The Pathology of Obstetric Shock

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THE name "obstetric shock" has a variety of interpretations, but not an exact definition. Clinically the concept of "pure obstetric shock" excludes all cases in which the aetiological factor is known; these are usually grouped as surgical shock, shock due to haemorrhage, and so on. A number of associated factors are recognized: pain, post-partum reduction of abdominal pressure, cold, malnutrition, and psychological distress. These factors have the support of a sufficient weight of clinical experience to be accepted but, as they are common to most hospital deliveries, any assessment of their exact significance is difficult. Undue emphasis must not be placed on them; otherwise the problem of why shock occurs after an occasional delivery becomes replaced by the problem of why it does not occur after every delivery.

From the standpoint of pathology, the essential requirement is that the patient shall have died of shock in connexion with obstetrics, no matter what the cause of the shock. Post-mortem investigation frequently discloses gross lesions which had not been suspected clinically, and it furnishes a few possible clues as to the mechanism of shock. But it does not explain how the lesion, such as inversion of the uterus, has produced the functional disturbance known as shock, nor does it explain the real mechanism of this functional disturbance. Bearing in mind these limitations of pathological anatomy, it seems nevertheless of value to summarize the pathological findings in obstetric shock, particularly as the literature on the subject is very meagre. The material studied here is restricted to those fatal cases of obstetric shock that have been examined post-mortem in this hospital during the past 5½ years. The criterion of obstetric shock that has been adopted is rather wider than that in common clinical use. It is that the patient died with the clinical appearances of shock during labour or within 24 hours after delivery, whether or not any explanation for the shock had been recognized before death. In many of the cases the patient was apparently well until about an hour or two after delivery, and then suddenly developed severe shock, which was fatal within 2 or 3 hours.

The initial difficulty is to differentiate the circulatory collapse due to shock proper from that due to haemorrhage; since both conditions are frequently co-existent in obstetric patients. These conditions are also related; not only can haemorrhage lead to shock but, in a badly shocked patient, a trivial haemorrhage can be fatal. Similarly, anaesthesia or operation may themselves be the cause of death or they may be merely accessory factors that precipitate a shock due primarily to some other cause. While any exact apportionment of responsibility is thus impossible, those cases are included in the present series in which shock played a major or important part in causing death, and cases are excluded in which death was essentially due to the anaesthetic or to severe haemorrhage, e.g. placenta praevia and ruptured aneurysms. To limit the subject further, a number of conditions related to shock has been excluded: sudden heart failures after delivery in patients with chronic valvular disease, circulatory collapse in certain cases of pre-eclampsia or eclampsia, acute pulmonary oedema after delivery (Hesse[1]), and all cases in the first half of pregnancy, chiefly collapse after surgical abortion for hyperemesis. Some of these exclusions are made on the grounds of the pathological findings; the patients were clinically suffering from obstetric shock of unknown origin. The cases finally selected as obstetric shock according to the present definition are grouped in Table I, according to the apparent aetiological factors; the more salient clinical features are separately noted for each group.

TABLE I.

Cause of shock	Number of cases
Dystocia	29
Ruptured uterus	13
Retained placenta	22
Utero-placental apoplexy	21
Uncomplicated Caesarean section	4
Complicating disease	8

Dystocia (29 *cases*).

This is the largest group; it consists of deaths from shock during or soon after a difficult delivery, usually within 8 hours.

The cause of the dystocia was simple disproportion, occipito-posterior presentation, or more rarely hydrocephalus or oblique lie: some of the cases had a slight degree of contraction of the pelvis. The average weight of the babies was slightly

over 9 pounds (an allowance of a half-pound has been made for craniotomy and of a quarter-pound for perforation). Labour was always prolonged, averaging 56 hours for the whole group. The pains were often irregular and unsatisfactory and there was always some pyrexia towards the end of labour, usually under 100°F. In 12 of the patients delivery by the forceps had failed during labour. The delivery was always artificial; version or difficult deliveries by the forceps in 19, perforation in 6, and craniotomy in 4. Despite the poor retraction of the uterus in nearly all cases, post-partum haemorrhage was not very frequent; only 2 severe and 4 moderate cases were noted. The usual cause of death was uncomplicated shock.

