use of the various muscle relaxants, and the severity of many surgical procedures, an automatic breathing device can be of the greatest value during periods of respiratory depression. It allows the anaesthetist both greater mobility and the freedom of his hands to attend to the general care and welfare of his patient.

I wish to thank Dr. H. Duncan Davies, medical superintendent, Morriston Hospital, and the surgical staff, both past and present, for their very kind co-operation during the development of this apparatus; Professor W. W. Mushin, Cardiff, for his invaluable advice and guidance; and Mr. Elwyn Williams, S.R.N., charge nurse to the anaesthetic department, for his most valued assistance. I am also grateful to Mr. A. Charles King, who before his retirement gave such whole-hearted encouragement, and to Mr. A. J. Juby, of Messrs. A. Charles King Ltd., for his practical suggestions and courtesy.

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ACQUIRED AFIBRINOGENAEMIA COMPLICATING PREGNANCY

REPORT OF TWO CASES OF CONCEALED ACCIDENTAL HAEMORRHAGE TREATED BY HUMAN FIBRINOGEN

BY

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Afibrinogenemia is a condition in which the blood is rendered incoagulable owing to the absence of one of the principal components of the normal blood-clotting mechanism. DeLee (1901) first described this condition in pregnancy when he referred to a haemophilia-like phenomenon complicating two of his cases. Williams (1925) reported a case in which bleeding continued for twelve hours after delivery, despite a well-contracted uterus and in the absence of any detectable traumatic lesion. Davis and McGee (1931) cited two fatal cases in which packing of the uterus and clamping of the uterine arteries failed to control haemorrhage. Dieckmann (1936) first suggested the true nature of the condition—a lack of circulating fibrinogen—and reported two cases of his own in which, in addition to uterine bleeding, there was haemorrhage from the gums and stomach and into the subcutaneous tissues. Tagnon *et al.* (1946) described a fatal case of post-partum haemorrhage in which the patient's blood was incoagulable, but clotted when fibrinogen was added to a freshly collected specimen. Weiner *et al.* (1950) reported three cases of defective coagulation mechanism in patients displaying evidence of rhesus incompatibility during their pregnancies.

Coagulation Reaction

The essential reaction in coagulation of the blood is the conversion of the soluble protein fibrinogen into the insoluble protein fibrin by means of the enzyme thrombin. Fibrinogen is a protein of high molecular weight and exists in the circulating blood as such. It is probably formed in the liver, and damage to this organ results in a rapid fall in the plasma fibrinogen concentration. This indicates that the circulating fibrinogen is rapidly destroyed or utilized by the body, but the manner of destruction or of utilization is unknown. Thrombin, an albumin, is not present in the circulating blood, but is formed from its inactive precursor prothrombin when blood is shed. Prothrombin, a globulin, is formed in the liver, and its synthesis is dependent upon an adequate supply of vitamin K. For the activation of prothrombin, free calcium ions and certain organic activators of obscure composition named thromboplastins are necessary. These thromboplastins are derived from damaged tissues, from disintegrating blood platelets, or from the plasma itself. The normal blood-clotting mechanism can be simply outlined thus:

All authorities are agreed that the essential factor in the incoagulability of the blood is a lack of circulating fibrinogen. The aetiology of the afibrinogenemia, however, remains speculative. Willson (1922) suggested that a toxin which he named "haemorrhagin" was responsible for the interference with the normal blood-coagulation mechanism. In 1929 Sakurai succeeded in defibrinating blood in experimental animals by injections of placental extracts, and in 1936 Eley *et al.* confirmed the antihaemophilic activity of these extracts and in addition produced evidence of intravascular coagulation in guinea-pigs and monkeys. Howell (1941) made a valuable contribution when he showed that injections of thromboplastins resulted in depletion of the blood fibrinogen and rendered the blood incoagulable. Furthermore, he suggested that these substances were liberated into the maternal circulation in cases manifesting the haemorrhagic diathesis following abruptio placentae.

