Pulmonary Distributions of Lead in Human Subjects

Erling A. Mylius and Egil M. Ophus
Department of Pathology,
Trondheim University Hospital
7000 Trondheim, Norway

The presence of lead in human lung tissue has been established by several investigators, and a comparative analysis of lead levels in lungs of different human beings with reference to age, sex, occupation and place of residence has recently been made (STRINGER et al. 1974). However, no information is available concerning the distribution of lead throughout the lung tissue. In a study of the distribution of some other trace elements, MOLOKHIA and SMITH (1967) found that the characteristic modes of distribution differed between essential and nonessential elements. It should be borne in mind that the deposition pattern of inhaled particles affects particle movement and pulmonary clearance and that the effective hazard of inhaled pollutants is influenced by the resultant regional distribution. This paper presents the concentration of lead in five different lung segments and in two pulmonary lymph nodes in ten cases at post mortem examination.

MATERIAL AND METHODS.

Tissue samples obtained by autopsies from ten patients aged from 20 to 69 years were analysed. All subjects, chosen at random, were from the city of Trondheim (population 120,000) and had no known occupational or otherwise abnormal exposure to lead.

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The analyses were carried out by the flameless atomic absorption technique described by STEGAVIK et al. (1976), which allows the analyses of small tissue samples. From each tissue sample, fixed and stored in formalin, ten replicas, each weighing approximately 2 mg, were analysed in the carbon rod atomic absorption spectrophotometer. In the right lung the following segments were analysed: upper lobe apical, middle lobe lateral and lower lobe superior. On the left side of the lung the upper lobe apical and the lower lobe superior lung segments were analysed. In addition, analysis was made of the lead content in right and left paratracheal hilar lymph nodes. Thus, regional distributions of lead in these lungs were determined by a thorough examination of each subjects by taking altogether 50 analyses from the lung tissue. The figures presented in Table 1 are the mean values of ten analyses from each of the five different lung segments. The variation in the values is due, both, to the method of analysis and to the heterogeneous tissue, although care was taken to avoid blood vessels and larger bronchi when sectioning the tissue.

RESULTS

The lead content in tissue samples from five lung segments and two lymph nodes of ten individuals is presented in Table 1. The average concentration of lead in the lungs of each individual ranged from 0,28 ppm (subject no 7) to 1,14 ppm (subject no 1) with an average of 0,56 ppm which is close to the average lung lead level found for the population in this city (unpublished study).

The average concentration of lead in the two lymph nodes analysed in each individual ranged from

0,93 ppm (subject no 3) to 2,60 ppm (subject no 10) with an average of 1,77 ppm. The average lead content of the lymph nodes in each individual exceeded that of the lung tissue from two to six times except for subject no 3 who showed approximately the same concentration of lead both in lymph nodes and in lung tissue.

There was no definite pattern in the distribution of lead between the right and left side neither for the lung segments nor for the pulmonary lymph nodes. It seemed, however, that the apex of the upper lobe tended to be more contaminated than the lower lobes.

In Table 2 the percent deviation of each lung segment from the mean lung lead content is presented as an average value for all individuals except no 2 (see discussion).

DISCUSSION

The concentration of lead in the lung segments, found by means of flameless atomic absorption, were all well below the levels which according to TIETZ et al. (1957), using spectrographic analysis, can be regarded as elevated or pathogenic. MOLOKHIA and SMITH (1967) found that essential trace metals exhibit less variation in concentration from one part of the lung to another than nonessential trace metals do. The lung lead level reflects the lead that has been absorbed by the body and redeposited in the tissue, in addition to any lead that has been deposited in the respiratory tract but not yet absorbed (STRINGER et al. 1974). A varying content and distribution in the lung tissue of this nonessential

TABLE 1.

Mean (±SE) lead concentration (ppm dry weight) in 5 lung segments and 2 pulmonary lymph nodes of ten individuals.

Subject no	1	2	3	4	5	6	7	8	9	10
Age	57	63	21	66	69	32	50	20	20	61
Sex	M	F	F	M	F	F	F	F	M	M
Lung segment										
Right:										
upper lobe apical	2,03 ±0,20	0,85 ±0,08	0,37 ±0,02	0,55 ±0,06	0,61 ±0,03	0,84 ±0,05	0,22 ±0,02	0,39 ±0,03	0,33 ±0,05	0,42 ±0,04
middle lobe lateral	0,72 ±0,06	0,61 ±0,06	0,85 ±0,08	0,32 ±0,05	0,73 ±0,04	0,40 ±0,02	0,19 ±0,02	0,49 ±0,05	0,29 ±0,03	0,50 ±0,07
lower lobe superior	0,78 ±0,06	1,15 ±0,09	0,68 ±0,04	0,23 ±0,02	0,66 ±0,03	0,53 ±0,02	0,21 ±0,02	0,31 ±0,03	0,29 ±0,04	0,60 ±0,03
Left:										
upper lobe apical	1,19 ±0,13	0,15* ±0,01	1,03 ±0,07	0,58 ±0,03	0,53 ±0,05	0,45 ±0,03	0,48 ±0,05	0,29 ±0,04	0,34 ±0,05	0,64 ±0,10
lower lobe superior	0,96 ±0,07	0,55 ±0,05	0,74 ±0,05	0,37 ±0,02	0,51 ±0,03	0,30 ±0,03	0,29 ±0,03	0,36 ±0,03	0,29 ±0,04	0,63 ±0,04
Lymph node										
Right:										
paratracheal hilar	2,88 ±0,25	1,61 ±0,16	0,87 ±0,05	1,89 ±0,15	2,16 ±0,22	1,03 ±0,07	1,87 ±0,17	2,10 ±0,20	1,96 ±0,10	2,06 ±0,21
Left:										
paratracheal hilar	2,20 ±0,15	1,38 ±0,15	0,99 ±0,15	1,04 ±0,08	1,28 ±0,13	1,07 ±0,08	1,79 ±0,20	2,23 ±0,20	1,78 ±0,17	3,14 ±0,24

* collapsed tissue

trace element should therefore be expected due to differences in exposure and physiological response.