It is of interest that of the 6 patients who died during the labour, only 1 was a primipara, the others died during their second to fifth labours. On the other hand, of the 23 patients who died after the delivery 20 were primiparae, the others died after their third, sixth, and ninth deliveries respectively.

Ruptured Uterus (13 *cases*).

This group is in certain ways associated with the previous group. Though it is usually excluded from discussions of obstetric shock, its practical importance is seen from the fact that in 7 of the cases the cause of the fatal shock was found only at post-mortem. Death was usually rapid; 5 of the patients died during the delivery and only 1 lived longer than 3 hours post-partum. There was always some haemorrhage, external or internal, but it was severe only in 4 cases, and the essential cause of the deaths appears to have been shock. The group falls into two divisions.

(*a*) Seven of the patients had dystocia requiring intra-uterine manipulations which were probably responsible for some of the ruptures. The average weight of the babies was $9\frac{1}{2}$ pounds, the average duration of labour was 20 hours, and the patients had from 3 to 7 children.

(*b*) The other 6 patients had spontaneous ruptures; one through an old Caesarean scar, the others for no obvious cause apart from probable old cervical tears. The average weight of the babies was $7\frac{1}{4}$ pounds, the average duration of labour 14 hours, and the patients had had from 2 to 12 children.

Retained Placenta (22 *cases*).

This group has two divisions.

(*a*) Six patients who died between 2 and 3 hours after deli-

very. These are border-line cases; all of them had severe post-partum haemorrhage which was probably of greater significance than the factor of shock in causing death. Three of the placentae were removed manually, the other 3 were undelivered and one of these was grossly adherent.

(*b*) There are 16 patients in this group of whom 11 died between 4 and 8 hours after delivery and the other 5 between 8 and 24 hours after delivery. There was some haemorrhage in most cases, but it was considered severe only in 4, and the essential cause of death appears to have been shock. Twelve of the patients had had frequently repeated but unsuccessful attempts to express the placenta; in 9 of these the placenta was finally removed manually and in 3 it was firmly adherent and remained undelivered. The other 4 patients had the placenta expressed with a good deal of force; 2 of these had inversion of the uterus, 1 died of shock and severe post-partum haemorrhage 4 hours after delivery, and the other of late shock nearly a day after delivery.

In the whole series there is no special relation to parity; 5 of the patients were primiparae and the others had had up to 12 children. The first and second stages of labour were often short, averaging 7 hours.

Utero-placental Apoplexy (21 *cases*).

This term is used here for cases of mixed or concealed accidental haemorrhage with retroplacental clot; it does not include external accidental haemorrhage. Though these cases are usually classified under the heading of haemorrhage, the usual cause of death is shock. There are two clinical groups:

(*a*) Twelve patients were between the thirty-sixth and fortieth week of gestation. Of these, 8 died undelivered; they were all advanced multiparae, having had from 8 to 15 children, and were usually fat. The other 4 were artificially delivered, either by the forceps or perforation, and died from $\frac{1}{2}$ to 4 hours later. These delivered women had had from 1 to 6 children; 3 of them are border-line cases, as they had severe atonic post-partum haemorrhage which certainly played an important part in causing death.

(*b*) Nine patients were between the twenty-seventh and thirty-third week of gestation. All but one of these patients were delivered, in each case spontaneously; they died from $\frac{1}{2}$ to 14 hours later. In 3 of the cases there was moderate haemorrhage. The patients had had from 1 to 9 children.

In the whole series 10 patients showed clinical or pathological evidence of hypertensive toxaemia.

The unusual tendency of the advanced multipara, not only to develop utero-placental apoplexy, but also to die of shock as a result is indicated in Table II. This shows the total mortality in this hospital during the years 1932 to 1936 among patients with mixed or concealed accidental haemorrhage with retro-placental clot.

TABLE II.

Mortality from mixed or concealed accidental haemorrhage.

Number of children	Total cases	Shock deaths	Puerperal deaths
1	88	3	2
2 and 3	128	3	4
4, 5, and 6	125	11	2
7 and over	103	15	4

Uncomplicated Caesarean section (4 *cases*).

These were prophylactic Caesarean sections before labour in primigravidae with contracted pelvis, followed by shock and death at 2 to 5 hours later. In one there was also fairly severe haemorrhage which certainly contributed to death; the other 3 had no obvious additional factor.

