OBSERVATIONS ON TRAUMA AS A CAUSE OF ACCIDENTAL HAEMORRHAGE

BY

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MUCH has been written over the past half century on the subject of accidental haemorrhage, yet we are to-day very little nearer to a complete understanding of its cause. An examination of the literature, however, does suggest a decreasing emphasis on the part played by trauma. Holmes (1901) listed 250 cases of accidental haemorrhage and ascribed an accident as cause in 67 of them. As causes of trauma he listed falls, jars, blows on the abdomen, violent exercise—including walking and running, and the lifting of heavy weights.

Even with his own figures in front of him Holmes expressed considerable reserve on the subject adding:

> Unquestionably all these diverse factors may and do have some influence in producing a separation of the placenta but I am of the belief that these causes *per se* have been most grossly exaggerated by all authors on this subject.

Writing again on the subject (1940) his reservations were even more marked for he stated:

> We cannot escape the effect of trauma, though our modern conception of cellular pathology relegates it to a lesser role. When trauma does occur it is tragic in effect.

Williams (1915) reviewing 2,000 labours at the Johns Hopkins Hospital noted premature separation of the placenta 17 times. In all but one instance there was external haemorrhage. Trauma did not figure in any of his cases but he commented:

> As a result of my investigations and study I am prepared to admit that trauma may occasionally be a causative factor.

Montgomery (1934) had a series of 32 cases of accidental haemorrhage out of which trauma was stated to have accounted definitely for 2 cases. Mahfouz and Magdi (1939) reported on a series of 83 cases of accidental haemorrhage, with trauma appearing to be a factor in 5. Vaginal bleeding occurred shortly after the patients had received a blow on the abdomen, the "victims" being otherwise healthy. They make, however, this reservation:

> Trauma may be a coincidence but not the cause or it may simply be the exciting factor in a patient on the verge of bleeding. Careful investigation may also reveal a few malingerers among women who attribute their symptoms to trauma.

Baens (1948) out of 78,828 deliveries diagnosed 80 cases of premature separation of the placenta but only 3 were, in his opinion, due to trauma. Kimbrough and Jones (1948) reported a series of 113 cases of accidental haemorrhage and stated:

> A definite history of trauma was elicited in only one instance; in this patient the co-existence of toxaemia was a more likely cause of the separation.

During the past 18 months at St. Mary Abbots Hospital there were just over 1,400 deliveries with 12 cases of accidental haemorrhage (one completely concealed). Trauma appears to have been definitely the cause in one case, the most likely cause in a second. There was no evidence of trauma in any of the other 10 cases.

CASE 1. Mrs. E. C., aged 25, had 2 full-time, normal deliveries in 1943 and 1948, the infants weighing 7 pounds and 8 pounds 14 ounces respectively. The pregnancy and puerperium each time was normal. Her past illnesses were irrelevant. She was a housewife. She was first seen during the 3rd pregnancy at the antenatal clinic at the 20th week. Nothing abnormal was detected. She attended the clinic regularly. On her last visit to the clinic before admission to the hospital she was 37 weeks pregnant, the presentation was right occipito posterior, the presenting part was free, the blood-pressure was 110/70 (as on previous examinations) and the urine was clear. Six days later she was brought into hospital by ambulance. She was groaning, pale and too confused to give an account of herself. She had been found by a neighbour collapsed on the second landing with a bucket of coal on one side of her and her 17-months old baby on the other. Next day the patient was able to state that she felt perfectly well until ascending to the second floor, on which she lived, with a heavy bucket of coal in one hand and her baby on the other arm. She suddenly experienced excruciating abdominal pain forcing her to the floor. She lost consciousness for a time.

On examination on admission her pulse was 120 per minute, of poor volume, blood-pressure 70/50, temperature 98°F. The uterus corresponded to a 38-weeks pregnancy and was so tense and tender that no foetal parts could be made out. The foetal heart could not be heard. There was no vaginal bleeding and a catheter specimen of urine showed nothing abnormal. Provisional diagnosis of a concealed accidental haemorrhage was made. She was given morphia ¼ gr. and intravenous transfusion with dextran was started. Three hours later the hardness of the uterus was less marked, the foetal heart could be heard for the first time and weak, infrequent uterine contractions could be felt. At this stage a surgical rupture of membranes was carried out and pinkish stained liquor escaped. Dextran was now replaced by group O Rh positive blood. Four hours after the induction she was delivered of an asphyxiated baby which responded quickly to routine resuscitation. Immediately after the birth of the baby she passed 3 large blood clots. The placenta was expressed and more clots and free blood came away. The total loss was estimated as 60 ounces. The mother made an uneventful recovery and was discharged on the 12th day with the baby weighing 6 pounds 8 ounces, a gain of 7 ounces.

