A comparison of concentrations of lead in human tissues¹

P. S. I. BARRY

Medical Department, The Associated Octel Co. Ltd., Ellesmere Port, Cheshire

Barry, P. S. I. (1975). *British Journal of Industrial Medicine*, 32, 119-139. **A comparison of concentrations of lead in human tissues.** This postmortem study of lead concentrations in the tissues of 129 subjects is an extension to a report by Barry and Mossman (1970). Lead concentrations in bone greatly exceeded the concentrations in soft tissues and were highest in the dense bones. Bone lead concentrations increased with age in both sexes, more especially in male subjects and in dense bone, varying between mean values of 2.16 ppm in the ribs of children to over 50 ppm in the dense petrous temporal bones of elderly male adults. Male adults contained over 30% more lead in their bones than females.

Mean concentrations of lead in the soft tissues varied from less than 0.1 ppm in organs such as muscle and heart to over 2 ppm in the aorta. In most tissues with lead values in excess of 0.2 ppm the male concentrations exceeded female values by about 30%. With the exception of the aorta, spleen, lung, and prostate, lead concentrations did not increase with age in the soft tissues of either sex after about the second decade of life.

Children showed concentrations of lead in their soft tissues comparable to female adults, but the concentrations in bone were much lower. It is suggested that children do not possess the same capacity as adults to retain lead in bone.

In male adults occupationally exposed to lead the concentrations of lead in bone exceeded the concentrations in unexposed male adults within the same age group by two- to three-fold. Soft tissue lead concentrations between the two groups were less divergent.

An assessment of the total body burden of lead revealed higher levels in adult male subjects than in females at mean values of 164.8 mg compared to 103.6 mg, respectively. Over 90% of the total body burden of lead in adults was in bone, of which over 70% was in dense bone. Male adults occupationally exposed to lead had mean total body burdens of 566.4 mg Pb, of which 97% was in bone.

The release of lead from bone in conjunction with calcium was not considered to be of physiological significance.

Lead concentrations in hair and nails were higher than soft tissue lead concentrations and varied widely. Hair lead measurements were not considered to provide a reliable assessment of lead absorption.

The concentrations of lead in the tissues of a mixed group of subjects with no known occupational exposure to lead have been shown to be comparable to the findings in earlier studies. Present levels of lead in the environment are not considered to be a hazard to the health of the population in general.

In the past few years much attention has been paid to the possible health effects of low level concen-

¹A full set of the data, some of which are not tabulated in this report, may be obtained from the author on request.

trations of lead in man. A great deal of investigatory work has been undertaken with a view to defining the pathways of lead metabolism and mechanisms of interference with physiological

function irrespective of whether such interference may be of significance in relation to health.

High levels of lead intake have been known for many centuries to cause clinical illness in man. The observations of Hippocrates (370 BC), Pliny (AD 23-79), and Dioscorides (AD 100) have been documented by Hunter (1957). In later times Ramazzini (1713), Sir George Baker (1941), Thomas Percival (1774), and Tanquerel des Planches (1848) described lead poisoning from various sources in people with differing occupations and habits.

However it was not until the publication by Kehoe, Thamann, and Cholak in 1933 'On the normal absorption and excretion of lead' that it was appreciated that lead was an inevitable constituent of body tissues by virtue of the ubiquitous presence of the metal on the surface of the earth. At the present time no useful function is known to be served by lead in the body.

The purpose of this study, an extension and follow-up to an earlier study by Barry and Mossman (1970), is to report the concentrations of lead found in the tissues of a largely urban contemporary population from a heavily industrial part of the north-west of England and to draw tentative conclusions from the data.

Study outline

Between May 1966 and May 1973 up to 35 different organs and tissues from a total of 129 cadavers were analysed for lead content. Out of this total 119 (60 male adults, 36 female adults, and 23 male and female children aged 16 years and under) had no history of occupational exposure to lead. Seven of the remaining 10 subjects, all male adults, had defined histories of occupational exposure to lead. It was concluded from a study of the data that the other three, from whom no history of occupation was obtained, had probably had an unusual past exposure to lead.

A wide distribution of age, limited only by availability of subjects, was obtained. Causes of death were varied, as were past illnesses and occupations during life. As all cases were subjected to postmortem examination for reasons associated with unusual or ill-defined causes of death, they might be considered unrepresentative of a normal population. Apart from this reservation however and considerations of age distribution and sex, no attempt was made to select cases for the study.

The preparation of samples and the analytical technique employed were the same as reported in the earlier study of Barry and Mossman (1970).

Results

For the most part results have been recorded in parts per million in wet weight of tissue on the samples as received. Exceptions included bones where a comparison was made in parts per million on wet weight and dry weight, and in the assessment of total body burden which was measured in milligrams.

A wide variation in lead concentrations in tissues between individual subjects was noted, particularly in bone.

The first section of this report is concerned with male and female subjects with no known occupational exposure to lead.

No known occupational exposure to lead

1. Sex difference A comparison of the concentrations of lead in the tissues of male and female adults over the age of 16 years is represented in Table 1. The mean concentrations of lead in the various bones examined exceeded mean individual soft tissue concentrations in both sexes by a considerable margin. Mean bone lead concentrations varied between 8.85 ppm in rib to 33.71 ppm in the petrous temporal bone in male subjects, compared with 6.77 ppm and 26.63 ppm respectively in females. These values represent a ratio of mean concentrations of lead in the bones of male adults compared with female adults of approximately 1.3:1. They agree quite closely with the mean values reported by Kehoe in 1961 and 1963 and confirm the difference between long and flat bones noted by Tompsett in 1936.

The concentrations of lead in male soft tissues exceeded the values in equivalent female tissues by about 30% in most of those tissues where mean lead values exceeded 0.2 ppm. In tissues with mean lead values of 0.2 ppm and less there was no difference between the sexes.

In both sexes the kidney cortex showed a higher mean concentration of lead than the kidney medulla by about 50%. Allcroft (1951) found that the kidney cortex contained higher concentrations of lead than the medulla in bovine animals poisoned by lead. Goyer and Rhyne (1973) have shown that lead tends to concentrate in the region of the proximal convoluted tubules, the straight parts of which extend into the renal cortex.

The lead concentration in male kidney tissue exceeded the concentration in female kidney by more than 30%, as also did the male liver and pancreas. Atheromatous aorta in male subjects at a mean lead concentration of 2.56 ppm was more than double the female value of 1.17 ppm, while non-atheromatous aortic mean values were approximately the same in both sexes, 1.82 ppm in males and 1.70 ppm in females.

The mean lead concentrations in the hilar lymphatic glands were approximately the same in both sexes, 0.50 ppm in males and 0.46 ppm in females. These values were about double the mean concentration of lead in lung in both sexes of 0.22 ppm, and suggest that pulmonary macrophage activity may have been responsible for the higher values found in the hilar glands.

Hair in female subjects showed mean lead values

TABLE 1
CONCENTRATIONS OF LEAD IN TISSUES OF SUBJECTS WITH NO KNOWN OCCUPATIONAL
EXPOSURE TO LEAD (ppm WET WEIGHT)

Tissue	Male adults				Female adults			
	No. of samples	Mean	SD	Range	No. of samples	Mean	SD	Range
Bone								
Petrous temporal	30	33.71	22.34	6.70- 89.00	16	26.63	15.42	7.50-62.00
Tibia	60	23.40	15.96	3.00- 73.00	36	15.99	12.49	1.50-48.00
Calvarium	31	20.17	16.00	3.90- 79.00	17	16.46	13.08	5.00-54.20
Rib	60	8.85	5.81	0.90- 28.50	36	6.77	5.08	0.85-22.60
Hair	33	6.56	5.05	1.00- 20.00	18	11.49	14.89	0.70-55.00
Nails	28	4.72	3.46	0.65- 15.00	11	5.58	4.22	1.50-15.00
Aorta								
Atheroma	32	2.56	3.49	0.21- 17.20	16	1.17	1.23	0.09- 4.20
Non-atheroma	42	1.82	2.47	0.09- 12.70	22	1.70	2.64	0.07-12.20
Liver	58	1.03	0.62	0.18- 3.13	36	0.66	0.38	0.19- 1.72
Dense connective tissue								
Cartilage	18	1.29	1.58	0.05- 4.80	12	0.56	0.53	0.08- 1.75
Ligamentum nuchae	16	0.33	0.29	0.03- 0.90	11	0.37	0.23	0.10- 0.86
Kidney								
Cortex	59	0.78	0.38	0.15- 1.85	35	0.55	0.39	0.10- 2.20
Medulla	59	0.50	0.25	0.13- 1.46	36	0.38	0.19	0.11- 0.97
Hilar lymphatics	56	0.50	0.57	0.06- 2.88	34	0.46	0.28	0.03- 1.20
Pancreas	58	0.37	0.26	0.09- 1.42	36	0.27	0.17	0.11- 0.95
Prostate	53	0.27	0.51	0.03- 2.82				
Ovary					25	0.26	0.31	0.03- 1.09
Skin	26	0.19	0.14	0.01- 0.60	20	0.15	0.11	0.03- 0.42
Spleen	59	0.23	0.25	0.03- 1.43	34	0.21	0.26	0.04- 1.40
Lung	59	0.22	0.11	0.05- 0.59	36	0.22	0.12	0.04- 0.55
Thyroid	55	0.19	0.23	0.05- 1.45	30	0.21	0.30	0.04- 1.73
Blood	53	0.20	0.12	0.03- 0.79	30	0.16	0.10	0.04- 0.41
Suprarenal	54	0.15	0.11	0.01- 0.62	27	0.17	0.11	0.05- 0.48
Brain								
Cortex	58	0.10	0.14	0.02- 0.78	34	0.12	0.15	0.01- 0.67
Basal ganglia	34	0.09	0.04	0.04- 0.19	17	0.11	0.05	0.05- 0.23
Fat								
Subcutaneous	23	0.08	0.08	0.01- 0.40	20	0.06	0.04	0.02- 0.16
Omentum	19	0.11	0.11	0.02- 0.52	14	0.08	0.03	0.03- 0.13
Gut								
Caecum	32	0.09	0.06	0.03- 0.27	27	0.14	0.14	0.02- 0.68
Midgut	31	0.12	0.06	0.05- 0.32	27	0.12	0.10	0.03- 0.48
Stomach	32	0.09	0.05	0.03- 0.25	26	0.11	0.10	0.02- 0.42
Testis	43	0.08	0.04	0.02- 0.28				
Heart	59	0.07	0.05	0.01- 0.30	36	0.08	0.06	0.02- 0.31
Muscle	35	0.05	0.04	0.02- 0.23	29	0.05	0.03	0.01- 0.15
Faeces ($\mu\text{g/g}$ ash)	50	17.39	12.40	2.20- 59.40	29	17.69	18.11	2.80-80.00
Urine ($\mu\text{g/l}$)	24	39.58	43.80	8.00-192.00	10	23.8	25.27	5.00-90.00

nearly twice those of males (11.49 ppm in females and 6.56 ppm in males), but nails showed nearer the same values (5.58 ppm in females and 4.72 ppm in males). These particular tissues will be discussed later in this report.