Complicating disease (8 *cases*).

These were clinically shock deaths, 1 died undelivered and the others within 8 hours after delivery, which was spontaneous in 5. Four of the patients had definite evidence of previous hypertension, the others had lobar pneumonia, acute haemorrhagic pancreatitis, renal disease, and influenza, respectively. The importance of previous hypertension as a factor in obstetric shock has been pointed out by Adair, Hunt and Arnell.[2]

Pure obstetric shock (0 *cases*).

The absence of any cases in this group is probably due to the fact that only patients with post-mortem examination have been considered. It is possible that pure obstetric shock may be fatal, but no case can be accepted without a careful post-mortem examination, including satisfactory histology.

PATHOLOGICAL FINDINGS.

In a recent review by Moon,[3] the tissue changes indicative of shock are described, and are said to be found not only in experimental shock but also in human beings dying of shock from a variety of causes. This picture does not, however, appear

to be characteristic of obstetric shock, as judged from the present series of cases. In particular, the following conditions were not found, congestion or petechial haemorrhages of the liver and gastro-intestinal tract, engorgement of peritoneal venules, effusion of blood-stained fluid into serous cavities with petechial haemorrhages in the serous membranes, and congestion, oedema and petechiae in the internal mucosae, except in cases in which they were obviously due to causes unrelated to shock. In this connexion it is important to remember that, in a patient with severe circulatory collapse, an electric blanket on the abdomen can produce lesions in the viscera without necessarily causing recognizable lesions in the overlying skin.

The actual findings in the present cases may be considered in sections according to the various organs; so far as possible, the functional interpretations will be avoided.

Vascular system in general.

The greatest caution is needed in making deductions about what the blood-flow was like during life from the post-mortem appearances such as congestion of viscera or fullness of blood-vessels. At death the pressure in the blood-vessels disappears and the tissue tension in certain organs squeezes the blood out of the capillaries; later, gravity and rigor mortis alter the distribution of the blood.

Certainly, marked circulatory differences in the manner of death can give very different post-mortem appearances. For instance, the bloodless viscera of a person who has been bled white are very different from the congested viscera of an asphyxiated person. In a post-mortem on a case of obstetric shock, one might, therefore, hope to find some such general evidence of a particular circulatory disturbance. However, in the post-mortem examination there is no finding of this kind. In cases of obstetric shock, as compared with death from other causes, there is not any obvious pooling of blood in the abdominal viscera, in the muscles of the thigh, or in the large veins. It must be emphasized again that these negative findings after death can not be interpreted as indications that during life there was not a pooling of blood in these sites. But it is of interest that obstetric surgeons who have performed laparotomy on shocked patients do not seem to have found any gross pooling of blood in the abdomen.

These limitations of pathology do not, however, cover the entire field. If there is a circulatory disturbance in a tissue,

certain changes can develop which do not disappear after death. Among these changes are oedema, areas of haemorrhage, marked capillary dilatation, or ischaemic necrosis. It is not necessary to discuss here the particular varieties of circulatory disturbances that may cause these changes; the primary requirement is to know their occurrence and position in obstetric shock.

Heart.

One of the commonest findings in obstetric shock is the presence of sub-endocardial haemorrhages on the left of the interventricular septum, and occasionally also at the base of the papillary muscles in the left ventricle. They may be small or large; they lie right over many of the branches of the auriculo-ventricular bundle (Figs. 1, 2, 3). In the present series of cases they were found in over a third of the hearts. The factor controlling their incidence appears to be the duration of the shock. If the patient dies after shock which has lasted for less than 2 or 3 hours, the sub-endocardial haemorrhages are very rare. On the other hand, they are very common when the patient has been shocked for 6 hours or more, and particularly in those cases in which the patient has a preliminary bad shock, then improves temporarily under treatment, and finally collapses again, will not respond to treatment, and dies.

