It plays a significant part in exacerbation of ulcer symptoms and is at least a contributory cause in one in eight cases of haematemesis, and in one in five of that group with no major dyspeptic history.

We wish to thank Dr. A. C. Aitkenhead, Dr. J. Bryant, and Mr. W. J. M. Brandon for permission to investigate patients in their wards, and Dr. R. MacAndrew for assistance in the examination of the specimens. We are indebted to Mr. R. G. Scott for the photographs and to Messrs. Reckitt & Colman, Ltd., for preparation of the hard aspirin tablets.

REFERENCES

- Douthwaite, A. H. (1938). *British Medical Journal*, 1, 1143.
 — (1954). *Lancet*, 2, 917.
 — and Lintott, C. A. M. (1938). *Ibid.*, 2, 1222.
 Forssell, G. (1938). *Fortschr. Röntgenstr.*, 57, 331.
 Hurst, A. (1943). *British Medical Journal*, 1, 768.
 — and Lintott, G. A. M. (1939). *Guy's Hosp. Rep.*, p. 173.
 Konietzky, G. E. (1930). *Die entzündliche Grundlage der typischen Geschwürsbildung im Magen und Duodenum*. Springer, Berlin.
 Magnus, H. A. (1937). *J. Path. Bact.*, 44, 389.
 — (1940). *British Medical Journal*, 1, 415.
 — (1946). *J. Path. Bact.*, 58, 431.
 Paul, W. D. (1943). *J. Iowa St. med. Soc.*, 33, 155.
 Publ. H. (1927). *Virchows Arch. path. Anat.*, 265, 160.
 Richmond, H. A. N. (1950). *British Medical Journal*, 1, 439.
 Walters, W., and Sebenius, W. (1932). *Minn. Med.*, 15, 579.
 Wolf, S., and Wolff, H. G. (1943). *Human Gastric Function*. Oxford Univ. Press, London.

ACCIDENTAL HAEMORRHAGE

BY

A. P. BARRY, M.D., M.A.O., F.R.C.O.G.
Master, National Maternity Hospital

J. K. FEENEY, M.D., M.Sc., M.A.O., D.P.H.
T.D.D.

Master, Coombe Lying-in Hospital

AND

F. J. GEOGHEGAN, M.D.
Pathologist, National Maternity Hospital, Dublin

During the past six years we have developed and maintained a special interest in accidental haemorrhage. In these years there have been 52,000 deliveries in the services of our two hospitals. Our investigations have included a study of the records of over 3,000 cases of accidental haemorrhage (including the necropsy findings in 100 cases) which were treated in the three Dublin Maternity Hospitals (Coombe, National Maternity, and Rotunda) over the period of 25 years from 1929 to 1953 inclusive.

A careful study of the case reports and statistics from Dublin and from other centres indicates that three complications render accidental haemorrhage one of the most serious happenings in present-day obstetrics. These complications are: (a) progressive and at times intractable haemorrhage, resulting from a deficiency in the clotting power of the blood; many of the hitherto imperfectly explained features of accidental haemorrhage are the natural results of this clotting defect; (b) anuric renal failure; and (c) foetal asphyxia. The foetal loss in all cases of accidental haemorrhage has been about 50%.

Deficiency in Clotting Power

We emphasize that the bleeding in a severe case of accidental haemorrhage is of a very special type. It is due to a fall in the level of the blood fibrinogen. Schneider has advanced an ingenious explanation of this occurrence. He has pointed out that the thromboplastin content of the decidua and placenta is very much higher than in most other tissues. When the placenta separates accidentally, the

disparity in the diameters of the arterial sinusoids of the endometrium and those of the retroplacental haematoma is such as to lead to an expanding force, activated in a manner comparable to that of a hydraulic pump. The pressure within the haematoma becomes very great and, bursting, its admixed tissue and decidual juices are forced into the intervillous lake. The liberation of thromboplastin in this way initiates coagulation. Thrombin is produced and the circulating fibrinogen is thereupon converted into fibrin. In our opinion, most of the fibrin formed in this way is deposited in the great vascular bed of the pregnant uterus.

