an epileptic seizure, one drowning, and one suicidal death from gunshot wounds. One death resulted from carcinoma of the bladder.

Infections were responsible for eight deaths: pneumonia accounted for four, and systemic infections for the remaining four.

Conclusions

Above all else, these 135 deaths of diabetic children within the last six years—11.2% from tuberculosis, 8.8% from diabetic coma, and 51.9% from renal disease—show that the diabetic with onset of disease in childhood should be followed more closely. Only in this way can needless deaths be avoided. Continuity of treatment is essential. Roentgenograms of the chest should be taken yearly. A constant awareness for evidences or incipient signs of renal disease must be maintained. Research should be concentrated on the kidney in the young diabetic. The importance of post-mortem examinations cannot be too strongly emphasized.

To preserve in comfort and prolong the lives of diabetic children the need is paramount for inexpensive and attractive opportunities, in children's and adolescents' camps, and especially in hospitals, of reviewing their physical condition and promoting morale and zeal for the continuing control of their diabetes.

ERYTHROCYTE SEDIMENTATION IN ANAEMIA

BY

RICHARD TERRY, M.D., M.R.C.P.

Senior Registrar, St. Bartholomew's Hospital

In 1921 Robin Fahraeus introduced his method of measuring the erythrocyte sedimentation rate (E.S.R.) and, after describing the various disorders in which it was increased, discussed the factors involved. Most of his findings are still widely accepted, especially the view that anaemia had an accelerating effect on blood sedimentation. Others studied this problem and also became convinced that anaemia, *per se*, increased the E.S.R. (De Courcy, 1925; Meeker, 1925; Rubin, 1926; Hubbard and Geiger, 1928; Cherry, 1934). It was soon suggested that the E.S.R. was so dependent on the red cell count that it was wholly unreliable in anaemia (Hunt, 1929) and that small increases in the E.S.R. should be discounted in the presence of anaemia (Bannick *et al.*, 1937). With few exceptions further studies have been in agreement on the effect of anaemia and on the need for correction of the increased rate, differing only over the best method of making this correction.

The object of this paper is to give certain theoretical, clinical, and experimental reasons for believing that the E.S.R. is not increased by anaemia, and therefore that the observed E.S.R. has the same significance in anaemia as in normal blood states.

Existing Correction Methods

Some recommend estimation of the E.S.R. in anaemia only after the packed cell volume (P.C.V.) has been adjusted to normal by manipulation of the blood sample (Hubbard and Geiger, 1928; Chung, 1935; Gibson, 1938), others after removal of enough plasma to raise the red cell count to 5 millions per c.mm. (Walton, 1933; Schuster, 1938). Charts have been devised to correct for various haemoglobin levels (Gram, 1929) or haematocrit readings (Ernsterne and Rourke, 1930). The latter method was adopted by Wintrobe and Landsberg (1935), who constructed a chart based on numerous readings on normal blood samples made "anaemic" by dilution with their own plasma. Certain impossible situations in this chart were pointed out by Hynes and Whitby (1938), whose modification is based on the same data. Other methods include simple subtraction based on the haemoglobin level (Gregg, 1939) and a proportional correction depending on the P.C.V. (Della Vida, 1947).

Contrary views have been expressed, but they have been weakened by inadequate evidence (Bouton, 1938; Vogt, 1941) or change of opinion (Gregg, 1937, 1939). Britton (1936) concluded that an increased E.S.R. in anaemia indicated a search for some accompanying disorder, but not only did he allow an upper limit of 15 mm. an hour (Westergren) for women, accounting for this high figure by their lower average red cell count, but he also lent support to a correction chart (Whitby and Britton, 1946). A number of cases of anaemia with normal E.S.R.s were reported by Sugarman (1939), but he gave few details and made no comment on correction. Without referring to clinical results, and believing that the red cells played an entirely passive part in sedimentation, Cutler *et al.* (1938) opposed correction.

