HAEMORRHAGES DURING THE LAST TRIMESTER OF PREGNANCY

by

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In the modern practice of Obstetrics, the major causes of maternal death are haemorrhages and toxaemia of pregnancy, comprising 42% of the total deaths. Advances in the antibiotic therapy have reduced the dangers of pelvic infection and given it a place next to the above two conditions in the list of the causes of maternal deaths. Inadequate ante-natal supervision is often the responsible factor for the avoidable deaths in the series. Older multiparas who have borne more than four children should receive special attention during the ante-natal supervision in order to avoid haemorrhagic complications in the ante-partum and post-partum periods. Most of the causes of post-partum haemorrhages are avoidable. Among the ante-partum haemorrhages, exluding the one due to placenta praevia with its unavoidable haemorrhage, the other important cause is the accidental haemorrhage which can to some extent be prevented by diligent ante-natal supervision and properly planned out treatment, as in the case of toxaemia of pregnancy. The proper preventive measures directed towards both these complications of pregnancy will contribute towards the reduction in the incidence of premature births and indirectly help to lower the perinatal mortality. In spite of the careful management by a well-organised ante-natal supervision, it is noted that over half the cases of accidental haemorrhages or abruptio placentae are admitted to the hospital with the foetal heart sounds absent. By the time patients deliver, 73% of the foetuses are lost and the total perinatal loss by the time the patients are discharged from the hospital is in the vicinity of 77.5%. Maternal loss, though reduced, continues to be in the vicinity of one per cent, with associated high morbidity created by the dangerous complications like oliguria, hypofibrinogenemia, anuria and puerperal pyrexia. Good many of these complications are preventable. However, the main incidence of accidental haemorrhage, which depends solely on uncertain factors as essential hypertension or immunological allergic reactions, as will be put forward by this paper, and not on pure toxaemia of pregnancy,

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is well beyond the control of antenatal supervision. It will continue to occur till more appropriate measures are discovered among hypotensive drugs or in the anti-allergic immunological substances. Placenta praevia with its unavoidable haemorrhage continues to take the toll of 2% of the mothers and one-third of the foetuses. The reorientation in the management of such cases by free use of caeserean section, and at the same time conservative management of the remaining cases, has improved the prognosis of the mother as well as that of foetus. The other clinically important haemorrhagic complication in the antepartum period is rupture of the uterus, the occurrence of which depends upon improper obstetric acumen and judgment in cases of cephalopelvic disproportion, neglect of the women in labour and injudicious use of forceps or pitocin or other oxytocic drugs and previous caesarean scar. Easy availability of expert operative facilities, blood transfusion and other anti-shock measures have no doubt improved the results to some extent. However, the gravity of the condition still persists and continues to keep up the high mortality of the foetus and the slightly reduced mortality and morbidity in the mother. The incidence of postpartum haemorrhage is greatly reduced by avoidance of uterine inertia through proper management of women in labour, prophylactic use of pitocin drip and the post-partum use of the more potent oxytocic drugs like methergin, pitocin, syntosinon etc.

TABLE I

Results in Ante-Partum Haemorrhage Cases
Total No. of Confinements: 80,419

A. P. H.	1028 (1.25%)	P. P. H.	Mortality		Preventable Foetal Loss	
			Maternal	Foetal	S.B.	Perinatal
Accidental haemorrhage	518 (0.63%)	40 (7.7%)	3 (0.57%)	407 (79.5%)	81/221 (36.6%)	108/221 (48.9%)
Placenta praevia ..	400 (0.49%)	33 (8.2%)	7 (1.75%)	140 (35.5%)	60.634 (17.9%)	74/334 (22.4%)
Unclassified	110 (0.13%)		NIL	33 (30.0%)		

Maternal Mortality in 80,419 was 0.34% (uncorrected).
Still-Birth Rate—29 per thousand.
Neonatal Death Rate—24 per thousand.
Perinatal Death Rate—53 per thousand.

