4. Campbell DJ, Banks AJ, Davis GD. The value of preliminary bone scanning in staging and assessing the prognosis of breast cancer. *Br J Surg* 1976; **63:** 811-816.
5. Bishop HM, Blamey RW, Morris AH. Bone scanning: its lack of value in the follow up of patients with breast cancer. *Br J Surg* 1979; **66:** 752-754.
6. Spencer GR, Khan M, Bird C, Seymour R, Brown TR, Collins CD. Is bone scanning of value in patients with breast cancer? *Acta Chir Scand* 1981; **147:** 247-248.
7. Ciatto S, Pacini P, Azzini V, *et al.* Preoperative staging of primary breast cancer. *Cancer* 1988; **61:** 1038-1040.
8. *Guide to Fees for Medical Services.* Cape Town: MASA Publications, 1991.
9. British Breast Group. Bone scanning in breast cancer: preliminary statement. *BMJ* 1978; **2:** 180-181.
10. Rossing N, Munck O, Pors Neilsen S, West Andersen K. What do early bone scans tell about breast cancer patients? *Br J Cancer Clin Oncol* 1982; **18:** 629-636.
11. Fisher B, Slack N, Kalbrych D, Woolmark N. Ten year follow up of patients with breast cancer in a co-operative clinical trial evaluating surgical adjuvant chemotherapy. *Surg Gynecol Obstet* 1975; **40:** 528-534.
12. Thompsen H, Lund JO, Munck O, Anderson KW. Experience with 7 604 bone scintigraphies at the time of operation for breast cancer 1977-1987. *Dan Med Bull* 1989; **36:** 481-483.
13. Forrest APM, Cant ELM, Roberts M, *et al.* Is the investigation of patients with breast cancer for occult metastases worth while? *Br J Surg* 1979; **66:** 749-751.
14. Kennedy H, Kennedy N, Barclay M, Horobin M. Cost efficiency of bone scans in breast cancer. *Clin Oncol* 1991; **3:** 73-77.

Accepted 2 Oct 1995.

Evidence of undue lead exposure in Cape Town before the advent of leaded petrol

S. R. Grobler, F. S. Theunissen, L. S. Maresky

Lead concentrations were determined in the exhumed teeth of 28 people who lived in the Cape Town area before the combustion of leaded petrol (i.e. before 1922). The lead content of circumpulpal dentine was analysed by graphite-furnace atomic absorption spectrometry. The mean lead level in the dentine of primary teeth (N = 6) was 109 µg/g, while that in secondary teeth (N = 22) was 315 µg/g. The current lead levels in circumpulpal dentine of Cape Town residents are reported to be 74 µg/g and 16 µg/g for primary and secondary teeth respectively. It was found that lead pollution of the human body during the period 1812 - 1922 in the Cape Town area was substantially higher than at present. We conclude that the main reasons for this were the widespread use of lead piping and soldering of water tanks, which resulted in a higher incidence of lead poisoning than that attributable to leaded petrol.

S Afr Med J 1996; **86:** 169-171.

Oral and Dental Research Institute, Faculty of Dentistry, University of Stellenbosch, Tygerberg, W. Cape

S. R. Grobler, B.SC. HONS, M.SC., PH.D., D.SC.

F. S. Theunissen, NAT.DIP.MED.TECH., ADV. DIPL. BUS.&ADMIN.

L. S. Maresky, B.D.S., M.CH.D.

Lead has no metabolic role in the human body and its presence is associated with various toxic effects.[1-3] The deposition of lead in calcified tooth structure is indelible evidence of exposure to and absorption of lead. Under normal circumstances there is no turnover of calcified tooth structure once its matrix is complete and mineralised. In man, this absence of tissue turnover is unique to the calcified tooth layers.[4] Therefore, tooth biopsies *in vivo* or after the removal of teeth from the body provide an absolutely reliable source of information about the deposition therein of substances such as lead, fluoride and tetracycline. Furthermore, the age at which the deposition took place can also be determined with a high degree of accuracy.

The deposition of lead in tooth structure is mainly the result of circulating blood lead, which is acquired largely via the inhalation of lead aerosol and, to a lesser extent, gastro-intestinal absorption.[1] Blood lead levels may fluctuate, dependent upon exposure to external sources and limited remobilisation of lead in bone and soft tissue during turnover.