2. Bone The distribution of frequencies of occurrence of lead in the bones (wet basis) of male and female subjects of all ages showed approximately 90% of the lead in rib values in both sexes to be less than 15 ppm, compared with nearly 60% for tibia and calvarium and nearly 40% for the petrous temporal

bone. Of the 20 results of lead in male tibia in excess of 30 ppm, 17 were from men over the age of 55 years; of the remaining 51 results of less than 30 ppm, 12 were from men in this same age group. The dense petrous temporal bone contained the highest concentrations of lead among the four bones examined in both sexes, and the vascular rib the lowest concentrations (Table 1).

It was reported by Barry and Mossman (1970) that the frequency distribution of the lead content of male tibia was biphasic. The present data, which included those of the earlier report, did not indicate evi-

dence of a biphasic distribution. It would seem that the greater number of incorporated values have smoothed out the previous pattern. The contention in the earlier report that the male subjects comprised two groups suggesting two distinct categories with respect to magnitude of exposure to lead, or that the opportunity for exposure may have been greater in the past, cannot be held tenable in the light of the present evidence.

The lead concentrations in the four varieties of bone examined were compared on a basis of ash weight versus wet weight in 46 subjects of both sexes. These results included those reported at the International Symposium on Environmental Health Aspects of Lead, Amsterdam, 1972 (Barry, 1973). On wet weight, for all subjects, the mean lead concentration of 5.9 ppm in rib was approximately one third of the values recorded in calvarium and tibia and less than a quarter of the concentration in the petrous temporal bone. Mean wet weight values for the 21 male adults showed rib at 7.4 ppm with just over a quarter of the lead concentrations of calvarium and tibia and less than one fifth of the petrous temporal bone. In 14 female adult ribs, with a mean lead concentration of 5.8 ppm, the corresponding values were less than a half and less than a quarter.

Wet weight values in 11 children showed the concentrations of lead in all four bones to be more approximate, with rib over half that of the petrous temporal bone, at 3.1 ppm and 5.6 ppm respectively.

On ash weight measurement the female adults showed a mean concentration of lead in rib which approximated to the mean concentrations in calvarium and tibia, i.e., between 16.9 ppm and 19.0 ppm, but about half that of the petrous temporal bone. In male adults the mean lead concentration in rib was more than 75% of the values recorded for calvarium and tibia, i.e., 27.7 ppm compared to 33.9 and 37.3 ppm respectively, and about half of the concentration in the petrous temporal bone of 54.2 ppm. The ash weight results for adult ribs related closely to those reported by Hislop *et al.* (1973). In children the mean ash weight concentration of lead in rib of 8.3 ppm equated with that of the petrous temporal bone but was approximately 25% greater than the values recorded for calvarium and tibia.

The ratios of ash weight to wet weight for the calvarium, tibia, and petrous temporal bones were nearly the same, between 1.3:1 and 1.6:1 for both male and female adults and children. The ash weight/wet weight ratio for rib was 4.1:1 in male adults and 2.9:1 and 2.7:1 respectively in female adults and children.

Assessment by ash weight brought the concentrations of lead in rib closer to those of the other

bones, but in adults a distinct difference remained between rib and the petrous temporal bone. Thus these findings do not support the view that analytical results reported on a basis of ash weight are necessarily uniform irrespective of the type of bone, as was suggested in the report of the National Research Council (1972).

3. Effect of age on tissue lead concentrations (a) Bone: The data in Figs 1 and 2 and in Tables 2 and 3 demonstrate that lead concentrations in the bones (wet weight) increased with age in both sexes. Male subjects showed a more pronounced increase than females. The petrous temporal bone, in addition to having the highest concentrations of lead, showed the steepest rise of lead concentration with age in both sexes, followed by the tibia and then the calvarium. Rib showed the lowest rate of increase in both sexes, particularly in females. There was no evidence of decrease of lead concentration with age in the bones of either sex. These findings agree with those of Morris (1940).

Schroeder and Tipton (1968) suggested that at the fifth decade of life the lead concentrations in bone levelled off and thereafter decreased. Their measurements were based upon concentrations of lead in rib in a mixed group of male and female subjects. In the 70 and 80-year-old age groups they amounted to a total of three results in female subjects only. Our findings indicate a definite difference in lead concentrations in comparable bones between sexes and that an extrapolation of results obtained from rib alone would not provide a valid representation of the lead content in the whole skeleton.

(b) Soft tissues: The data presented in Tables 2 and 3 demonstrate that the majority of the soft tissues did not show an increase of lead concentration with age after about the second decade of life. Exceptions were the aorta and to a lesser extent the spleen and lung in both sexes, as shown in Figs 3 and 4. In addition, the prostate, and also the cartilage in females, showed minimal evidence of increase of lead concentration with age. None of the other tissues examined showed evidence of increased lead concentration with age in either sex, but the skin, subcutaneous fat, and muscle showed a marginal decrease of lead concentrations with age in both sexes, as did the testis and faeces in male subjects.

Children

The tissues from 23 infants and children up to 16 years of age were analysed for lead content. Of this number, 14 were male subjects and nine female; 18 were under the age of 10 years (nine of each sex). There was no apparent difference in tissue lead concentrations between the sexes.

The lead concentrations in all of the tissues examined are given in Table 4. Mean bone lead

FIG. 1. Males. Lead concentrations in bones—non-occupational exposure (95% confidence limits).

FIG. 2. Females. Lead concentrations in bones—non-occupational exposure (95% confidence limits).

TABLE 2
AGE GROUPS—MALES
TISSUE LEAD CONCENTRATIONS—ppm WET WEIGHT

<i>Tissue</i>	<i>Age (years)</i>					
	<i>0-9</i>		<i>10-19</i>		<i>20-29</i>	
	<i>No.</i>	<i>Mean (Range)</i>	<i>No.</i>	<i>Mean (Range)</i>	<i>No.</i>	<i>Mean (Range)</i>
Bone						
Petrous temporal	3	5.50 (1.20-8.10)	4	7.95 (3.40-16.00)	7	9.86 (6.70-15.40)
Tibia	7	2.41 (0.21- 5.50)	7	4.55 (1.50-12.50)	9	6.91 (3.00-12.30)
Calvarium	3	5.10 (2.00- 6.70)	4	4.90 (1.90- 9.00)	8	6.49 (4.10-11.80)
Rib	9	1.83 (0.01- 3.90)	8	3.09 (1.00- 6.96)	9	4.03 (1.75- 5.60)
Hair	2	6.10 (3.20- 9.00)	5	8.66 (2.30-19.00)	7	9.28 (1.50-19.00)
Nails	1	55.00	3	12.00 (6.00-15.00)	4	7.48 (5.30- 9.00)
Aorta						
Atheroma	0		3	0.06 (0.03- 0.09)	4	0.23 (0.21- 0.24)
Non-atheroma	4	0.19 (0.04- 0.29)	6	1.83 (0.04- 9.90)	9	0.41 (0.10- 1.04)
Liver	9	0.65 (0.08- 1.24)	8	0.82 (0.34- 1.36)	8	1.11 (0.44- 1.80)
Dense connective tissue						
Cartilage	3	0.21 (0.06- 0.44)	3	0.13 (0.04- 0.22)	2	0.09 (0.05- 0.12)
Ligamentum nuchae... .. .	1	0.15	3	0.43 (0.09- 1.00)	2	0.26 (0.20- 0.31)
Kidney						
Cortex	8	0.40 (0.01- 1.05)	8	0.57 (0.34- 1.00)	9	1.07 (0.55- 1.85)
Medulla	8	0.29 (0.01- 0.80)	8	0.43 (0.24- 0.81)	9	0.52 (0.27- 0.71)
Hilar lymphatics	3	0.66 (0.04- 1.40)	7	0.61 (0.10- 1.70)	7	0.46 (0.15- 1.75)
Pancreas	6	0.41 (0.26- 0.68)	8	0.36 (0.22- 0.61)	9	0.50 (0.30- 1.20)
Prostate	3	0.35 (0.10- 0.50)	6	0.13 (0.03- 0.25)	9	0.09 (0.04- 0.05)
Skin	3	0.29 (0.07- 0.69)	4	0.36 (0.15- 0.70)	3	0.39 (0.28- 0.60)
Spleen	9	0.12 (0.06- 0.18)	7	0.14 (0.07- 0.21)	9	0.12 (0.05- 0.24)
Lung	9	0.11 (0.01- 0.42)	8	0.18 (0.09- 0.40)	9	0.16 (0.11- 0.20)
Thyroid	6	0.18 (0.01- 0.42)	6	0.20 (0.07- 0.41)	8	0.13 (0.08- 0.21)
Blood	6	0.09 (0.04- 0.13)	6	0.30 (0.07- 0.79)	8	0.19 (0.08- 0.25)
Suprarenal	5	0.31 (0.01- 0.87)	6	0.17 (0.04- 0.27)	8	0.18 (0.08- 0.38)
Brain						
Cortex	9	0.06 (0.01- 0.21)	8	0.09 (0.02- 0.17)	9	0.07 (0.04- 0.09)
Basal ganglia	3	0.15 (0.08- 0.24)	5	0.13 (0.04- 0.18)	8	0.09 (0.05- 0.16)
Fat						
Subcutaneous... .. .	3	0.27 (0.18- 0.35)	4	0.30 (0.03- 0.58)	3	0.10 (0.04- 0.18)
Omentum	2	0.13 (0.08- 0.17)	3	0.11 (0.05- 0.16)	3	0.10 (0.06- 0.14)
Gut						
Caecum	6	0.11 (0.01- 0.21)	4	0.15 (0.04- 0.21)	2	0.06 (0.04- 0.07)
Midgut	6	0.09 (0.03- 0.17)	4	0.15 (0.06- 0.19)	2	0.09 (0.05- 0.12)
Stomach	7	0.07 (0.01- 0.20)	4	0.12 (0.04- 0.25)	2	0.13 (0.06- 0.20)
Testis	3	0.18 (0.03- 0.30)	6	0.09 (0.03- 0.16)	9	0.09 (0.06 0.12)
Heart	8	0.05 (0.01- 0.11)	8	0.12 (0.03- 0.24)	9	0.08 (0.03- 0.19)
Muscle	6	0.08 (0.02- 0.16)	4	0.10 (0.02- 0.23)	3	0.05 (0.04- 0.07)
Faeces (μ g/g ash)	3	38.49 (3.46-77.00)	8	31.61 (5.50-59.40)	5	16.13 (3.15-23.00)
Urine (μ g/l)	3	27.43 (8.30-50.00)	5	39.20 (13.00-71.00)		