The present paper deals with antepartum haemorrhages and is based on the study of 80,419 confinements that took place in Nowrosji Wadia Maternity Hospital, Bombay, India, during the period of 7 years (1956 to 1962 inclusive). During this period the overall maternal mortality was 0.34% in 80,419 deliveries with still-birth rate of 29 per thousand, neonatal death-rate of 24 per thousand and

perinatal death-rate of 53 per thousand. These represent the uncorrected percentages and should be used for comparison with the rest of the figures given in this article.

There were 1028 cases of ante-partum haemorrhage, giving an incidence of 1.25%. Table I gives percentage incidence of accidental haemorrhage, placenta praevia and unclassified ante-partum haemorrhages with the maternal and foetal mortality, as also incidence of post-partum haemorrhage. The overall foetal mortality is further subgrouped into preventable foetal loss by taking into consideration those patients who were admitted with foetal heart sounds present and determining the still-birth rate and perinatal mortality in that group. The improvement of results in this group is the only possibility left for the obstetricians in the problem of ante-partum haemorrhages. As will be evident from the table, the maternal mortality in both accidental haemorrhage and placenta praevia is reasonably low with the modern methods of treatment. The incidence of post-partum haemorrhage is the same in both the conditions, but there is a marked contrast in the preventable foetal loss; the overall foetal loss by three-fourths of the mothers with accidental haemorrhage and by one-third of the mothers with placenta praevia would indicate investigations to find out means to prevent the occurrence of these conditions, while the loss of half the number of babies in accidental haemorrhage and one-fourth in placenta praevia, when the pregnant mothers are admitted with living foetuses in the uterus, would definitely indicate the revaluation in our mode of management and treatment of these cases during the intra and post-natal periods. A careful consideration of Table 2 brings to light the invalidity of subdivision of accidental haemorrhage into three groups, namely revealed, concealed and mixed, as there is hardly any difference in the figures of the last two groups. Further it is noted that even though the initial still-birth loss is low in revealed variety the neonatal loss is four to eight times that in other varieties bringing the perinatal mortality in the vicinity of the other two conditions. This will indicate an

TABLE II

Foetal Outcome in Accidental Haemorrhage

Type of Acc. haemorrhage	Cases	No. F.H.S. S.B.	F.H.S. Present S.B.	Total S.B.	Neonatal Loss	Perinatal Mortality	Live Birth
Revealed ..	82	29	6	35 (42.6%)	15 (18.3%)	50 (60.9%)	47 (57.4%)
Concealed	111	60	27	87 (78.3%)	5 (4.5%)	92 (82.8%)	24 (21.7%)
Mixed ..	327	210	48	258 (78.8%)	7 (2.1%)	265 (81.0%)	69 (21.2%)

F.H.S.—Foetal Heart Sounds. S.B.—Still Births.

early action in the concealed and mixed variety by carrying out a quick rupture of membranes to reduce the intra-uterine tension and to avoid the complications of still-births, coagulation defects in the mother produced by absorption of enzymes from the uterus into the maternal blood stream and the occurrence of oliguria and anuria. To get three times the live-births in the revealed type of haemorrhage compared to the concealed and mixed types and then to lose one-third of them in the puerperium may be due to the prematurity at which the children are born and to the condition of varying degree of anoxaemia during labour that favours lung, liver, stomach and brain complications in the newly-born. This will further stress the importance of avoiding the development of abruptio placentae rather than treating it. At present a lot of attention is paid to the effects of ablatio placentae and the treatment of its complications e.g. anuria or oliguria, lung congestion and secondary cardiac failure, cerebral complication from hypertension and much dreaded and also much investigated condition of hypofibrinogenemia. Along with these complications the fear of renal failure is present in every case, as it develops insideously during the stage when the attending obstetrician is waiting for the natural delivery to take place, particularly if he delays the rupture of membranes to reduce the intra-uterine tension.