This opposition has been too indecisive, and the field is now firmly held by the supporters of correction. The present position remains as described in the exhaustive review of blood sedimentation by Ham and Curtis (1938), who concluded: "It has been clearly established that decrease in erythrocyte concentration causes acceleration of the sedimentation rate."

Fallacies of Correction

Anaemia is not a simple entity. Alterations occur in the red cells affecting their number, mass, size, shape, uniformity, and haemoglobin content, and these alterations vary with the cause of the anaemia, as do changes in the plasma proteins. It follows that a single correction factor will not be suitable for all forms of anaemia, and that consideration of one aspect of the anaemia will lead to other aspects being ignored or distorted. Thus adjustment of the haemoglobin to 100% in an anaemic sample may well increase the red cell count to 10 millions per c.mm. and the P.C.V. to 60%, while manipulating the red cell count of the same specimen to 5 millions per c.mm. may leave the haemoglobin at 40% and the P.C.V. at 30%; similarly, adjusting the P.C.V. to normal will not produce normality in other respects.

These methods of "correcting" the blood sample before estimating the E.S.R. also ignore other changes in the red cells. Cutler *et al.* (1938) maintained that the red cells played an entirely passive part in sedimentation. It seems certain that this view is incorrect. Ham and Curtis (1938), by suspending washed red cells of varying size in the same plasma, showed that large cells sediment faster than small cells. The experiments of Bunting (1939) with cases of sickle-cell anaemia are convincing. He found that their E.S.R. was greatly increased after reducing the sickling by bubbling oxygen through the blood, whereas when about 50% sickling was produced by using carbon dioxide, rouleaux formation and sedimentation were almost abolished.

Plasma protein changes affecting the absolute and relative amounts of albumin/globulin fractions and fibrinogen vary with the cause of the anaemia, but they are not considered in any correction method.

That anaemia is not a simple entity therefore casts doubt on the whole system of correction.

Further doubt arises when one examines the construction of the most widely used correction chart, that of Wintrobe and Landsberg (1935). They diluted specimens of normal blood to produce various degrees of "anaemia." The E.S.R. of each "anaemic" sample was then estimated and the chart constructed from the results. Their argument was simply that if a blood sample of normal P.C.V. with an E.S.R. of 8 mm. an hour was diluted to a P.C.V. of 25% and the E.S.R. thereby increased to 42 mm. an hour, then an E.S.R. of 42 mm. an hour in an anaemic patient whose blood had a P.C.V. of 25% could be regarded as normal. It is apparent that the "anaemic" blood samples on which the chart was based consisted in fact of normal plasma and cells unaltered in any way except concentration. It is therefore difficult to see how this chart is applicable to anaemia.

Thus there seem to be reasonable grounds for doubting the validity of existing correction methods and also for believing that, even if correction were necessary, the complexity of the anaemias would make it impossible. The necessity for correction must now be examined.

Correction methods rest on the assumption that anaemia increases sedimentation. The grounds for this assumption appear to be flimsy. Fahraeus (1921) introduced the concept of anaemia increasing the E.S.R. at the same time that he published his method of measuring sedimentation, and it seems that the great value of the test and the excellence of his report have conferred immunity on his views on the effect of anaemia. He had been impressed by the frequent association of anaemia and increased sedimentation and, not perceiving that both had a common origin, concluded that the increase was due to the anaemia. The absence of sedimentation in polycythaemia seemed to confirm this view. When he found that he could increase the E.S.R. of normal blood by diluting the sample with its own plasma, he regarded the accelerating effect of anaemia as proved. Thus was pseudo-anaemia originated, and others, repeating the same error, have perpetuated the belief that anaemia increases sedimentation.

Evidence against any accelerating effect of anaemia will now be described. Unless otherwise stated, all E.S.R.s mentioned will have been estimated by the Westergren technique, using 0.4 ml. of 3.8% sodium citrate to 1.6 ml. of blood in a 200-mm. tube. The advantages of the Wintrobe technique are appreciated, but the Westergren method is still the most widely used. For similar reasons haemoglobin levels are given as % Haldane.