The placenta (Fig. 1) measured 9 inches by 7 inches and showed a saucer-shaped depression extending over half the maternal surface. The cord was 20 inches long. Fig. 2 shows how blood clots filled the depression. The area of the depression was sectioned and showed marked congestion with villi packed closely together, suggestive of infarction (Fig. 3). There were also some areas of hyalinization.

CASE 2. Mrs. A. H., aged 31 years, primigravida, was first seen at the antenatal clinic when 16 weeks pregnant. Nothing abnormal was detected Blood-pressure was 120/70, urine normal. Her past illnesses were irrelevant. She was a housewife. She attended the clinic regularly. At the 32nd week the presentation was right sacro-anterior with legs extended. Her blood-pressure was 120/80 and the urine still clear. After the presentation had been confirmed by X-rays an external version without an anaesthetic failed. Next day an external version under gas, oxygen and ether was carried out successfully, but immediately after this the patient started a brisk vaginal haemorrhage. Her general condition remained good but the uterus became tense and tender. The foetal heart was heard at a rate of 160 per minute. She was given ¼ gr. morphine and the bleeding became less, persisting in a scanty form for the next 3 days and then ceased. The day after the version the blood-pressure had risen to 140/90 and remained at this figure until the ninth day when it rose to 145/100. There was also some oedema of the legs but the urine was still clear. The patient remained in hospital for 20 days after the version, her blood-pressure being

constantly between 140/90 and 150/100. She became impatient and insisted on taking her discharge. On each of the next 2 weeks she visited the antenatal clinic when her blood-pressure was 140/90 and the urine still clear. The foetal heart was heard. At the end of the third week, however, the blood-pressure had risen to 160/120, the urine contained a cloud of albumen and there was marked oedema of the legs. The foetal heart could be heard. She was immediately re-admitted and a surgical induction was carried out. This was followed by a normal delivery of a live infant weighing 5 pounds 8 ounces. Patient and baby made an uneventful recovery and were discharged on the 12th day. Her blood-pressure on discharge dropped to 110/70 and the urine was clear. The placenta measured 8 inches by 6 inches, and on the maternal surface there were several white infarcts. Two old blood clots, the size of a half-crown subtended the infarcted areas. The clots were pale and could be indented like clay.

Discussion

In Case 1 the onset of symptoms and clinical signs following immediately the marked exertion make it difficult not to accept trauma as the cause of the partial separation of the placenta. It is also interesting to note that the infant was born alive despite the extensive separation.

Gibberd (1948) stated: "The prognosis of the foetus depends more upon the presence or absence of an 'associated toxaemia', than upon the amount of the bleeding. So often in a case of 'toxaemia' the foetus has little or no placental reserve and the sudden loss of even a small part of its effective placental exchange is enough to kill it." In Case 1 it was possible to exclude pre-eclamptic toxaemia and this, coupled with the rapid onset of labour, would account for the survival of the foetus.

In the Case 2, the external version was undoubtedly responsible for the premature separation. In assessing the risks of this *manoeuvre* published figures cannot be taken as an accurate guide for the results obtained vary in extraordinary fashion. Adair (1940), in a collected review from 9 authors covering 1,105 attempted external versions, reported only 2 cases of slight vaginal bleeding. Observations reported in Britain on the same subject, however, make one feel that Adair's authors were unusually successful. Gibberd (1927), for instance, had one case of slight bleeding in 179 external versions, without consequence to mother or child. Macafee and McClure (1937) had one antepartum haemorrhage out of 134 external versions. Wrigley (1934) reported 76 attempted versions under anaesthesia. In 45 cases the version was successful but brisk vaginal haemorrhage occurred in 3 mothers due to detachment of the placenta. Their children were stillborn, due, the author believed, to asphyxia. White (1933), reviewing 300 cases of external version at University College Hospital, noted bleeding in 10 cases. Ninety-two of the versions, he points out, were under anaesthesia.