Schneider (1947) concluded, following extensive experiments, that thromboplastins were in fact the toxic elements which, when liberated into the blood stream, resulted in afibrinogenemia. Reid (1951) suggested that a fibrolytic enzyme (first referred to by Tagnon *et al.* in 1946) was responsible for the depletion of fibrinogen. He states that this enzyme is normally present in the circulating blood, but that its activity is held in abeyance by an antifibrolytic substance and it is liberated in excessive amounts only in cases of shock or haemorrhage. An antithrombin substance (Ratnoff and Vosburgh, 1952) has also been cited as the cause of the haemorrhagic diathesis; this, however, would appear to be unlikely, as antithrombin does not cause a depletion of fibrinogen—a constant biochemical finding. However, it may well be that the production of antithrombin is of secondary importance in producing defective haemostasis, as suggested by Weiner *et al.* (1953). Liver damage has also been suggested as the underlying cause of the afibrinogenemia, but necropsy examination has failed to show any evidence of hepatic insufficiency.

The Thromboplastin Theory

However, the consensus of opinion undoubtedly favours the "thromboplastin theory," and it would appear from a survey of the literature that it is most acceptable.

Thromboplastins, as stated above, are substances of obscure composition. They are a normal component of all tissues, but some tissues contain a higher concentration than others. The uterus, placenta, lung, and brain are known to contain large quantities.

Reid *et al.* (1953), following up previous experiments by Steiner and Lushbaugh (1951), confirmed that amniotic fluid emboli can occur during pregnancy. Furthermore, they ascertained that amniotic fluid is a rich source of thromboplastins, which it derives normally from foetal exfoliation. In addition, the thromboplastin content of the amniotic fluid is greatly enriched from the decidua.

In pregnancies in which there is premature separation of the placenta (abruptio placentae) it has been shown by Schneider (1952) that rupture of the basal decidua plate occurs, resulting in the liberation of thromboplastins into the maternal circulation.

Weiner *et al.* (1950) reported that afibrinogenaemia may develop in an occasional Rh-negative patient in which the foetus has died and is retained *in utero*. They suggest that in these cases the most likely cause of the incoagulability of the blood is resultant on the death of the foetus; its membranes and the decidua disintegrate and allow the absorption of thromboplastins into the maternal circulation.

To summarize, therefore, the present hypothesis of afibrinogenaemia is as follows: thromboplastins are liberated from the uterus or its contents and gain access to the maternal circulation, where they set in motion the normal blood coagulation mechanism, resulting in extensive intravascular clotting. Consequent on this there is a marked depletion of the circulating fibrinogen, and the liver is unable to make good the deficiency in time to prevent the haemorrhagic diathesis manifesting itself.

It is suggested that a fibrolysin is produced in the maternal blood as a counter-measure to combat the intravascular deposition of fibrin, and that this in turn aggravates the condition rather than alleviating it, by producing fibrin emboli (see below).

Conditions Associated with Afibrinogenaemia

Clinically, so far as is known, afibrinogenaemia may manifest itself in three conditions complicating pregnancy: (1) abruptio placentae; (2) amniotic fluid embolism syndrome (Reid *et al.*, 1953); and (3) rhesus incompatibility resulting in intrauterine death and retention of the foetus *in utero* (Weiner *et al.*, 1950). In these conditions, if depletion of the fibrinogen has taken place, the patient's blood fails to clot, and haemorrhage, uncontrollable by ordinary measures, will take place from any raw surface—that is, placental site or episiotomy incision. This is illustrated by the cases reported here, in which, despite a well-contracted uterus and the absence of any apparent traumatic lesion, uterine bleeding continued unabated and the blood which was shed failed to clot.

Bleeding may even occur from the intact mucous membranes and subperitoneally; these latter are frequently apparent at caesarean sections performed on cases of abruptio placentae, and constitute the classical Couvelaire lesions. According to A. P. Barry and to K. Feeney (personal communications), these internal haemorrhages are one of the chief causes of death in accidental haemorrhage.