Range of Normal E.S.R.

There is considerable variation in the figures given for the normal E.S.R., but an upper limit of 10 mm. an hour agrees with most accepted figures. Fig. 1 shows the percentage distribution of E.S.R.s of 10 mm. per hour or less among 367 healthy women and 228 healthy men encountered during the present investigation, both groups having haemoglobin values above 86%.

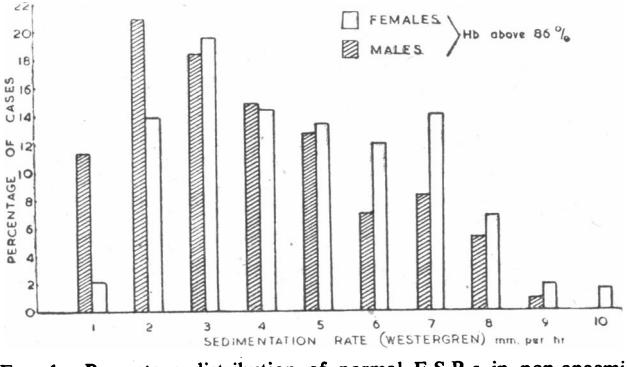

FIG. 1.—Percentage distribution of normal E.S.R.s in non-anaemic men and women.

The mean E.S.R. for the women is 4.72 mm. an hour (twice standard error = ± 0.22), with a modal E.S.R. of 3 mm. an hour; while for the men the mean E.S.R. is 3.82 mm. an hour (twice standard error = ±0.26) with a modal E.S.R. of 2 mm. an hour. Thus there is a highly significant difference.

Range of E.S.R. in Simple Hypochromic Anaemia

The percentage distribution of E.S.R.s between 1 and 10 mm an hour found in 367 women with Hb values above 86% and in 160 women with Hb levels of 86% and less is shown in Fig 2

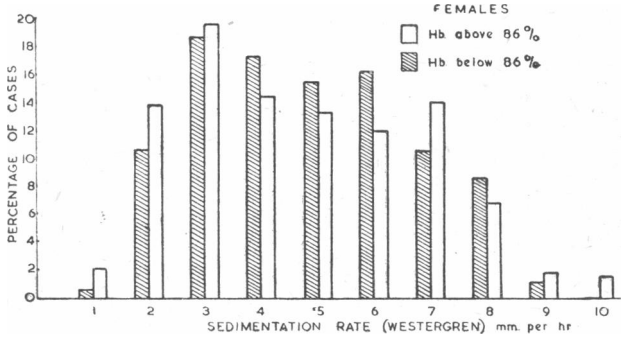

FIG. 2.—Percentage distribution of normal E.S.R.s in anaemic and non-anaemic women.

Of the 160 cases, the Hb levels were between 86% and 77% inclusive in 86, between 76% and 67% inclusive in 27, and 66% or less in 47. The anaemia was of a benign origin in every instance. The mean E.S.R. of the non-anaemic group is 4.72 mm. an hour (twice standard error = ±0.22) and of the anaemic group 4.79 mm. an hour (twice standard error = ±0.30). The modal E.S.R. is 3 mm. an hour in both groups.

The absence of any significant difference is considered to be evidence against anaemia having any accelerating effect on the E.S.R.

A further conclusion suggested by these two distribution charts is that the higher E.S.R. of women is due to factors other than their lower average red cell count.

A similar comparison between anaemic and non-anaemic groups of males is not presented, simply because insufficient cases are available, anaemia of benign origin being so much less common in men.