Odell (1943) collected 3 cases of premature separation following external versions and described one case in detail. His patient, however, had a high blood-pressure before the version was attempted, and labour coincided with the bleeding, and it is, therefore, impossible to compare the clinical data with that of my second case. Here we had a gradual increase in the blood pressure, following the version, over a period of 6 weeks, culminating in a severe pre-eclamptic toxaemia. The question arises: was the rise in blood-pressure a mere coincidence or was it the sequelae to the separation? I have been unable to find any similar observation in the literature, except, indirectly by Young (1914). He states that the partial placental separation gives rise to toxaemia by liberating poisonous autolytic products from the

separated parts. He adds: "In accidental haemorrhage we obviously have a corresponding part of the placenta cut off from its blood supply. If the haemorrhage develops and extends so rapidly that it quickly kills the patient or determines a complete separation of the placenta, either naturally or by the interference necessitated in the patient's interest, there will be no opportunity for the necrotic changes to develop and there will be no toxaemia. But, if the placenta remains attached at one part for some hours or days, the circulation there will remain undisturbed and there will be an opportunity for the discharging into the maternal blood of the toxic ingredients quickly elaborated by the disintegration of the separated portion. Only in such cases will an albuminuria or an eclampsia develop."

Young and Walker (1947) found further support for this theory in reporting a case in which there had been an attempt to procure an abortion by forcible injection into the birth passage of a mixture of soap and dettol. The changes produced in the uterus and kidneys resembled those found in the graver forms of concealed accidental haemorrhage. The writers were of the opinion that much of the uterine placental injury was caused by the inoculum but that the permanent kidney changes which led to the patient's death were due to materials escaping from the damaged tissue.

In my Case 2, undoubtedly all factors were in favour of the "toxic ingredients" passing into the maternal blood stream and becoming responsible for the gradual development of the pre-eclamptic toxaemia.

Summary

Two cases of premature separation of the placenta are examined in detail and the part played by trauma is discussed.

A review is made of the literature concerning trauma as a cause of premature separation of the placenta.

The possible relationship between the premature separation of the placenta and pre-eclamptic toxaemia is briefly discussed.

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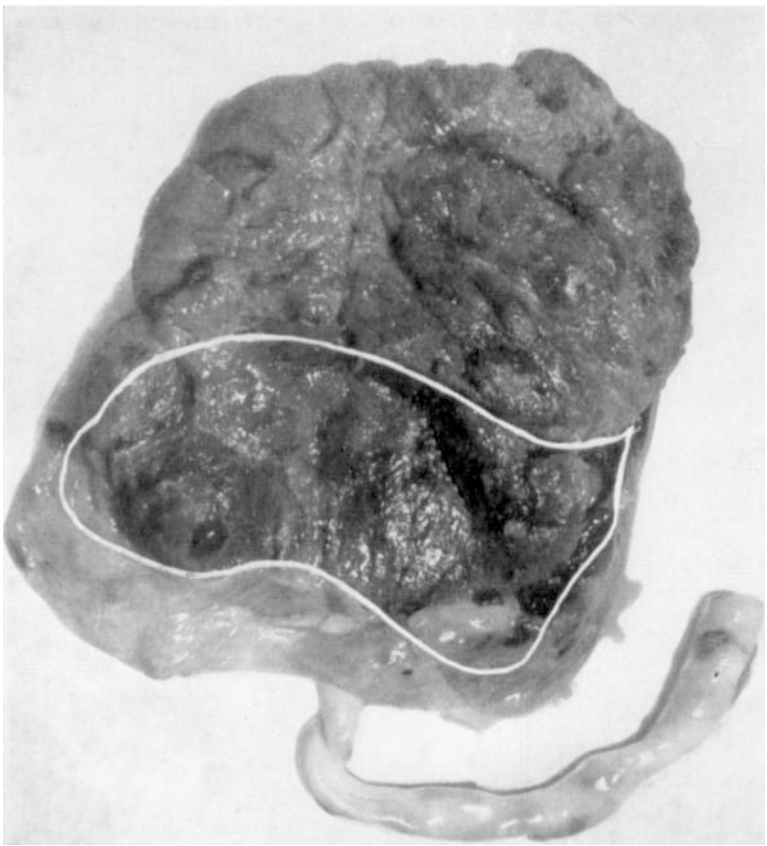

FIG. 1

Placenta with saucer-shaped depression indicated by wire.