TABLE 2

Average percent deviation in 5 lung segments from mean values of lung lead content of 9 individuals.

Right:	
upper lobe apical	32
middel lobe lateral	22
lower lobe superior	17
Left:	
upper lobe apical	25
lower lobe superior	12

Although the number of individuals in this investigation is too small to give the average pulmonary distribution of lead in this population, it is evident (Table 1) that 1) the concentration of lead in human lungs has no fixed value throughout the tissue and 2) there is a considerable variation in the pulmonary distribution of this metal from one individual to another. In addition, there is a tendency towards increased accumulation of lead in the upper lobe segments, this tendency being most prominent in the most heavily exposed individual (no 1). The tendency of the upper lobes' apical segments to be more contaminated than the segments of the lower lobes is an accordance with the findings of MOLOKHIA and SMITH (1967) for some other nonessential trace elements. They stated that this tendency is due to the fact that the source of these elements accumulated in the lungs is airborne dust particles. It is known (MITCHELL 1960) that the mechanisms involved in the deposition of inhaled particles

are related to the physical and chemical properties of the dust as well as to the anatomy and physiology of the respiratory tract. In addition, differences in distribution pattern may be due to the fact that pulmonary function varies with such factors as age, sex, and exercise and that the deposition of particles will depend on tidal volume and breathing rate (CLAYTON 1975).

Atmospheric lead, mainly derived from automobile exhaust, is present predominantly in particles that can penetrate and be retained in both the tracheobronchial and alveolar regions of the respiratory tract (LEE et al. 1968). Both theoretic and experimental considerations have shown that about 50% of inspired lead is retained by the lungs (HICKS 1972). Dependant on particle size, the chief mechanisms of lung deposition are sedimentation and diffusion by Brownian motion (DAVIES 1963). People living in urban areas are exposed to coarser lead particles than subjects living in remote areas with low traffic density, because the lead content in the air and the particle size decrease as the distance from the source of emission increases (DAINES 1970). Thus, in urban areas the sedimentation process in lung deposition is more prominent than in remote areas, and this, surely, will influence the deposition pattern in the lungs.

Physiologically, air ventilation and blood perfusion increases towards the lower part of the lung, while the ventilation-perfusion ratio is highest in the apical segments (BRYANS et al. 1964). This may explain the tendency for the increased deposition of lead in the upper regions of the lungs (Table 1). Evidence has been presented suggesting that the uneven ventilation in the normal lung is the consequence of regional differences

in pleural pressure (MILIC-EMILI et al. 1966). The concentration of lead in lung samples will clearly reflect the functional condition of the tissue. Impaired function in one lung segment may possibly increase the lead burden in other parts of the lung. In subject no 2 (Table 1) the left upper lobe apical lung segment was collapsed, due to pulmonary interstitial fibrosis and areas of atelectasis. The low lead level in this lung segment obviously reflects an impaired state of function. The left paratracheal hilar lymph node from the same subject was enlarged and partly fibrous.

Studies of anthracosis and of coalminers' lungs have shown that there is a tendency for the upper lobes to acquire the largest deposits of dust (DAVIES et al. 1963). The greater retention in the upper parts of the lung of air contaminants may in fact explain why there is often an increased frequency of remnants of pathological conditions in the upper part of the lung. Table 2 confirms that this part of the lung shows a greater variation in the concentration of lead than the lower part. When analysis of entire lungs is not practical due to the limitation of the method, it seems that tissue specimens taken from the lower parts of the lung best reflect the actual lung lead level. We therefore suggest that samples be taken from the segments of the lower lobes when comparative analyses of the lead content in lungs of different populations are made.

Clearance of inhaled lead particles from the lungs, as any other inhaled material, may take place partly by removal via alveolar macrophages. It is also possible, however, that lead particles may penetrate the alveolar wall in the free state and pass to the hilar nodes where

they are trapped (OGATA et al. 1973). In both cases, an increased lead level in local lymph nodes is expected. The higher concentration of lead in local lymph nodes found in our investigation confirms that clearance via the lymphatic vessels is an important mechanism in the handling of lead particles by the human lungs.

SUMMARY

The concentration of lead in five lung segments and in regional lymph nodes was determined post mortem in ten subjects by atomic absorption technique. In each case there was a considerable variation in the concentration of lead in different lung segments, and, in general, the apical segment in the upper lobe tended to be more contaminated than segments in the lower lobes. With one exception, the lead levels in the regional lymph nodes exceeded those of the lung segments. Individual differences in the distribution of lead in the lungs may be due to differences both in exposure and physiological response. When comparative analyses are made, we suggest that samples be taken from the same and preferably the basal segments of the lung.

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