Table 2 continued

Age (years)											
30-39		40-49		50-59		60-69		70-79		80-89	
No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)
2	15.15 (10.30-20.00)	3	29.67 (19.00- 48.00)	3	35.00 (28.00- 45.00)	10	44.20 (16.00-65.00)	4	56.50 (22.00-89.00)	1	50.00
5	16.40 (3.30-38.00)	9	18.22 (4.40- 38.00)	9	22.39 (11.20- 43.00)	15	31.67 (13.00-55.00)	5	31.76 (11.00-49.00)	5	47.94 (38.00- 73.00)
2	7.95 (3.90-12.00)	3	16.83 (9.50- 22.00)	3	20.17 (15.00- 24.00)	10	23.95 (14.00-41.00)	4	32.00 (12.00-47.00)	1	79.00
5	6.10 (0.90-13.30)	9	8.31 (2.60- 21.00)	9	11.75 (7.00- 20.40)	15	10.31 (3.20-28.50)	5	8.68 (4.20-15.00)	5	14.34 (4.10- 24.00)
2	2.95 (2.90- 3.00)	4	10.39 (8.00- 16.00)	3	12.00 (6.00- 20.00)	11	4.04 (1.00- 8.20)	4	4.95 (1.10-12.20)	1	4.00
1	2.00	4	4.34 (1.80- 6.25)	3	6.58 (3.60 12.50)	10	2.62 (0.80- 5.00)	4	3.09 (0.65- 6.40)	1	6.00
2	2.01 (0.26- 3.76)	3	1.72 (0.33- 3.85)	6	1.66 (0.55- 2.90)	12	3.33 (0.46-17.20)	3	4.12 (0.42-10.20)	2	4.81 (2.60- 7.02)
4	0.71 (0.22- 1.56)	7	1.21 (0.33- 4.26)	7	1.69 (0.57- 2.85)	8	3.03 (0.90-81.10)	4	5.52 (0.28-2.70)	1	3.10
5	0.88 (0.18- 1.93)	9	0.76 (0.34- 1.25)	9	0.98 (0.34- 2.01)	15	1.19 (0.28- 3.00)	5	0.70 (0.42-1.17)	4	1.47 (0.56- 3.13)
2	0.33 (0.10- 0.56)	2	4.25 (3.70- 4.00)	1	0.30	7	1.10 (0.20- 4.67)	3	1.45 (0.34- 2.70)	1	1.50
1	0.20	1	0.10	1	0.22	7	0.26 (0.03- 0.70)	3	0.53 (0.20- 0.90)	1	0.80
5	0.73 (0.35- 0.91)	9	0.80 (0.25- 1.28)	9	0.69 (0.15- 1.80)	15	0.72 (0.18- 1.08)	5	0.60 (0.22- 1.25)	4	0.91 (0.40- 1.66)
5	0.64 (0.22- 1.46)	9	0.46 (0.22- 0.87)	9	0.51 (0.13- 1.00)	15	0.42 (0.19- 0.84)	5	0.42 (0.19- 0.90)	4	0.71 (0.37- 1.08)
5	0.32 (0.18- 0.50)	9	0.35 (0.06- 0.80)	9	2.23 (0.06- 0.50)	15	0.74 (0.11- 2.88)	5	0.33 (0.09- 0.75)	4	0.66 (0.43- 1.10)
5	0.53 (0.12- 0.94)	9	0.35 (0.17- 0.65)	9	0.36 (0.09- 1.42)	14	0.27 (0.10- 0.49)	5	0.22 (0.10- 0.32)	4	0.53 (0.19- 0.92)
4	0.08 (0.05- 0.11)	8	0.12 (0.04- 0.23)	9	0.15 (0.04- 0.58)	15	0.46 (0.03- 2.82)	3	0.17 (0.07- 0.33)	4	1.04 (0.08- 2.51)
2	0.22 (0.20- 0.24)	3	0.28 (0.03- 0.42)	3	0.17 (0.07- 0.24)	9	0.07 (0.01- 0.20)	3	0.20 (0.16- 0.25)	2	0.27 (0.19- 0.35)
5	0.19 (0.07- 0.40)	9	0.06 (0.06- 0.31)	9	0.28 (0.06- 0.82)	15	0.36 (0.03- 1.43)	5	0.23 (0.06- 0.65)	4	0.27 (0.17- 0.38)
5	0.23 (0.10- 0.37)	9	0.20 (0.12- 0.32)	9	0.22 (0.10- 0.47)	15	0.24 (0.05- 0.59)	5	0.22 (0.06- 0.40)	4	0.28 (0.14- 0.51)
5	0.11 (0.08- 0.16)	9	0.12 (0.06- 0.26)	8	0.12 (0.05- 0.25)	15	0.25 (0.06- 1.45)	5	0.35 (0.07- 1.00)	3	0.30 (0.19- 0.38)
5	0.22 (0.08- 0.28)	8	0.16 (0.08- 0.24)	7	0.21 (0.03- 0.43)	15	0.18 (0.04- 0.31)	5	0.11 (0.08- 0.17)	3	0.23 (0.18- 0.31)
5	0.13 (0.09- 0.19)	9	0.17 (0.01- 0.62)	7	0.11 (0.07- 0.13)	15	0.14 (0.03- 0.35)	5	0.19 (0.04- 0.46)	3	0.14 (0.12- 0.16)
4	0.06 (0.05- 0.08)	9	0.13 (0.03- 0.78)	9	0.06 (0.02- 0.12)	15	0.09 (0.03- 0.30)	5	0.12 (0.04- 0.27)	4	0.25 (0.08- 0.73)
2	0.06 (0.05- 0.07)	5	0.08 (0.05- 0.12)	3	0.08 (0.04- 0.13)	10	0.11 (0.04- 0.19)	4	0.08 (0.04- 0.12)	1	0.17
2	0.06 (0.04- 0.07)	3	0.06 (0.03- 0.11)	3	0.07 (0.03- 0.11)	8	0.06 (0.01- 0.14)	2	0.09 (0.04- 0.13)	1	0.07
3	0.03 (0.02- 0.04)	2	0.14 (0.04- 0.23)	2	0.04 (0.04- 0.04)	6	0.07 (0.03- 0.13)	3	0.25 (0.11- 0.52)	1	0.15
2	0.05 (0.04- 0.05)	5	0.08 (0.03- 0.17)	4	0.06 (0.04- 0.11)	10	0.09 (0.04- 0.27)	4	0.12 (0.10- 0.17)	4	0.11 (0.04- 0.18)
2	0.09 (0.05- 0.13)	4	0.14 (0.09- 0.17)	4	0.09 (0.05- 0.14)	10	0.12 (0.05- 0.18)	4	0.16 (0.07- 0.32)	4	0.10 (0.06- 0.14)
2	0.09 (0.03- 0.15)	5	0.07 (0.03- 0.11)	4	0.08 (0.04- 0.13)	10	0.08 (0.04- 0.12)	4	0.11 (0.06- 0.14)	4	0.09 (0.06- 0.12)
2	0.21 (0.13- 0.28)	6	0.06 (0.04- 0.07)	6	0.07 (0.03- 0.11)	12	0.08 (0.02- 0.19)	5	0.07 (0.05- 0.11)	1	0.06
5	0.10 (0.04- 0.30)	9	0.05 (0.03- 0.09)	9	0.05 (0.03- 0.08)	15	0.05 (0.01- 0.17)	5	0.07 (0.03- 0.10)	4	0.10 (0.05- 0.21)
3	0.04 (0.03- 0.05)	5	0.06 (0.02- 0.16)	5	0.05 (0.02- 0.08)	10	0.05 (0.02- 0.12)	4	0.06 (0.02- 0.09)	4	0.05 (0.04- 0.06)
5	19.22 (4.70-51.40)	8	17.04 (2.20- 31.00)	7	15.82 (7.10- 31.00)	15	14.70 (3.40-39.00)	4	15.65 (5.10-36.50)	3	17.73 (12.00- 23.00)
2	38.50 (35.00-42.00)	4	51.50 (15.00-132.00)	3	51.67 (10.00-110.00)	9	26.22 (13.00-43.00)			2	102.00 (12.00-192.00)

TABLE 3
AGE GROUPS—FEMALES
TISSUE LEAD CONCENTRATIONS—ppm WET WEIGHT

Tissue	Age (years)							
	0-9		10-19		20-29		30-39	
	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)
Bone								
Petrous temporal	5	4.27 (0.25- 6.70)	1	7.50	2	9.40 (7.80-11.00)	2	23.50 (17.00-30.00)
Tibia	7	2.99 (0.62- 6.20)	1	5.90	5	4.46 (2.50- 6.30)	2	17.50 (14.00-21.00)
Calvarium	5	4.58 (1.10- 6.50)	1	5.20	2	7.00 (6.50- 7.50)	2	22.50 (12.00-33.00)
Rib	9	2.48 (0.01- 5.50)	1	10.00	5	3.23 (1.50- 4.90)	2	10.50 (9.00-12.00)
Hair	5	16.14 (7.20-25.00)	1	8.60	3	12.10 (7.70-19.00)	2	5.18 (1.36- 9.00)
Nails	2	72.50 (45.00-100.0)	0		2	13.00 (11.00-15.00)	2	3.59 (3.57- 3.60)
Aorta								
Atheroma	0		1	0.14	1	0.09	0	
Non-atheroma	6	0.34 (0.07- 1.10)	1	0.26	5	0.27 (0.07- 0.64)	2	0.44 (0.41- 0.47)
Liver	9	0.63 (0.10- 1.37)	1	0.67	5	0.64 (0.42- 0.80)	2	0.43 (0.25- 0.61)
Dense connective tissue								
Cartilage	5	0.29 (0.18- 0.50)	1	0.26	2	0.14 (0.08- 0.20)	0	
Ligamentum nuchae	2	0.34 (0.19- 0.48)	0		2	0.30 (0.20- 0.40)	0	
Kidney								
Cortex	9	0.42 (0.03- 1.20)	1	0.57	5	0.59 (0.26- 1.35)	2	0.24 (0.20- 0.28)
Medulla	9	0.29 (0.03- 0.73)	1	0.59	5	0.49 (0.21- 0.97)	2	0.19 (0.13- 0.24)
Hilar lymphatics	4	0.51 (0.38- 1.60)	0		5	0.33 (0.07- 0.65)	2	0.09 (0.03- 0.15)
Pancreas	8	0.25 (0.01- 0.58)	1	0.36	5	0.28 (0.24- 0.33)	2	0.15 (0.11- 0.18)
Ovary	5	0.54 (0.10- 1.50)	1	0.14	5	0.11 (0.03- 0.34)	2	0.10 (0.05- 0.15)
Skin	5	0.73 (0.25- 1.80)	0		2	0.14 (0.07- 0.21)	2	0.25 (0.08- 0.42)
Spleen	9	0.12 (0.02- 0.23)	1	0.11	5	0.11 (0.06- 0.14)	1	0.04
Lung	9	0.15 (0.02- 0.32)	1	0.33	5	0.19 (0.06- 0.44)	2	0.15 (0.14- 0.15)
Thyroid	7	0.28 (0.10- 0.80)	1	0.12	5	0.10 (0.04- 0.20)	2	0.13 (0.08- 0.71)
Blood	6	0.12 (0.01- 0.40)	1	0.25	5	0.18 (0.04- 0.32)	2	0.14 (0.12- 0.15)
Suprarenal	6	0.39 (0.14- 1.20)	1	0.22	5	0.14 (0.09- 0.19)	1	0.27
Brain								
Cortex	8	0.07 (0.01- 0.19)	1	0.08	5	0.06 (0.04- 0.07)	1	0.08
Basal ganglia	5	0.13 (0.06- 0.33)	0		3	0.08 (0.05- 0.10)	2	0.09 (0.08- 0.09)
Fat								
Subcutaneous	5	0.48 (0.09- 1.30)	1	0.05	2	0.05 (0.04- 0.06)	2	0.04 (0.03- 0.05)
Omentum	5	0.25 (0.11- 0.67)	1	0.10	2	0.06 (0.05- 0.06)	2	0.06 (0.06- 0.06)
Gut								
Caecum	7	0.15 (0.01- 0.25)	1	0.29	2	0.06 (0.05- 0.06)	0	
Midgut	7	0.19 (0.03- 0.52)	1	0.17	2	0.07 (0.05- 0.08)	0	
Stomach	7	0.08 (0.03- 0.14)	1	0.40	2	0.07 (0.05- 0.08)	0	
Heart	9	0.08 (0.01- 0.14)	1	0.15	5	0.07 (0.04- 0.13)	2	0.05 (0.04- 0.05)
Muscle	7	0.10 (0.03- 0.30)	1	0.04	2	0.05 (0.04- 0.05)	2	0.03 (0.03- 0.03)
Faeces (µg/g ash)	6	29.95 (1.70-75.00)	1	31.00	3	32.50 (12.50-65.00)	2	13.00 (12.00-14.00)
Urine (µg/l)	1	27.00	1	36.00	0		2	12.50 (5.0 -20.00)