At a certain level of falling blood fibrinogen (200 mg. per 100 ml.) haemorrhages are likely to occur. Liver replacement, which may take from 12 to 24 hours, fails to keep pace with fibrinogen conversion, so that hypofibrinogenemia results, with the onset of widespread haemorrhages, occurring spontaneously or induced by trauma. The uterus is not paralysed and incapable of contracting, but clotting power is lost. In spite of massage, oxytocic substances, contraction and retraction, blood transfusion, uterine plugs, and hysterectomy, haemorrhage continues from every traumatized point in the genital tract until death occurs or the clotting defect is corrected. If blood should lose its clotting power after premature separation of the placenta, more and more blood is poured out behind the detached placenta to overstretch the uterus. Shock is thereby induced from simple blood loss and from distension of the organ. The non-clotting blood can infiltrate between the uterine muscle fibres and escape into the broad ligaments and into and behind the peritoneal cavity. Thus there is still more shock from further blood loss and from tissue trauma and haematoma formation. The Couvelaire uterus might well be described as a diffuse haematoma of the uterus.

Once the maternal blood escapes without clotting, each tiny vascular breach will open wider, because it is not sealed off in the ordinary way. The stage is thereby set for the well-known clinical features of severe accidental haemorrhage: the tense, tender, Couvelaire uterus; the retroplacental and intraligamentous collections of blood; the haemorrhages under the capsule of the liver; the death undelivered; the death from intractable post-partum haemorrhage in spite of exploration, plugging, replugging, or hysterectomy; the death following caesarean section or caesarean hysterectomy. We believe that there is no unusual element of shock in these disasters. The patient dies from haemorrhage which is occurring not only from her genital tract, but into and about her abdominal organs, especially at the site of trauma, no matter how slight. The major loss may not be external. The condition is directly due to fibrinogenopenia, resulting from the absorption into the circulation of thromboplastin.

This clotting defect is to be found in the majority if not in all cases of combined and concealed accidental haemorrhage. Once it develops, bleeding must continue until the clotting power of the blood has been restored to normal. Serious loss of blood from the circulation is a constant feature of these cases. In a recent letter (to J. K. F.) Fletcher Shaw wrote: "I believe that hypofibrinogenemia gives me the answer, which I have so long desired, to a remarkable series of 11 cases of accidental haemorrhage treated 40 years ago by Clifford and myself in St. Mary's Hospital, Manchester. In all cases the uterus was markedly discoloured. One or both broad ligaments contained large effusions of blood which extended upwards to the lower pole of the kidney in two instances. One uterus showed minute cracks exuding blood like the splitting of a ripe plum. The pelves were filled by liquid blood. I can still visualize the dreadful picture as clearly as when I opened the abdomen. Five of the 11 cases survived caesarean hysterectomy. I came to the conclusion that the poor nutrition of wartime was a factor." Malnutrition and anaemia are indeed important associated conditions.

Since the amount of blood-coagulation substances in transfused fresh or citrated blood is relatively small (each pint of transfused blood has a fibrinogen content of about

200 mg. per 100 ml.), transfusion can be effective only if given early and generously before marked defibrination of blood yields about 1 g. of fibrinogen, but when a rapid prior to or early in the development of this progressive condition. It should be accompanied by early puncture of the membranes and adequate drainage of the liquor in an attempt to reduce thromboplastin absorption and uterine distension. This step should be carried out at the onset of treatment. The best laboratory methods to determine the presence of fibrinogenopenia are the Schneider and the Shea tests, both of which may be easily performed in any small clinical laboratory. The Schneider test can be done within 15 to 20 minutes. The Shea test, after 45 minutes, gives a sufficiently accurate figure for plasma fibrinogen. The Lee and White clotting test, although useful in severe cases, does not detect minor degrees of the condition.

