

## Blood Lead and Haemoglobin in Lead Absorption

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In an analysis of blood lead and haemoglobin estimations from 655 lead workers, there was no indication of any change in the haemoglobin until the blood lead exceeded 110  $\mu\text{g./100 ml.}$ ; the slight fall at higher levels of blood lead was not significant at the 5% level of confidence.

Sixty-seven men who had blood leads greater than 90  $\mu\text{g./100 ml.}$  were examined clinically. One had a low haemoglobin but none had symptoms or signs that were likely to be due to lead absorption. A further 18 men had haemoglobins of 89% (13 g./100 ml.) or less. None was thought to be low due to lead absorption.

The absence of symptoms, signs, and low haemoglobins in association with relatively high blood leads is unlikely to be due to errors in blood lead or haemoglobin estimation. Alternative possibilities are that there were no susceptible workers among those studied; or that the stable conditions of exposure in this population allowed the development of relatively high blood leads without other evidence of high lead absorption.

When the haemoglobin is abnormal, blood lead observations may be more meaningful if a correction factor, approximately equal to  $\frac{100}{\text{Hb}\%}$ , is used.

It is concluded that in a population where sophisticated lead control is practised no purpose is served by estimating haemoglobins in all lead workers every three months, but only in those whose blood lead is likely to be in excess of 110  $\mu\text{g./100 ml.}$  It may also be of value in the first year of exposure to detect susceptible workers.

Previous reports have been concerned mainly with the levels of blood lead concentration and haemoglobin which are 'normal' and the levels found in cases of lead poisoning. There is no direct evidence showing at what level of blood lead the anaemia of lead poisoning may begin.

The purpose of this paper is to examine the relationship between lead-in-blood and haemoglobin in 655 workers in an electric accumulator factory.

Cantarow and Trumper (1944) quoted the normal ranges of lead-in-blood suggested by 11 authorities and noted that their upper limits varied from 10  $\mu\text{g./100 ml.}$  to 130  $\mu\text{g./100 ml.}$  Much of this discrepancy was thought to be due to differences in analytic method, and they proposed 80  $\mu\text{g./100 ml.}$  as the upper limit of normal.

Kehoe (1961) stated that the blood lead concentration in normal individuals varies between 15 and

40  $\mu\text{g./100 ml.}$  and averages 'somewhat less than 30  $\mu\text{g./100 ml.}$ '.

Hofreuter, Catcott, Keenan, and Xintaras (1961) found that mean blood lead varied between 11 and 21  $\mu\text{g./100 g.}$ , depending on such factors as sex, smoking habits, and urban or rural exposure.

Similarly, large differences existed in the early literature concerning the level of blood lead at which symptoms of lead poisoning develop. Cantarow and Trumper (1944) quoted six authorities who suggested 100  $\mu\text{g./100 ml.}$ , and others who gave lower levels of 30 to 60  $\mu\text{g./100 ml.}$  More recently, Kehoe (1961) stated that anyone with a blood lead of over 80  $\mu\text{g./100 ml.}$  is in danger of developing an episode of lead intoxication without warning and should be removed from exposure.

Cantarow and Trumper (1944) noted that the anaemia produced by excessive lead absorption has been recognized since Laennec in 1831 described the pallor and 'thinness of the blood' at necropsy. They noted also that most authorities thought that

with increasing lead absorption there is an initial rise in the haemoglobin and red cell count, followed by an anaemia which is rarely severe; nevertheless, an overwhelming majority of observers did not believe that a decreased haemoglobin is the first sign of lead poisoning.

In his Milroy lectures, Lane (1949) said that anaemia is found in lead workers after heavy exposure, and a haemoglobin persistently below 85% should be investigated.

King and Thompson (1961) studied the relation between lead-in-blood and haemoglobin in 540 workers in the motor-car industry. They found little, if any, variation in haemoglobin with blood leads up to 120  $\mu\text{g./100 ml.}$  However, Williams, Matthews, and Judd (1962) have reported cases of lead poisoning with blood leads of 100 to 130  $\mu\text{g./100 ml.}$  and haemoglobins of less than 90%.

Kehoe (1962) bases industrial monitoring and control on measurements of lead-in-blood and lead-in-urine without haemoglobin estimation.