The internal haemorrhagic manifestations are explained by (1) the ischaemic necrosis of the small vessels due to fibrin emboli resulting from the action of fibrolytic enzymes on the intravascular deposits of fibrin; and (2) the associated incoagulability of the blood.

The diagnosis of the condition is not difficult in the established case. Purpura haemorrhagica may simulate the condition, but can be distinguished by the fact that the blood-coagulation time is normal and bleeding does not characteristically come from the uterus but rather from lacerations of the cervix, vagina, or perineum. Obvious traumatic causes of haemorrhage must, of course, be excluded by vaginal examination. Rupture of the uterus is more difficult to exclude without laparotomy; but the nature of the labour and delivery will suggest whether or not its possibility should be seriously considered, and any blood issuing from the vagina will be seen to clot in the container. Absolute diagnosis of afibrinogenaemia can

only be made biochemically, a method which will show the depletion or absence of fibrinogen. The condition should always be kept in mind when dealing with cases of abruptio placentae, with retention of a dead foetus *in utero* or with premature rupture of membranes. Potential and established cases can be detected by the "clot observation test" (W. Weiner, 1953, personal communication).

Treatment

The treatment of the condition falls under two headings: prophylaxis and established cases.

Prophylaxis.—This is directed towards early detection and prompt correction of the afibrinogenaemia. In the three varieties of cases already outlined, but chiefly in cases of abruptio placentae—for these are most likely to manifest the haemorrhagic diathesis—the possibility of the complication of incoagulability of the blood must be borne in mind. Blood should be drawn from any suitable vein, and Weiner's "clot observation test" be carried out at hourly intervals. When the fibrinogen concentration of the blood has decreased to a critical level the blood withdrawn from the vein will fail to coagulate; when the fibrinogen level falls to a lesser degree clotting-time will be delayed beyond the normal maximum of ten minutes, and when it does occur the coagulum will fragment and dissolve within one hour when kept at a temperature of 38° C.

Determination of the blood fibrinogen level may be carried out, where facilities are available, when the "clot observation test" indicates a defective clotting mechanism. The normal fibrinogen content of the blood varies between 190 and 330 mg. per ml. The minimal level for haemostasis is considered to be 100 mg. per ml. (Reid, 1951). When the findings suggest a fall or depletion of the fibrinogen, immediate replacement must be made by intravenous administration of human fibrinogen. The amount of replacement required depends upon the clotting behaviour of the blood as displayed by the "clot observation test." In cases of abruptio placentae blood transfusion should also be begun, to counter the shock which will undoubtedly manifest itself owing to the concealed loss of blood (O'Donel Browne, 1952).

Established Cases.—Moloney *et al.* (1949) were the first to treat successfully an established case by combining fibrinogen replacement and blood transfusion. In these cases the patient manifests varying degrees of shock, depending on the amount of internal and external blood loss. Therefore, in addition to correcting the afibrinogenaemia, antishock therapy must be immediately started. Restoration of blood volume is of prime importance, but it must be clearly understood that the condition cannot be corrected by multiple transfusions of stored blood alone. The amount of fibrinogen required in established cases is still debatable; it has, however, been established that between 8 and 12 g. of fibrinogen is present in the blood of a normal person; therefore theoretically in a case of complete defibrinogenaemia an equivalent amount should be given intravenously. Reid (1951) suggested that the initial dose of fibrinogen should be 4 to 6 g., and that repeated injections of 1 g. at half-hourly intervals should be given until the blood-clotting mechanism has returned to normal. Moore (1954) has successfully treated a case of afibrinogenaemia associated with abruptio placentae by the administration of human fibrinogen (2 g.) and transfusions of whole fresh blood. However, in our opinion far smaller doses of fibrinogen are effective, as illustrated in the cases presented, and we agree with Weiner that there is no doubt that fibrinogen is the most effective therapeutic agent in the cure of these cases.