Normal Sedimentation in Anaemia

Whatever theoretical grounds exist for thinking that anaemia has no accelerating effect on sedimentation, it is essential to determine whether normal E.S.R.s do in fact occur in significant anaemia. During this study 60 instances have been encountered and are detailed in Tables I and II. Table I shows the full blood counts of 30 patients with haemoglobin levels below 60% and E.S.R.s of 9 mm. an hour or less. Table II shows a similar group in which the indices were not estimated. All the cases were women except Nos. 58, 59, and 60. Without exception the anaemias were secondary to simple blood loss, usually menstrual and never neoplastic.

Clearly hypochromic anaemia does not inevitably increase sedimentation.

TABLE I.—*Full Blood Counts of Patients with Haemoglobin Levels Below 60% and E.S.R. of 9 mm./hr. or Less*

Case No.	E.S.R.	Hb%	R.B.C.	P.C.V.	M.C.H.C.	M.C.V.
1	8	21	3·2	18	16	56
2	2	24	2·6	17	20	65
3	5	36	3·2	22	16	69
4	6	36	3·6	25	20	69
5	3	38	5·3	28	19	53
6	5	38	3·0	25	21	83
7	6	38	4·2	26	20	62
8	8	40	4·8	29	19	61
9	2	44	4·3	30	21	68
10	2	44	4·1	31	20	76
11	3	44	4·3	31	20	74
12	5	44	4·1	26	24	64
13	7	44	4·3	28	22	64
14	9	44	4·9	27	22	55
15	1	46	3·9	28	22	71
16	2	46	4·7	33	19	72
17	3	46	5·0	30	21	60
18	5	46	3·7	32	20	86
19	5	46	5·1	34	19	68
20	3	48	3·7	27	24	72
21	6	48	5·0	32	20	64
22	3	50	3·8	34	20	90
23	4	50	3·5	29	23	82
24	7	50	3·3	32	21	98
25	2	54	4·3	31	24	72
26	4	54	3·7	29	26	78
27	2	56	3·1	27	29	87
28	4	56	5·0	41	19	80
29	8	56	4·7	35	22	74
30	3	58	4·3	35	23	80

TABLE II.—*Group of Patients Similar to Table I, But in Which the Indices Were Not Estimated*

Patient	E.S.R.	Hb%	R.B.C.	Patient	E.S.R.	Hb%	R.B.C.
31	3	42	5·1	46	6	58	—
32	6	44	2·3	47	8	58	5·2
33	8	44	—	48	9	58	—
34	2	46	3·4	49	4	60	5·1
35	5	46	3·4	50	5	60	4·3
36	6	48	2·5	51	5	60	4·9
37	7	48	4·2	52	5	60	4·5
38	6	50	4·9	53	6	60	4·9
39	8	50	4·7	54	7	60	4·8
40	6	52	—	55	2	64	—
41	3	54	4·8	56	3	64	—
42	7	54	3·8	57	3	64	—
43	6	56	—	58	5	38	—
44	7	56	4·5	59	6	39	—
45	5	58	—	60	1	50	—

Slightly Increased Sedimentation in Anaemia

If these arguments are correct, slight increases in the E.S.R. should be significant even when there is marked anaemia. Table III is presented in support of this view.