On clinical grounds, we believe that fibrinogenopenia can be assumed to be present if the uterus is tender. The uterus becomes tender when blood is infiltrating between its muscle fibres. This does not occur unless the coagulation defect is already established. A severe degree of fibrinogenopenia can be reversed only by the intravenous administration of pure fibrinogen or of plasma solids. One pint (568 ml.) of transfused blood yields about 1 g. of fibrinogen, but when a rapid coagulation effect is essential, pure human fibrinogen (4 to 6 g.) or total plasma solids (80 to 120 g.) should be administered intravenously. 18 g. of plasma solids, a single dose as supplied by our National Blood Transfusion Association, contains just under 1 g. of fibrinogen.

To attempt to overcome severe fibrinogenopenia by transfusion alone may lead to the situation so ably described by Kellogg (1948) when referring to the treatment of severe cases of accidental haemorrhage: "This has on occasions led to the unpleasant situation in which more and more foreign blood has been given to a patient only to be washed out in what remains of the patient's own blood, her condition being such that one did not dare to stop the transfusion."

Anuric Renal Failure

O'Driscoll and Quinn (1951) studied maternal mortality in Dublin in the decade 1940-9. During these years, as a result of improvement in the treatment of haemorrhage, the incidence of death from renal failure increased. Of 32 mothers who died of accidental haemorrhage, 15 survived the acute phase but succumbed to anuric renal failure. Transfusion and restorative treatment will save patients, but they must be rescued from the anuria which may follow. This is caused by renal vasospasm, which has been so ably investigated by Sheehan and Moore (1952).

The utero-renal reflex of Sophian, combined with the haemex effect of Franklin, adequately explains the intense vasospasm which frequently occurs. In every case of accidental haemorrhage, renal output must be recorded accurately, and if it falls below the arbitrary figure of 1 oz. (28 ml.) an hour, steps must be immediately taken to cause relaxation of the renal vasospasm. These consist of energetic treatment of the shock by the administration of morphine and blood transfusions, immediate puncture of the membranes and drainage of the liquor in order to abolish the utero-renal reflex and to diminish the absorption of thromboplastin, and the performance of renal pelvic nerve block. The beneficial effect of block has been demonstrated by the insertion of ureteric catheters immediately before its performance.

If the anuria should persist after the block and if delivery should not be imminent, lower-segment caesarean section under local analgesia should be performed. If treatment has not been carried out in good time and if renal failure continues into the puerperium, the management of the case should be according to the method advocated by Bull *et al.* (1949). When advanced cortical necrosis has already occurred, this regime will not prove successful. If due consideration is paid to these two factors—that is, the clotting defect and the anuric renal failure—the following treatment for accidental haemorrhage should be carried out.

There has been no maternal mortality in 600 consecutive cases of accidental haemorrhage treated on these lines in our two hospitals.

All cases may be classified as mild, moderate, or severe.

Mild Cases

Mild cases are those with no symptoms or signs apart from slight vaginal bleeding, with or without an associated toxæmia. In general, they require no special treatment other than bed rest, sedation, and attention to the toxæmia if present. If the bleeding should continue or recur, the membranes should be punctured. There are many cases on record of mild accidental haemorrhage with temporary recovery and discharge from hospital, but with the onset of grave concealed and sometimes even fatal haemorrhage at a later date. If an associated toxæmia is present in these mild cases and if it does not clear up at once, the pregnancy should be terminated because of the danger of recurring haemorrhage and worsening toxæmia. In some mild cases, factors such as elderly first pregnancy, foetal distress, recurrent bleeding, exacerbation of toxæmia, etc., may indicate caesarean section. There are no dangers in the mild case other than those of actual blood loss, which can be seen and restored if necessary, and the possibility that the case, if not treated promptly, may pass into the next group. Early puncture of the membranes will avoid this misfortune. The only way to reduce foetal mortality in accidental haemorrhage is by energetic treatment of all mild cases. There is a tendency to underestimate their significance.