Case 1

A 33-year-old 7-gravida, whose estimated date of delivery was October 25, 1953, had maintained excellent health during her pregnancy except for a moderate degree of pyelitis at the eighth month, which responded to chemotherapy. Her past and family histories were free of any evidence of abnormal bleeding tendency. Her previous

pregnancies were uneventful and the blood loss in the third stage had always been within normal limits.

The patient was admitted to hospital at 2.15 a.m. on October 23 as a case of abruptio placentae. She gave a history of having had continuous abdominal discomfort for five hours and slight vaginal bleeding for two hours before admission. Foetal movements had not been appreciable since the onset of vaginal bleeding. Labour had begun on the way to hospital, and the membranes had ruptured immediately before admission.

On examination her general condition was good, the pulse rate 84 a minute and of good volume, temperature 97.8° F. (36.6° C.), and blood pressure 115/70. There was no oedema or albuminuria. The uterus corresponded in size to the period of gestation; it was tense and tender on palpation, but not markedly so. Foetal parts were difficult to define clearly, but the lie was longitudinal, with the back on the left side and the head engaged in the pelvis. The foetal heart was not heard. There was a slight amount of dark non-clotting blood coming from the vagina; a rectal examination showed the presenting part to be at the level of the ischial spines, and the cervix to be three-quarters dilated. The patient was in labour.

She had a spontaneous delivery of a full-term stillborn male infant at 3 a.m., and the placenta together with 1 pint (570 ml.) of old blood clot was immediately expressed from the uterus. Ergometrine, 0.5 mg., was given intramuscularly and the uterus contracted firmly. There was no perineal tear, nor was episiotomy necessary. The placenta, when examined, displayed all the typical findings associated with accidental haemorrhage. Immediately after delivery the patient's condition was good, pulse rate 92 and blood pressure 120/70.

After the delivery there was a very slight blood loss per vaginam despite a well-contracted uterus; the midwife in charge was not worried, regarding the loss as being within normal limits. At 3.55 a.m. she became anxious owing to the persistence of the slight bleeding, which now amounted to 1 pint (570 ml.). The patient's pulse rate was 105 and thready, the blood pressure was 95/45, and, in addition, her colour was pale and her skin clammy. It was observed that the blood which was shed failed completely to coagulate in the container.

An intravenous drip of "plasmosan" was begun at 4.5 a.m. and arrangements were made for immediate cross-matching of the patient's blood. A speculum examination failed to reveal any apparent traumatic lesion. In view of this and the incoagulability of the blood passed per vaginam, a definite diagnosis of afibrinogenaemia was made. Dr. Weiner, of the Birmingham Transfusion Service, was contacted, and he kindly provided a supply of fibrinogen. At 4.20 a.m. blood was substituted for the plasmosan.

Despite rapid blood transfusion the patient's condition continued to deteriorate, and the vaginal bleeding persisted. At 5.55 a.m. the fourth pint of blood was started, and the apparent blood loss up to that time was measured at 5 pints (2.8 litres). The pulse rate was 115; its volume was weak but was regular; the blood pressure was 90/40.

At 6.5 a.m. 20 ml. of human fibrinogen (404 mg.) was administered intravenously, and the vaginal bleeding ceased at 6.25 a.m. The patient's condition improved rapidly thereafter; in fact, the improvement was more rapid than one would expect from cessation of the external bleeding alone.

From this time the patient's condition continued to improve. A total of 6 pints (3.4 litres) of blood was transfused, and there was no recurrence of the bleeding. The puerperium was apyrexial, the uterus involuted normally, and the patient was allowed out of bed on the second day, being discharged home on the tenth day.

Blood which was collected from the patient during the "bleeding phase" failed to coagulate while under observation for 48 hours, and the pathological report on it was as follows: "Serum prothrombin time, 5 minutes +; tiny ill-formed clot at 100 seconds (normal control, 24 seconds).

Plasma prothrombin time, 30 minutes + (normal control, 14 seconds). Microelectrophoresis of plasma shows absence of fibrinogen bands. Twelve hours later, after administration of the fibrinogen, clotting and bleeding times were normal and platelets were seen in a film to be present, but appeared large in size and reduced in numbers; count, 120,000 per c.mm."