The Lead Processes (Medical Examinations) Regulations, requiring three-monthly haemoglobin estimations in lead workers, were introduced in Great Britain in 1964. The following levels of haemoglobin and lead-in-blood, indicating asymptomatic and overt lead poisoning, were given as a guide to Appointed Factory Doctors undertaking the examinations (Ministry of Labour, 1964) (Table I).

TABLE I  
NORMAL VALUES AND VALUES TO BE EXPECTED IN LEAD INTOXICATION

	Normal	Asymptomatic Poisoning	Poisoning
Haemoglobin (g./100 ml.)	14.6	12-13	< 12
% Haldane	100	82-89	< 81
Blood lead ( $\mu\text{g./100 ml.}$ )	20-40	60-80	> 80

#### Material and Methods

In an electric accumulator factory in Lancashire, employing about 1,000 workers exposed to varying degrees of lead dust and fume, blood lead and haemoglobin estimations are carried out routinely, the frequency of the test depending on the workman's exposure.

Those in more exposed jobs have blood leads and haemoglobins estimated four times a year. Those with very low exposure have haemoglobins measured once a year; and at the same time blood lead is measured on a small group of workers who serve as monitors.

Whenever blood is taken by venepuncture for lead estimation, a sample is taken from the lobe of the ear for haemoglobin measurement.

The samples for lead estimation are sent to the Occupational Hygiene Service, Manchester University, where they are analysed by a modification (to be published) of the method of King and Thompson (1961). The Hygiene Service also supplies syringes and de-aired containers.

The haemoglobins are measured by M.R.C. Grey Wedge Photometer at the factory by an experienced nurse or technician. Anyone who has a high blood lead or a low haemoglobin is examined clinically. He is told why he is being seen, and a history is taken if he has medical complaints. If he has no complaints he is asked directly if he feels tired, lacks energy, or has constipation, diarrhoea, indigestion, or abdominal discomfort. He is examined physically as indicated by the history, and possibly for Burton's blue line and wrist drop. Since neither of the latter classical signs have been seen in more than 40,000 statutory monthly examinations they are no longer looked for as a routine. The man's job is then discussed and he is advised how to reduce his lead absorption. He may be suspended temporarily or permanently from lead work.

**Blood Lead and Haemoglobin** The first blood lead estimation and its paired haemoglobin measurement were taken from the records of 656 workers tested in the first six months of 1965. No one was included more than once. A few were not tested because they were absent or refused. Thus nearly all the most exposed lead workers, most with medium exposures and a few with low exposures, were included.

**Clinical Examinations** The results of clinical examinations of men whose blood leads were 90  $\mu\text{g. lead/100 ml. blood}$  or more and/or whose haemoglobins were 89% (13 g./100 ml.) or less were taken from the records.

#### Results

**Blood Lead and Haemoglobin** Six hundred and fifty-six pairs of readings were collected, of which one pair was rejected because of the unreliability of the haemoglobin level. No pair was rejected because of unusual blood lead. One very high reading of 350  $\mu\text{g./100 ml.}$  was thought to be true because preceding and succeeding estimations were also high.

Of the 655 pairs of readings analysed, the blood lead mean was 69.3  $\mu\text{g./100 ml.}$ , range 3-350  $\mu\text{g./100 ml.}$ ; the haemoglobin mean was 104.4%, range 80-123%.

Scatter diagrams were made by plotting the co-ordinate points of each blood lead and haemoglobin pair. This showed that there was no statistical relation between the two indices. However, for blood leads above 110  $\mu\text{g./100 ml.}$  the haemoglobins were slightly below the group average. The blood leads were then grouped in 10  $\mu\text{g./100 ml.}$  intervals, and

the mean haemoglobin and its 95% confidence limits (based on Student's t-test using the pooled variance) were calculated for each group. At the upper extreme of the blood lead distribution the numbers in the groups were very small and were combined. Blood leads under 40  $\mu\text{g.}/100\text{ ml.}$  were also combined. The results (Table II; Figure) give no indication of any fall in haemoglobin until the blood lead exceeds 110  $\mu\text{g.}/100\text{ ml.}$ , and the slight fall then is not significant at the 5% level of confidence; nor is there any indication of a rise in haemoglobin at lower levels of blood lead.