Table 3 continued

Age (years)											
40-49		50-59		60-69		70-79		80-89		90-99	
No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)	No.	Mean (Range)
3	21.17 (14.00-34.00)	2	22.00 (20.00-24.00)	2	33.50 (29.00-38.00)	0		3	43.33 (26.00-62.00)	1	48.50
7	10.94 (1.50-22.00)	4	12.38 (4.70-17.10)	8	18.84 (7.71-43.20)	4	19.85 (10.50-44.40)	4	24.59 (6.35-42.00)	1	48.00
4	14.73 (5.60-31.50)	2	10.50 (5.00-16.00)	2	13.65 (11.30-16.00)	0		3	18.07 (10.70-27.00)	1	54.20
7	3.92 (1.20-10.00)	4	5.56 (2.50-9.10)	8	8.16 (0.85-22.60)	4	7.57 (3.50-13.10)	4	8.55 (3.60-16.50)	1	17.10
3	5.97 (4.70-8.00)	3	8.35 (1.30-21.10)	2	28.65 (2.30-55.00)	0		3	16.27 (0.70-43.00)	1	2.57
1	7.00	2	1.50 (1.50-1.50)	2	3.13 (1.50-5.50)	0		2	4.35 (2.00-6.70)	0	
4	0.64 (0.32-0.95)	2	0.89 (0.98-0.97)	3	1.04 (0.98-1.10)	1	4.11	3	1.77 (0.47-4.20)	1	1.60
3	0.58 (0.32-0.88)	3	1.24 (0.69-1.65)	3	2.21 (0.98-4.10)	1	4.11	3	2.15 (1.46-3.50)	1	12.20
7	0.70 (0.19-1.25)	4	0.37 (0.23-1.43)	8	0.84 (0.23-1.49)	4	0.51 (0.33-0.71)	4	0.57 (0.20-1.00)	1	1.72
3	0.29 (0.28-0.30)	2	0.80 (0.30-1.30)	2	0.73 (0.35-1.10)	0		2	1.13 (0.51-1.75)	0	
3	0.48 (0.24-0.86)	2	0.48 (0.20-0.75)	2	0.20 (0.10-0.30)	0		2	0.33 (0.30-0.36)	0	
7	0.86 (0.37-2.20)	4	0.43 (0.30-0.77)	7	0.52 (0.21-0.85)	4	0.43 (0.30-0.68)	4	0.34 (0.10-0.57)	1	0.63
7	0.42 (0.25-0.70)	4	0.23 (0.17-0.30)	8	0.43 (0.14-0.77)	4	0.37 (0.26-0.54)	4	0.27 (0.11-0.48)	1	0.57
7	0.43 (0.10-1.05)	3	0.46 (0.32-0.60)	8	0.48 (0.15-0.81)	4	0.71 (0.36-0.20)	4	0.49 (0.35-0.60)	1	0.64
7	0.26 (0.12-0.39)	4	0.37 (0.11-0.95)	8	0.29 (0.16-0.73)	4	0.26 (0.13-0.43)	4	0.12 (0.12-0.25)	1	0.45
4	0.25 (0.05-0.62)	3	0.13 (0.11-0.15)	4	0.26 (0.05-0.78)	1	0.89	4	0.32 (0.07-0.84)	1	1.09
4	0.16 (0.07-0.30)	2	0.15 (0.10-0.20)	6	0.15 (0.04-0.35)	2	0.12 (0.03-0.20)	2	0.08 (0.07-0.08)	0	
6	0.14 (0.09-0.17)	4	0.15 (0.07-0.28)	8	0.33 (0.08-1.40)	4	0.20 (0.07-0.39)	4	0.36 (0.07-0.88)	1	0.36
7	0.23 (0.18-0.30)	4	0.15 (0.06-0.20)	8	0.18 (0.04-0.30)	4	0.33 (0.16-0.53)	4	0.25 (0.13-0.33)	1	0.50
6	0.14 (0.06-0.26)	4	0.16 (0.10-1.22)	4	0.17 (0.07-0.27)	3	0.16 (0.11-0.25)	4	0.54 (0.06-0.55)	1	1.73
6	0.17 (0.06-0.30)	3	0.07 (0.05-0.10)	7	0.19 (0.04-0.41)	3	0.14 (0.07-0.26)	2	0.19 (0.15-0.22)	1	0.20
5	0.18 (0.06-0.35)	4	0.18 (0.06-0.48)	3	0.19 (0.08-0.25)	3	0.13 (0.05-0.28)	4	0.09 (0.05-0.13)	1	0.32
7	0.08 (0.04-0.18)	3	0.05 (0.01-0.08)	8	0.27 (0.05-0.67)	4	0.10 (0.06-0.19)	4	0.06 (0.03-0.09)	1	0.28
4	0.13 (0.09-0.16)	2	0.09 (0.05-0.13)	2	0.12 (0.11-0.13)	0		3	0.12 (0.06-0.23)	1	0.16
4	0.07 (0.03-0.11)	2	0.03 (0.02-0.03)	5	0.06 (0.02-0.16)	2	0.06 (0.05-0.06)	2	0.06 (0.02-0.09)	0	
3	0.05 (0.05-0.13)	2	0.08 (0.03-0.13)	2	0.10 (0.08-0.12)	0		2	0.07 (0.04-0.10)	0	
6	0.20 (0.05-0.68)	3	0.05 (0.03-0.07)	8	0.13 (0.04-0.23)	4	0.17 (0.02-0.46)	3	0.07 (0.04-0.09)	0	
6	0.09 (0.03-0.16)	3	0.08 (0.04-0.11)	8	0.17 (0.04-0.48)	4	0.15 (0.06-0.36)	3	0.09 (0.06-0.13)	0	
6	0.15 (0.05-0.42)	3	0.11 (0.03-0.24)	7	0.09 (0.06-0.14)	4	0.06 (0.02-0.16)	3	0.05 (0.05-0.06)	0	
7	0.09 (0.02-0.31)	4	0.05 (0.04-0.05)	8	0.07 (0.03-0.14)	4	0.08 (0.05-0.14)	4	0.05 (0.03-0.07)	1	0.23
6	0.07 (0.01-0.15)	3	0.03 (0.01-0.05)	8	0.06 (0.02-0.10)	4	0.05 (0.03-0.07)	3	0.03 (0.01-0.04)	0	
6	13.80 (3.40-39.00)	2	6.00 (3.00-9.00)	7	18.97 (2.80-80.00)	4	14.65 (6.99-32.10)	3	16.33 (5.00-31.00)	1	23.60
4	17.00 (7.00-26.0)	0		1	90.00	0		1	7.00	1	12.00

FIG. 3. Lead concentrations in soft tissues—non-occupational exposure (95% confidence limits).

FIG. 4. Lead concentrations in soft tissues—non-occupational exposure (95% confidence limits).

concentrations were considerably lower than those of adults, varying between 2.16 ppm for rib and 5.80 ppm for the petrous temporal bone. The concentrations of lead in the soft tissues were mostly comparable to those in corresponding female adult tissues; aorta, cartilage, kidney, pancreas, lung, spleen, and blood had rather lower mean values, and skin, suprarenal gland, ovary, and fat had higher values. Also the testes showed mean lead concentrations a little in excess of the mean of the adult values.

The concentrations of lead in the tissues of three age groups of children are shown in Table 5. In eight infants of less than 1 year of age the mean lead concentrations in the tissues were all lower than those in the corresponding tissues of 10 children aged between 2 and 9 years and of five children aged between 13 and 16 years. Bone lead concentrations were similar in the latter two age groups, but the soft tissue lead concentrations in the children aged 2 to 9 years were higher than in the 13 to 16-year-old

group, with the exception of heart, spleen, and blood in which the values were comparable.

The mean faecal lead concentration of 14 of the children was 31.26 $\mu\text{g/g}$ ash (Table 4). On the basis of an excretion mass of 15 g per day dry weight (Ter Haar and Aranow, 1974) and an estimated ratio of ash weight to dry weight of 2.5:1 this would amount to a mean daily excretion of 187.56 μg of lead. Chisolm and Harrison (1956) estimated a normal faecal excretion of lead in children of 132 μg per day, and Barltrop and Killala (1967) of 123 μg per day. The mean faecal lead concentration in the adult subjects with no occupational exposure to lead was shown to lie between 17 and 18 $\mu\text{g/g}$ ash (Table 1). At an estimated dry weight daily faecal mass excretion of 40 g, the daily lead excretion of the adults would approximate to 280 μg , a figure in accord with the findings of Kehoe (1961) and of Thompson (1971) for the normal daily excretion of lead in adults.