TABLE III

Preventable Foetal Mortality in Accidental Haemorrhage

Type of Acc. Haemorrhage	Cases	Still-Birth	% Mortality	Perinatal Mortality
Revealed ..	53	6	11.3	
Concealed ..	51	27	52.9	81 plus 27 = 108
Mixed ..	117	48	41.0	
Total ..	221	81	36.6	48.9%

TABLE IV

Foetal Outcome in 400 Cases of Placenta Praevia
Total Foetal Mortality 35.0%

Grade	Total Cases	Mortality	% Mortality
I	66	22	33.3
II	195	54	27.1
III	73	39	53.4
IV	66	25	38.4
Total	400	140	35.3

As stated above the change in the coagulation of the blood is secondary to the damage to the placenta. It is partly a normal physiological process and partly due to the failure of normal physiology. The damage to the placenta liberates thrombokynase into maternal circulation which uses up all the available fibrinogen in the blood and liberates small fibrin clots

which, when carried by the blood stream, may precipitate fine widespread embolism in the important organs e.g. lungs, kidneys and brain. As a physiological response to the thrombotic emboli nature favours production of heparin and fibrinolysin. This is investigated clinically by the bed-side clot observation test as suggested by Wainer. The failure of the formation of clot indicates absence of fibrinogen. If the normal blood, when mixed with patient's blood, fails to clot it indicates the presence of heparin-like factor. If the clot from normal blood dissolves on adding patient's blood it indicates the presence of fibrinolysin. All these conditions lead to haemorrhagic tendency in the patient and each one requires a different mode of treatment as will be discussed later. This process of locking up of the fibrinogen and fall in the blood fibrinogen level may be either a gradual and progressive process in some of the cases of essential hypertension or it may come on as a sudden catestrophy. The premature onset of labour is not the causative factor, as the microscopic studies of placentae from cases of premature delivery following accidental haemorrhage do not show the same changes as are seen in the spontaneously delivered premature placentae which show minor abnormalities of implantation.

In accidental haemorrhage the placental infarction is immunological in origin, of the nature of the Sanarelli-Shwartzman phenomenon. The spiral arterioles of the decidua may bring on by their spasm more subtle changes in the placental villi e.g. the ischaemic necrosis of the villi, adhesions of the villi and white and red infarcts of the placenta. These changes take place progressively in cases of accidental haemorrhage. If fibrin is deposited on the villi, they develop vesicular swollen cells of syncytio-trophoblastic origin. There are enzymes normally present in the placenta e.g. alkaline and acid phosphotase, 5 nucleotidase, ATP, Leucin-amino-peptidase and various dehydrogenases, and their production is likely to be affected and impaired. Normally a pregnant woman with a normally functioning placenta and a living foetus of $5\frac{1}{2}$ lb. in weight excretes 12 mgm. of oestriol per day in the urine. When the placental insufficiency developes the oestriol excretion drops. With the drop below 30% the foetus is in serious jeopardy. Disappearance of histidine from the urine also indicates the placental damage and foetal death and can be detected by comparatively simple chemical test that can be carried out by any clinician. The fall in the fibrinogen does not develop earlier than 5 weeks after the intrauterine foetal death. During the phase of hypofibrinogenemia if an injection of fibrinogen is given, the fibrinogen level in the blood returns to the pre-injection level within 24 hours. Since termination of pregnancy follows as a rule within three weeks of the death of the foetus an attempt to evacuate the uterus should be undertaken after the 3rd week but before the 5th week.

The separation of the placenta and the haemorrhage appear to be immunological reactions in the decidua locally, while the lack of fibrinogen production appears to be due to the

TABLE V

Fibrinogen (mg) in Normal Pregnancy, Labour and Puerperium

Normal Pregnancy (15)	STAGE 1st (11)	2nd (9)	3rd (13)	1st day (8)	2nd day (2)	3rd day (2)
428	490	533	481	527	395	553