TABLE II

MEAN HAEMOGLOBIN WITH 95% CONFIDENCE LIMITS IN 655 MEN GROUPED BY THE LEVEL OF BLOOD LEAD

Blood Lead ( $\mu\text{g.}/100\text{ ml.}$ )	No. of Men	Mean Hb (%)	95% Confidence Limits
0-9	1	105.2	102.9-107.4
10-	2		
20-	16		
30-	28		
40-	82	104.7	103.0-106.4
50-	132	104.3	102.9-105.6
60-	115	104.6	103.2-106.1
70-	112	104.1	102.7-105.6
80-	64	105.3	103.4-107.3
90-	38	104.6	102.1-107.2
100-	25	104.7	101.5-107.9
110-	9	100.9	94.9-106.8
120-	8	103.1	97.1-109.2
130-	2	101.7	98.4-105.0
140-	8		
150-	3		
160-	3		
170-	4		
180-189	2		
350	1		

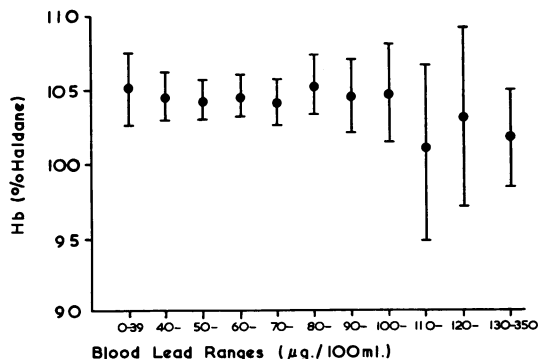


FIGURE. Mean haemoglobin and 95% confidence limits in various blood lead ranges.

Clinical Examinations

**High Blood Leads** One hundred and three (15.7%) men had blood leads of 90  $\mu\text{g.}/100\text{ ml.}$  or more; 67 of these were examined by the author. Nine had positive findings as indicated by symptoms, signs, or a low haemoglobin (Table III). One man with a blood lead of 110  $\mu\text{g.}/100\text{ ml.}$ , who had no symptoms but a low haemoglobin (85%), was suspended from lead work because it was thought that his low haemoglobin was due to lead. Of the remaining eight who had symptoms, one had headaches following a severe head injury. Relevant extracts from the medical records of the remaining seven men who had clinical findings possibly due to lead are shown in Appendix 1. It will be seen that four men (cases 4, 5, 6, and 7) had epigastric pain very suggestive of peptic ulceration (or

TABLE III

POSITIVE CLINICAL FINDINGS IN 67 MEN WITH BLOOD LEADS (BL) OF 90  $\mu\text{G.}/100\text{ ML.}$  OR MORE

Probably due to Lead	Possibly due to Lead	Not due to Lead
1. BL 110; Hb 85% No symptoms	1. BL 147; Hb 98% Slight indigestion and tiredness on 6-2 shift only	1. BL 99; Hb 98% Headaches from head injury
	2. BL 158; Hb 102% Tired. Well 2 weeks later when BL 117	
	3. BL 120; Hb 104% Slight tiredness (BL probably contaminated because in 2 weeks BL 54)	
	4. BL 170; Hb 93% Epigastric pain $\Delta$ Peptic ulcer	
	5. BL 160; Hb 94% Occasional epigastric pain $\Delta$ Peptic ulcer and anxiety neurosis	
	6. BL 157; Hb 99% Epigastric pain after eating onions $\Delta$ ? Peptic ulcer	
	7. BL 116; Hb 95% Epigastric pain $\Delta$ ? Peptic ulcer	

gastritis) rather than of lead colic. This is 6.0% of the original 67, which is similar to the incidence of 5.8% of peptic ulcer in London males estimated by Doll and Avery Jones (1951).

It is clear therefore that among the 67 men with blood leads of 90  $\mu\text{g./100 ml.}$  or more, 59 had no clinical evidence of excessive lead absorption, seven possibly had such evidence, and only one definitely had clinical evidence of excessive absorption.

**Low Haemoglobins** Of the 655 readings of haemoglobin, 19 (3%) were 89% (13 g./100 ml.) or less. Eight of these men were examined clinically but none had symptoms.

One man with a haemoglobin of 85% and a blood lead of 110  $\mu\text{g./100 ml.}$  is included in Table III. He was thought to have a low haemoglobin due to lead and was suspended.

In the other seven men seen clinically, the low haemoglobins were regarded as being not due to lead for the following reasons:

In two the blood leads were low (48 and 79  $\mu\text{g./100 ml.}$ ).