TABLE III.—*Slightly Abnormal E.S.R.s in Anaemia*

Case No.	E.S.R.	Hb%	R.B.C. (mills.)	P.C.V. (%)	M.C.H.C. (%)	M.C.V. (μ^3)	Diagnosis
61	15	23	3·5	20	16	56	Atrophic gastritis
62	12	26	3·2	20	18	62	Bronchitis. T. 99·8° F. (37·7° C.)
63	12	32	3·9	26	16	67	Dental sepsis. T. 99° F. (37·2° C.)
64	9	36	4·5	27	19	60	Ulcerative colitis
65	18	38	3·4	25	21	74	Carcinoma of colon (early)
66	15	40	3·9	24	23	62	Leucocytosis (? cause). 17,500
67	15	44	4·1	30	20	73	Bilateral phthisis
68	11	46	4·7	29	21	61	Chronic phthisis. T. 99·6° F. (37·55° C.)
69	11	48	3·5	26	24	74	Melaena. W.B.C. 13,000
70	19	48	3·5	—	—	—	Thrombophlebitis migrans
71	11	50	3·2	26	26	81	Cirrhosis hepatis
72	12	50	4·1	28	24	71	Pregnancy (5th month)
73	12	50	3·8	30	23	76	Post-gastrectomy syndrome
74	13	50	4·2	31	22	73	T. 99° F.(? cause)
75	12	54	4·8	35	21	73	Recent infective hepatitis
76	14	56	5·0	34	22	62	Plummer–Vinson syndrome
77	15	60	3·8	31	27	80	Pregnancy (3rd month)
78	18	60	5·0	36	23	72	*Bact. coli* pyelitis
79	16	62	3·8	33	26	85	Subacute appendicitis
80	15	64	4·5	—	—	—	Advanced phthisis

In each case an abnormality usually associated with an increased E.S.R. was present, though the cause was sometimes obscure. It is emphasized that during this investigation no case of hypochromic anaemia with raised E.S.R. has been seen in which there was not good cause for that increase.

Grossly Increased Sedimentation in Anaemia

When there is gross acceleration of the E.S.R., anaemia with its reduced P.C.V. will delay the packing phase of sedimentation and so permit a greater fall over a period of time than if the P.C.V. were normal. The longer the packing phase is delayed the more accurate the reading with the Westergren and Wintrobe methods of estimating the fall in a specific time. But when the E.S.R. is assessed by the time required for a fall of specific length (Linzenmeier, 1920), or by the rate during the phase of constant sedimentation (Cutler, 1932), the effect of the packing phase is specifically eliminated. Thus there seems to be no indication for correction of high E.S.R.s in anaemia.

Antisedimenting Effect of Anaemia

Among the cases shown in Table III were many in which the E.S.R.s were less than might have been anticipated from the severity of the associated disorder, especially Cases 68 and 80, both of which had extensive bilateral phthisis, and Case 79, in which ulcerative colitis, though healing, was still causing four to six loose stools a day.

Further support for this antisedimenting effect is given by the behaviour of the E.S.R. in Cases 61, 63, 69, and 76. In these patients as the anaemia responded to treatment the E.S.R. increased, without clinical evidence of increased activity of the disorder responsible for the abnormal sedimentation (see Table IV).

TABLE IV.—*Behaviour of E.S.R. During Treatment of Anaemia*

Case No.	Before Treatment		During Treatment	
	E.S.R.	Hb%	E.S.R.	Hb%
61	15	23	19	44
63	12	32	25	78
69	11	48	35	70
76	14	56	18	68

Consideration of the effect of the sodium citrate content of the Westergren blood sample also suggests that the suspension stability of the red cells in anaemia is increased. Ham and Curtis (1938) demonstrated in experiments on haemophiliac blood that sodium citrate has a marked retarding effect on sedimentation. Now, the addition of one part of 3.8% sodium citrate to four parts of normal blood (P.C.V. = 45%, say) in the Westergren technique results in a concentration of 1.2% sodium citrate in the plasma. In the case of anaemic blood (P.C.V. = 25%, say) there is a final concentration of 0.95% in the plasma of the Westergren specimen. Thus when a normal E.S.R. is present in anaemia (see Tables III and IV) it occurs in spite of a lower sodium citrate concentration than in normal specimens and therefore in spite of a smaller citrate retarding effect.

During this study observations have been made on the effect of diluting Westergren blood samples with their own citrated plasma. The retarding effect of sodium citrate was best seen in dilution experiments on polycythaemic blood (P.C.V. = 75%, say), in which the sodium citrate concentration in the Westergren specimen is 1.9% and the E.S.R. is normally zero. In one representative dilution experiment on polycythaemic blood, 75% dilution of the sample with the citrated plasma resulted in an increase of the E.S.R. to 3 mm. in one hour and 11 mm. in two hours, compared with 40 mm. in one hour and 95 mm. in two hours when the same dilution experiment was carried out with a Wintrobe sample. Wintrobe's dry oxalate mixture produces no alteration in sedimentation rate (Wintrobe and Landsberg, 1935; Ham and Curtis, 1938).

