ACCIDENTAL HAEMORRHAGE

A Review

by

Y. PINTO ROSARIO,* M.D.

Accidental haemorrhage,—abruptio or ablatio placentae, denotes the premature separation of a normally situated placenta and is potentially one of the most dangerous forms of haemorrhage.

History

It was Rigby who in 1776 first differentiated it from the haemorrhage of placenta praevia. Baudeloque in 1790, followed by Goodall in 1875 drew attention to the tremendous maternal complications and mortality which followed this condition. Holmes in 1901 clarified the picture, and about the same time DeLee (1901) made his monumental discovery of the bleeding tendency which is often found in these patients. Wilson in 1922 confirmed this observation, and in 1936 Dieckmann defin-

*Prof. of Obst. & Gynec., Lady Hardinge Medical College, New Delhi.

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ed this condition as a decrease in the blood fibrinogen. Since then much progress has been made specially on the American continent. Weiner et al and Schneider (1958) further worked and improved on the knowledge that a clotting defect occurs in these patients. In 1947 it was Trueta (1947), Sophian (1955) and Sheehan and Moore (1952) who noted their findings on the involvement of the kidney in cases of abruptio placentae.

Incidence

Enormous amount of literature has been published on this subject and the incidence varies according to the severity of the cases included. Hester and Saley, in 1957, reported 1.3% by excluding grade 0 type. Kimbrough (1959), reviewing 383 cases from 1944-57, gives an incidence of 1 in 109 deliveries. While Burger and Gotz (1960) (Weirzberg) in a 10 year study found an incidence of 0.93%, and Douglas et al (1955)

give an incidence of 0.55% in New York. Dyer and McCoughey (1959), again in a 10 year survey, noted an incidence of 0.5%, while Pritchard and Brekhen (1967), from Dallas, studying only severe cases found the incidence to be 0.2%. Hendelman and Fraser (1960) from Montreal, during 1950-57, found an incidence of 0.48%, while Perlin and Stewart (1963) reported 0.7% from Halifax. Potter (1960) found 283 cases among 54,286 deliveries. In the United Kingdom, Hibbard and Jeffcoate (1966) studied the occurrence of abruptio at Millroad Maternity Hospital, Liverpool, from 1952 to 1964 and found the incidence to be 1.16% of all deliveries and 31% of all antepartum haemorrhages. Paintin in 1962 noted an incidence of 0.7% in 30,383 deliveries in Aberdeen, while Brown (1952) reported the same figure of 0.7% from Dublin, though he took only the severe cases.

In Europe, from Helsinki, Kotsalo in 1958 gave an incidence of 0.58%, while Rouchy et al (1965) found 150 cases in 27,447 deliveries from 1948-63. Figures from the Orient also vary. Thambu and Llewellyn Jones (1966) gave an incidence of 1.9% in Malaysia and Hsu et al (1960) found in Taipei Hospital an incidence of 2.09% of total deliveries and in Taipei city only 0.16%. In India, Naidu et al (1961), from Hyderabad, reported an incidence of 0.56%, closely resembling Poddar's (1960-61) incidence of 0.59% from Calcutta. Parikh and Masani from Bombay give the incidence as 1 in 90, Dass and Vohra (1961), from Delhi, as 1 in 132, while Krishna Menon gives a higher incidence of 1 in 55 deliveries. In 1968,

Ashar and Purandare (1968) reported an incidence of 0.86% from Bombay.

Aetiology

The etiology of this interesting condition is not yet known but several causative factors have been mentioned. No contributory cause, not even trauma, could be found in a 10 year survey by Dyer and MaCaughey (1959). Toxaemia was implicated from the time Chantreuil originally noticed albuminuria in patients with abruptio placentae. Gaifami (1959) found albuminuria in 80% and Potter (1960) stated that 93.3% were associated with toxaemia, while Dieckmann (1936) found 63% in his series and Munro Kerr (1954) 30%. Bartholomew et al (1949) could correlate toxaemia with abruptio in 52.5%, Goethal (1928) with 24.2%, Hester and Saley (1963) with 45% and Torup and Weilandt (1960) with 31.2%, with no relation between the degree of separation and severity of toxaemia. Bewis (1958), deCunha (1959), Douglas et al (1955), and Hendlman and Fraser (1960) found a 25% correlation. Eastman in 1956 found a 47% association with toxaemia and commented that the signs of toxaemia preceded the blood loss.