In five the blood leads were less than 90  $\mu\text{g./100 ml.}$ , except in one man, and subsequent haemoglobins were all above 89%.

Of the 11 men who were not examined clinically, one man was absent from work because of a painful shoulder, and the remaining 10 all had blood leads of less than 90  $\mu\text{g./100 ml.}$

Thus, of the 655 men tested, probably only one had a low haemoglobin due to lead.

### Discussion

It is important to consider possible reasons for the failure to find lowered haemoglobins and symptoms of excessive lead absorption in men with high blood leads.

**Errors in Haemoglobin and Blood Lead Measurement** It is possible that the absence in the present data of a fall in haemoglobin with blood leads of less than 110  $\mu\text{g./100 ml.}$  could be due to errors in measuring the haemoglobin or the blood lead. Errors in haemoglobin estimation could be responsible only if low haemoglobins were read high, while normal and high haemoglobins were read correctly. This possibility seems extremely unlikely. Errors in blood lead estimation could be responsible only if the higher blood leads obtained were false and if truly high blood leads were either not occurring in the factory or were being misread to such an extent that they were randomly distributed throughout all levels in the series. This also seems extremely unlikely.

**Individual Susceptibility** Not all individual workers have an equal susceptibility to lead (Legge and Goadby, 1912; Lane, 1949). There is variation in the quantity of lead absorbed, due to differences in several factors such as cleanliness, respiratory tract shape, and minute volume, and there is variation in the metabolic effect of the lead absorbed due to racial, familial, dietary, and other factors (Legge and Goadby, 1912). Agreement differs on the degree of the susceptibility and on the frequency of its distribution throughout the population (Kehoe, 1963).

The present findings indicate that susceptibility to lead, which produces either a fall in haemoglobin or symptoms or signs of lead poisoning, is either so infrequent that it has not occurred to a noticeable extent in any individual with a blood lead less than 110  $\mu\text{g./100 ml.}$  or that susceptibility is of such a degree that it is only apparent with blood leads higher than 110  $\mu\text{g./100 ml.}$

**Blood Lead Correction for Low Haemoglobin** The greater part of the total lead in the blood is in the red cells (Cantarow and Trumper, 1944). Thus if the total red cell volume is for any reason low, the observed total lead-in-blood would be lower than expected, assuming that there is no qualitative change in the red cell affecting its affinity for lead.

A correction factor has been deduced (see Appendix 2), which is approximately equal to the reciprocal of the haemoglobin, by which the observed blood lead might be multiplied to get a corrected figure (Table IV).

TABLE IV  
EXAMPLES OF A CORRECTION FACTOR (CF) FOR VARIOUS VALUES OF HAEMOGLOBIN

Hb	CF	$\frac{100}{CF}$
110	0.924	108
100	1.000	100
95	1.041	96
90	1.089	92
85	1.137	88
80	1.195	84
75	1.252	80
70	1.318	76

Note that  $\frac{100}{CF}$  approximately equals the haemoglobin.

This correction factor will be important in standardizing toxic threshold levels of blood lead derived from observations on workers with low haemoglobin. For example, applying the correction factor to the man in the present series with a low haemoglobin (85%) probably due to excessive lead

absorption (blood lead 110  $\mu\text{g.}/100\text{ ml.}$ ) (Table III),  
corrected blood lead =  $110 \times 1.137$   
= 125  $\mu\text{g.}/100\text{ ml.}$

If this correction factor was applied to the present data, the level of blood lead at which the haemoglobin begins to fall would be little affected; but the rate of fall of the haemoglobin with further increasing blood lead would be reduced.

**Conditions of Exposure** Harashima's (1958) experiments with dogs indicate that lead absorbed primarily on the surface of the red cell may gradually move into the internal fluid to be retained there in a non-ionic state. Also Haeger-Aronsen (1960) suggests that the red cell lead concentration may depend partly on the concentration in the marrow when the cell was formed. Thus lead workers suddenly exposed to very high levels of lead exposure may suffer lead poisoning before the blood lead has reached a steady level. The blood lead will then appear optimistically low. Thus the use of frequent routine haemoglobin estimations might well be justified for lead workers in unstable conditions, for example, for men occasionally involved in burning through lead paint, as in ship breaking or dismantling heavily leaded machinery.