This may be illustrated from the individual clinical groups. In ruptured uterus or retained placenta, most of the patients die soon after the shock develops and do not show any changes in the heart; it is only in the few late shock deaths at 6 to 12 or more hours' post-partum that the sub-endocardial haemorrhages are found. The dystocia patients are usually in poor condition at delivery, as in most of them attempts to deliver by the forceps have failed earlier; the sub-endocardial haemorrhages are rare if the patient dies in the first hour or two after delivery, but are common if she dies later than this. In the accidental haemorrhages the patient is badly shocked some time before delivery; the shock lasts for several hours in nearly all cases. The sub-endocardial haemorrhages are usually present, whether the patient is delivered or not, and the amount of blood lost does not seem to be a factor of importance. In any of these conditions, if the patient recovers from severe and prolonged shock but dies from some other cause during the first few days of the puerperium, the left interventricular septum may show rather old and fading sub-endocardial haemorrhages which probably date from about the time of the shock.

FIG. 1.
SMALL SUB-ENDOCARDIAL HAEMORRHAGES.
Case of retained placenta shock.

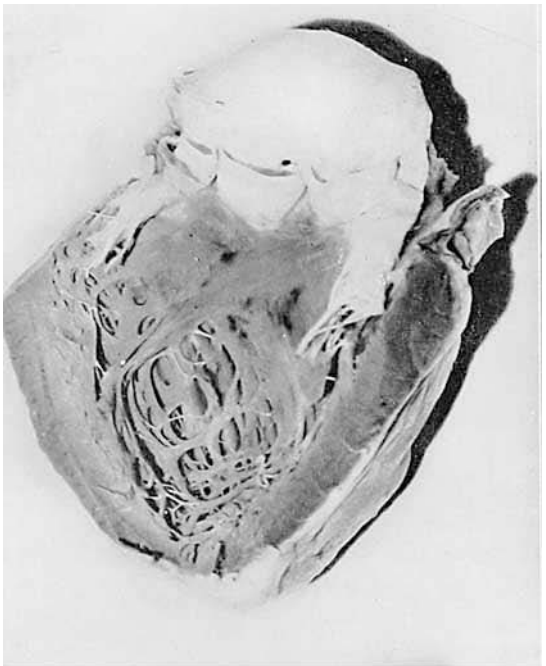
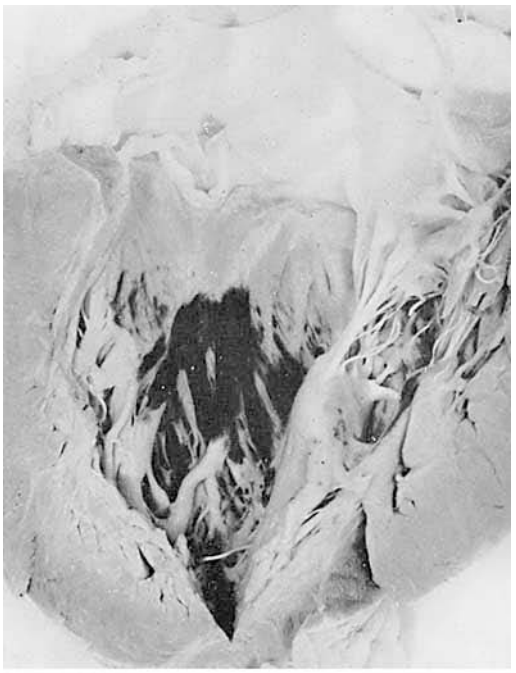


FIG. 2.
LARGE SUB-ENDOCARDIAL HAEMORRHAGES.
Case of utero-placental apoplexy.



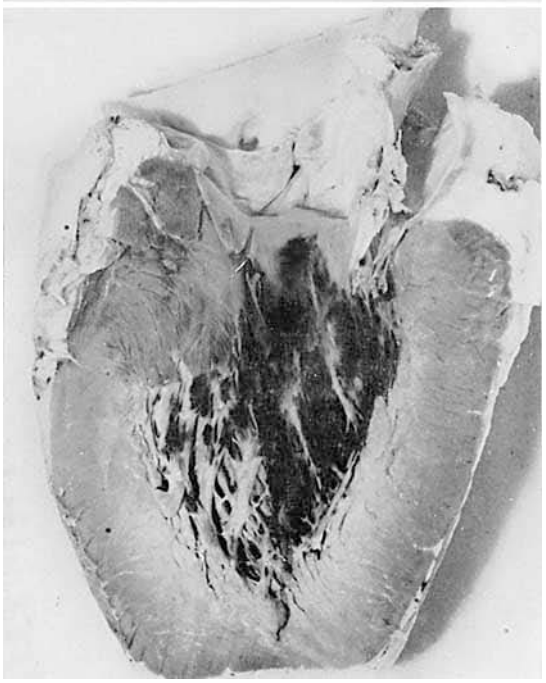


FIG. 3.
LARGE SUB-ENDOCARDIAL HAEMORRHAGES.
Case of dystocia shock.