Moderate Cases

Moderate cases are those showing some shock or moderate external loss and some internal bleeding, as indicated by hypertonicity of the uterus and abdominal tenderness. As we have already pointed out, we believe that abdominal tenderness indicates some degree of clotting defect. The Schneider and Shea tests should be done. Such cases should be treated by immediate puncture of the membranes and drainage of the liquor, combined with blood transfusion of two or more pints (1 litre or over). Labour should be stimulated by the administration of pitocin, because early delivery plays a very important part in the prevention of the complications already mentioned. Both renal and blood complications depend to a great extent upon the length of time which elapses between the collection of blood beneath the placental site and delivery. We recommend the use of pitocin in these cases in spite of experimental animal evidence that it leads to vascular constriction. A catheter must be inserted at once and constant observation of the urinary output maintained. Clotting-time should be observed from hour to hour. If progress is not rapid, or if the complication of clotting defect or suppression of urine should ensue, treatment should be carried out as recommended for the severe case and lower-segment section performed, if delivery is not imminent. Even with great care, an occasional case may pass into the severe group, or, as not infrequently happens, the patient is admitted already in this category.

Severe Cases

Severe cases are those exhibiting shock and a tender distended uterus of board-like consistency together with an absence of foetal heart sounds. The maternal deaths occur in this group. Such patients should receive a sedative in order to allay apprehension and to ease pain—morphine, $\frac{1}{4}$ gr. (16 mg.). A rubber catheter must be placed in the bladder and renal secretion recorded. The simple clot-observation and Schneider tests should be performed at hourly intervals. Blood transfusion of at least 3 pints (1.7 litres) should be administered. Blood provides fibrinogen, relieves shock, improves the circulation to organs such as the kidney and myocardium, and helps to counteract pre-existing anaemia. The transfusion should be rapid, as restoration of renal blood flow lags considerably behind the re-establishment of normal blood pressure and cardiac output. This is explained by the nature of the utero-renal reflex and of the haemex effect. The membranes must be

punctured as soon as possible so as to reduce intra-amniotic and retroplacental tension. This is one of the most important steps in treatment and should never be neglected, even though the patient may still be in a state of shock and the uterus still inactive. It helps to reduce shock, to restore renal secretion, to stimulate uterine contractions, and to delay or prevent the further absorption of thromboplastin. If the clotting defect has not been controlled by brisk transfusion, pure fibrinogen in a dose of 4 to 6 g., or plasma solids, 72 to 108 g., must be given intravenously.

The best clinical evidence of control of the clotting defect is an improvement in the general condition of the patient and the absence of increasing uterine distension and intra-uterine tension. If urinary secretion does not rapidly return to an amount of at least 1 oz. (28 ml.) an hour, bilateral splanchnic block should be performed without delay. Indeed, when the systolic blood pressure has reached 100, block should be carried out at once if the kidneys are not secreting as noted. If haemorrhage has been controlled, if the patient's general condition is improving, and if the kidneys are secreting not less than 1 oz. (28 ml.) an hour, vaginal delivery may be awaited and its advent may be hastened by the administration of pitocin. If, however, bleeding continues or begins again, as evidenced by worsening of the patient's general condition, by an increase in the size of the uterus, or by a fall in blood pressure, or if the kidneys have failed to respond to block, then the uterus should be emptied immediately by lower-segment caesarean section, under local or expertly administered general anaesthesia. The administration of fibrinogen or plasma solids should be continued during the operation in order to prevent free loss from all traumatized points. There is no necessity to remove the uterus, no matter how black and damaged it may be. Post-partum haemorrhage will not occur if the clotting defect has been corrected. As already stated, if oliguria or anuria persists after delivery, the management should be by Bull's method. If one is aware of the possibility of renal vasospasm, and if one treats the condition adequately before delivery, Bull's regime should be rarely required.

