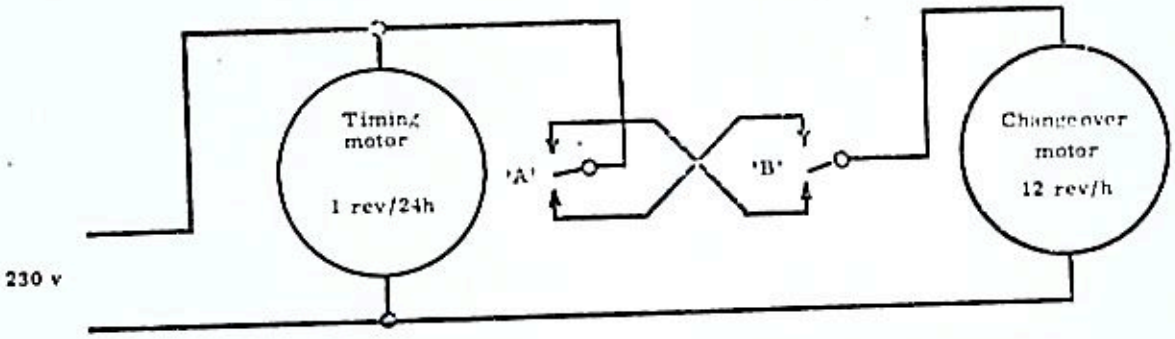
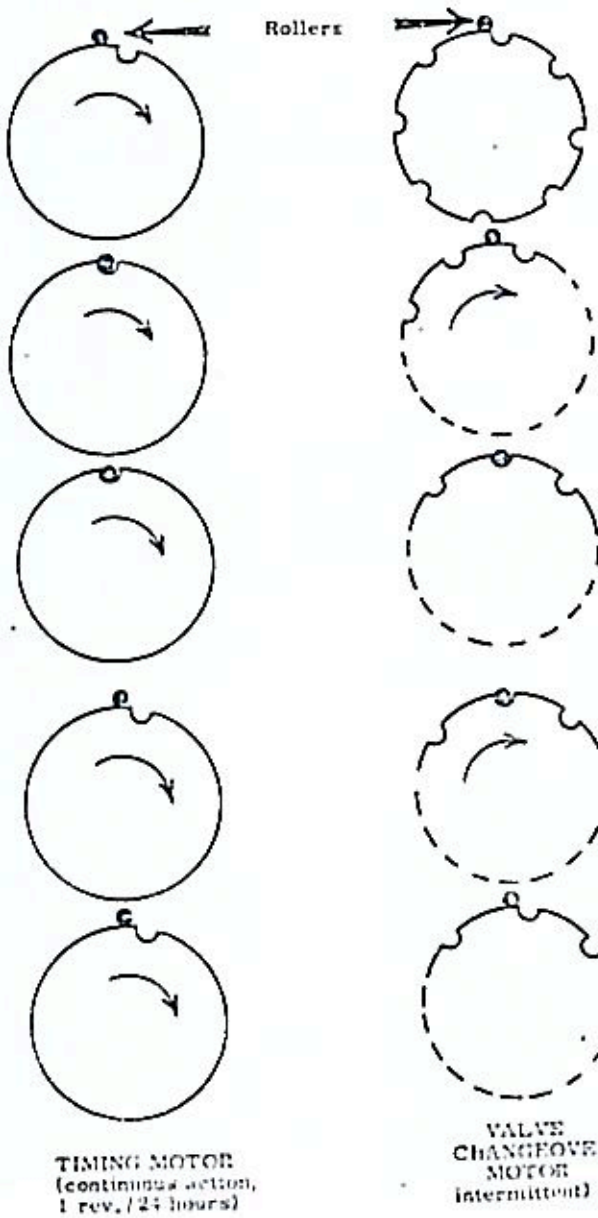


Fig. 3

Position of microswitch rollers

Effect of position of rollers



1. Both microswitch rollers are out of grooves during greater part of day.

   Valve changeover motor is off, disc stationary. Sampling goes on via port No. 1 (not shown).

2. After 24 hours first roller enters groove so that one is in, one out.

   Valve changeover motor starts; disc rotates through 45° until next groove reaches roller.

3. Second microswitch roller also enters groove; both are now in grooves.

   Valve changeover motor stops. Sampling begins through port no. 2. (not shown).