In every dilution experiment in this study on normal blood, dilution has resulted in marked acceleration of sedimentation, confirming the findings of Fahraeus (1921) and many others. However, in every experiment on anaemic blood similar dilution has resulted in less acceleration of the E.S.R. than in the normal specimens (comparing samples of identical E.S.R. before dilution). Now in these anaemic samples there is a smaller concentration of red cells and dilution is carried out with plasma containing a lower concentration of sodium citrate (see above). For these two reasons one would expect that dilution of anaemic samples would show a greater acceleration of sedimentation than dilution of non-anaemic samples. That there is in fact less acceleration is felt to be further evidence of the increased suspension stability of the red cells in anaemia.

Table V shows the increases in sedimentation with various dilutions of blood samples with haemoglobin levels of 136%, 100%, and 58%. These results are representative of repeated similar experiments.

TABLE V.—*Effect of Dilution on Westergren E.S.R.*

Hb%	E.S.R.	Dilution			
		Nil	25%	50%	75%
136	In one hour	0	0	2	3
100		4	9	15	20
58		4	7	10	15
136	In two hours	0	1	3	5
100		10	23	40	55
58		12	20	30	40

Discussion

With a wealth of authoritative opinion in support, correction of the E.S.R. is now generally believed to be necessary and is widely practised. When such corrections are made, minor abnormalities of the E.S.R. are converted to normal and any possible significance is lost. When the E.S.R. is raised in the presence of anaemia the elevation is usually disregarded because "the E.S.R. goes up in anaemia." When a patient is found to be anaemic the E.S.R. is often not estimated, since "it will be up anyway."

On the contrary, this study suggests that the E.S.R. in hypochromic anaemia of simple origin has the same limits of normal as in non-anaemic blood and that minor increases in the E.S.R. in anaemia are of at least the same significance as similar increases in normal blood. The E.S.R. has its limitations like any other clinical test, but anaemia is not one of them.

It is, however, a little unexpected to find normal sedimentation rates associated with marked anaemia, since dilution of normal blood undoubtedly increases the rate. It has been pointed out that the red cells are not entirely passive in the processes of sedimentation. It may be that the small abnormal cells of hypochromic anaemia resist rouleaux formation, or that the plasma of anaemia is deficient in rouleaux-forming properties.

Summary and Conclusion

Reasons are given for doubting the current view that the E.S.R. in anaemia requires correction.

The impossibility of correction and the fallacies of correction methods are discussed.

The distribution of E.S.R.s in 367 normal women was found to be identical with that in 160 women suffering from hypochromic anaemia of benign origin. The modal E.S.R. in both groups was 3 mm. an hour.

The modal E.S.R. in 273 normal men was found to be 2 mm. an hour. This finding is thought to indicate that the higher average E.S.R. of women is due to some factor other than their lower average red cell count.

In 60 patients with haemoglobin values between 21% and 60%, E.S.R.s ranged between 1 and 9 mm. an hour. It is therefore evident that anaemia does not increase the E.S.R.

In 20 patients with haemoglobin levels between 23% and 64%, the E.S.R.s were between 9 and 19 mm. an hour. Adequate reasons for the raised E.S.R.s were present in all, indicating the significance of small increases in sedimentation in anaemia and emphasizing the dangers of correction.

Evidence is outlined for believing that the suspension stability of red cells is increased in anaemia.

These findings refer to the Westergren E.S.R. Similar but insufficient observations suggest that they also apply to the Wintrobe E.S.R.

Hypochromic anaemia neither invalidates the Westergren E.S.R. nor necessitates correction.