The presence of lead in different tissues of various South African populations has been studied since the early 1980s.[5-12] However, the exhumation of human remains necessitated by urban redevelopment in the Cape Town area afforded a unique opportunity to determine lead concentrations in calcified tooth structures of people who lived in the Cape Town area during the period 1810 - 1922. Prior to 1921, minimal combustion of petrol took place in South Africa. Furthermore, leaded fuel was not utilised during that time. However, prior to 1922 the population was exposed to lead contamination in water reticulation systems, ceramic utensils, pewterware and minimal amounts of industrial and occupational waste. From 1922, an ever-increasing number of vehicles dependent upon leaded petrol for their fuel combustion came into use in South Africa.

The object of this study was to determine the lead concentrations in the circumpulpal dentine of the teeth of a population group who lived in the Cape Town area in the period before the combustion of leaded petroleum.

Materials and methods

The teeth of 28 people, buried between 1863 and 1922 and exhumed recently, were analysed. The circumpulpal dentine was separated mechanically from the rest of each tooth with the help of a diamond disc cutter.[13] The collected pieces of dentine of each tooth were etched with 1.0M hydrochloric acid to remove contamination, washed with lead-free water, dried, weighed[5] and ashed in nitric acid as described by Keating *et al.*[14] The lead in the final solution, which contained 5% nitric acid, was determined by means of graphite-furnace atomic absorption spectrometry, basically according to the method of Keating *et al.*[14]

Results

The average age of the subjects was 43.6 years (SD = 29.3; median = 53.5; range 0.5 - 89 years). Table I shows the circumpulpal lead concentration (µg/g) in the exhumed teeth. The influence of age at the date of death on the lead levels is summarised in Table II. In general the lead concentration

increased significantly with the age of the donor ($P < 0.05$). The regression line for $y = ax^b$ was 0.73. Fig. 1 summarises the results of various workers and compares them with the result of this study.

Table I. Lead values in the circumpulpal dentine of teeth

N	28
Mean	303.7 µg/g
SD	177.7 µg/g
Range	54.5 - 764 µg/g

Table II. Lead accumulation in circumpulpal dentine of excavated teeth in correlation with age at the date of death

	Lead levels (µg/g dry weight)		
Age range (yrs)	Mean (SD)	Range	*N*
0,5 - 10	106 (42)	65 - 122	5
11 - 20	262 (209)	124 - 503	2
21 - 30	219	219	1
31 - 40	173 (89)	110 - 237	2
41 - 50	254 (95)	116 - 310	3
51 - 60	367 (113)	217 - 583	6
61 - 70	354 (108)	200 - 454	4
71 - 80	482 (190)	259 - 697	4
81 - 90	646 (167)	528 - 764	1

Discussion

No difference in the lead content was reported in various studies of deciduous teeth.[15-17] Bercovitz and Laufer[18] also reported no significant difference in the lead level of different tooth types. However, Rabinowitz *et al.*[19] reported that differences in lead levels among tooth types are unlikely to reflect physiological factors such as blood supply to teeth or mineralisation rates. These differences are much more likely to depend on age-related, and consequently tooth-related, differences in exposure patterns. Thus, the general level of lead uptake could vary considerably during the various stages of tooth formation and at different time periods. In this study it was not possible to select the type of tooth from the excavated remains because of a lack of availability, while from the above results this also did not seem to be necessary.

Table Mountain served as the main catchment area for the water supply of Cape Town and surrounding areas for many years. Because of the presence of organic material, the water has a high acid content and is also soft (T. W. Timoney, Water Engineers Division, Cape Town City Council (retired) — personal communication). Therefore, a higher than normal dissolution of lead from the lead piping could have been expected. During 1812, the first reticulation system, linking the reservoirs on Table Mountain with Cape Town households was installed; the main pipes were constructed of cast iron while lead pipes linked each household to these.

Many homes were equipped with wooden storage tanks, which were lead-lined and had lead-soldered joints in order to render them watertight. Such domestic water supply systems remained in use for over a century, until the Cape Town municipality appointed a special committee to investigate the problem of plumbo solvency in 1931.