While most authorities are willing to concede that there is a correlation, their figures fall far short of those given by Eastman in 1956. Hibbard and Jeffcoate (1966) found only 6.12% and 5.14% cases of abruptio placentae associated with pre-eclampsia and chronic hypertension respectively, while 1 in 54 cases of eclampsia had abruptio. Eastman in his most recent edition agrees with

Hibbard's (1964) findings and noted at King's Country Hospital that abruptio was complicated by pre-eclampsia and chronic hypertension in 6.4% and 9.2% respectively. The modern tendency is to view the hypertension and albuminuria as a protective reaction following accidental haemorrhage. Hendelman and Fraser (1960) found a low incidence of 11% associated with toxaemia and this is in close agreement with Haynes' (1966) figure of 12.6%. Kimbrough (1959) found only 8.8%, and Macafee's combined figure of 12.5% is in agreement with Hibbard and Hibbard (1963), who found abruptio and pre-eclampsia together in 7.5% and with essential hypertension in 4.9%. Waddington (1957) reports 16.5% which is three times his hospital incidence.

In India, Krishna Menon (1961) noted in Madras an association in 22% with toxaemia, while Parikh and Masani (1960-61), that of 31.6% in Bombay, a figure closely agreeing with 31% of Dass and Vohra (1961) from Delhi. Poddar (1960-61), in Calcutta, noted a correlation of 42.1% and Naidu et al found, however, a very high figure of 62% associated with toxaemia in Hyderabad.

Trauma seems to play a very small part, only 1% according to Grieg (1951), and 4.7% according to Poddar (1960-61). Short cord and traction also play an insignificant role. Gough (1959) points out that with hydramnios there is a risk of abruptio placentae after membranes rupture and, when surgical induction of labour is indicated, advises abdominal paracentesis or hind-water rupture preferably to fore-water rupture.

The association of circumvallate placenta and accidental haemorrhage was studied by Scott in 1960. He called it placenta extrachorialis and gave an incidence of 18% with haemorrhage, mostly revealed in 47%.

Age and Parity

Paintin (1962) noted that accidental haemorrhage may result from external version or coitus. He did not find any correlation with increasing age but with parity. Age according to Eastman (1966), unlike that in placenta praevia, does not play a significant role, though Hibbard and Jeffcoate (1966) noted that while the incidence rose with age, being 0.79% in those under 20 years and 1.84% in those over 35 years, it was parity that was more important. Robinson in 1930 stated that it commonly attacked old multiparae.

Kotsalo (1958), however, disagrees diametrically and found that accidental haemorrhage has no relation with parity but increases with age, being twice as frequent after 30 years and 4 times as frequent in twin pregnancy.

The relationship to parity was noted in 1934 by Solomons who found 130 out of 140 patients with accidental haemorrhage to be "dangerous multiparas". Gordon in 1921 and George and Power in 1949 also found abruptio placentae more in multipara who were para 5 or more and Bieber (1963) found in his series that 28.5% were gravida 6 or more. Hibbard and Hibbard (1963) found that the incidence of this condition rose with increasing parity and was 3 times more common in women who were para 5 or more as in primipara, while

Fuchs and Peretz (1961) noted that it was 9 times and Chientienshu (1960) that it was 10 times as common in grand multiparas. In Hendelman's (1960) series 62.7% occurred in multiparas of which 10% were grand multiparas. Potter (1960) noted that 232 out of 283 cases of abruptio occurred in multiparas, and Kerr (1964) and Quinlivan (1964) also substantiated this claim. In India, Ashar and Purandare (1968) showed that 86.6% occurred in multipara, 28.3% being para 6 and above, the hospital incidence being 17%. Krishna Menon (1961) found that 228 out of 450 cases occurred in gravida 5 and over, the ratio of primipara to multipara being 1:9.7. Parikh and Masani (1960-61) also noted this relation with multipara and Poddar (1960-61) found accidental haemorrhage in only 10.9% of primipara.

