when the vaginal walls were retracted. My house-surgeon was proceeding to apply forceps when I entered the labour room with my students. When I examined the outlet I knew the head could not be extracted. I divided the symphysis, following which my house-surgeon was able to complete the delivery with the greatest ease. The kyphotic pelvis in which the interischial diameters are so narrow that the head cannot be delivered is comparatively rarely encountered. In most instances the deformity is not extreme, and generally the child is small.

In my experience the worst cases of outlet dystocia have been those associated with "deep transverse arrest" of the head. It is inevitable if the sacral curve is very flat (the "flat non-rachitic pelvis"). I have seen several examples of this most troublesome complication, and on occasion have found it impossible to effect rotation manually and deliver with forceps, or to rotate the head with forceps. There is another type of pelvis which those who have studied outlet dystocia by means of radiography claim to be a common cause of "deep transverse arrest"—namely, the android pelvis. I can well understand this; but I cannot speak from personal experience because I had retired from practice before I was able to make many observations with this advanced pelvic radiography as a diagnostic aid.

Those who have had considerable experience of pelvic radiography give us this comforting information—and I am convinced they are right—that the anthropoid type of pelvis need cause little concern. The incidence of this type of pelvis has not been assessed for our country as a whole, or for any locality. Thoms (1937), Caldwell and Molloy, and others in the United States have found the incidence to be 15 to 17 per cent. for the white population and over 30 per cent. for the negro population. In a large proportion (30 to 40 per cent.) of patients with pelves of anthropoid form an occipito-posterior position develops and becomes persistent. Unless, however, the fore-pelvis is very narrowed, spontaneous delivery "face to pubes" is frequent, and if forceps is necessary delivery "face to pubes" is easy. My clinical experience goes to support this—not the association with anthropoid pelvis, but the fact that some deliveries face to pubes are easy and others difficult irrespective of the factor of size of foetal head. Now, there is a very important practical deduction from this. If an anthropoid pelvis is known to exist from the pelvic radiograph there is no need to rotate the head manually or by forceps—indeed, it may be a mistake to do so. The interests of mother, and especially of child, may be better served by leaving the case to Nature as in the ordinary gynaecoid type, in which long rotation forwards occurs in over 90 per cent. of cases.

And now I am going to spring a surprise—I advise the *judicious employment of symphysiotomy or pubiotomy*. It is preferable to Caesarean section where the head is fixed at the pelvic outlet. You cannot employ the lower segment incision in this event because of the difficulty in removing the child. Besides, the labour has been going on for long and possibly several vaginal examinations have been made, and manual corrections may even have been attempted. By all means choose Caesarean section if beforehand you know it is almost a certainty that there will be great difficulty in the delivery per vaginam because of the outlet deformity. Let us consider symphysiotomy for a moment. I know the operation is looked upon with disfavour by most obstetricians, but I have satisfied myself that *it is a wise procedure in certain cases of outlet dystocia*: please note, outlet dystocia, not inlet dystocia. I have had personal experience of this operation: few of

my critics can say that they have ever performed the operation or seen it performed. If symphysis or pubic bone is divided, every centimetre of separation of pubic bones increases to a like extent all the transverse diameters of the pelvis. This greatly facilitates intrapelvic manipulations and forceps delivery.

There is one other matter to which I feel I must make reference—breech presentations with known pelvic distortion. Unless the distortion is of a very minor degree I advise Caesarean section. No attempt at external version before labour should be made.

So you see—and on this note I conclude these two lectures—how clear and well defined is the treatment of pelvic disproportion, provided there is exactness in diagnosis. This can now be ensured—to a degree never before possible—by ordinary old-time methods, supplemented by judicious employment of radiography. And just one word more. It is satisfactory to learn that the exact observations of pelvic formation by radiography carried out by those who have had experience of them have not lessened the employment of "conservative methods" of delivery. On the contrary, the rates for Caesarean section and for forceps delivery have fallen in the clinics in which meticulous exactness of diagnosis has been striven after.

REFERENCES

- Jeffcoate, T. N. A. (1938). *J. Obstet. Gynaec. Brit. Emp.*, **45**, 893.
Simpson, Sir J. Y. (1847). *Prov. med. surg. J.*, **9**, 673.
Thoms, H. (1937). *Surg. Gynec. Obstet.*, **64**, 700.