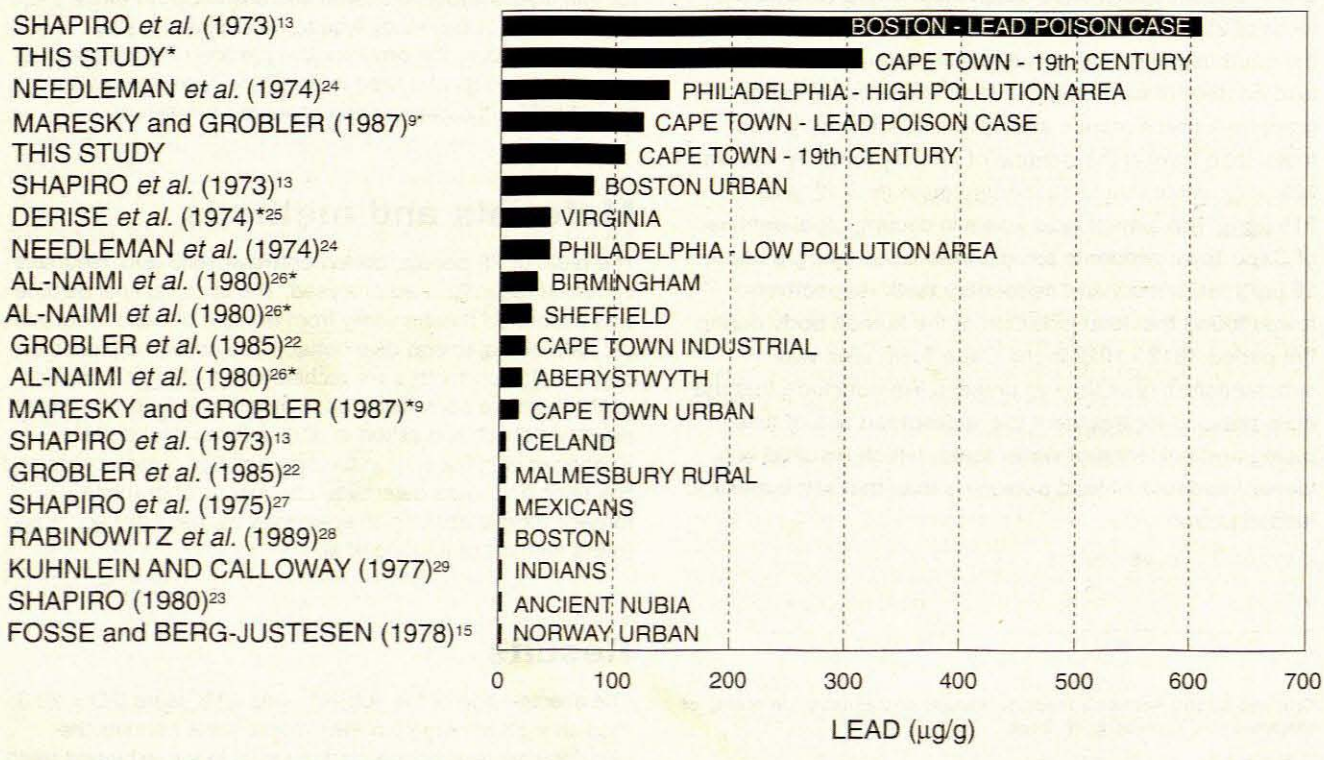

Fig. 1. Comparison of various results.

In 1914 a report by the medical advisor, Dr J. Anderson, showed that domestic water supplies in Cape Town contained lead in concentrations ranging from 0.05 to 0.4 ppm. Furthermore, raw bulk supplies, allowed to stand overnight in new lead pipes, reached levels of 2.3 ppm.[20] In 1917, samples of domestic water from Cape Town households tested by the State Health Laboratories showed levels ranging between 0.5 and 7.63 ppm. In 1926 three cases of lead-poisoning were reported. In Rosebank, a suburb of Cape Town, lead levels of 5.75 ppm were found. In New Church Street in the city, the level was found to be 0.5 ppm and in nearby Hof Street a level of 4 ppm was recorded. In all three cases, lead plumbing was found. In December 1928, a shop in Sir Lowry Road, equipped for soda water production, showed lead levels of 25.2 ppm. The soda water was prepared in copper tanks with lead-soldered joints. From there it was distributed to other outlets in the city.