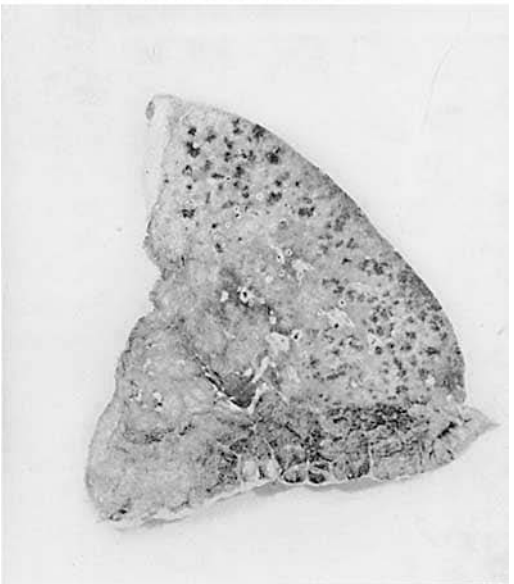


FIG. 4.
MULTIPLE PETECHIAL HAEMORRHAGES IN LUNG.
Case of inversion of uterus.

Though they are common in prolonged obstetric shock they are not specific to this condition. Thus, in a series of 1,000 post-mortems on non-obstetrical cases they were noted 25 times. The chief conditions in which they are reported, apart from obstetric shock, may be grouped as follows:

General shock. Death from burns (Shennan[4]), in soldiers dying of shock due to wounds (Moon and Kennedy,[5] Kulbs and Strauss[6]), and after the intravenous injection of cardiac stimulants which may have been used in treating shock.

Lesions of brain and vagus. Most of the examples in a general hospital are seen in deaths from fractured skull, meningitis or cerebral haemorrhage. It is said that the lesion can be produced experimentally by stimulation of the vagus in the neck either mechanically or electrically, or by raising the intracranial pressure (Monckeberg[7]). I have been unsuccessful in producing them in rabbits or guinea-pigs by these methods.

Purpuric diseases, such as leukaemia or septicaemia, are of little importance in the present connexion as the haemorrhages are merely local manifestations of a general disease. The haemorrhages are also seen in certain poisonings such as delayed chloroform poisoning, phosphorus poisoning, or poisoning by diphtheria toxin.

Hypertensive toxaemia. The haemorrhages are sometimes found in patients dying of eclampsia, more commonly in those with cerebral haemorrhage. Here there are the complications of the cerebral condition and the not infrequent termination in collapse; the hypertension cannot be accepted as more than a predisposing factor. The frequency of subendocardial haemorrhages in utero-placental apoplexy is in the same way not satisfactorily explained as due to the hypertension as such.

Other conditions. It is of interest to note that in deaths from valvular lesions of the heart, the sub-endocardial haemorrhages are not found.

The last question is whether these are important lesions which have a serious effect on cardiac function, or are just trivial post-mortem findings of the terminal variety. They lie over or among the fibres of the conducting system of the heart, but they are usually quite superficial and probably do not in themselves exert any gross mechanical influence. They do, however, constitute a post-mortem indication that during life there has been some capillary damage, probably due to a local circulatory disturbance. Any circulatory disturbance in this very important region of the heart must be regarded as potentially serious.

Clinically, one of the most striking signs in nearly all cases of obstetric shock is the great rapidity of the pulse-rate and its uneven force; in many of the cases which recover, the tachycardia persists for days or even weeks. But there is no definite evidence that the presence or cause of the sub-endocardial haemorrhages is responsible for these disturbances of cardiac rate, and undue emphasis must not be placed on them. Blalock[8] says that it has been proved in many experiments that the heart itself is not at fault in shock.

Lungs.

The chief interest in the lungs lies in the comparison of the lungs in death from shock with those in deaths from haemorrhage. In both cases it is often considered that there is an insufficient return of the blood to the heart, and in theory the lungs should, therefore, be similar in the two conditions.

The first complication is that the findings in the lungs in cases of death from haemorrhage vary according to the type of haemorrhage. (See Table III.)