Technique of Splanchnic Nerve Block

The patient is placed sitting upright with her legs over the side of the bed. This is the most suitable position for easy insertion of the needles and is reasonably comfortable for the woman suffering from accidental haemorrhage with its associated abdominal pain. Four long needles, 12 to 16 cm., are selected. After sterile preparation the following sites are marked out with skin blebs of local analgesia without hyaluronidase: two points on each side, 8 cm. (3½ in.) lateral to the level of the twelfth thoracic and first lumbar spines. The lower points lie just below the outer end of the twelfth rib. The long needles are directed inwards at an angle of 45 degrees and slightly upwards in order to strike the antero-lateral aspect of the vertebral body. It is better to direct the upper needles slightly upwards, instead of straight in, as in the usual paravertebral injection. When the tip of the needle strikes the antero-lateral aspect of the vertebral body it should be withdrawn slightly and the direction changed in order to allow the point of the needle to pass round the maximum circumference of the vertebral body. When the tip of each needle finally passes clear of the vertebral body, it should be introduced about a centimetre further forwards, when it will be in the correct position. At this stage, in a patient of average girth and bulk, a 16-cm. needle will be at a depth of almost 12 or 13 cm. Careful aspiration for blood and cerebrospinal fluid must be carried out before injection is begun. When the four needles are placed in position, each site should be injected with 20 ml. of 1% procaine containing 1,000 Berger units of hyalase per 100 ml. of solution.

Schneider Method for the Rapid Assay of Plasma Fibrinogen

Schneider (1952a) has devised a method for the rapid assay of plasma fibrinogen. We have used the test extensively, and find it useful in the detection of degrees of fibrinogen

depletion which are insufficient to disturb the coagulation time of whole blood. Essentially, the method consists in making a serial dilution of the patient's blood in a diluent such as Ringer's solution (which will act as a source of calcium ions), adding preformed thrombin, and observing the highest dilution in which a discernible fibrin coagulum appears. For details of the technique the reader is referred to Schneider's original description.

Rapid Fibrinogen Estimation (Shea)

The following is an account of the practical details of this biuret method for fibrinogen estimation.

Reagent:

Sodium potassium tartrate ..	9 g.
CuSO ₄ .5H ₂ O	3 g.
Potassium iodide	5 g.
0.1N NaOH	to 1 litre
Distilled water	to 1.9 litres

Thrombin Solution:

P. D. & Co. "thrombin topical" in 50% glycerol-saline.

Procedure.—Take blood in the Heller-Paul (Wintrobe) oxalate mixture. If possible, secure 3 ml. of plasma. Add 20 units of thrombin per ml. of plasma. Clotting should occur within 5 seconds, and rapidly even at 5° C. Provided that the plasma clots within 60 seconds, upwards of 97% of the fibrinogen will be precipitated in one hour, even if clotting occurs and is allowed to continue at a temperature as low as 5° C. After one hour at 5° C. or 37° C., the clot is spilled out on to a filter paper, or a paper towel (preferable), and the plasma expressed in three or more stages. The clot is washed in distilled water, and the fluid again expressed. The clot is then transferred to a boiling tube containing 0.5 ml. of N NaOH, and dissolved by heating in a boiling-water bath for two to three minutes. Then 9.5 ml. of the biuret reagent is added and the colour developed by heating in a water bath at 30 to 32° C. for 30 minutes. A reagent blank is prepared by the addition of 9.5 ml. of the reagent to 0.5 ml. of N NaOH, with similar incubation at 30° C. for 30 minutes. The optical density of the test is read against the reagent blank in a photo-electric colorimeter at 555 mμ, each tube being at about 30° C. The instrument is calibrated by duplicate estimations by the Kjeldahl method. The reagent described gives a colour which follows Beer's law.

Statistical Notes

The following is a summary of the statistics relating to accidental haemorrhage in Dublin during the 25-year period under review.

1. In that period 288,412 women were delivered in the service of Coombe, National Maternity, and Rotunda hospitals. There were 3,256 cases of accidental haemorrhage (1.1% of all deliveries) and 106 maternal deaths, a case mortality of 3.3%.

2. As an indication of the general trend: in the five-year period 1929 to 1933 inclusive, 42,639 women were delivered, with 369 cases of accidental haemorrhage (0.9%) and 23 deaths (6.2%); in the five-year period 1949-53 inclusive, the number of deliveries had increased to 71,171, with 1.8% of the patients suffering from accidental haemorrhage, and a case mortality of 1.2%. Note the increasing number of deliveries in the service of the three hospitals, representing an advance of 70% between the first and the fifth five-year period. Over 20 years the actual number of cases of accidental haemorrhage treated in the hospitals increased fourfold and the percentage of patients treated for accidental haemorrhage twofold. This does not indicate that accidental haemorrhage has become more common, but that doctors in practice are sending into hospital their ante-partum haemorrhage cases, including the mild ones. Note, too, that mortality in the last period was only one-fifth of that in the first period. From 1939, earlier admission to hospital, more effective replacement of fluid loss and alleviation of shock by glucose and blood rather than by saline, early puncture of the membranes, and the judicious employment of caesarean section all helped to reduce mortality.