It is with great pleasure that I record my gratitude to Dr. R. Bodley Scott for his advice and encouragement, to Dr. Geoffrey Bourne and others for access to their clinical material, to Dr. H. F. Brewer for facilities in the Department of Clinical Pathology, to Mr. M. P. Curwen, of the Statistics Department, and to Mr. Norman K. Harrison, of the Photographic Department, St. Bartholomew's Hospital.

REFERENCES

Bannick, E. G., Gregg, R. O., and Guernsey, C. M. (1937). *J. Amer. med. Ass.*, **109**, 1257.
Bouton, S. M. (1938). *J. Lab. clin. Med.*, **23**, 519.
Britton, C. J. C. (1936). *N.Z. med. J.*, **35**, 310.
Bunting, H. (1939). *Amer. J. med. Sci.*, **198**, 191.
Cherry, T. H. (1934). *J. Lab. clin. Med.*, **20**, 257.
Chung, H. (1935). Ibid., **20**, 633.
Cutler, J. W. (1932). *Amer. J. med. Sci.*, **183**, 643.
—— Park, F. R., and Herr, B. S. (1938). Ibid., **195**, 734.
De Courcy, J. L. (1925). *Amer. J. Surg.*, **39**, 129.
Della Vida, B. L. (1947). In Dyke's *Recent Advances in Clinical Pathology*. Churchill, London.
Ernsterne, A. C., and Rourke, M. D. (1930). *J. clin. Invest.*, **8**, 545.
Fahraeus, R. (1921). *Acta med. scand.*, **55**, 1.
Gibson, H. J. (1938). *Proc. R. Soc. Med.*, **31**, 309.
Gram, H. C. (1929). *Acta med. scand.*, **70**, 242.
Gregg, R. O. (1937). *J. Lab. clin. Med.*, **22**, 786.
—— (1939). *Proc. Mayo Clin.*, **14**, 600.
Ham, T. H., and Curtis, F. C. (1938). *Medicine*, **17**, 447.
Hubbard, R. S., and Geiger, H. B. (1928). *J. Lab. clin. Med.*, **13**, 322.
Hunt, H. F. (1929). Ibid., **14**, 1061.
Hynes, M., and Whitby, L. E. H. (1938). *Lancet*, **2**, 249.
Linzenmeier, G. (1920). *Arch. ges. Physiol.*, **181**, 169.
Meeker, D. O. (1925). *Clifton med. Bull.*, **11**, 72.
Rubin, E. H. (1926). *Arch. intern. Med.*, **37**, 848.
Schuster, N. H. (1938). *Tubercle*, **19**, 529.
Sugarman, H. (1939). *Canad. med. Ass. J.*, **40**, 65.
Vogt, C. J. (1941). *Amer. J. Obstet. Gynec.*, **41**, 206.
Walton, A. C. R. (1933). *Quart. J. Med.* n.s., **2**, 79.
Whitby, L. E. H., and Britton, C. J. C. (1946). *Disorders of the Blood*, 5th ed. Churchill, London.
Wintrobe, M. M., and Landsberg, J. W. (1935). *Amer. J. med. Sci.*, **189**, 102.

Nearly 15,000 more hospital beds are now available for patients in England and Wales than before the National Health Service began in July, 1948. This brings the number of staffed beds to 470,000, out of a total of about 514,000. Hospital nursing and midwifery staff has increased by 24,000—16,000 more full-time and 8,000 more part-time. Increases have been recorded among all grades, including student nurses, midwives, and pupil midwives. There are now 7,000 more student nurses, the total having risen to 49,000. This is a new record. Total full-time hospital nursing and midwifery staff is now nearly 135,000, and part-time 25,000. The increase in staff has enabled many more hospitals not only to bring empty beds back into use but to introduce as well a 96-hour fortnight and to improve working conditions generally. These figures were issued by the Ministry of Health early in November, and cover the period July 5, 1948, to June 30, 1950—the first 24 months of the Service.