TABLE III.

Combined weight of lungs in obstetric cases.

Cause of death	300 to 550 grammes	550 to 900 grammes	900 grammes and over
Bled to death			
Single haemorrhage	8	—	—
Successive haemorrhages ...	—	9	4
Death from shock following			
Single haemorrhage	7	2	3
Successive haemorrhages ...	2	6	2
Little or no haemorrhage ...	8	14	7

A person who bleeds to death as a result of a single large haemorrhage has dry anaemic lungs. This is what is seen in rupture of arteries or very great post-partum haemorrhage. On the other hand, a person who bleeds to death from a haemorrhage which is the last of a series of lesser haemorrhages, has quite marked oedema and congestion of the lungs. This is the typical finding in deaths from placenta praevia.

In death from shock there is no constant finding. Speaking quite generally there is a tendency for shock which comes on after a single haemorrhage to produce dry anaemic lungs, and for shock which follows a series of haemorrhages to produce congested oedematous lungs. In shock when there has not been

much haemorrhage the appearances depend on the time of survival. If the patient dies undelivered or within 1 or 2 hours post-partum the lungs are usually very congested and oedomatous, whereas if she dies 4 to 8 hours post-partum, the lungs are usually dry and anaemic. Without going into explanations, it is sufficient to say that deductions as to the condition of the circulation based on the presence or absence of oedema and congestion in the lungs are apt to be somewhat uncertain.

Any oedema of the lungs is usually not hypostatic; it usually affects one or two lobes, which may be upper lobes almost as often as lower. Patients who have had intravenous glucose-saline before death have dry anaemic lungs. In the series of post-mortems studied there is not a single case of pulmonary oedema due to intravenous saline. This is in agreement with the experimental findings of Davis.[9]

Occasionally there are enormous numbers of small petechial haemorrhages scattered throughout the lung (Fig. 4). These are of two types. The first are true haemorrhages into the alveoli, similar to those described by Moon[8] and Davis.[9] It seems probable that they are a manifestation of shock, but they are sometimes seen in patients dying of other conditions. The only obvious factor linking these cases together is that all the patients were made to inhale high concentrations of oxygen for some time before death. The second type are really very early haemorrhagic bronchopneumonias; they are more common in the shocks that follows dystocia than in the other types.

Uterus.

The appearance of the uterus depends on the clinical condition. *Rupture* is an obvious lesion; the tearing of the parametrial tissues appears at first glance to account satisfactorily for the shock. The haemorrhage is usually insufficient to account for death, though in certain cases it may spread widely in the retroperitoneal tissues or open into the peritoneal cavity. It is not clear why in such severe shock subtotal hysterectomy should have a relatively good effect; the operation does not deal with the damaged pelvic tissues and in itself it should add greatly to the shock. Nevertheless the only early deaths after hysterectomy for ruptured uterus in the present series appear to have been due to continuing haemorrhage internally rather than to shock. In *accidental haemorrhage* with retroplacental clot, the standard finding is, of course, the multiple gross haemorrhages under the peritoneum and in the outer layers of the uterine

muscle. An interesting aspect of this subject lies in the minor subperitoneal haemorrhages which are sometimes found near the cornua in conditions other than accidental haemorrhage. A few of the cases are found in eclampsia in patients with 2 or 3 children, and may be an indication of an incipient accidental haemorrhage that failed to eventuate. Nearly all the other uteri showing these small sub-peritoneal haemorrhages near the cornua are cases of obstetric shock, particularly those following dystocia or uncomplicated Caesarean section. This raises the possibility that in these shocks there may be a minor degree of the same circulatory disturbance to the uterus as occurs in accidental haemorrhage.

Dystocia. In the early deaths from shock after dystocia the uterus is usually not well retracted. It is large, soft and oedematous and weighs 1100 to 1300 gm., which is big even allowing for the large size of the baby. In addition, there may be signs of an early endometritis. In 2 of the late cases of shock in which the patient died 18 and 24 hours after a dystocic delivery, the uterus showed a striking lesion; it was very large and reddish, owing to an infiltration with bloodstained fluid, and weighed 1600 and 1800 gm. Microscopically there was necrosis of the entire wall of the uterus, the nuclei had lost their staining reaction and the muscle fibres were separated by oedema fluid. Such a gross lesion could naturally produce severe general toxic symptoms and be responsible for these late shocks. The question arises as to whether any of the heavy oedematous uteri of the early dystocia shock deaths may be very early stages of this complete necrosis. If so, it can be only in occasional cases in view of the rarity of the late necrosis. The possibility of a fulminant infection in these cases must also be borne in mind.