Fibrinogen (mg) in Pathologic Conditions during Pregnancy

Threatened Abortion (13)	Vesicular Mole (6)	Placenta Praevia (9)	Accidental Haemorrhage Revealed (2)	Mixed (7)	Concealed (1)
418	473	362	290	5 Afibrinogenemia	1

liver failure produced by the same reaction in that organ. It may not be due to a local effect as it becomes evident during the investigations that the fibrinogen content of the blood is very rarely seen to fall even when retroplacental haematoma was produced in placenta praevia by its separation. See Table V. If the mere occurrence of bleeding and haematoma formation had any causative action one should have seen the coagulation defect and a fibrinogenemia with equal frequency in placenta praevia as in accidental haemorrhage. In fact the fibrinogen content of the blood of patients with placenta praevia was never seen to be altered to the same extent in spite of bleeding. Toxaemia of pregnancy, including pre-eclampsia and eclampsia, as a causative factor for accidental haemorrhage is doubtful, as the fibrinogen content of our series was seen to increase with the onset of pre-eclampsia and during the eclampsia the figure rose as high as over thousand mgm. See Table VI. The essential hypertension cases, however, showed a progressive tendency towards a fall in fibrinogen values and in 6-8% of the cases it may reach a critical level below 100 mgm. so as to give rise to a clinical manifestation of bleeding tendency and failure of blood clotting. A clinician is rightly scared about the haemorrhage produced by hypo or afibrinogenemia as it is noted that even though its incidence varies from

TABLE VI

Blood Fibrinogen (mg.%)

	Normal Non-Pregnant (30)	Normal Pregnant (15)	Pre-Eclampsia (23)	Eclampsia (10)
Mean	317	428	468	615
Maximum	492	547	713	806
Minimum	215	344	307	437

4—8%, the mortality rate in the mothers is 12% as reported by Longo et al.[1] in their 48 cases and the foetal mortality as high as 80%. Thus the possible causes of fibrinogenopenia are (1) failure of the fibrinogen synthesis as a result of anoxic liver damage probably produced as immunological reaction, (2) depletion of circulating fibrinogen by the formation of retroplacental clot, (3) blood loss, (4) thromboplastic and fibrinolytic agents from the placental or decidual fragments entering the maternal circulation and the production of heparin-like substances by the liver. Hypofibrinogenemia is frequently associated with slight to moderate reduction of plasma prothrombin activity by 60 to 90% of the normal. There is also a fall in platelets and incease in the circulating plasma fibrinolytic activity. Plasma fibrinogen level of 150 mgm. per 100 c.c. or above is ample for effective coagulation during pregnancy, labour and the puerperium. Phillips et al.[3] in 1956, reported activators, a so-called fibrinolytic activator and profibrinolysin, present in normal placenta and they advocated the theory that in afibrinogenemia the fibrinolytic activity may be due to "activator intoxication." The human placenta possesses considerable fibrinolytic activity. The activators disappear from the endometrium when it is transformed into decidual tissue.

Thus it will be evident that even though the various mechanisms for disturbing the blood coagulation in the ante and intra-partum periods are known, the actual substance or substances which bring about the decidual destruction and initiation of the separation of the normally implanted placenta are yet to be found out so as to prevent the very occurrence of abruptio placentae. Hypotensive and tranquiliser drugs will help to relieve the spasm of the decidual vessels and anti-allergic substances may help to neutralise the allergens precipitating its occurrence.

The avoidance of the development of placenta praevia is beyond the scope of preventive therapy. Perhaps the judicious use of progesterone during the early months of pregnancy in a patient with previous history of placenta praevia may favour production of proper decidual reaction at the fundus. Solace lies in the proper and effective management of the case once it has developed the bleeding episode. The same will apply to the bleeding coming from the circular sinus of the placenta.

TABLE VII

Maternal Mortality in Ante-Partum Haemorrhages

Accidental Haemorrhage	Placenta Praevia
3 in 318 Cases (0.57%)	7 in 400 Cases (1.75%)
Causes:	Causes:
1. Hypofibrinogenemia	1. Pulmonary Embolism
2. Anuria	2. Hypofibrinogenemia
3. Anaemia—Congestive Cardiac Failure	3. Infective Hepatitis
	4. Severe Haemorrhage with Anaemia (4 Cases).