3. The 106 maternal deaths which occurred in the 25-year period were studied as carefully as the case records permitted. It was considered that haemorrhage (before delivery, during caesarean section or caesarean hysterectomy, or early in the post-partum period) was responsible for one-half of the deaths; renal failure for one-quarter; and other causes, including eclampsia, puerperal infection, and apoplexy, for the remaining quarter. In a noteworthy proportion of the haemorrhage cases it was stated that the blood which had escaped per vaginam failed to clot or that non-clotting blood was found in the wall of the uterus, in the peritoneal cavity, between the layers of the broad ligament, or behind the peritoneum. Although shock was stated to have occurred in many of the fatal cases, it is obvious, in the light of recent knowledge, that haemorrhage was the most important factor in the causation of this condition.

4. Age in relation to maternal death.—Under 20 years, 1; 20 to 24, 3; 25 to 29, 19; 30 to 34, 27; 35 to 39, 37; 40 and over, 19.

5. Parity in the fatal cases.—Primigravidae, 19; multiparae 2 to 5 inclusive, 28; multiparae 6 to 9 inclusive, 33; tenth and later pregnancy, 26.

6. Fate of the foetus.—3,310 cases yielded 3,256 foetuses. The fate of 3,271 was noted in the records: 1,445 stillbirths and 176 neonatal deaths. The foetal loss, then, was 1,621 in 3,271, or just 50%. The loss in severe accidental haemorrhage was 100%.

Summary

Haemorrhage, renal failure, and foetal asphyxia render accidental haemorrhage a serious or potentially serious condition.

In the severe case of accidental haemorrhage the bleeding is of a special type—that is, due to a fall in blood fibrinogen. The clinical manifestations of this clotting defect are described.

The pathogenesis of anuric renal failure is presented in brief.

A scheme of treatment for the mild, moderate, and severe case of accidental haemorrhage is suggested. In Dublin, over the past two years, the results of this treatment have been most encouraging.

Notes relating to splanchnic block and to the Schneider and Shea tests for fibrinogen deficiency are included.

A summary of the statistics of accidental haemorrhage in Dublin over the 25-year period 1929–53 is presented.

BIBLIOGRAPHY

- Annual Clinical Reports of the Three Dublin Maternity Hospitals*, by various authors: 1929–53.
- Barnett, V. H., and Cussen, C. A. (1954). *British Medical Journal*, 2, 676.
- Barry, A. P., and Geoghegan, F. (1954). *Irish J. med. Sci.*, p. 189.
- Browne, McClure (1954). *Cold Spr. Harb. Symp. quant. Biol.*, 19, 60.
- Browne, O'D. (1952). *J. Obstet. Gynaec. Brit. Emp.*, 59, 153.
- Bull, G. M., Joekas, A. M., and Lowe, K. G. (1949). *Lancet*, 2, 229.
- Dieckmann, W. J. (1936). *Amer. J. Obstet. Gynec.*, 31, 734.
- Feeney, J. K. (1954). *J. Irish med. Ass.*, 35, 310.
- (1955). *Irish J. med. Sci.*, p. 195.
- Franklin, K. J. (1952). *Lancet*, 2, 1141.
- (1955). *Irish J. med. Sci.*, p. 214.
- Hodgkinson, C. P., Margulis, R. R., and Luzadre, J. H. (1954). *J. Amer. med. Ass.*, 154, 557.
- Kellogg, F. S. (1948). *Obstet. Gynec. Surv.*, 3, 746.
- Moore, H. C. (1954). *British Medical Journal*, 2, 277.
- (1955). *Irish J. med. Sci.*, p. 227.
- O'Driscoll, K., and Quinn, B. (1951). *J. Obstet. Gynaec. Brit. Emp.*, 58, 961.
- Page, E. W., Fulton, L. D., and Glendening, M. B. (1951). *Amer. J. Obstet. Gynec.*, 61, 1116.
- Reid, D. E., Weiner, A. E., and Roby, C. G. (1953a). *J. Amer. med. Ass.*, 152, 227.
- (1953b). *Amer. J. Obstet. Gynec.*, 66, 475.
- Schneider, C. L. (1951). *Surg. Gynec. Obstet.*, 92, 27.
- (1952a). *Amer. J. Obstet. Gynec.*, 63, 1078.
- (1952b). *Ibid.*, 64, 141.
- (1955). *Ibid.*, 69, 758.
- Shaw, W. Fletcher (1921). *J. Obstet. Gynaec. Brit. Emp.*, 28, 135.
- Shea, S. M. (1954). *Irish J. med. Sci.*, p. 36.
- (1955). *Ibid.*, p. 233.
- Sheehan, H. L., and Moore, H. C. (1952). *Renal Cortical Necrosis and the Kidney of Concealed Accidental Haemorrhage*. Blackwell, Oxford.
- Sophian, G. J. (1955). *Irish J. med. Sci.*, p. 220.
- (1953). *Toxaemias of Pregnancy*. Butterworth, London.
- Year Book of Obstetrics and Gynaecology* (1954). Edited by J. P. Greenhill. Year Book Publishers, Chicago.