### Conclusion

It seems that in a population where sophisticated lead control is practised no purpose is served by estimating haemoglobins in all lead process workers as frequently as once every three months, but only in those whose lead-in-blood is likely to be in excess of 110  $\mu\text{g.}/100\text{ ml.}$

It may also be of value in the first year of exposure to detect susceptible workers, or in a population of a different race, nutritional status, or sex.

I would like to thank Professor R. S. F. Schilling and members of the Department of Occupational Health and Applied Physiology at the London School of Hygiene and Tropical Medicine, and Professor R. E. Lane for advice in preparing this paper. I am indebted to Miss Joan Walford for statistical help; to Mr. E. King at the University of Manchester for the lead-in-blood estimations; to Dr. D. Malcolm and the Directors of Electric Power Storage Ltd. for encouragement and permission to publish this paper; and to Sister E. O. Jones, Sister N. Bush, and Mrs. Barbara Roughley for technical assistance.

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### Appendix I

**Case Notes** The abbreviations used are: BL = blood lead ( $\mu\text{g.}/100\text{ ml.}$ ); UL = urinary lead ( $\mu\text{g.}/\text{litre}$ ); SG = specific gravity of the urinary lead sample; UCP = urinary coproporphyrin, measured on the Donath (1956) semi-quantitative scale 1-8; PBC = punctate basophil count (punctate cells/million red cells) [Dark ground, binocular microscopy, 10,000 punctate cells/million red cells is considered safe upper limit].

- Case 1** Age 35, Forming Department since 1959  
12.10.64 PBC 1,900, Hb 102%  
4.2.65 BL 147, Hb 98%  
22.2.65 Examined re high BL. Complains of slight indigestion and tiredness on 6.00 a.m.-2.00 p.m. shift but not on the other shifts. Diagnosis—almost certainly not due to lead, but stop lead burning for two months  
6.4.65 BL 74, Hb 108%  
**Case 2** Age 37, Pasting Department since 1962  
18.9.64 PBC 3,100, Hb 108%  
21.12.64 Absent sick. 'Nervous debility' (mother died)  
2.1.65 BL 158, Hb 102%  
26.1.65 Examined re high BL ? tired for some time  
Repeat BL 117, Hb 113%, UCP 2  
8.2.65 Well except appetite poor one week; repeat BL in one month  
8.3.65 BL 78  
**Case 3** Age 43, Lead Smelting Furnace since 1944  
6.12.64 BL 69  
3.3.65 BL 120, Hb 104%  
15.3.65 Examined re high BL; well except for slight tiredness; repeat BL  
19.3.65 BL 54  
**Case 4** Age 43, Plate Cleaning Department since 1957  
12.6.63 UL 289, SG 1.022, UCP 2, PBC 2,600, Hb 98%

- 12.7.63 Complains of epigastric pains twice daily for three months, especially several hours after meals but not relieved by food (?). Appetite good. On examination tender in epigastrium. Wt. 10 st. 3 lb. (65 kg.). Diagnosis—peptic ulcer. Treatment—extra food breaks and refer to general practitioner. Repeat UL
- 23.11.64 PBC 1,700, Hb 103%
- 19.1.65 BL 170, Hb 93%
- 1.2.65 UCP 4
- 8.2.65 Examined re high BL. Complains of recurrence of peptic ulcer pain (above). Treatment—extra food breaks and refer to general practitioner. Discuss with foreman time spent on breakpart machine. Repeat BL
- 22.2.65 BL 82, Hb 108%
- Case 5* Age 41, Pasting Department since 1957
- 18.11.59 Abdominal pain relieved by food. On examination epigastric tenderness. Under treatment by general practitioner
- 5.2.60 Duodenal ulcer and domestic troubles
- 9.4.62 Recurrence of abdominal pain. Refer to general practitioner
- 11.4.62 Domestic troubles ++. Attending general practitioner
- 2.9.63- Hospital investigation for 'renal colic'
- 2.10.63
- 15.10.63 He says 'nothing was found except a duodenal ulcer'
- 27.5.64 Request from general practitioner (letter) that he continues day work because of peptic ulcer
- 21.1.65 BL 160, Hb 94%
- 29.1.65 Examined re high BL. Feels better than he has for years. Only occasional epigastric pain
- 1.2.65 BL 117, Hb 90%, UCP 4
- Case 6* Age 27, Pasting Department since February 1964
- 9.9.64 PBC 0, Hb 98%
- 12.1.65 BL 157, Hb 99%
- 25.1.65 Examined re high BL. Feels very well except complains of epigastric pain after eating onions. On examination no abdominal tenderness. Diagnosis—? peptic ulcer
- 8.2.65 BL 58, Hb 108%
- Case 7* Age 33, Forming Department since February 1964
- 16.4.64 PBC 2,500, Hb 90%
- 24.4.64 Examined re low Hb. Complains of irregular pains lower chest bilaterally. On examination of respiratory system and gastro-intestinal system nothing abnormal detected. UL 95, SG 1.020, UCP 3, Hb 108%
- 16.9.64 Complains of recurrent retrosternal and sub-costal pains lasting a few hours, relieved by lying down and sleeping, not related to food, exercise, or alkalis, but appetite reduced during pain. No nausea. Bowels open regularly. *Had pain slightly before present employment.* Hb 109%. Diagnosis—? peptic ulcer. Refer to general practitioner
- 18.9.64 UL 161, SG 1.016, UCP 2, PBC 5,900, Hb 109%
- 23.9.64 Pain better
- 2.10.64 PBC 5,800, Hb 110%
- 20.10.64 UL 116, SG 1.020, UCP 5
- 2.2.65 BL 116, Hb 93%
- 17.2.65 Examined re high BL. Well, except still getting pains (above). Says radiograph was 'clear'. Refer to general practitioner again. Advised re working methods.
- 29.3.65 BL 72, Hb 105%