C.G.

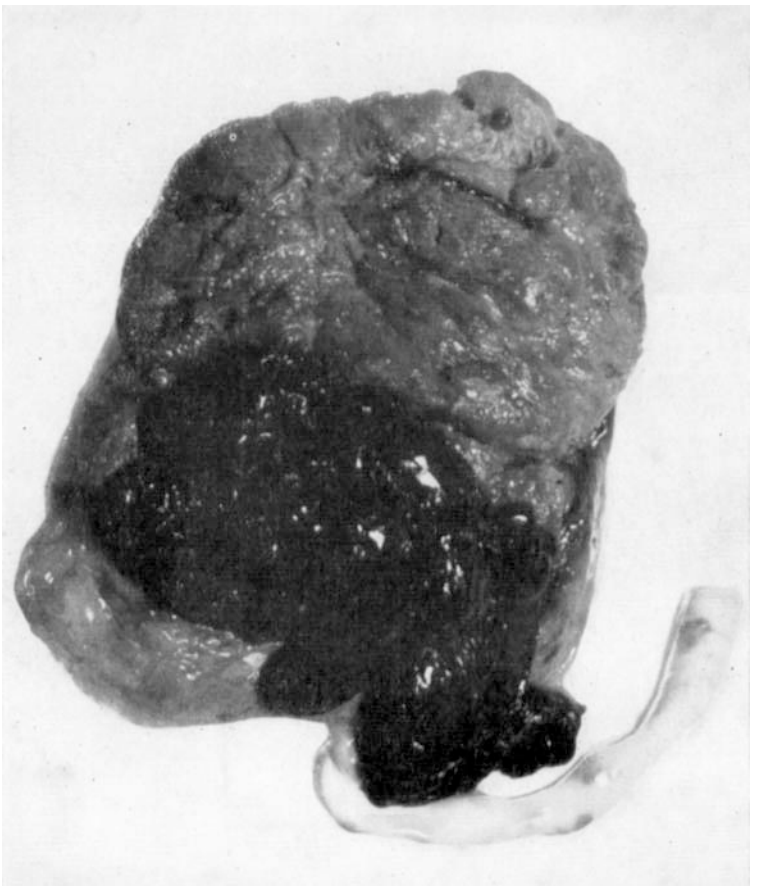

FIG. 2

Placenta showing blood clots filling depression.

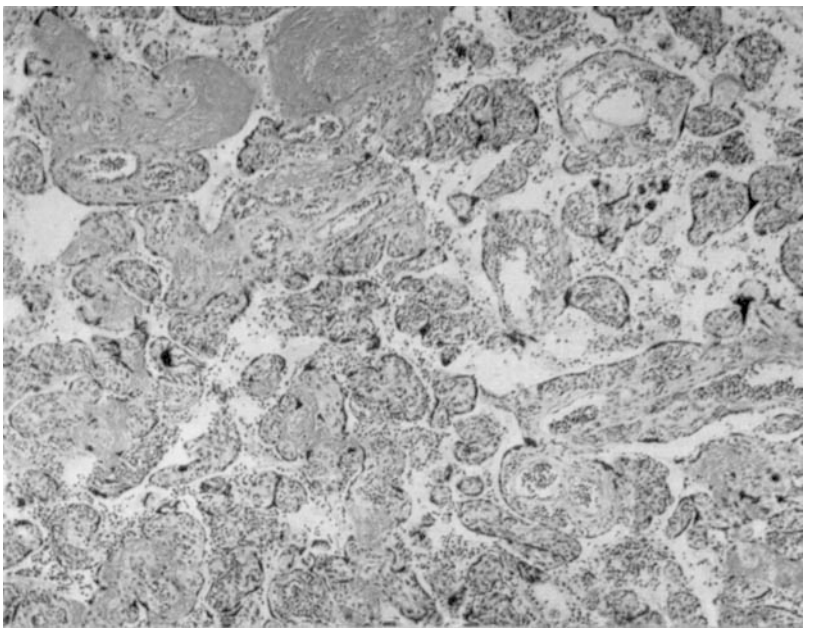

FIG. 3

Section of placental depression showing villi packed together. × 110