HAEMATEMESIS AND MELAENA

OBSERVATIONS ON THE USE OF CONTINUOUS
DRIP TRANSFUSIONS

BY

F. AVERY JONES, M.D., M.R.C.P.

*Second Assistant to the Medical Professorial Unit,
St. Bartholomew's Hospital*

This report illustrates the results which may be obtained in severe and repeated haematemesis by the prevention of dehydration, by liberal feeding, and by the use of continuous-drip blood transfusions. During the past four years all cases of haematemesis and melaena admitted to the medical professorial unit at St. Bartholomew's Hospital have been treated medically on the lines instituted by Professor Witts in 1935 and described by him in 1937; in no case was surgical intervention advised for arrest of haemorrhage. This series consisted of sixty-five consecutive cases, including the twenty-six already described by Witts (1937). Of the total, seven were due to cirrhosis of the liver and two to carcinoma of the stomach, and six patients left hospital before an accurate diagnosis could be established. The remaining fifty patients bled from acute or chronic ulceration; they form the subjects of this report, and include thirteen with severe and repeated haemorrhage. The severity of these thirteen cases may be judged from the details given in Table I.

Recurrence here refers to signs and symptoms of acute haemorrhage not less than twenty-four hours after a previous haemorrhage. In some cases there was also persistent slow bleeding. Two comments may be made on the table. In one patient the lowest haemoglobin recorded was 50 per cent. This was estimated before full dilution of the blood could have occurred, because the rise in the haemoglobin after the transfusion was 20 per cent. less than expected. The lowest haemoglobin reading

in the table is 24 per cent. Lower figures were prevented in all probability by the drip transfusions, which were usually given if the haemoglobin fell below 40 per cent.

TABLE I.—*Particulars of Thirteen Cases of Severe Haematemesis*

Sex	Age	Diagnosis	Lowest Hb %	Highest Blood Urea mg. per 100 c.cm.	Recur- rences	Blood Transfusion c.cm.
Female	69	Erosive gastritis	29	280	4	3,950
"	50	Subacute G.U.	33	45	6	Died 6,735
Male	53	Acute ulcer	24	84	3	(Chart) 6,700
"	49	Chronic D.U.	27	108	3	7,150
"	61	Acute ulcer	26	132	3	2,200
"	48	"	50	116	2	2,810
"	51	Chronic D.U.	33	96	3	600
Female	44	Acute ulcer	30	84	1	1,050
Male	48	" "	33	78	4	600
"	39	" "	41	86	2	600
Female	47	" "	39	78	1	—
Male	54	Chronic G.U.	40	80	2	—
"	49	Chronic D.U.	33	168	2	—

Out of the fifty patients with simple peptic ulceration one only died. She was a woman of 69, and at necropsy a severe erosive gastritis was found. Although perhaps it is not permissible to draw conclusions from so small a series, one death in fifty cases of bleeding from peptic ulceration, including thirteen with severe and repeated haemorrhage, of which ten had three or more haematemeses, compares favourably with the results in other series treated medically or surgically: in cases from the same hospital Cullinan and Price (1932) recorded twelve deaths out of twenty-one cases of haematemesis that recurred more than once. In the present series patients received at least 2,700 c.cm. of fluid daily, and, in addition, semi-solids were allowed from the time of admission. Dehydration, so common under the old regime, was almost entirely eliminated, and there is no evidence to suggest that the liberal feeding was anything but beneficial. This aspect has been discussed at length by Witts (1937).

Indications for Transfusion

It is the experience with drip blood transfusions that I wish to place on record. There have been two main indications for transfusion. If there was severe shock, with the systolic blood pressure below 90 mm. Hg and the pulse above 120, a transfusion was given, whatever the haemoglobin percentage might be; for such patients, if left too long, may pass into a state of secondary haemorrhagic shock from which transfusion may fail to save them. Again, if the haemoglobin fell below 40 per cent. the patient was transfused, since the clinical condition may deteriorate rapidly if haemorrhage recurs in a patient with this degree of anaemia. If the haemoglobin fell below 40 per cent. but there were no symptoms to suggest continued bleeding, then 600 to 1,200 c.cm. of blood were given by the drip technique. If the haemoglobin was below 40 per cent. and there were indications of continued bleeding, then the drip was maintained until all signs of bleeding had ceased and this could be proved by a rising haemoglobin. In this way 1,500 to 3,000 c.cm. of blood have been given.