### Appendix 2

**Deduction of Blood Lead Correction Factor for Low Haemoglobin** Assume that (1) the total red cell volume equals the haematocrit ( $h$ ); (2) the concentration of cell lead is 10 times that of plasma lead (Cantarow and Trumper, 1944); and (3) the ratio in (2) above is approximately independent of  $h$ : for  $h$  equals the mean corpuscular volume (M.C.V.) multiplied by the red blood count (R.B.C.); in lead anaemia the M.C.V. is normal (Fullerton, 1952); and since in the steady state the concentrations of lead in the cells and in the plasma are in equilibrium with each other it is probable that the concentration of lead in a given cell will not vary with the proximity of an adjacent cell (R.B.C.).

Consider 100 ml. of blood, centrifuged and having haematocrit  $h$ . The volume distribution between cells and plasma is

$h$	$100-h$
cells	plasma

Let  $x$  = concentration of lead in plasma ( $\mu\text{g. lead in plasma/ml. plasma}$ )

$y$  = concentration of lead in cells ( $\mu\text{g. lead in cells/ml. cells}$ )

$\text{BL}_{\text{obs}}$  = observed total blood lead

$\text{BL}_{\text{exp}}$  = expected total blood lead

Then  $\text{BL}_{\text{obs}} = yh + x(100-h)$

and if the normal haematocrit =  $h_n$

$\text{BL}_{\text{exp}} = yh_n + x(100-h_n)$ .

Assuming  $y = 10x$  (from (2) above)

$\text{BL}_{\text{obs}} = x(9h + 100)$

$\text{BL}_{\text{exp}} = x(9h_n + 100)$

Then  $\frac{\text{BL}_{\text{exp}}}{\text{BL}_{\text{obs}}} = \frac{9h_n + 100}{9h + 100}$

Now  $h = \frac{\text{Hb} \times 100}{\text{M.C.H.C.}}$

where Hb = haemoglobin in g./100 ml. blood

M.C.H.C. = mean corpuscular haemoglobin concentration

In normals, haematocrit = 46% } (Documenta  
M.C.H.C. = 34.1% } Geigy)

Also in eight cases of lead anaemia (Fullerton, 1952) the mean M.C.H.C. was normal.

Thus, substituting

$$\frac{BL_{exp}}{BL_{obs}} = \frac{(9)(46) + 100}{(9)(Hb)(100) + 100} \quad (34.1)$$

which 
$$= \frac{1}{0.00805 Hb + 0.195} \quad \text{for Hb in \% Haldane}$$

This is the correction factor (CF) by which the observed blood lead must be multiplied to get the expected (or corrected) blood lead. Examples of the correction factor for various values of haemoglobin are shown in Table IV, which also shows the reciprocal of the correction factor, expressed as a percentage. The latter can be seen to be very approximately equal to the haemoglobin. Thus for clinical work sufficient accuracy would be obtained if the observed blood lead was corrected by dividing it by the haemoglobin expressed as a percentage.