Three of the children in the age group 2 to 9 years

TABLE 4
TISSUE LEAD CONCENTRATIONS IN CHILDREN AGED 16 YEARS AND UNDER
(ppm WET WEIGHT)

Tissue	No. samples	Mean	SD	Range
Bone				
Petrous temporal	12	5.80	4.02	0.25- 16.00
Tibia	18	2.71	1.94	0.21- 6.20
Calvarium	12	4.82	2.32	1.10- 9.00
Rib	23	2.16	1.48	0.01- 5.50
Hair	11	10.65	7.36	2.30- 25.00
Nails	5	44.20	37.23	6.00-100.00
Aorta				
Atheroma	4	0.07	0.04	0.03- 0.11
Non-atheroma	14	0.97	2.59	0.04- 9.90
Liver	23	0.64	0.41	0.08- 1.40
Dense connective tissue				
Cartilage	11	0.23	0.15	0.04- 0.50
Ligamentum nuchae	6	0.35	0.35	0.09- 1.00
Kidney				
Cortex	22	0.45	0.36	0.01- 1.20
Medulla	22	0.31	0.24	0.01- 0.80
Hilar lymphatics	11	0.62	0.49	0.04- 1.60
Pancreas	19	0.33	0.17	0.01- 0.68
Ovary	5	0.54	0.56	0.10- 1.50
Prostate	7	0.22	0.19	0.03- 0.50
Skin	11	0.52	0.48	0.07- 1.80
Spleen	22	0.13	0.06	0.02- 0.23
Lung	23	0.14	0.10	0.01- 0.42
Thyroid	17	0.22	0.18	0.01- 0.80
Blood	16	0.12	0.10	0.01- 0.40
Suprarenal	15	0.30	0.32	0.01- 1.20
Brain				
Cortex	22	0.07	0.05	0.01- 0.21
Basal ganglia	12	0.13	0.09	0.04- 0.33
Fat				
Subcutaneous	11	0.36	0.35	0.03- 1.30
Omentum	10	0.18	0.18	0.05- 0.67
Gut				
Caecum	16	0.12	0.07	0.01- 0.25
Midgut	16	0.14	0.12	0.03- 0.52
Stomach	17	0.07	0.05	0.01- 0.20
Testis	7	0.14	0.10	0.03- 0.30
Heart	22	0.07	0.05	0.01- 0.18
Muscle	16	0.08	0.07	0.02- 0.30
Faeces ($\mu\text{g/g}$ ash)	14	31.26	27.85	1.70- 77.00
Urine ($\mu\text{g/l}$)	7	35.90	23.90	8.3 - 71.00

showed a mean faecal lead concentration of 73.33 $\mu\text{g/g}$ ash, equivalent to a mean daily excretion of 440 μg of lead. Exclusion of their faecal values resulted in a mean faecal lead concentration in the remaining 11 children from whom faecal samples were obtained of 20.42 $\mu\text{g/g}$ ash, equivalent to a daily excretion of 122.52 μg of lead.

The concentrations of lead in the tissues of the three children with a high faecal lead content were compared with those of six children less than 8 years of age, from whom faecal samples were obtained with a mean faecal value of 12.53 $\mu\text{g/g}$ ash, equivalent to a daily excretion of 75.18 μg of lead. Their tissue lead concentrations were for the most part in excess of those of the six children with a low faecal

lead content. Their soft tissue concentrations were comparable to adult male soft tissue lead values, but their bone lead values were much less than those of adults of either sex, although in excess of those of the six children with low faecal lead. The raised tissue lead concentrations observed in the three children with high faecal lead suggest an excess of lead intake by ingestion, which probably occurred over a period of time and may have been associated with pica.

One of the children with high faecal lead, a boy aged 9 years at the time of death, was mentally retarded and had suffered from idiopathic hypercalcaemia since infancy for which he had been taking a low calcium diet. Death was due to a cerebrovascular accident caused by hypertension. In view of

TABLE 5
TISSUE LEAD CONCENTRATIONS (ppm WET WEIGHT) IN CHILDREN OF VARYING AGE GROUPS

Tissue	8 infants < 1 year old (4 of each sex)			10 children aged 2-9 years (5 of each sex)			5 children aged 13-16 years (all male sex)		
	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range
Bone									
Petrous temporal	0			8	4.73	0.25- 8.10	4	7.95	3.40-16.00
Calvarium	0			8	4.78	1.10- 6.70	4	4.90	1.90- 9.00
Tibia	5	0.91	0.21-1.53	9	3.70	0.96- 6.20	4	2.73	1.50- 4.40
Rib	8	1.09	0.01-2.71	10	3.01	0.01- 5.50	5	2.18	1.00- 3.30
Aorta	1	0.11		9	0.30	0.04- 1.10	5	2.16	0.04- 9.90
Liver	8	0.35	0.08-1.37	10	0.87	0.44- 1.40	5	0.63	0.34- 0.92
Kidney									
Cortex	8	0.09	0.01-0.22	9	0.70	0.21- 1.20	5	0.58	0.34- 1.00
Medulla	8	0.07	0.01-0.22	9	0.49	0.22- 0.80	5	0.37	0.24- 0.65
Hilar lymphatics	1	0.41		6	0.73	0.04- 1.60	4	0.50	0.10- 0.80
Lung	8	0.06	0.01-0.18	10	0.19	0.05- 0.42	5	0.15	0.10- 0.25
Skin	1	0.10		7	0.63	0.07- 1.80	3	0.39	0.15- 0.70
Subcutaneous fat	1	0.28		7	0.42	0.09- 1.30	3	0.27	0.03- 0.58
Prostate	1	0.10		2	0.48	0.45- 0.50	4	0.13	0.03- 0.25
Pancreas	4	0.15	0.01-0.31	10	0.38	0.17- 0.68	5	0.36	0.22- 0.61
Thyroid	4	0.13	0.01-0.24	9	0.28	0.07- 0.80	4	0.17	0.07- 0.26
Suprarenal	3	0.10	0.01-0.14	8	0.45	0.29- 1.20	4	0.15	0.04- 0.27
Spleen	8	0.10	0.02-0.18	10	0.13	0.08- 0.23	4	0.16	0.13- 0.21
Blood	4	0.03	0.01-0.05	8	0.14	0.05- 0.40	4	0.15	0.07- 0.28
Gut									
Stomach	6	0.03	0.01-0.06	8	0.11	0.05- 0.20	3	0.07	0.04- 0.10
Midgut	5	0.05	0.03-0.08	8	0.20	0.11- 0.52	3	0.14	0.06- 0.18
Caecum	5	0.06	0.01-0.10	8	0.15	0.06- 0.25	3	0.13	0.06- 0.20
Brain cortex	7	0.03	0.01-0.06	10	0.09	0.03- 0.21	5	0.07	0.02- 0.13
Muscle	5	0.06	0.02-0.13	8	0.11	0.03- 0.30	3	0.06	0.02- 0.11
Heart	8	0.03	0.01-0.09	9	0.09	0.03- 0.17	5	0.10	0.03- 0.18
Faeces ($\mu\text{g/g}$ ash)	2	2.58	1.70-3.46	7	41.43	4.00-77.00	5	28.50	9.00-52.00

the reported association of excessive lead intake and nephropathy (Nye, 1929; Fairley, 1934; Henderson, 1954; Emmerson, 1963), a postmortem report on the renal pathology would have been of considerable interest, but unfortunately this was not obtained. The concentrations of lead in the tissues of this child approximated to the concentrations in the tissues of the two other children with a similar faecal lead content, both female and 2 and 3 years of age respectively, one of whom had died from asphyxia in a polythene bag and the other from status asthmaticus; in neither was there a history of hypercalcaemia. These findings suggest that the elevated tissue lead concentrations in the hypercalcaemic child were not influenced by calcium metabolism. The concentrations of lead in the kidneys of the three children were similar, i.e., 1.05 ppm in the cortex and 0.80 ppm in the medulla of the hypercalcaemic child, and 0.74 ppm and 1.20 ppm in the cortex and 0.73 ppm and 0.57 ppm in the medulla of the other two children; these values were in excess of those of the children with a lower faecal lead content.

Occupational exposure to lead

Seven male adults among the total of subjects investigated had past histories of occupational exposure to lead. A further three, for whom no history of occupation was obtained, showed tissue lead concentrations that were strongly suggestive of past occupational exposure, and for this reason these were included among the occupational exposure group. Clinical lead poisoning was not suspected in any of the subjects during life.

The concentrations of lead in the tissues of the 10 subjects are given in Table 6. The mean bone lead concentrations exceeded those of the non-occupationally exposed male adults by about three-fold, ranging between 85.50 ppm in the petrous temporal bone and 29.92 ppm in the rib. Mean soft tissue lead levels were comparable to those in the unexposed group, with the exception of aorta, liver, brain, blood, skin, pancreas, and prostate which showed higher values.

An age-related comparison of nine of the occupationally exposed group with 29 unexposed male subjects, all of whom were over the age of 55 years,

TABLE 6
CONCENTRATIONS OF LEAD (ppm WET WEIGHT) IN TISSUE OF 10 MALE
ADULTS AGED 29-82 YEARS WITH OCCUPATIONAL EXPOSURE TO LEAD

<i>Tissue</i>	<i>No. samples</i>	<i>Mean</i>	<i>SD</i>	<i>Range</i>
Bone				
Petrous temporal ..	2	85.50	6.36	81.00- 90.00
Tibia	9	74.01	58.60	16.60-221.00
Calvarium	2	59.75	6.72	55.00- 64.50
Rib	10	29.92	15.91	8.70- 61.00
Hair	3	65.67	44.61	15.00- 99.00
Nails	3	28.63	15.06	13.90- 44.00
Aorta				
Atheroma	3	4.88	4.40	2.10- 9.95
Non-atheroma ..	4	9.93	12.69	0.32- 27.90
Liver	10	1.93	1.22	0.87- 5.10
Kidney				
Cortex	10	0.66	0.56	0.33- 2.20
Medulla	10	0.63	0.42	0.23- 1.60
Hilar lymphatics ..	8	0.36	0.22	0.02- 0.61
Pancreas	8	0.49	0.37	0.13- 1.20
Prostate	7	0.52	0.88	0.07- 2.50
Skin	4	0.53	0.98	0.01- 2.00
Spleen	9	0.32	0.18	0.11- 0.57
Lung	10	0.31	0.16	0.14- 0.64
Thyroid	9	0.18	0.16	0.03- 0.50
Blood	9	0.31	0.19	0.02- 0.72
Suprarenal	8	0.18	0.15	0.07- 0.52
Brain				
Cortex	10	0.65	1.25	0.03- 4.17
Basal ganglia ..	3	0.29	0.28	0.03- 0.58
Fat				
Subcutaneous ..	5	0.10	0.12	0.01- 0.03
Omentum	1	0.15		
Gut				
Caecum	6	0.18	0.19	0.03- 0.44
Midgut	6	0.12	0.09	0.03- 0.22
Stomach	5	0.10	0.07	0.04- 0.20
Testis	3	0.11	0.03	0.08- 0.14
Heart	9	0.10	0.11	0.01- 0.35
Muscle	5	0.04	0.15	0.02- 0.06
Faeces ($\mu\text{g/g}$ ash) ..	6	24.32	13.00	7.40- 52.20
Urine ($\mu\text{g/l}$)	1	72.0		

showed mean bone lead concentrations that were two to three times greater in the occupationally exposed group. Mean soft tissue lead concentrations were comparable, with the exception of the tissues mentioned above which showed higher values in the group exposed to lead.

The highest values for tibia, rib, aorta, heart, and brain cortex were from an individual who had spent 43 years of his life working in a white lead factory. He had retired from work at the age of 65 and died at the age of 82 from coronary thrombosis (Barry and Mossman, 1970). The remainder of his soft tissue lead concentrations were similar to those of the non-occupationally exposed group. Exclusion of his tissues with high values reduced the soft tissue lead concentrations of the exposed group to levels comparable with those of the unexposed group, but the lead values for rib and tibia remained at more

than double those of the non-occupational exposure group.

Total body burden of lead

The total body burden of lead was assessed in milligrams for each of the groups of adult male and female subjects with no history of occupational exposure to lead, comprising 60 and 36 subjects respectively, for the group of 10 male adults with occupational exposure to lead, and for the 23 children of both sexes under the age of 16 years.

The assessment was made using a table of percentages of organ weights relative to the actual body weight of each subject and also to a standard body weight of 70 kg (Barry and Mossman, 1970). Where an organ was missing the mean lead concentration for that organ in the age group of the subject was substituted, or failing this, the mean value of the

organ for the whole group was taken. The skeleton was represented by the tibia and rib, it being assumed for purposes of the assessment that dense bone and erythropoietic bone are present in the body in a ratio of equal weight.