A strong prejudice exists against transfusion for gastro-duodenal haemorrhage. There is evidence to suggest

that an ordinary transfusion given soon after the haemorrhage will provoke further bleeding in 50 per cent. of the cases; if, however, it is given when bleeding has stopped, there is no tendency for further haemorrhage to ensue (Cullinan and Price, 1932). An ordinary transfusion of 300 to 600 c.cm. of blood is made in fifteen to thirty minutes. To give the same amount by the drip technique of Marriott and Kekwick (1935-7) will take three to six hours. It is of importance to discover whether or not this is an advantage in transfusing for gastro-duodenal haemorrhage. Two aspects of the problem must be considered. Is a drip transfusion less likely to provoke further bleeding than an ordinary transfusion, and is it safe to give several pints? Does bleeding stop if a drip transfusion is begun?

Blood-pressure Changes after Haemorrhage and Transfusion

Recurrence of haemorrhage may be due to further erosion of the bleeding vessel or to displacement of the clot, possibly by an increase in blood pressure. Attention has therefore been paid to the blood-pressure changes after haemorrhage and after transfusion. In my experience, after a small haematemesis the blood pressure was usually within normal limits, or even slightly raised. This is in accordance with experimental work in animals, where the blood pressure is at first well maintained after haemorrhage (Meek and Eyster, 1921). With a rapid moderately severe bleeding the patient passes into a shocked state, sweating freely. The systolic blood pressure is below 90 mm. Hg and the pulse often above 110. If given the usual treatment, after one-half to one hour the blood pressure begins to rise and the pulse rate falls, and within six to twenty-four hours there is usually a noticeable increase in pallor. By this time the percentage of haemoglobin is falling, from dilution of the blood and return to the normal blood volume. In some cases the pallor is out of proportion to the haemoglobin reading, and it is at this time that an increase in blood pressure may be found: almost without exception the highest blood-pressure readings recorded in these patients have been observed within six to twenty-four hours of a haemorrhage. In over a quarter of the cases the blood pressure was apparently raised. The blood pressure is so labile in normal people that this observation might be discounted as an emotional reaction to the sphygmomanometer. It is difficult, however, to do so in these cases, for when the blood pressure is taken at frequent intervals a steady rise and fall extending over many hours may be found, as in the case of a man aged 42 with acute ulcer, who was admitted one day after haemorrhage with a blood pressure of 160/65, which on subsequent days was 160/65, 120/70, 120/70, and 120/70. This rise and fall is not due to the journey to hospital, for it is seen after recurrent bleeding in the wards, as shown in the following case:

A man aged 35 with duodenal ulcer had a haematemesis in the out-patient department. His blood pressure one hour later was 106/70, and at midnight, after a second haemorrhage, it was 95/50. At 2 a.m. it had increased to 136/85, and at 10 a.m. to 150/88. On the following days it fell to 140/68, 120/65, and 118/64.

The phase of reactive hypertension is well seen in the accompanying chart. It shows that the blood pressure rose sharply within an hour of the start of the first drip transfusion to a peak of 150 mm. Hg. This might have been attributed to the transfusion, but a similar transient hypertension occurred on the eighth day, again after haemorrhage, this time without transfusion. On the

thirteenth day there was further severe bleeding, and a drip transfusion was begun. Although 2,650 c.cm. of blood was then given the blood pressure did not rise above 118 mm. Hg. It is possible that the large transfusion prevented the phase of hypertension, which had been observed twice previously.

There have been few opportunities for observing the blood-pressure changes following ordinary transfusions

tend to provoke further immediate haemorrhage, which occurred in one case only, and there is no evidence that it is haemostatic in action, but the signs of bleeding may cease while the patient is undergoing drip transfusion.

The observation that some of the patients bled three or more times and on each occasion had to be transfused must not condemn this therapeutic measure. In the past the mortality in cases of recurrent haemorrhage has been

TABLE II

Case No.	Volume of Blood Given	Time Taken	Blood Pressure	
			Before	After
1	2,810 c.cm.	25 hours	104/63	102/60
2	750 "	4 "	112/52	110/60
	2,300 "	14 "	84/46	120/64
	1,300 "	8 "	118/60	120/64
	2,800 "	18 "	120/64	110/60
3	1,200 "	7½ "	60/42	115/68
4	1,200 "	8 "	98/54	128/72
5	3,100 "	28 "	58/?	100/56
6	1,135 "	19½ "	118/60	142/76
	1,700 "	21 "	120/64	120/60
	1,250 "	14 "	116/64	110/70
7	2,650 "	23 "	76/36	110/68
	1,200 "	19 "	124/60	130/60
8	1,200 "	6½ "	110/	105/
	1,800 "	15 "	120/	140/
	3,000 "	20 "	106/	135/

Chart of a severe case of haematemesis given continuous-drip transfusion.

given soon after a haematemesis. It has been mentioned that such transfusions tend to provoke further bleeding. A possible reason for this would be a sudden increase in blood pressure associated with the injection of 300 to 600 c.cm. of blood in fifteen to thirty minutes. Such a sudden increase was actually observed in one case.