Contrary to this Miller in 1954 found an incidence of 2.1% and Schram (1954) in the same year also noted only a 2.39% correlation with multiparity. Neither did Kerb (1956) or Ziel (1962) substantiate this theory of multiparity.

Paintin (1962), however, not only found that multiparity played a significant part but also the low socio-economic status; and Martin et al (1957) found an increased incidence of abruptio with low levels of vitamin C in the last trimester. Hibbard and Hibbard (1963) also noticed the poor nutrition and the low social class of these patients. The above authors found an association between anaemia, megaloblastic erythropoiesis and abruptio placentae and are in agreement with Hourihane et al (1960). They found that 72 out of 73 had megaloblastic erythropoiesis and said that there was relationship between abruptio placentae and folic acid deficiency though they do not deny that both conditions may have a common aetiological factor. Hibbard and Jeffcoate (1966) found abnormal morphology in the bone marrow in 63% of their cases. One hundered and sixty-one cases of abruption were studied for foliate status and 97.5% showed folic acid deficiency. Also 10% of women with folic acid deficiency later developed abruption. They assumed that the foetus needs more than the mother and any defect in the folic acid metabolism will cause foetal abnormality, abortion or abruption. Coyle and Geoghegan (1962) reported that 35 of the 77 cases of severe abruptio placentae had megaloblastic bone marrow and the majority came from the low socio-economic class.

Thambu and Llewellyn Jones (1966) contradicted this finding and reported in Malaysia that only 35% of their cases with abruption had megaloblastic erythropoiesis and this compared favourably with their control cases and hence they concluded that there was no increased incidence of megaloblastic erythropoiesis with abruption and that if malnutrition is the · primary cause of abruptio placenta then it was due to multiple nutritional deficiencies rather than due to specific deficiency of folic acid.

Krishna Menon et al (1961), studying folic acid status in accidental haemorrhage, found serum folic acid to be less than 3 mμg per ml in very few cases in their series. Bone marrow studies on 112 cases of accidental haemorrhage showed megaloblastic

changes in only 10.7% against 64% noted by Hibbard (1964). Krishna Menon 1961) concluded that there was no increased incidence of folic acid deficiency or megaloblastic erythropoesis in abruptio placentae and that pregnant women with megaloblastic anaemia and folic acid deficiency are not more vulnerable to accidental haemorrhage.

Pathology

The basic pathology is the infusion of blood into the basalis, either from a red infarct or from changes in the wall of the uterine arterioles. This splits the decidua and separates the placenta off its bed and destroys its function. The degree of haemorrhage and separation finally decides the ultimate picture of tetany, shock and the maternal and foetal prognosis. Poddar (1960-61) found that the size of the clot varied from 8 oz in 28 cases to over 1 lb. in 10 cases, and histological examination showed crowding of villi, prominent syncytial masses with either patchy or massive fibrin deposition and areas of infarction.

Kiss and Tarjan (1960), from Budapest, found a subperitoneal plexus which plays an important part in the blood supply to the gravid uterus and in detachment of the placenta. In 90% of all abruptio placentae blood was found in the peritoneal cavity.

Clinical Features

Time of Occurrance of Abruption: While Kimbrough (1959) found that 3/5 of the separation occurred during labour and therefore did not constitute the greatest hazard, others do not subscribe to this view. Separation occurred in the majority prior to 36 weeks, as it did in 54.7% in Hibbard's (1964) series and 66% and 70.3% in Krishna Menon's and Poddar's (1960-61) series respectively. This has a tremendous bearing on the perinatal mortality.