The total body burden of lead in each group of subjects is given in Table 7. The mean total body burden of lead in female adults was less than that of unexposed male adults, i.e., 103.6 mg compared to 164.8 mg. The mean soft tissue lead totals were 6.2 mg in female adults and 9.3 mg in male adults. The lead in bone amounted to 94% of the total body burden in both sexes, of which over 70% was in dense bone. At a standard body weight of 70 kg the difference in the mean total body burden of lead between the sexes was reduced, i.e., 118.9 mg in female adults and 164.8 mg in male adults; the respective soft tissue values were 7.1 mg and 9.3 mg.

The total body burden of lead in children was much lower than the body burden in either male or female adults. On a basis of a mean actual body weight of 23 kg they showed a mean total body burden of 12.3 mg of lead, which on conversion to a standard body weight of 70 kg became 37.2 mg. Their lead in bone accounted for 72% of the total body burden with small differences between bones. The mean lead content in the soft tissues was 3.4 mg on actual weight, compared to 10.2 mg when assessed on a standard body weight of 70 kg, a figure marginally in excess of the equivalent measurement in adults. In contrast, the content of lead in the bones of the children was proportionately much less than in adults.

An age-related comparison of the total body burdens of lead in children showed a mean lead content in bone of 1.02 mg in eight infants of less than 1 year of age (mean weight 6 kg) compared to 9.36 mg in 10 children aged 2 to 9 years (mean weight 19 kg) and 20.58 mg in five children aged 13 to 16

years (mean weight 58 kg). The soft tissues showed mean lead values of 0.75 mg, 3.45 mg, and 7.37 mg in the respective age groups. The percentage of lead in the bones of the total body burden of lead in the three groups was 57% in the infant group and 73% in each of the other two groups.

When assessed on the basis of a standard body weight of 70 kg, the bone lead content in the infant group was less than half that of the other two groups at 12.95 mg, compared to 32.63 mg and 26.41 mg respectively. The soft tissue lead content in the infant group was 8.75 mg, compared to 13.15 mg and 9.33 mg respectively in the other two groups. The children aged 2 to 9 years showed the highest lead content in both bone and soft tissues of the three groups of children, on a standard body weight assessment of 70 kg.

The occupational exposure group of 10 male adults had mean total body burdens of lead over three times those of the unexposed male group, i.e., 566.4 mg Pb, compared to 164.8 mg (Table 7). Of the total body burden of lead in each group, 97% of the lead was in the bones of the occupationally exposed subjects and 94% in the bones of the unexposed subjects. The mean soft tissue values of the occupational group exceeded the mean soft tissue values of the unexposed group at 15.7 mg and 9.3 mg respectively.

A comparison of the occupational exposure and non-occupational exposure groups of male adults over the age of 55 years showed the mean total body burden of lead of the occupational group to be nearly three times that of the non-occupational group, i.e., 611.3 mg and 213.9 mg. The percentages of lead in bone in each group were 97% and 96% respectively. The mean soft tissue lead values in the occupationally exposed group were nearly double those of the unexposed group, at 16.0 mg and 8.5 mg respectively. When assessed on a standard body

TABLE 7
TOTAL BODY BURDENS OF LEAD IN MILLIGRAMS

Tissue	60 Male adults Non-occupational exposure (mean actual wt 70 kg)			10 Male adults Occupational exposure (Mean actual wt 76 kg)			36 Female adults (Mean actual wt 61 kg)			23 Children (Mean actual wt 23 kg)		
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
Bone	155.5	89.39	21.8-342.0	550.8	411.21	149.7-1668.0	97.4	119.7	12.1-237.4	8.9	8.63	0.2-25.4
Soft tissue	9.3	3.12	2.7- 20.3	15.7	12.11	5.6- 46.3	6.2	1.68	3.2- 9.7	3.4	2.71	0.3- 8.8
Total	164.8	89.58	27.2-353.5	566.4	414.52	162.4-1691.5	103.6	67.03	18.5-244.5	12.3	11.11	0.5-33.7
% Pb in bone ..	94.4			97.2			94.0			72.5		
Standard weight 70 kg												
Bone	← As above →			507.3	126.6	141.1-4	111.8	19.0	329.3	27.0	3.3	58.6
Soft tissue				14.4	5.6	36.4	7.1	4.6	10.3	10.2	7.1	24.9
Total				521.7	137.4	1431.3	118.9	29.0	339.2	37.2	10.4	83.5
% Pb in bone ..				97.2			94.0			72.5		

TABLE 8
TOTAL BODY BURDENS OF LEAD (mg) IN AGE GROUPS—NON-OCCUPATIONAL EXPOSURE

Age group (actual wt)	Male						Total	% Bone
	No. samples	Bone		Soft tissues				
		Mean	Range	Mean	Range			
0- 9	9	5.19	0.17- 19.95	1.82	0.26- 4.87	7.01	74.04	
10-19	8	33.98	18.20- 88.54	9.86	5.28-19.66	43.84	77.51	
20-29	9	53.25	27.30- 77.08	8.95	5.32-11.12	62.20	85.61	
30-39	5	121.58	21.98-256.76	8.77	5.17-11.16	130.35	93.27	
40-49	9	151.94	57.33-335.56	11.20	6.51-20.27	163.14	93.13	
50-59	9	178.43	97.77-342.04	8.57	6.38-11.74	187.00	95.42	
60-69	5	196.17	36.40-259.12	7.85	2.67-13.62	204.02	96.15	
70-79	5	174.77	85.81-262.08	9.36	5.47-10.73	184.13	94.92	
80-89	5	256.89	219.64-295.30	9.56	5.56-14.31	266.45	96.41	
90-99								
(Standard wt 70 kg)								
0- 9	9	20.58	3.27- 46.54	9.74	7.12-12.65	30.32	67.88	
10-19	8	38.25	12.51- 97.39	11.16	5.05-21.63	49.41	77.41	
20-29	9	54.75	29.28- 84.34	9.26	7.32-12.23	64.01	85.53	
30-39	5	112.95	21.02-256.76	8.02	4.95-11.16	120.97	93.37	
40-49	9	132.77	35.14-295.29	9.47	6.51-17.84	142.24	93.34	
50-59	9	170.87	97.59-317.31	8.29	6.42-10.63	179.16	95.37	
60-69	15	210.08	85.08-338.34	8.27	3.33-11.45	218.35	96.21	
70-79	5	202.38	82.07-320.32	10.66	6.33-13.67	213.04	95.68	
80-89	5	311.71	281.28-385.88	11.15	9.41-14.31	322.86	96.55	
90-99								

Age group (actual wt)	Female						Total	% Bone
	No. samples	Bone		Soft tissues				
		Mean	Range	Mean	Range			
0- 9	9	6.00	1.18- 14.16	2.68	0.69- 5.79	8.68	69.12	
10-19	1	72.35		6.54		78.89	91.71	
20-29	5	37.58	21.34- 52.07	5.73	3.34- 7.75	43.31	85.80	
30-39	2	152.88	125.58-180.18	6.51	5.99- 7.03	159.39	95.92	
40-49	7	73.22	12.10-160.16	7.44	5.26- 8.95	80.66	90.78	
50-59	4	80.92	31.12-131.13	5.13	3.35- 6.53	86.05	94.04	
60-69	8	103.63	27.27-237.37	6.61	3.18- 9.74	110.24	94.00	
70-79	4	114.17	58.57-235.46	5.09	3.19- 6.61	119.16	95.73	
80-89	4	145.18	36.25-229.32	5.39	4.20- 6.93	150.57	96.42	
90-99	1	236.97		6.88		243.85	97.18	
(Standard wt 70 kg)								
0- 9	9	27.18	8.13- 58.57	12.65	7.57-24.90	39.83	68.24	
10-19	1	79.59		7.19		86.78	91.71	
20-29	5	40.48	24.02- 54.54	6.17	5.25- 7.75	46.65	86.77	
30-39	2	140.12	115.11-165.16	5.97	5.49- 6.44	146.09	95.91	
40-49	7	74.37	19.01-160.16	7.77	5.26-10.03	82.14	90.54	
50-59	4	92.48	36.03-131.13	5.60	4.91- 6.23	98.08	94.29	
60-69	8	132.99	42.84-329.32	8.39	5.38-10.34	141.38	94.07	
70-79	4	149.74	85.09-287.78	6.65	5.40- 8.08	156.39	95.75	
80-89	4	165.86	49.84-242.74	6.47	4.60- 9.15	172.33	96.25	
90-99	1	325.83		9.46		335.29	97.18	

weight of 70 kg, the occupational group showed a mean total body burden of lead of 570.5 mg compared to 226.8 mg in the unexposed group, a ratio of 2.52:1. The mean soft tissue lead values were 14.9 mg and 9.0 mg respectively, a ratio of 1.66:1.

The total soft tissue body burdens of lead were greater in the occupationally exposed group than in the unexposed group, but bone lead body burdens were proportionately greater still.

The total body burdens of lead are shown on an

age-related basis for male and female subjects with no occupational exposure to lead in Table 8. The mean bone lead content represented more than 90% of the total body burden of lead in both sexes in each age group after the third decade of life. When assessed against the actual weight of each subject, the amount of lead in bone ranged from a mean of 5.19 mg in the first decade up to 256.89 mg in the ninth decade in male subjects, and in female subjects 6.00 mg in the first decade up to 163.53 mg in those over 80 years of age. The mean of the soft tissue lead values in each age group ranged between 1.82 mg and 11.20 mg in male subjects, and between 2.68 mg and 7.44 mg in female subjects. At a standard body weight for all subjects of 70 kg the mean bone lead content in males ranged between 20.58 mg in the youngest age group and 311.71 mg in the oldest, and in females between 27.18 mg in the youngest and 197.85 mg for those over 80 years of age. The mean soft tissue lead content in males varied between 8.02 mg and 11.16 mg, and in females between 5.60 mg and 12.65 mg, the higher values coming from the childhood age groups.

Figure 5 shows, on a basis of actual weight, that the bone lead content increased with age in both sexes but more substantially in the males. The soft tissue lead content was generally greater in males and in both sexes showed a regression with a peak

lead content in the 30 to 40-year-old age group, followed by a small reduction and a further small rise after the seventh decade.

Hair and nails

The postmortem studies provided evidence that lead was concentrated in hair and nails to a greater degree than in soft tissues in both male and female adults and in children. Wide variations in lead concentrations between samples were observed, particularly in hair.

A comparison of results for male and female adults and children is shown in Table 9. The mean values for 65 subjects from whom samples were obtained were 11.4 ppm in hair and 10.7 ppm in nails. Lead in hair values of 1 000 ppm in a male adult with no occupational exposure to lead and 325 ppm in a female adult were excluded from the above results. If these are added to the total the mean value for lead in hair becomes 30.8 ppm. In this female subject it was noted that the initial dark colouration of the hair became progressively lighter during the preliminary washing process with a detergent solution, suggesting that a dye, probably containing lead, had been used during life.