The pathological report on the blood on October 25 was as follows: "Serum prothrombin time, 30 seconds (normal control, 11 seconds); plasma prothrombin time, 11 seconds (normal control, 11 seconds). Therefore prothrombin consumption was within normal limits. Microelectrophoresis of plasma shows fibrinogen band to be present, but in much reduced proportion. Bleeding-time, 6 minutes, clotting-time (Lee and White's method), 6 minutes (normal, 6 to 15 minutes); clot retraction, normal; platelets (film), within normal limits."

Case 2

A 37-year-old 6-gravida whose expected date of delivery was June 19, 1954, had been in good health throughout her present pregnancy, and had been seen antenatally by her general practitioner and the district nurse. Her past and family histories were free of any evidence of abnormal bleeding tendency. All her previous pregnancies and labours had been normal, the last having taken place in 1947.

The patient was admitted on May 1, at the thirty-third week of pregnancy, having a history of accidental antepartum haemorrhage, with considerable external blood loss. She was sent in by her own doctor from an outlying district, and was seen by us at 6 p.m. The following were the findings. Oedema of ankles; albuminuria ++; blood pressure, 120/70; tense tender uterus; foetal parts very difficult to determine with accuracy; vertex presentation; foetal heart inaudible; brisk haemorrhage per vaginam; catheter urine $\frac{1}{2}$ oz. (14 ml.).

6.20 p.m.: Clot observation test demonstrated incoagulability, and subsequent micro-electrophoresis showed absence of fibrinogen bands. 6.30 p.m.: Blood transfusion started. 7.30 p.m.: Fibrinogen, 0.13 g., obtained from Birmingham, was given intravenously. 8 p.m.: Test demonstrated clotting; the membranes ruptured surgically; 2.5 units of "pitocin" injected. 8.30 p.m.: 2.5 units of pitocin injected. 9 p.m.: Labour started; blood pressure, 130/75. 11.45 p.m.: Spontaneous delivery of a dead foetus was followed immediately by delivery of the placenta. There was evidence of abundant retroplacental bleeding having taken place previously.

The immediate post-partum bleeding amounted to 24 oz. (680 ml.), and, despite a well-contracted uterus and absence of trauma, uterine bleeding continued. The blood was obviously incoagulable; blood pressure, 115/60.

May 2.—12.20 a.m.: A further 0.13 g. of fibrinogen was given (second dose). 1 a.m.: Test again showed clot formation. A catheter specimen of urine taken at this time showed marked haematuria. The total amount of catheter urine collected between 6 p.m. of the day of admission (not including the first catheterization) and 1 a.m. was 5 oz. (140 ml.). 1.30 a.m.: Blood loss now much less, but coagulation still entirely absent. 5 pints (2.8 litres) of blood had now been given by transfusion. 2 a.m.: Blood loss slight, but no evidence of coagulation yet seen; blood pressure, 104/60. 2.45 a.m.: 0.13 g. of fibrinogen given (third dose); blood loss still slight, but continuous and without clot formation. 3.15 a.m.: Blood loss ceased entirely. Clot rapidly formed. Six pints (3.4 litres) of blood had now been given by transfusion; blood pressure, 140/90. Total urinary output since 6 p.m. on the day of admission was 8½ oz. (240 ml.).

The subsequent recovery of the patient was uneventful.

Summary and Conclusions

The suggestion that internal haemorrhage might be dependent on a defect in the clotting mechanism of the blood was first made when DeLee described a haemo-

philia-like phenomenon complicating accidental ante-partum haemorrhage. Dieckmann first suggested the true nature of this condition—a lack of circulating fibrinogen.

The essential reactions in blood coagulation are described, and shown in diagrammatic form.

Every case of accidental ante-partum haemorrhage must be suspected of internal haemorrhage, which can prove fatal; and the possibility of a severe post-partum bleeding, uncontrollable by ordinary means due to blood incoagulability, must also be kept in mind.