The clinical picture is sudden abdominal pain, either localised or generalised, often associated with vaginal bleeding and sometimes followed by uterine tetany, foetal death and maternal shock. Kotsalo (1958), grading his cases by the severity, found 10% were severe, 30% moderate and 60% mild. Using Page's classification Krishna Menon (1961) found that in grades II and III the incidence of toxaemia was only in 26.8% and suggests that the severe type of accidental haemorrhage is common even in the absence of hypertensive toxaemia. Dyer and Mc-Caughey (1959), in 204 patients, found vaginal bleeding in 168, abdominal pain in 103, a tetanic uterus in 52 and albuminuria in 66, while 35 patients had none of these signs on admission.

The older concept that shock in abruptio placentae is out of proportion to blood loss even with a prior estimate of blood in the haematoma has been challenged recently. Weiner et al found the blood clot volume to be only 40% of the blood lost and that blood extravasates into the broad ligament and retroperitoneum cannot be measured exactly, while Eastman (1956) states that the shock is in direct relationship to the over-all blood loss. Reid et al (1953) comments on the shock not being obstetric but haemorrhagic and that 1/3 of

the blood volume has been lost by the time the blood pressure begins to fall. In Poddar's (1960-61) series shock was present in 15.6% which compares favourably with Kotsalo's (1958) figure of 17.5%. The latter also noticed that in 39 the bleeding was concealed. In Torup and Weilandt's (1960) series shock was present in 5%. Gibberd (1948) and Love (1962) noted that even in severe shock the pulse is slow.

Madan et al (1967), studying blood volume in accidental haemorrhage using Evan's Blue, found a drop of 25% compared to normal pregnant women. When the loss was 20 ml. per kg. or more severe shock occurred, and renal failure when the loss was over 50% of the original blood volume. Tovey and Lennon (1963) also noticed that a loss above 40 ml/kg. indicated renal failure and 60 ml/kg. a critical state.

Recently Baker and Dewhurst (1963) have drawn attention to the classical picture of abruptio placentae with pain, shock and uterine tetany in the mid-trimester and suggest that these cases be treated as abruption, not abortion. Couvelaire uterus, caused by the extravasation of blood into the uterine musculature and beneath the peritoneum into the connective tissue of broad ligament and ovaries and giving the entire uterus a bluish purple look, is seen in severe degrees of abruption. It was first described by Couvelaire (1912). The dissociation of the fibres interferes with contraction and gives rise to grave postpartum haemorrhage, as in 2 cases in Dyer and McCaughey's (1959) series.

Eastman (1966) noted that signi-ficant utero-placental apoplexy was seen only in 5% and this is in keeping with Kaltreider's figure of 2.3%. Krishna Menon (1961) found 22 cases in his series of 450 and Dyer and McCaughey (1959), in 41 (20%). Seventeen uteri had to be removed in their series for this condition and 2 due to atony and severe post-partum haemorrhage after vaginal delivery. Douglas et al (1955) found the incidence of Couvelaire uterus as 7.9%.

In view of the modern knowledge it seems likely that incoagulability of the blood is an important aetiological factor in the causation of utero-placental apoplexy. Brown (1952) and Douglas (1955) theorise that the patient must have a phase of hypofibrinogenaemia to allow blood to infiltrate through the musculature. According to Nötling et al (1960), haemorrhage which has its origin in incoagulability may be the result not of retroplacental haemorrhage but of insufficient blood supply during the bleeding stage and which may not cause external but sufficient internal haemorrhage. Similarly, with oliguria and anuria which are the ultimate results of insufficient blood supply to the kidneys.

Diagnosis

In a typical case of abruptio placenta there is shock with a variable amount of bleeding, a tightly contracted uterus and board-like rigidity, generalised or localised uterine tenderness, with or without the absence of foetal heart sounds—the latter depending on the amount of placenta separated. The amniotic fluid may be blood-stained. In con-

cealed haemorrhage there is no vaginal bleeding, but tenderness and uterine rigidity are more. This rigidity, with the uterus not relaxing properly, is the one salient dependable feature and can be found in both severe and in mild cases. Poddar found the severe variety in 49 out of 64 cases, with concealed haemorrhage in 11% and shock in 15.6%. In milder cases the diagnosis becomes difficult and can only be made by a process of exclusion. Not all cases complain of pain. In the series of Hendelman *et al* (1960), of 126 cases studied, 58 came in labour with absence of pain in 32% and slight bleeding only in the majority. Dyer and McCaughey (1956) found vaginal bleeding in 168 out of 214 cases, while pain was present only in 103 and a tetanic uterus in 52. No signs and symptoms were present in 35 cases. Persistent third trimester bleeding incriminates placenta praevia and often the diagnosis is not made till after delivery. Acute hdyramnios of sudden onset with distension and rapid pulse and inability to outline foetal parts makes diagnosis difficult. Diagnosis then depends on the tenderness of the uterus, its board-like rigidity and shock.