TABLE VIII

Maternal Morbidity in Ante-Partum Haemorrhages

Accidental Haemorrhage		Placenta Praevia	
Causes:		Causes:	
1. Puerperal Pyrexia	20	1. Puerperal Pyrexia	16
2. Pyelitis	2	2. Thrombophlebitis	1
3. Psychosis	1	3. Oliguria	1
4. Anuria	6	4. Cystitis	1
5. Hypofibrinogenemia ..	2	5. Hepatitis	1
		6. Gangrene Leg	1
		7. Burst Abdomen	1
		8. Cervical Tear	1

The mode of management of the accidental haemorrhage and placenta praevia is given in Table IX. Very few new methods have been added as is evident from the list. If at all some of the procedures formerly used very frequently are either not used or used very occosionally. What is really achieved is accumulation of the experience and the knowledge when to avoid the various methods in the light of the acquired results both in the mother and the child. Any method of treatment adopted should give rise to minimum complications in the mother and the child and there should be as little morbidity as possible. In the case of accidental haemorrhages the importance of interference as early as possible with minimum operative procedure appears to give very good results. Early rupture of the membranes and free use of pitocin is indicated and only in the exceptional cases caesarean section is resorted to, as vaginal delivery follows in majority of cases. One should be ready for post-partum haemorrhage which will require blood transfusion, plasma, etc. and powerful oxytocic drugs. If afibrinogenaemia develops, use of 4 gms. of fibrinogen when available, double concentration plasma transfusion or

TABLE IX

Line of Treatment in Ante-Partum Haemorrhages

Accidental Haemorrhage 518 Cases			Placenta Praevia 400 Cases		
	Cases	%		Cases	%
1. Spontaneous Delivery ..	238	(45.9)	1. Spontaneous Delivery ..	122	(30.5)
2. Artificial Rupture of Membranes	173	(33.4)	2. Artificial Rupture of Membranes	70	(17.5)
3. A.R.M. with Pitocin ..	79	(15.2)	3. L.S. Caesarean Section ..	142	(35.5)
4. Pitocin alone	9	(1.70)	4. Classical Caesarean ..	5	(1.25)
5. A.R.M. with Pulling down leg	3	(00.56)	5. A.R.M. & Pulling down leg	28	(6.5)
6. Internal Podalic Version	4	(0.74)	6. Internal Podalic Version	12	(3.0)
7. L. S. Caesarean Section	11	(2.10)	7. Willett Forceps	14	(3.5)
8. A.R.M., Pitocin & L.S.C.S.	1	(0.18)	8. Perforating Placenta ..	5	(1.25)
			9. Hysterectomy	2	(0.05)

blood transfusion is indicated. Whole blood contains 1.5 gms. per litre while the plasma contains double that amount. When the clotting defect is due to fibrinolysin, 100 to 200 gms. hydrocortisone should be administered intravenously or intramuscularly. Epsilon - Amino - Caproic Acid (EACA) is advocated with the intention of inhibiting fibrinolysis. Leroux et. al. have suggested the use of enzyme-inhibiting substance 9921 RP or Zymofren.

To counteract heparin-like factor, Protamine Sulphate 20-50 mgs. given intravenously slowly and without a repetition is also advocated. There is no harm in giving pitocin drip provided previous rupture of the membranes is carried out. The use of pitocin during labour does not produce a drop in the fibrinogen in the blood nor is there any significant change in fibrinolytic activity. Blood coagulation improves following the emptying of the uterine contents and there is not sufficient evidence to prove that the maternal mortality can be reduced by more frequent use of caesarean section.

There is a possibility of repetition of the accidental haemorrhage in subsequent pregnancy. In the majority of cases, the severity of the disease is as grave as on the previous occasion and sometimes even worse, along with progressive damage to the kidneys. It is imperative to advise the stoppage of future pregnancies in multiparas. In the cases of placenta praevia the policy of judicious use of caesarean section to the extent of 50% of the cases, and conservative expectant therapy in the remaining, gives very satisfactory results both from the point of view of the mother and the child.

In the light of the present study it is evident that further investigations directed towards finding out the etiological factor in the production of accidental haemorrhage and a proper assessment of the various methods in the management of placenta praevia are indicated in getting over the important problem of ante-partum haemorrhages during the last trimenster of pregnancy. Detailed study of genuine cases of essential hypertension without toxaemia of pregnancy would go a long way towards finding out the real etiological factor in the production of ablatio placentae.

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