Excluding the two highest values, the mean lead in hair concentration was lower in 33 non-occupationally exposed male adults than in 18 female adults

FIG. 5. Total body burdens of lead (95% confidence limits): actual weight.

TABLE 9
LEAD CONCENTRATIONS IN HAIR AND NAILS (ppm) IN POSTMORTEM SUBJECTS

	Total	Male adults		Female adults	Children
		Non-occupational exposure	Occupational exposure		
<i>Hair</i>					
No. samples	65 (67) ¹	33 (34)	3	18 (19)	11
Mean	11.4 (30.8)	6.6 (35.8)	65.7	11.5 (28.0)	10.7
SD	17.1	5.1	44.6	14.9	7.4
Range	0.7-99.0 (0.7-1000.0)	1.0-20.0 (1.0-1000.0)	15.0-99.0	0.7-55.0 (0.7-325.0)	2.3-25.0
<i>Nails</i>					
No. samples	47	28	3	11	5
Mean	10.7	4.7	28.6	5.6	44.2
SD	17.6	3.5	15.1	4.2	37.2
Range	0.7-100.0	0.7-15.0	13.9-44.0	1.5-15.0	6.0-100.0

¹Two highest hair lead values included in parentheses

at 6.6 ppm and 11.5 ppm respectively. The concentrations of lead in nails were lower than in hair and were less divergent at mean values of 4.7 ppm in 28 males and 5.6 ppm in 11 females. The mean concentration of lead in the hair of 11 children approximated to the adult female value of 10.7 ppm, but the mean lead in nails concentration of 44.2 ppm in samples obtained from five children exceeded equivalent adult values by more than eightfold. In the report by Barry and Mossman (1970) a mean concentration of 19.9 ppm lead was recorded in the hair of eight living children, which included three who gave nail samples with a mean lead concentration of 35.0 ppm.

Barltrop *et al.* (1974) found significant differences in hair lead content among two groups of children living in separate districts with high and low lead contents in the soil (10 000 ppm and 500 ppm). Children with pica for soil living in the high soil lead area showed mean hair lead values of 21 ppm compared to 16 ppm for children without pica. In children living in the low soil lead area no significant difference was noted in hair lead concentrations between those with pica and those without, i.e., 10 ppm and 9 ppm respectively.

Hair lead concentrations showed no evidence of regression with age in either sex. Nails showed high lead values in the young age groups in both sexes, but by the third decade of life the values had reduced and showed no evidence of regression thereafter.

Only three postmortem samples of hair and nails were obtained from the occupationally exposed subjects. These showed mean lead values of 65.7 ppm in hair and 28.6 ppm in nails, both much higher than equivalent values in non-occupationally exposed

adult subjects. Two of the three occupationally exposed subjects were still actively engaged at work prior to their deaths, one as a painter and the other as a scrapyard worker.

To determine whether a correlation might exist between the concentration of lead in hair and lead in blood and urine, 32 adult male lead workers were examined, whose prime exposure was to inorganic lead with an additional lesser exposure to organo-lead. Hair samples were obtained from each worker and divided so as to represent hair proximal and hair distal to the scalp. The samples underwent a triple wash with detergent solution prior to analysis.

The results showed that hair distal to the scalp contained higher lead concentrations than hair proximal to the scalp, at mean concentrations of 517 ppm and 402 ppm respectively. The mean blood lead concentration was relatively low at 30 µg/100 g blood. The highest value of 67 µg Pb/100 g blood was within acceptable limits for occupational exposure (Lane *et al.*, 1968). The mean urinary lead concentration was 82 µg/litre at a specific gravity of 1.019.

The concentrations of lead in hair were disproportionately high compared to the concentrations of lead in blood and urine. They greatly exceeded the concentrations in the postmortem subjects.

There was no correlation between lead in hair and the time each individual spent in the industry, or with concentrations of lead in blood and in urine. Regression analysis showed no evidence of increase of lead concentration in hair with age. Blood lead and urinary lead concentrations did not relate to age nor to the period of time that the individuals were employed in the industry.

Discussion

A study of lead concentrations in the tissues from a series of postmortem subjects has shown a difference between the sexes and between children and adults. Male adults were found to have more lead in their tissues than female adults, and in both sexes bone was shown to accumulate lead with age. The accumulation of lead in bone was far from uniform, which suggests that the intake of lead in food and beverages and of airborne lead inhaled during the course of various occupations at home and at work varied widely between individuals. An explanation for the observed difference between the sexes could be that male adults consume a greater bulk of food and therefore of lead than female adults, and may also be involved in a greater variety of occupational experiences which carry an enhanced exposure to lead.

The results suggest that lead is retained in the dense bones of the body and does not become available for subsequent release. Osteoporosis with decalcification of the skeleton is a common physiological accompaniment of old age. If loss of lead from bone accompanied decalcification, then it would be reasonable to expect the lead concentrations in the bones of the elderly, irrespective of sex, to show evidence of decrease. The findings do not provide such evidence but tend to support the view that lead deposited in dense bones does not subsequently become mobilized to affect physiological function irrespective of the effects on body metabolism of disease processes. These observations run counter to those of Hardy (1965; 1966) who considered that the mobilization of lead from the skeleton of the aging may be a cause for serious concern. A preliminary assessment of our data did not suggest that a correlation would be found in respect of tissue lead concentrations and disease states, or causes of death.

A significant point with respect to any effect which might result from the combined release of lead and calcium from bone would be the concentration in bone of lead relative to calcium. At a total body burden of lead in bone of 220 mg, assessed as the amount present in a mature man of 70 kg, and of calcium in the skeleton of 1.1 kg, the ratio of lead to calcium would approximate to one part in 5 000. If the amount of lead released from bone was determined by the rate of release of calcium, then a concentration of lead in blood sufficient to cause concern in relation to health would necessitate a proportionately much larger release of calcium. Assuming a total blood volume of 5 litres, to increase the concentration of lead in blood from 20 $\mu\text{g}/100$ ml to 40 $\mu\text{g}/100$ ml would necessitate an addition of 1 mg of lead to the total blood volume. This would be equivalent to 5 g of calcium, i.e., 100 mg Ca/100

ml blood, a concentration 10 times in excess of normal blood calcium levels. In considerations of possible effects of the release of lead from bone in conjunction with calcium, the large difference in ratio of lead to calcium in bone should not be overlooked.

The concentrations of lead in the majority of the soft tissues appeared to achieve equilibrium during the second decade of life in both sexes and thereafter were maintained, irrespective of age or of concentrations of lead in bone. The retentive capacity of bone for lead and its unavailability for release into soft tissues is exemplified by the findings in the tissues of the group of occupationally exposed male adults.

In the case of children the results indicated that concentrations of lead in their soft tissues approximated to the values found in adults and in some tissues exceeded adult values. In contrast, bone lead concentrations were very much lower than in adults. These findings suggest that the immature skeletal structure of children is less able to absorb and retain lead than is the mature dense bone of adults. In some measure this might account for a higher concentration of lead to be found in the soft tissues of children exposed to excessive lead intake than in the same tissues of mature adults exposed to an equivalent intake, bearing in mind differences of weight and surface area.

Thompson (1971), in a lead balance study, estimated that individuals retain an average of approximately 10 μg of lead per day. This would be equivalent to 219 mg of lead retained after 60 years, virtually the same as the postmortem finding of 218 mg assessed on a standard body weight of 70 kg in the bones of the group of unexposed male subjects over the age of 55 years whose average age was 68. However the accumulation of lead in bone will depend upon past exposure which may fluctuate widely in the course of a life span. It is unlikely that a quantitative assessment of retention can be accurately determined on a day to day basis.

It has been suggested that the lead content of hair may represent the level of lead exposure and absorption to which an individual might have been subjected (Kopito, Briley, and Schwachman, 1969; Hammer *et al.*, 1971; Dakhakhny and Sadik, 1972; Renshaw, Pounds, and Pearson, 1972; Weiss, Whitten and Leddy, 1972; Barltrop *et al.*, 1974).

The results from the 32 lead workers in the present study suggest that much of the lead found in hair came from sources external to the body and was not representative of the body burden. The facility of lead for adsorption onto keratin together with the presence of sulphhydryl groups in hair which have a strong affinity for lead would argue that the lead present in the atmosphere, much greater in a lead industry than in the general ambient air, may have

contributed directly to the concentrations of lead that were found. The difference in lead concentrations between hair proximal to the scalp and hair distal to the scalp lends support to this hypothesis.

Dakhakhny and Sadik (1972) found that in hair samples from a group of lead workers, washing with detergent solution alone was insufficient to remove all lead contaminating the hair, and they incorporated a wash with 1% hot nitric acid. The average of their results was less than 20 ppm of lead in hair and none exceeded 81 ppm, values very much lower than those found in our own group of lead workers. By contrast the mean lead concentration of 61 µg/100g in their group of workers was twice that of our group. Treatment with nitric acid would appear to reduce surface contamination to a marked degree but might also result in a loss of lead from hair that may have been incorporated by physiological absorption as opposed to surface attachment.

The concentrations of lead in nails were generally less divergent and lower than those found in hair in the group of postmortem subjects, with the exception of the children where the reverse held true. Samples were obtained from three of the occupationally exposed male adults and these showed higher values for both hair and nails than the unexposed group.

There did not appear to be a correlation between the concentrations of lead in hair or nails and the total body burden of lead in the postmortem group of subjects not occupationally exposed to lead. In their study, Schroeder and Tipton (1968) considered it doubtful whether hair lead levels reflected body burdens.

The concentration of lead in hair does not seem to afford a good parameter upon which to base an accurate estimate of lead absorption. It may provide broad evidence of environmental exposure of relatively recent origin, but because of the probability of the firm adherence of lead from external sources, hair lead cannot be regarded as a reliable indicator of lead intake which could supersede the measurement of lead in blood and urine or of ALA and coproporphyrin in urine.

The findings in this investigation in which the study by Barry and Mossman (1970) was extended by additional data did not differ fundamentally from those of the earlier report. An exception was the earlier observation of a biphasic frequency distribution of lead in the male tibia which was not confirmed and did not warrant the explanation put forward in the original study.

Lead is an inevitable constituent in the body of man where in soft tissues it is in balance in dynamic equilibrium, as was demonstrated by Kehoe (1961). The equilibrium may be altered by change of intake which may be excessive in the case of workers exposed to lead, and in children among deprived

families living in poor socio-economic circumstances in whom the habit of pica may be prevalent. Chisolm (1971), Sachs (1974), Guinee (1971), Emmerson (1963), Henderson (1954), Laurer *et al.* (1973), and Barltrop (1971; 1972) have clearly defined those situations which give rise to a childhood risk, practically all associated with the ingestion of peeling flecks of lead-containing paint from the surfaces of old dilapidated houses. Other unusual causes giving rise to high lead intake include the drinking of alcoholic and other beverages contaminated with lead, the use of cooking utensils and improperly glazed ceramic tableware containing lead, the ingestion by children of lead in toys and exposure to the burning of scrap lead batteries, water supplies contaminated by lead piping, cosmetics and factory emissions (Conway, 1940; Cantarow and Trumper, 1944; Travers, Rendle-Short, and Harvey, 1956; Lane and Lawrence, 1961; Morgan, Hartley, and Miller, 1966; Harris and Elsea, 1967; Walls, 1969; Klein *et al.*, 1970; Hickman, 1970; Beattie *et al.*, 1972a; Beattie *et al.*, 1972b; Fugaš *et al.*, 1973; Martin *et al.*, 1974; and McNeil and Ptasnik, 1974).