Blood-pressure Changes following Continuous-drip Transfusion

A number of observations have been made on the blood-pressure changes with continuous-drip blood transfusions, and they are recorded below (Table II). Except in the one case already discussed, any change in blood pressure has been gradual, and there have been no sudden fluctuations during any transfusion when detailed records have been made. It will be noticed that many of the transfusions were given rather more rapidly than advised by Marriott and Kekwick.

These observations suggest that a drip transfusion does not produce any rise of blood pressure other than might occur without transfusion. Drip transfusion does not

extremely high. It is interesting to note that in three cases in which haemorrhage had already occurred several times a slow transfusion of over 2,400 c.cm. of blood was not followed by further bleeding and the patient made a rapid convalescence. It may be necessary to persevere and give several transfusions, but in most cases the bleeding will ultimately stop. In three patients it was necessary to give over 6,000 c.cm. of blood.

Conclusion

The technique of Marriott and Kekwick is so simple and the apparatus so inexpensive that with it institutional treatment is not necessary. It is not even essential to cut down on the veins if the special needle introduced by Waring (1938) is used. The difficulty, of course, is to secure a sufficient number of donors. This has been achieved by the full use of relatives and fellow employees, and by the generous assistance of the British Red Cross Society Transfusion Service. It may be argued that the larger drip transfusions may increase the blood volume. If begun soon after the haemorrhage it is probable that this increase is small, for it must be remembered that the blood lost in these severe cases is about two or three litres. One of my observations, nevertheless, suggests that there was a slight rise in the blood volume after a drip transfusion of 3,000 c.cm. The haemoglobin percentage increased from 28 to 75 during the transfusion, but rose a further 8 per cent. in the following twenty-four hours, and so during this time there must have been a decrease of plasma volume, associated with the 8 per cent. rise in haemoglobin. As there was no abnormal rise in blood pressure and no cardiac embarrassment it is difficult to see how the temporarily increased blood volume was harmful.

It was found that the clinical response of the patient to the haemorrhage or to the transfusion could be well followed by charting the pulse frequency, the blood pressure, the haemoglobin percentage, and the blood-urea values. It was thus possible to obtain an indication of the severity of the haemorrhage, and to tell whether or not the haemorrhage had ceased. Much emphasis has recently been laid on the value of blood-volume estimation in determining these two points (Izod Bennett *et al.*, 1938). These estimations are of great interest, and may be of practical value, but for planning treatment and judging the clinical response it would appear that enough information may be gained by the simple measures described above.

Summary

A series of fifty cases of haemorrhage from simple peptic ulceration is reported. There was only one death, although there were thirteen cases of severe repeated bleeding.

This low mortality rate is attributed to the liberal feeding of the patients and to the use of continuous-drip blood transfusions.

Drip blood transfusion does not provoke further immediate haemorrhage.

After haemorrhage there may be a transient phase of reactive hypertension, but there is no evidence that a drip transfusion produces any abnormal increase in the blood pressure.

It is suggested that a sufficiently accurate indication of the progress of a patient may be gained by charting the blood pressure, pulse frequency, haemoglobin percentage, and possibly the blood-urea values.

My thanks are due to Professor L. J. Witts and Professor R. V. Christie, on whose patients these observations were made, for their interest and assistance in this paper. I am grateful for helpful criticism from Dr. G. Graham, Dr. Geoffrey Evans, Dr. H. L. Marriott, and Dr. R. Bodley Scott.

REFERENCES

- Bennett, T. I., Dow, J., Lander, F. P. L., and Wright, S. (1938). *Lancet*, **2**, 651.
Cullinan, E. R., and Price, R. K. (1932). *St. Bart's Hosp. Rep.*, **65**, 185.
Marriott, H. L., and Kekwick, A. (1935). *Lancet*, **1**, 977.
— (1937). *Practitioner*, **139**, 250.
Meek, W. J., and Eyster, J. A. E. (1921). *Amer. J. Physiol.*, **56**, 1.
Waring, J. (1938). *Lancet*, **2**, 1180.
Witts, L. J. (1937). *British Medical Journal*, **1**, 847.