Strong representations by Dr J. A. Mitchell (Assistant Medical Officer, Department of Home Affairs and later Secretary of the Department of Health) led to the Cape Town municipality's instituting a thorough investigation in early 1931 by a special committee (Cape Town Archives, 3/CT; 3A/10). In 1932, a decision was made to establish the first full-scale water treatment works at Constantia. This chemical treatment resulted in a 90% reduction in the leaching of lead into domestic water supplies. By 1938 several other treatment plants had been constructed and all Table Mountain's water was treated before it entered the domestic supplies of Cape Town (T. W. Timoney — personal communication). From about that time, lead pipes were gradually replaced by copper in all plumbing systems. However, many old houses in the Cape Town area had lead pipes for much longer, as was found in the District Six area during the 1970s.

In general we found an increase in tooth lead content with age (Table II). This has been reported by various authors.[16,21] It must be remembered that lead was analysed in circumpulpal dentine, which is laid down continuously throughout life. A higher correlation between age and lead levels was therefore to be expected. The above reasons could also explain the high standard deviation (Table I). Furthermore, during the collection of circumpulpal dentine, the amount of other dentine which could not be physically separated would vary. Therefore, the lead levels in the circumpulpal dentine could be masked to a greater or lesser extent in different cases.

In this study we analysed the lead levels in the teeth of 6 children under the age of 15 years. The mean lead level was 109 µg/g. This is markedly higher than the 74 µg/g reported for children living in an industrialised urban area in Cape Town[22] before the reduction of lead in petroleum (Fig. 1). The 74 µg/g level was attributed mainly to the degree of industrialisation and the petrol lead level of 0.8 g/l at that stage. The average lead level in the teeth of the adults over 15 years is 315 µg/g (N = 22). This is much higher than the 126 µg/g reported[8] in a person who had been employed continuously as a motor technician in South Africa for 32 years, and had exhibited signs and symptoms typically manifested by people suffering from lead intoxication. However, an average level of 650 µg/g in the circumpulpal dentine of unequivocally lead-poisoned children from Philadelphia and Boston in the USA was reported.[13] The lead level in this study (Table I) contrasts strongly with the low level of 2.5 µg/g reported for children in ancient Nubia where there was minimal lead exposure.[23] The annual lead retention in dentine decreased with age up to an age of about 33 years, after which it stabilised. The Nubian children[23] also showed a decrease in lead retention with increased age. There are many factors which may influence the lead levels of circumpulpal dentine, such as the period of exposure, the exposure level, the type of lead exposure and the diet during exposure.

In conclusion this study clearly demonstrates that lead pollution of the human body in the Cape Town area was unacceptably high before the era of the combustion of leaded petroleum. The main cause was the pollution of drinking water by lead plumbing and solder of water tanks.

Finally, the tooth lead levels associated with the combustion of petrol (0.8 g Pb/litre petrol before 1983),[6] were substantially lower than those attributable to the lead pollutants in early Cape Town's drinking water supplies.