THE NATURE AND SOME HAZARDS OF OBSTETRICS IN GENERAL PRACTICE

BY

K. DOUGLAS SALZMANN, M.D. M.R.C.P.Ed.
D.Obst.R.C.O.G.

*Practitioner in charge of Dellwood General-Practitioner
Maternity Unit, Reading*

Few details have been published of the midwifery of general practitioners. For information concerning its nature and results I analysed the records for seventeen years (1935–51) of a group practice. Although five partners dealt with most of the abnormal cases, altogether eight principals and four assistants were concerned in the obstetrics.

Nature of the Work

Quantity of Midwifery.—During the seventeen years 2,911 confinements were attended (this figure ignores attendances made solely to suture perineal tears)—2,241 were “booked” cases, the patients having engaged us to attend, and 670 were “emergency” ones, which we attended in response to requests from midwives.

Ratio of Booked to Emergency Cases.—Before 1948 two-thirds of the confinements were booked and one-third emergency, but since the financial obstacle to engaging a doctor was removed that year by the National Health Service our booked cases have been doubled and the emergencies decimated. The ratio of booked to emergency cases is now 50 to 1. The total midwifery undertaken now corresponds to 16 confinements per 1,000 patients.

Parity of Cases.—The mothers in the analysis were divided into three classes—primiparae, secundiparae, and “other” multiparae. Ordinarily, secundiparae are included with other multiparae, but, though they may behave similarly, they are not “mothers of many children.” The analysis provided an opportunity of comparing secundiparae with “other” multiparae. The analysis showed that, excluding a few cases in which parity was not recorded, 48.5% of the booked cases and 71.9% of the emergencies were primiparae. Secundiparae (14.0%) and other multiparae (14.1%) contributed equally to the emergency total, but more mothers booked a doctor for their second confinements than for subsequent ones (Table I).

TABLE I

	Primiparae	Secundiparae	Other Multiparae
Booked cases (2,241)	48.5%	34.7%	16.8%
Emergency cases (670)	71.9%	14.0%	14.1%

Parity and Abnormality.—By taking “normal” to mean a spontaneous vertex delivery of a live baby, with no haemorrhage or other intrapartum complication, and recording as “abnormal” all deliveries not satisfying these criteria, it was found that primiparous confinements were abnormal more than twice as often as multiparous ones (Table II). Abnormal labour occurred among the booked patients in

TABLE II.—Percentage of Normals in 2,241 Booked Cases

	Primigravidae	Secundiparae	Other Multiparae
Normal with no perineal tear	49.7%	68.9%	76.9%
Normal but with tear or episiotomy	23.2%	19.2%	11.0%
Total normal	72.9%	88.1%	87.9%