Prognosis

The prognosis depends upon various factors, the chief being the degree of separation which is reflected on the clinical picture and is characterised by shock, uterine tetany, absent foetal heart sounds, on the degree of blood loss and whether it is revealed or concealed, the utero-placental apoplexy and associated hypertension, as these latter factors increase the maternal hazard. The next factor is the availability of fresh blood; 28% in Parikh and Masani's (1960-61) series and 56.2% in Poddar's series required transfusion. The interval between separation and beginning of treatment and emptying of the uterus play a significant part. In Hayne's (1966) series delivery was accomplished in 12 hours in 87% and oxytocin was employed in 20.4%. The longer the placenta and haemorrhagic fluid are retained the greater the chances of coagulation failure and renal involvement. In Krishna Menon's series of 450 cases, in 99 (22%) there was coagulation defect and in 81 of these the delivery occurred after 8 hours.

The chief complications are coagulation and renal failure. Coagulation failure was first recognised by DeLee (1901) over 60 years ago and for many years it was assumed that postpartum haemorrhage which often followed a major degree of abruptio was due to uterine atony as the uterus was disorganised and not capable of firm retraction and hence the older practice of removing the Couvelaire uterus. Weiner, *et al* have shown that there is an 'acute defibrination syndrome' with depletion of circulating fibrinogen and even afibrinogenaemia in cases of severe accidental haemorrhage. Beller *et al* (1961) distinguish between a quantitative deficiency (hypofibrinogenaemia) and a qualitative change (dysfibrinogenemia) and suggest that it is fibrinogenolysis rather than defibrination which is the primary cause of hypofibrinogenaemia and hence very difficult to treat.

According to Schneider (1958), thromboplastins from the placental site enter the maternal circulation because of increased intrauterine pressure and start intravenous clotting, causing hypofibrinogenaemia and hence bleeding starts from various sites. Further fibrinolysins are formed which dissolve any clot. Pritchard and Wright (1959), studying the pathogenesis of hypofibrinogenaemia in 7 cases of total abruption, noticed that 2/3 of the fibrinogen was found in the clot as fibrin and concluded that continued haemorrhage was the primary contributing factor, i.e. haemorrhage with coagulation and deposition of fibrin at the placental site and progressive hypofibrinogenaemia. Adas et al (1959) noted that the condition increased with non-operative management as the increasing intrauterine pressure continued forcing the thromboplastin into the circulation. According to them clot observation tests every hour till the patient delivered and 4 hourly postpartum for 24 hours were strongly advised, and should the delivery not occur in 4 hours then a caesarean section be done.

Dyer and McCaughey (1959) found that 7.4% developed afibrinogenaemia in their series, but Kimbrough (1959) only found 4 patients in 5 years and Poddar (1960-61), only 3 out of their 64 cases. In Krishna Menon's (1961) series, (99) 22% developed coagulation failure, with none occurring in grade I cases. Forty-three had fibrinogen levels below 100 mg.% and 6 had afibrinogenaemia; 20% with coagulation failure were associated with toxaemia. Eastman (1956) found hypo-

fibrinogenaemia in 10% of cases with abruptio placentae, but Pritchard and Biekhan (1967), studying only severe cases in which the foetus was dead, noted that 35% had levels less than 150 mg./100 ml. and 28% less than 100 mg/100. Hsu et al (1960), in Taipei, give a very high figure of 62.5% developing afibrinogenaemia.