The April issue of *The Mission Hospital* has an article on pioneering among the Nuers of Zeraf Island in the Southern Sudan, which reveals how the struggle for existence is being eased by public health activities and, more particularly, by an intensive campaign against mosquitos, flies, and white ants, associated with the prevention of such diseases as trachoma; this work is carried on in dispensaries built of mud and surrounded by a most apathetic and ignorant population. In contrast there comes news from Travancore of the opening of a fine new rural medical mission, replacing a tiny thatched shed, which will now serve as a ward with one or two beds, the scheme being the forerunner of a hospital compound, including a child welfare centre. The uphill work going on in Palestine, in the face of increasing unemployment and poverty caused by political strife, is also described by the medical officer at Nablus, which has been for long a stronghold of Moslem Arabs. These and other articles illustrate how the days of pioneering are not over for medical missionaries, and the possibilities of conducting a large practice with the aid of tents, lorries for hospitals, and river boats for dispensaries are indicated. The jubilee of medical missions as a component part of the Church Missionary Society is due in two years' time.

THE MORTALITY OF CHILDREN IN ENGLAND AND WALES AND GERMANY

A STATISTICAL COMPARISON

BY

GUSTAV TUGENDREICH, M.D.

It has long been recognized that study of rates of mortality in different countries at approximately the same levels of sanitary development is an important duty of the intelligence service of a Public Health Department. No two systems of public health services are identical, and variations in rates of mortality may throw light upon their respective efficiencies. These considerations apply particularly to rates of mortality in early life, when it is reasonable to believe that good or bad environmental factors exert their greatest power.

While working in the Statistical Department of the London School of Hygiene and Tropical Medicine I was struck by the excessive mortality from measles which characterized England and Wales as compared with Germany. Although the population of England and Wales is little more than 60 per cent. of that of Germany, in the years 1921–30, 42,450 deaths were assigned to measles in England and Wales and 39,740 in Germany, while in the quinquennium 1931–5 England and Wales had 13,750 deaths and Germany only 7,506. I was, of course, aware that the periodicity of epidemic measles makes comparison of short series of years dangerous, while differences in the practice of certification and classification to single causes in different countries are always stumbling-blocks of international comparisons. But examination of the official rules for classification in use in the respective Government Departments did not suggest that there was any gross fallacy of comparison, and led me to conclude that Sieveking was justified in his remark (*Der öffentliche Gesundheitsdienst*, **2**, 1936–7, p. 380) that measles has not in Germany the serious character which causes the disease to be feared in England. On working out the actual rates of mortality for each of the five years 1931–5, it appeared that, although in every age group the English rate of mortality was the higher, the excess in the age group 1–5 was particularly striking. At the best (1935) it was nearly twice as high as the German rate, at the worst (1931, 1932, and 1934) some five times as high. This especially unfavourable position of children of pre-school age in England and Wales led me to examine the statistics of child mortality in further detail. I propose here to give a summary of the most striking features, reserving a detailed examination for another occasion.

I begin with rates of mortality from all causes in the first year of life. As within the quinquennium 1931–5 there were relatively large variations in the numbers of births, particularly in Germany, the conventional method of statement of mortality rates in terms of annual live births is not quite satisfactory. But as recalculation of the English rates in terms of quarterly births did not seriously affect the results the usual method may stand.

TABLE I.—Rates of Mortality from All Causes per 1,000 Live Births in England and Wales and in Germany

Months of Life	1931		1932		1933		1934		1935	
	E.&W.	G.	E.&W.	G.	E.&W.	G.	E.&W.	G.	E.&W.	G.
0–1	31.6	42.9	31.6	41.9	32.2	41.5	31.3	36.6	30.4	36.2
1–3	10.9	13.9	10.8	14.6	9.9	12.7	8.8	11.8	9.1	11.4
3–6	9.9	13.3	9.2	12.4	8.8	11.9	7.5	11.0	7.7	10.9
6–9	7.4	8.2	7.2	7.9	6.3	7.7	5.8	7.1	5.4	6.9
9–12	6.8	5.5	6.3	5.2	6.0	4.9	5.1	5.0	4.3	4.6