Retained placenta. In the early deaths from shock there is little or no abnormality in the uterus. In the later deaths from shock, at 6 to 10 hours post-partum, the uterus sometimes shows the large, soft oedematous appearance and weighs 1100 or 1200 gm. In one late death from shock, 21 hours after a delivery with a retained placenta, the uterus showed the same complete necrosis as in the two dystocia cases and weighed 1600 gm.

Ovaries.

Haemorrhages into the ovaries are sometimes seen in cases of obstetric shock, usually in cases in which there are also small subperitoneal haemorrhages near the cornua of the uterus. They are also seen in the cases with complete necrosis

of the uterus, but, rather surprisingly, in only one of the cases of utero-placental apoplexy. The haemorrhage spreads throughout the stroma, but only rarely affects the follicles and never the corpus luteum. It does not appear to be due to manual trauma—it was seen at laparotomy in one case of spontaneous rupture of the uterus.

While it is not common, it has only been found in cases of obstetric shock, and could presumably play a part in producing the shock.

Pelvic Floor.

Oedema of the parametrium and pelvic tissues is an occasional finding which does not appear to be very closely related to shock. It is present in patients with gross necrosis of the uterus and in certain other cases of shock, particularly those associated with hypertensive toxaemia: it is also sometimes found in uncomplicated cases of eclampsia. Even when the oedema seems quite considerable, the total weight of the parametria and pelvic connective tissues is only about 300 or 400 gm. This amount of fluid loss is not sufficient to be used as an explanation for obstetric shock along the lines of the work of Blalock and others.

Before leaving this subject, mention must be made of a suggestion of Miles Phillips[10] that there might be unsuspected tears in the muscles of the pelvic floor. Unfortunately the pelvic floor is rarely examined in great detail at post-mortem, but any damage sufficient to produce local haemorrhage is easily recognizable. The only places where this has been seen are around ruptures of the lower uterine segment and occasionally between the bladder and the symphysis pubis.

Congestion and oedema or ulceration of the trigone of the bladder are standard findings in the patients dying from dystocia, and are often associated with an early acute pyelonephritis affecting both kidneys.

Stomach.

Acute dilatation of the stomach with air is a rather common feature in deaths from shock after a dystocic delivery; it is less frequently seen in the other types of obstetric shock. Its significance is not clear; presumably the vagus is implicated and the condition is to be regarded as an effect rather than as a cause of the shock. The upper part of the duodenum is not affected.

Pituitary Gland.

A patient who dies of obstetric shock during the first 12 or 14 hours post-partum does not show any recognizable lesion, but if she survives the shock for a day or two and then dies, there is usually necrosis of the anterior lobe of the pituitary gland. This is particularly common if the shock is associated with much haemorrhage. The necrosis may be large or small, depending on the severity of the shock. It appears to be only a result of the general circulatory collapse of the patient. The lesion probably begins at about the time of the shock, but it takes several hours to become histologically recognizable.

During the initial stages, before the necrosis can be found histologically, there is presumably some functional disturbance of the anterior lobe. The question arises whether, in this incipient phase, the lesion can produce any immediate bad effects. Certainly many patients with almost complete necrosis of the anterior lobe recover from their shock and either die in the puerperium or survive. But is the preliminary functional disturbance a factor assisting in the death of some of the obstetric shock patients? The only relevant evidence comes from rats. In the normal rat, hypophysectomy is a simple operation with a negligible mortality. If, however, the hypophysectomy is done on rats at the time of delivery, all the rats pass into a state of collapse and die. This has been shown by Jeffers[11] and confirmed by Oastler and Black working in Glasgow (unpublished observations). While this is very suggestive, any analogy between rats and human beings must of course be made with the greatest caution.

Other Organs.

In view of the findings in other types of shock, it may be noted that in obstetric shock specific lesions have not been found in the adrenals or the brain. The spleen may be quite large, but this is a common finding in multiparae who die from any cause.

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