4. First microswitch roller leaves groove, so that one is in, one out

   Valve changeover motor starts but at once disengages roller from groove and therefore stops.

5. When second roller leaves groove both are out and ready for another cycle

   Sampling is not affected by (4) and continues via port no. 2 until cycle is repeated

Fig. 4

The timing motor runs continuously but the valve change-over motor is switched on only when the two micro-switches are in opposite positions, i.e. one in a groove and the other out.

The sequence of operations is as follows:-

1. During quiescent period while sampling is taking place A and B are both out of their groove.

2. At the completion of a 24 hour sampling period A drops into its groove and B remains out. This causes the valve change-over motor to rotate the disc with 8 grooves until B drops into the next groove.

3. Since A is still in its groove, the valve change-over motor stops. B remains in its groove for a short time, but then the continuous rotation of the timing motor disc causes A to come out of its groove.

4. The valve change-over motor starts but stops again almost at once because B then comes out too. This operation therefore causes no further change in the "live" port but the system is "cocked" ready for the next 24 hour change.

Action of timing mechanism

Although an understanding of the working of the timing mechanism is not necessary for those using the valve, the system is likely to be of interest to those with some knowledge of this type of mechanism and this explanation is therefore provided.

The valve assembly contains two motors, a timing motor and a valve changeover motor.

The timing motor shaft carries a disc with one groove in its periphery. The roller of a microswitch presses on to the periphery of this disc and as the latter rotates continuously the roller passes into and out of the groove, one complete rotation taking 24 hours.

This roller operates in conjunction with a similar microswitch roller on a corresponding disc attached to the shaft of the valve changeover motor.

This second disc has however not one but 8 equally spaced grooves round its periphery. It is held by springs to the slotted plate (See Fig. 1) and both rotate together when the valve changeover motor operates; this happens only when either one of the two microswitch rollers is in a groove and the other is not. The sequence of events is illustrated above (Fig. 4)

APPENDIX III

Analysis of Blood - Determination of Lead Using an Extraction Procedure

SCOPE

This method describes the determination of lead in blood using an extraction procedure, and may be applicable to other elements as well.

REAGENTS

Triton^{R} X-100 (TX; an alkyl phenoxy polyethoxy ethanol, Rohm & Haas, Philadelphia, Pa.), solution, 5% (w/v) in deionized water.

Ammonium pyrrolidine dithiocarbamate, APDC, solution, 2% (w/v) in deionized water. Solutions stored in a refrigerated amber glass bottle remain fresh for up to one month. If desired, the APDC solution can be prepared with the 5% TX solution instead of deionized water.

Methyl isobutyl ketone (MIBK), water-saturated.

STANDARD SOLUTIONS

Prepare an aqueous 10 µg/ml lead standard solution by diluting the 1000 µg/ml stock lead standard (described under the Standard Conditions for Pb) with deionized water. Prepare working standards by the addition of 0.00, 0.15, 0.30, and 0.50 ml of the 10 µg/ml Pb standard to 5.0 ml aliquots of pooled human blood using an Ostwald-Folin pipette. Mix thoroughly on a vortex mixer. Add 1 ml of 5% TX solution and treat as described under SAMPLE PREPARATION from the addition of 1 ml of APDC solution. These standards contain the equivalent lead added concentrations of 30, 60, and 100 µg/100 ml (µg%).

SAMPLE PREPARATION

To a 5-ml sample of blood, add 1 ml of 5% TX solution and mix thoroughly on a vortex mixer. Add 1 ml of the APDC solution, and again mix thoroughly on a vortex mixer. Add 5 ml of MIBK, seal the tube with a rubber stopper, shake vigorously about 60 times, and centrifuge for five minutes at about 2000 rpm.

ANALYSIS

Aspirate the organic supernatant directly and compare versus the organic supernatant obtained from the lead working standards. Use the Routine Procedure described in the General Information section. Refer also to the section on Organic Solvents for operating parameters.

CALCULATIONS

Calculate the lead concentration of the blood samples directly from a working curve prepared as follows: Subtract the reading obtained

CALCULATIONS (cont.)

for blood standards with added lead. This corrects for any lead present in the pooled blood. Prepare the calibration curve by plotting the corrected readings versus the equivalent concentrations of added lead (30, 60, and 100 µg/100 ml). Read the concentration of lead in the samples in µg/100 ml directly from the calibration curve.

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D. W. Hessel, At. Absorp. Newsl. 7, 55 (1968).