Renal failure is sometimes found with very severe forms of abruptio, though oliguria may often be seen. The exact pathology is not known but infiltration of blood into the uterus associated with concealed haemorrhage damages muscle tissue with increasing shock and decreasing the blood flow to the kidneys. Eastman (1956) found 1% acute renal failure in abruptio placentae and Krishna Menon (1961) 6.3%, but established anuria with no urinary output for 24 hours was found in 25 (5.5%), with 10 belonging to grade II and 25 to grade III and 15 being associated with coagulation failure. Except for 2 patients, no case developed anuria when the bleeding delivery interval was less than 10 hours. There were no cases of renal failure in Poddar's series.

Treatment

While the treatment for placenta praevia with caesarean section is tending to increase, the figures for the treatment of abruptio placentae by caesarean section are far lower than a generation ago. Over the years there is a swing of the pendulum from the radical to the conservative, back to radical and now conservative again. The modern trend is to replace blood loss, estimate fibrinogen levels with the clot observation test and re-

place it if it is low, undertake no obstetrical management except an artificial rupture of membranes until blood has been replaced and shock treated, and to deliver the patient within 6 to 8 hours, and if this has not occurred or is not imminent, abdominal delivery to be undertaken. Vaginal delivery, if possible, is the better approach as in the majority the conservative measures produce better results. The indication for caesarean section seems to be more when the case is complicated with severe toxaemia or with a non-dilated cervix and severe bleeding, or when artificial rupture of membranes does not lead to delivery within 6 hours. Recently, however, several English and American obstetricians have advised caesarean section for concealed accidental haemorrhage with the idea of preventing irreversible shock and coagulation and renal failure.

Macafee (1963), after controlling shock with blood transfusion, advises caesarean section after eight hours if the patient is not in labour and so does Crichton (1950), after a few hours of conservative treatment; while Eastman (1956) also advises it after control of shock. Douglas et al (1955) sectioned 40% of their cases and Kotsalo (1958), 33.5% in severe cases with the cervix closed, regardless of the foetal condition. Donald (1964) advises it if the cervix is closed and the foetus alive or in cases not responding to transfusion. Artificial rupture of membranes and the giving of oxytocics are controversial as there are authorities who believe that this precipitates coagulation failure and throws thromboplas-

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tins into the blood. Others believe that early resort to artificial rupture of membranes before the uterine tone has returned increases the internal haemorrhage. However, most authorities feel that artificial rupture of the membranes relieves the intrauterine pressure and expedites the delivery and hence prevents coagulation failure. Adam et al (1959) found with oxytocin that they could reduce their incidence of caesarean section from 50% in 1953 to 6% during 1955-57 and conversely, the incidence of intravenous oxytocic rose from 12% to 38%. Spontaneous labour also was more common in 1955-57 because of early artificial rupture of membranes. Dyer and McCaughey (1959), out of 214 cases, delivered 133 vaginally, while 23 had hysterectomies. Hendelman and Fraser from 1950-57 used artificial rupture of membranes and blood replacement, only 6 patients requiring pitocin, and a caesarean section rate of 15%, while Potter (1960) had a section rate of only 2.5%.

The trend in previous years was to do caesarean hysterectomies especially with the Couvelaire uterus, but in recent years this has been replaced by a conservative approach and now only caesarean section is being done unless the uterine contractility cannot be obtained, which is fortunately rare. Parikh and Masani (1960-61) found only 4 cases with caesarean hysterectomy in 190 cases, and Naidu et al (1961) had 18 caesarean sections and 1 caesarean hysterectomy in 161 cases, while Krishna Menon (1961) recorded 23 caesarean sections and 3 caesarean hysterectomies in 450 cases giving a caesarean sec-

tion rate of 5.7%. Potter (1960) studied 283 cases of which 100 were moderate and 102 severe; labour was stimulated in 172 out of 202 moderate and severe cases with artificial rupture of membranes and pitocin, while caesarean section was done only in 7. Nötling *et al* (1960) had a caesarean section rate of 8% and they advised uterine exploration to remove all clots and get good retraction. Haynes' (1966) caesarean section rate was higher, 16.4%, with maternal indications in 28 of the 33 cases done. Huber (1966) noted that caesarean section should be done only for maternal indications and not to salvage a distressed and damaged baby. Because of the threat of coagulation and renal failure, many clinics treat the severe type primarily with caesarean section if the patient is not in labour. The pathos of accidental haemorrhage is that with severe cases the foetal loss is nearly 100% and in milder cases, 20-50%.