The majority of the findings in this report compare quite closely with those of Tompsett (1936), Morris (1940), Kehoe (1961), Nusbaum *et al.* (1965), and Schroeder and Tipton (1968). They do not suggest that levels of lead in the environment, with the exception of unusual circumstances of exposure, have caused an increase of lead uptake in body tissues in recent decades. The physiological capacity of man for the disposal of varying quantities of absorbed lead would appear to be highly developed and well adapted in protecting him from its adverse effects. The evidence does not support the conjecture that present-day levels of lead in the environment are a cause for concern in relation to the health of the population in general.

I wish to acknowledge the invaluable contribution of Dr. D. B. Mossman who provided the bulk of the tissue samples from the postmortem subjects, and the work of Mr. E. H. Lowe and his staff at the Lostock Green biological laboratory of The Associated Ocel Company Limited who undertook the analysis of the tissues. I record also my appreciation of Mr. J. F. Church who was responsible for the statistical analysis of the data, of Miss B. Baker who provided information on case histories and occupations of the subjects investigated, and of Mr. F. Murray and Mrs. D. E. Lowe for the assembly of data and secretarial assistance.

References

- Allcroft, R. (1951). Lead poisoning in cattle and sheep. *Veterinary Record*, **63**, 583-590.
- Baker, G. (1941). An enquiry concerning the cause of the endemial colic of Devonshire. Paper presented at the

- College of Physicians, London 1767. Reproduced in *Industrial Medicine*, **10**, 260-266.
- Barltrop, D. (1971). Lead poisoning. *Archives of Diseases in Childhood*, **46**, 233-235.
- (1972). Children and environmental lead. *Proceedings of Conference on Lead in the Environment* held at the Zoological Society, London, pp 52-59.
- and Killala, N. J. P. (1967). Faecal excretion of lead by children. *Lancet*, **2**, 1017-1019.
- , Strehlow, C. D., Thornton, I., and Webb, J. S. (1974). Significance of high soil lead concentrations for childhood lead burdens. *Environmental Health Perspectives*, Experimental Issue No. 7, pp. 75-82.
- Barry, P. S. I. (1973). A comparison of lead concentrations in human bones and in soft tissues. In *International Symposium on Environmental Health Aspects of Lead*, p. 415. Luxembourg, Commission of the European Communities (EURATOM 5004).
- and Mossman, D. B. (1970). Lead concentrations in human tissues. *British Journal of Industrial Medicine*, **27**, 339-351.
- Beattie, A. D., Dagg, J. H., Goldberg, A., Wang, I., and Ronald, J. (1972a). Lead poisoning in rural Scotland. *British Medical Journal*, **2**, 488-491.
- , Moore, W. R., Devenay, W. T., Millar, A. R., and Goldberg, A. (1972b). Environmental lead pollution in an urban soft-water area. *British Medical Journal*, **2**, 491-493.
- Cantarow, A. and Trumper, M. (1944). *Lead Poisoning*, pp. 168-170. Williams and Wilkins, Baltimore.
- Chisolm, J. J. (1971). Lead poisoning. *Scientific American*, **224**, 15-23.
- and Harrison, H. E. (1956). The exposure of children to lead. *Paediatrics*, **18**, 943-957.
- Conway, N. (1940). Lead poisoning from unusual causes. *Industrial Medicine*, **9**, 471-476.
- Dakhakhny, A. A. and Sadik, Y. M. (1972). Lead in hair among exposed workers. *American Industrial Hygiene Association Journal*, **33**, 31-34.
- Emmerson, B. T. (1963). Chronic lead nephropathy; the diagnostic use of calcium EDTA and the association with gout. *Australasian Annals of Medicine*, **12**, 310-324.
- Fairley, K. D. (1934). A review of the evidence relating to lead as an etiological agent in chronic nephritis in Queensland. *Medical Journal of Australia*, **1**, 600.
- Fugaš, M., Wilder, B., Pauković, R., Hršak, J., and Steiner-Škreb, D. (1973). Concentration levels and particle size distribution of lead in the air of an urban and an industrial area as a basis for the calculation of population exposure. In *International Symposium on the Environmental Health Aspects of Lead*. Luxembourg, Commission of the European Communities (EURATOM 5004).
- Goyer, R. A. and Rhyne, B. C. (1973). Pathological effects of lead. *International Review of Experimental Pathology*, **12**, 35.
- Guinee, V. F. (1971). Lead poisoning in New York City. *Transactions of the New York Academy of Sciences*, **33**, 539-545.
- Hammer, D. I., Finklea, J. F., Hendricks, R. H., Shy, C. M., and Horton, R. J. M. (1971). Hair trace metal levels and environmental exposure. *American Journal of Epidemiology*, **93**, 84-92.
- Hardy, H. L. (1965). Lead: a review. *US Public Health Service Symposium on Environmental Health*, pp. 73-83.
- (1966). What is the status of knowledge of the toxic effect of lead on identifiable groups in the population? *Clinical Pharmacology and Therapeutics*, **7**, 713-722.
- Harris, R. W. and Elsea, W. R. (1967). Ceramic glaze as a source of lead poisoning. *Journal of the American Medical Association*, **202**, 544-546.
- Henderson, D. A. (1954). A follow-up of cases of plumbism in children. *Australasian Annals of Medicine*, **3**, 219-224.
- Hickman, J. R. (1970). Lead poisoning: Pottery glazes, an often ignored hazard. Paper presented to the Canadian Safety Council, Fredericton, N.B., Canada, May 26.
- Hislop, J. S., Parker, A., Spicer, G. S., and Webb, M. S. W. (1973). The Determination of Lead in Human Rib Bone. Report of the Analytical Sciences Division of UK Atomic Energy Authority Research Group, AERE, Harwell, England.
- Hunter, D. (1957). *The Diseases of Occupations*, 2nd edition, p. 219. The English Universities Press, London.
- Kehoe, R. A. (1961). The Harben Lectures, 1960. The metabolism of lead in man in health and disease. *Journal of the Royal Institute of Public Health and Hygiene*, **24**, 81-96, 101-120, 129-143 and 177-203.
- (1963). Industrial lead poisoning. In *Industrial Hygiene and Toxicology*, 2nd revised edition, edited by F. A. Patty, vol. 2, pp. 941-985. Interscience Publishers, New York.
- , Thamann, F., and Cholak, J. (1933). On the normal absorption and excretion of lead. *Journal of Industrial Hygiene*, **15**, 257-272.
- Klein, M., Namer, R., Harpur, E., and Corbin, R. (1970). Earthenware containers as a source of fatal lead poisoning. Case study and public health considerations. *New England Journal of Medicine*, **283**, 669.
- Kopito, L., Briley, A. M., and Schwachman, H. (1969). Chronic plumbism in children: diagnosis by hair analysis. *Journal of the American Medical Association*, **209**, 243-248.
- Lane, C. R. and Lawrence, A. (1961). Home-made wine as a cause of lead poisoning. *British Medical Journal*, **2**, 939-940.
- Lane, R. E. *et al.* (1968). Diagnosis of inorganic lead poisoning: a statement. *British Medical Journal*, **4**, 501.
- Laurer, G. R., Albert, R. E., Kneip, T. J., Pasternack, B., Strehlow, C. D., Nelson, N., and Kent, F. S. (1973). The distribution of lead paint in New York City tenement buildings. *American Journal of Public Health*, **63**, 163-168.
- Martin, A. E., Fairweather, F. A., St. J. Buxton, R., and Roots, I. M. (1974). Recent epidemiological studies on environmental lead of industrial origin. *International Symposium on Recent Advances in the Assessment of the Health Effects of Environmental Pollution*, Paris, June 24-28.
- McNeil, J. L. and Ptasnik, J. A. (1974). Evaluation of long-term effects of elevated blood lead concentrations in asymptomatic children. *International Symposium on Recent Advances in the Assessment of the*

- Health Effects of Environmental Pollution, Paris, June 24-28.*
- Morgan, J. M., Hartley, M. W., and Miller, R. E. (1966). Nephropathy in chronic lead poisoning. *Archives of Internal Medicine*, **118**, 17-29.
- Morris, H. P. (1940). Age and the lead content of certain human bones: a compilation and statistical analysis of recently published data. *Journal of Industrial Hygiene and Toxicology*, **22**, 100-103.
- National Research Council (1972). Airborne Lead in Perspective. A report by the Committee on Biological Effects of Atmospheric Pollutants of the Division of Medical Sciences, p. 119. Washington, N.A.S.
- Nusbaum, R. E., Butt, E. M., Gilmour, T. C., and Didio, S. L. (1965). Relation of air pollutants to trace metals in bone. *Archives of Environmental Health*, **10**, 227-232.
- Nye, L. J. J. (1929). An investigation of the extraordinary incidence of chronic nephritis in young people in Queensland. *Medical Journal of Australia*, **2**, 145.
- Percival, T. (1774). *Observations and Experiments on the Poison of Lead*. J. Johnson, St. Paul's Church-Yard, London.
- Ramazzini, B. (1713). *De Morbis Artificum Bernardini Ramazzini, Diatriba (Diseases of Workers)*. The Latin text of 1713. Revised. With translation and notes by W. C. Wright, University Chicago Press, Chicago, 1940.
- Renshaw, G. D., Pounds, C. A., and Pearson, E. F. (1972). Variation in lead concentration along single hairs as measured by non-flame atomic absorption spectrophotometry. *Nature*, **238**, 162-163.
- Sachs, H. K. (1974). Effects of a screening program on changing patterns of lead poisoning. *Environmental Health Perspectives*, Experimental Issue No. 7, pp. 41-45.
- Schroeder, H. A. and Tipton, I. H. (1968). The human body burden of lead. *Archives of Environmental Health*, **18**, 965-978.
- Tanquerel des Planches, L. (1848). *Lead Diseases—a Treatise*, translated by Samuel L. Dana. (a) pp. 31-36; (b) p. 43; (c) p. 45. Daniel Bixby Lowell, Mass.
- Ter Haar, G. and Aranow, R. (1974). New information on lead in dirt and dust as related to the childhood lead problem. *Environmental Health Perspectives*, Experimental Issue No. 7, pp. 83-89.
- Thompson, J. A. (1971). Balance between intake and output of lead in normal individuals. *British Journal of Industrial Medicine*, **28**, 189-194.
- Tompsett, S. L. (1936). The distribution of lead in human bones. *The Biochemical Journal*, **30**, 345-346.
- Travers, E., Rendle-Short, J., and Harvey, C. C. (1956). The Rotherham lead-poisoning outbreak. *Lancet*, **2**, 113-116.
- Walls, A. D. F. (1969). Home-made cyder: source of lead poisoning. *British Medical Journal*, **1**, 98.
- Weiss, D., Whitten, B., and Leddy, D. (1972). Lead content of human hair (1871-1971). *Science*, **178**, 69-70.

Received for publication 1 April 1974.

Accepted for publication 13 June 1974.