The clot observation test as described by Weiner in 1953, carried out at hourly intervals, gives clear warning of developing fibrinogen deficiency with all its dangers, and enables treatment to be given at an early stage when the prognosis is good.

The administration of pure human fibrinogen is essential, should severe afibrinogenaemia develop, if life is to be saved. The administration of whole fresh blood, although it contains fibrinogen, is by no means as efficient, since it is not possible to give the fibrinogen it contains quickly enough to prevent the rapid conversion of the latter to fibrin.

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The Universities Federation for Animal Welfare, in association with the Animal Welfare Institute of New York, has appointed Dr. W. M. S. RUSSELL, D.Phil., as Research Fellow to study the history and progress of the introduction of humane techniques into experimentation on animals, with a view to assisting further progress. Examples of progress already achieved are the use of anaesthetics in experiments involving operations, the reduction of the requisite number of animals by means of modern small-sample statistical theory, and in the assay of drugs the substitution of painless (e.g., chemical) criteria for various criteria entailing discomfort.

THE NATURE OF BELL'S PALSY

BY

R. WYBURN-MASON, M.A., M.D.

The anterior primary divisions of the second and third cervical nerve roots divide into two and the adjacent parts of each root join to form the great auricular, lesser occipital, and cutaneous cervical nerves beneath the sternomastoid muscle. The latter appear at the mid-point of the posterior border of the muscle, where they pierce the deep fascia, usually immediately above and behind the external jugular vein, which crosses the posterior border of the muscle at the same point. The great auricular nerve then ascends towards the lobe of the ear, crossing the muscle obliquely, and, as it does so, divides into anterior and posterior branches. The posterior branch joins the posterior auricular branch of the facial nerve behind the ear, communicates with the lesser occipital nerve and the auricular branch of the vagus, and supplies the skin over the mastoid process. The anterior branch unites with the various branches of the facial nerve as they lie on and within the substance of the parotid gland. Other branches supply the lobe of the ear, part of the posterior and lower part of the auricle, and the skin over the angle of the jaw and parotid gland. The great auricular nerve is purely sensory and some of its fibres join the facial nerve and with it pass to all parts of the face. The above facts and the procedure for alcohol block and section of the great auricular nerve have been previously described (Wyburn-Mason, 1953).

Some Causes of Lower-motor-neurone Facial Palsy

A lower-motor-neurone lesion of the facial nerve may be produced in many ways. I am not here considering cases in which the facial palsy is due to an intracranial lesion, to disease or fracture of the petrous bone, or to middle-ear disease, but only those due to certain infratemporal causes. A palsy arising for no apparent cause is usually given the term Bell's palsy, but one with identical features may be produced in a number of ways, of which the following are of interest.

(a) Facial Palsy and Zoster of the Second and Third Cervical Dermatomes

Cases of herpes zoster of C2 and 3 zonal areas, of the trigeminal area, or of so-called "otic zoster" are not infrequently complicated by a facial weakness similar to that occurring in cases of Bell's palsy (see Case 1), which may be transient or permanent. As shown by Lewis (1927), "herpetic and herpetiform eruptions occur as sequels to lesions of the sensory nerve tracts; not only do they follow irritative lesions of the [root] ganglion, but they are produced also by lesions of those tracts distal to the ganglia themselves." Such irritation of nerve roots or their branches may follow mechanical, physical, or chemical trauma, gunshot wounds, x-irradiation, primary or metastatic tumours, or handling of the nerve roots at operation. The irritation causes the passage of antidromic impulses peripherally. These liberate histamine (or H-substance) and possibly acetylcholine (Grant, Pearson, and Comeau, 1936), and they cause not only vasodilatation, but exudation of fluid from the blood vessels, leading to oedema, small haemorrhages, and also the signs of inflammation (see Lewis, 1927). The inflammatory changes are found in the skin and subcutaneous tissues and also in deeper tissues and their contained structures. When C2 and 3 nerve roots are irritated, giving rise to herpes zoster in the affected skin segments, the antidromic impulses pass peripherally in those fibres (among