Maternal Mortality

Maternal mortality all over the world is dropping and according to Eastman (1956), with adequate treatment and blood transfusions the maternal mortality should be nil. Kimbrough (1959) had 1 maternal death in 383 cases and Dyer and Mc-Caughey (1959), in a series of 214, had 6 maternal deaths earlier in their study, with 66% being due to renal failure, while after 1954 with a change in treatment and early delivery there were no deaths. Tennent (1959), treating 162 cases with hypertension and accidental haemorrhage, had a mortality of 6.9% in the first 3 years and after the introduction of bromethol per rectum it dropped to 0.96%. There were no maternal deaths in Townsend's series in Australia, nor in 126 cases in Hendelman and Fraser's (1960) series, nor any in the 211 cases reported in the Scandinavian countries by Torup and Wielandt, nor in Nötling's (1960) series. Nilsen (1958), from Oslo, recorded 2.5% maternal mortality from 1932-56 and 0% more recently, and by raising the incidence of caesarean section from 3.6% to 7.7%, lowered the mortality figures which were 14 times higher than the general incidence.

In India, Krishna Menon from Madras reports 20 (4.4%) deaths in 450 cases, with only 1 in grade I and 19 in grades II and III. His corrected mortality is 4%; 75% of the deaths were para 5 and over and 75% when abruption had occurred prior to 36 weeks. In Calcutta, Poddar (1960-61) reports a maternal mortality of 6.2%, while Dass and Vohra (1961), 2.7% in Delhi, and in Bombay Parikh and Masani (1960-61) give a mortality rate of 2.1%. Naidu *et al* (1961), from Hyderabad, report a mortality of 4.4%. The lowest Indian figures are 1.66% reported from Bombay by Ashar and Purandare (1968).

The advantage of section over conservative measures is yet to be seen. Browne (1952) had a mortality rate of 6.4% with artificial rupture of membranes and oxytocin and 31% with caesarean section, but it must be remembered that sections are usually done on very ill patients. Recently, in 20 more patients operated on before the onset of shock, a

mortality rate of 10% was obtained. Douglas et al (1955) reported 164 caesarean sections in 398 cases. Their mortality rate was 1.7%, while Kotsalo's (1958) caesarean section rate was 33.5% and his mortality rate, 3%.

Perinatal Mortality

Perinatal mortality studies reveal that antepartum haemorrhage is an important contributory factor and whereas recently, with the expectant line of management, blood transfusions and pediatric care, the loss due to placenta praevia has been reduced, that due to accidental haemorrhage still remains high, and hence it is one of the most tragic incidences that occur in pregnancy. Perinatal mortality varies from 20% to 100% depending upon the severity of the condition. In Burger's (1960) series foetal loss was 82%, but 23% were dead on admission, while from Australia, Beischer gives a perinatal mortality of 885% and Potter (1960), of 65%. Kimbrough's (1959) figure of 24.6% compares favourably with 31% of Hendelman and Fraser (1960). Torup and Wielandt (1960) reported a loss of 48.5% which closely resembles Kotsalo's (1959) figure of 45%. Viller (1959), from South Africa, recorded a foetal loss of 61.8%. Indian figures vary—Naidu et al (1961) had a perinatal loss of 56.6%, Poddar (1960-61), of 59.3%, Dass and Vohra (1961), of 68% and Parikh and Masani (1960-61), 82.6%, Ashar and Purandare (1968) salvaged only 22.2%.