REFERENCES

1. Harrison RM, Laxen DPH, eds. *Lead Pollution: Causes and Control.* New York: Chapman and Hall, 1981: 133-158.
2. Braghurst PA, McMichael AJ, Wigg NR. Environmental exposure to lead and children's intelligence at the age of seven years. *N Engl J Med* 1992; **327:** 1279-1284.
3. Needleman HL, Gunnoe C, Leviton A, *et al.* Deficits in psychological and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979; **300:** 689-695.
4. Needleman H, Tuncay OC, Shapiro IM. Lead levels in deciduous teeth of urban and suburban American children. *Nature* 1972; **235:** 111-112.
5. Van Wyk CW, Grobler SR. Lead levels in deciduous teeth of children from selected urban areas in the Cape Peninsula. *S Afr Med J* 1983; **63:** 559-562.
6. Grobler SR, Maresky LS, Kotze TJ. Lead reduction of petrol and blood lead concentrations of athletes. *Arch Environ Health* 1992; **47:** 139-142.
7. Grobler SR, Maresky LS, Rossouw RJ. Blood lead levels of South African long-distance road-runners. *Arch Environ Health* 1986; **41:** 155-158.
8. Disler PB, Mets JT, Rip MR, Watermeyer GS. Effects of lead on health: A review; Implications of environmental lead in South Africa. *S Afr J Sci* 1984; **80:** 300-304.
9. Maresky LS, Grobler SR. Unusually high circumpulpal lead concentrations as valid evidence of lead intoxication. *Hum Toxicol* 1987; **6:** 475-477.
10. Maresky LS, Grobler SR. Effect of the reduction of petrol lead on the blood lead levels of South Africans. *Sci Total Environ* 1993; **136:** 43-48.
11. Von Schirnding Y, Bradshaw D, Fuggle R, Stokol M. Blood lead levels in South African inner-city children. *Environ Health Perspect* 1991; **94:** 125-130.
12. Grobler SR, Van Wyk CW, Kotze D. Lead levels in deciduous teeth of children from a rural area in the Cape Province, South Africa. *S Afr J Sci* 1984; **80:** 331-332.
13. Shapiro IM, Dobbin B, Tuncay OC, Needleman HL. Lead levels in dentine and circumpulpal dentine of deciduous teeth of normal and lead poisoned children. *Clin Chim Acta* 1973; **46:** 119-123.
14. Keating AD, Keating JL, Halls DJ, Gordon SF. Determination of lead in teeth by atomic absorption spectrometry with electrothermal atomisation. *Analyst* 1987; **112:** 1381-1385.
15. Fosse G, Berg-Justesen N. Lead in deciduous teeth of Norwegian children. *Arch Environ Health* 1978; **33:** 166-175.
16. Moore MR, Campbell BC, Meredith PA, Beattie AD, Goldberg A, Campbell D. The association between lead concentrations. *Clin Chim Acta* 1978; **87:** 77-83.
17. Chatman T, Wilson DJ. Lead levels in human deciduous teeth in Tennessee. *Environ Lett* 1975; **8:** 173-183.
18. Bercovitz K, Laufer D. Tooth type as indicator of exposure to lead of adults and children. *Arch Oral Biol* 1991; **35:** 895-897.
19. Rabinowitz MB, Bellinger D, Leviton A, Wang J. Lead levels among various deciduous tooth types. *Bull Environ Contam Toxicol* 1991; **47:** 602-608.
20. Steyn ER. Lood as bedryfs- en openbare gesondheidsgevaar in Suid-Afrika. MD thesis, University of Pretoria, 1976.
21. Drasch GA, Ott J. Lead in human bones. Investigations on an occupationally non-exposed population in southern Bavaria (F.R.G.) II. *Sci Tot Environ* 1988; **68:** 61-69.
22. Grobler SR, Rossouw RJ, Kotze D. Lead levels in circumpulpal dentine of children from different geographical areas. *Arch Oral Biol* 1985; **30:** 819-820.
23. Shapiro IM, Grandjean P, Nielsen OV. Lead levels in bones and teeth of children in ancient Nubia. In: Needleman HL, ed. *Clinical Implications of Current Research.* Philadelphia: Raven Press, 1980: 35-41.
24. Needleman HL, Davidson I, Sewell EM, Shapiro IM. Subclinical lead exposure in Philadelphia school children. Identification by dentine lead analyses. *N Engl J Med* 1974; **290:** 245-248.
25. Derise NL, Ritchey SJ. Mineral composition of normal human enamel and dentine and the relation of composition to dental caries. *J Dent Res* 1974; **53:** 853-858.
26. Al-Naimi T, Edmonds MI, Fremlin JH. The distribution of lead in human teeth, using charged particle activation analysis. *Phys Med Biol* 1980; **25:** 719-726.
27. Shapiro IM, Mitchell G, Davidson I, Katz SH. The lead content of teeth. Evidence establishing new minimal levels of exposure in a living preindustrialized human population. *Arch Environ Health* 1975; **30:** 483-486.
28. Rabinowitz M, Bellinger D, Leviton A. The blood lead-tooth relationship among Boston children. *Bull Environ Contam Toxicol* 1989; **43:** 485-492.
29. Kuhnlein HV, Calloway DH. Minerals in human teeth: differences between preindustrial trial and contemporary Hopi Indians. *Am J Clin Nutr* 1977; **30:** 883-886.

Accepted 28 Dec 1993.