Perinatal loss is heavily weighed down by problems involving prematurity, toxaemia and severity of the accidental haemorrhage and mode of delivery. Bieber (1963) stated that the foetal mortality was directly proportional to the degree of separation of the placenta, the highest being when the separation was 70% or more. Fortunately, in his series, in 50% of cases there was less than 45% separation, and amazingly 4 live babies were born with 100% premature separation; in these cases the baby and placenta were born together, with very little foetal anoxia. With minor separation, Studdiford (1953) reported 28.3% foetal mortality, mostly due to prematurity, and with major degrees it was 100%. Krishna Menon (1961), from Madras, got a foetal loss of 87% in grades II and III, but 60% were dead on arrival. In grade I the mortality was 31.6%. Torup and Weilandt (1960) graded foetal loss with the type:—grade I having a loss of 30.5% and severe types 70%. Thambu and Llewellyn Jones (1966) correlated the mortality with the severity of haemorrhage; with mild haemorrhage the corrected foetal loss was 8.7% and with moderate loss and mature babies it was 73.5%. Bevis (1958), reporting on the relationship of foetal loss and toxaemia, found 22% loss in the non-toxaemia group and 67% loss with pre-eclampsia; while with pre-eclampsia, Brown found a loss of 80% and Daro et al (1956), 68%. Thambu and Llewellyn Jones (1966), however, disagree with the above authors and found that toxamia has no effect on foetal loss.

On the effect of prior antenatal care there seems to be a divergence of opinion. One would assume that with antenatal care and control of

severe toxaemias the incidence of placental separation would fall with a resultant reduction in foetal loss. This view is subscribed to by Naidu *et al* (1961) and Parikh and Masani (1960-61) who noted that 58.4% were emergency cases. However, Bevis (1958), da Cunha (1959), and Kerr (1964), all found the same foetal loss in booked and in unbooked cases, while Brown reported that the overall maternal and foetal loss had not improved with antenatal care. However, it cannot be denied that antenatal care has an important place in the control of toxaemia.

Foetal loss is also related to onset of labour. Brown found 90% of foetal deaths occurred when the mother was not in labour on admission. Review of literature has provided a wealth of information on the type of treatment. While conservative treatment with artificial rupture of membranes and pitocin seems to give good results, some obstetricians advise liberal use of caesarean section in the interest of both the mother and the foetus. A comparative study of foetal loss in vaginal delivery and by caesarean section in Dyer and McCaughey's (1959) series show that foetal salvage was 49% by vaginal delivery and 21% by caesarean section. Haynes (1966) found a perinatal mortality of 46.8%—44.2% after vaginal delivery and 66.6% after caesarean section.

The protagonists of caesarean section, like Verschoof (1960), had a significant lower foetal mortality with active treatment and they recommend it both in the interest of the mother and the foetus.

Prencel *et al* found an uncorrected foetal salvage of 71%. In the moderate group 89% of all infants delivered vaginally were saved and the salvage rate of those born by section was 100%, while in severe cases only 50% of infants delivered vaginally lived, whereas 100% of those delivered by caesarean section lived. Hernandez (1966), from Venezuela, also found foetal mortality after vaginal delivery as 66.3% and after caesarean section as 3.5%. Nötling *et al* (1960) found a perinatal mortality of 76% after vaginal delivery and none after caesarean section. Kotsalo (1958) had a foetal mortality rate of 36% with caesarean section and 63% with vaginal delivery. Douglas *et al* (1955) pointed out that if the section was done within 3 hours of premature separation there was a good chance of foetal survival if the baby was mature. Bysshe (1957) reduced foetal mortality from 35% to 23.6% by increasing his caesarean section rate from 12% to 30%. Page *et al* (1954) also subscribe to the above view. Daro *et al* (1956) found a similarity in the foetal survival in both groups—75.7% by the expectant line and 77.8% in the group treated by caesarean section. Perlin and Stewart (1968) noted that the increased caesarean section rate in the latter years did not reveal an increased foetal salvage.

Accidental haemorrhage is, therefore, a condition obscure in its aetiology, dangerous in its effect on the mother and hazardous for the foetus. The critical and progressive nature of the condition claims the highest clinical judgement on the part of the

obstetrician. The to and fro swing in the line of treatment shows that most obstetricians are not satisfied with their results, particularly in the field of foetal salvage, and aspire to bring down mortality, both maternal and perinatal, to lower levels.

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