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RENAL FAILURE AFTER UTERO-PLACENTAL DAMAGE

BY

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There are two conditions in obstetrics in which massive damage affecting the placenta, uterine muscle, or other pelvic tissues may be followed by renal insufficiency and azotaemia—accidental (retroplacental) haemorrhage (Young, 1942) and the trauma of labour (Young and McMichael, 1941). They show the same clinical sequence. (1) There is the initial tissue damage. (2) In severe cases there is "shock," which may be immediately fatal. In milder cases there may be an absence of shock. In cases which survive the shock there are (3) anuria or oliguria, with a urine containing hyaline and granular casts and leucocytes, and (4) a rising blood urea which reaches its maximum usually between the 5th and 9th days and which is followed by death or by an increasing diuresis and eventual recovery. These two clinical states resemble the "crush syndrome" (Bywaters and Beall, 1941). In the latter condition the chief histological changes are found in the kidney; these have recently been fully described by Bywaters and Dible (1942). They consist of a degenerative and necrotic lesion specially affecting the ascending limb of Henle and the second convoluted tubule, usually with the presence of pigmented (myohaemoglobin) casts. We have recently drawn attention to the similarity between the shock-azotaemic state following the trauma of difficult labour and the crush syndrome (Young and McMichael, 1941). Further evidence, to be published later, suggests that this state is by no means a rare sequel of difficult obstetrics.

The other obstetrical condition in which shock and renal failure are relatively frequent—namely, concealed accidental (retroplacental) haemorrhage—is the especial concern of the present communication. The evidence to be adduced again indicates that in the aetiology of the renal failure the same two factors emerge for consideration—the liberation into the general circulation of blood pigment which is excreted by the kidneys, and tubular damage. The clinical syndrome in concealed haemorrhage is confused by the eclamptic phenomenon (pre-eclampsia and eclampsia) which invariably accompanies it, generally in the form of severe albuminuria, hypertension, oedema, blurred vision, etc. This association is no mere haphazard one. For it has long been known that haemoglobinaemia and haemoglobinuria not infrequently occur in eclampsia associated sometimes with renal failure and with a renal lesion exhibiting blood-pigment casts and tubular degeneration. The accidental-haemorrhage syndrome is to be regarded as a variant of the pre-eclamptic and eclamptic picture, with the renal failure element thrust into the foreground.

The purpose of the following communication is to discover how far these similarities between the crush and obstetrical syndromes rest upon any basic identity in regard to genesis. In this connexion the massive ischaemic lesion in the placenta and uterine wall found in the obstetrical state calls for special consideration.

The manner in which tissue trauma leads to renal failure is uncertain. It has been suggested that the blood pigment released from the autolysed muscle may act by causing damage to the tubular epithelium or blockage of the tubular lumina. Alternatively, it is possible that the tubular degeneration is determined by the toxic action of some other material derived from the autolysis of the damaged muscle and that the blood-pigment merely represents the myohaemoglobin liberated at the same time and precipitated in the tubules during excretion. There is also to be considered the possible influence of the shock phenomena (circulatory collapse, vasoconstriction) acting alone or in combination with the other factors.

The Accidental-haemorrhage Syndrome

In some cases the patient is known to have been previously suffering from pregnancy toxaemia, and the accidental haemorrhage then forms the critical and terminal phase. In other cases the pregnancy is normal up to the acute onset. This consists of abdominal discomfort and pain, often acute. Faintness and vomiting are common, with pallor, shivering, sweating, and rapidity (sometimes slowing) of the pulse. There may be vaginal bleeding. The blood pressure may be lowered to shock levels, but this is not usual and it is apt to be followed by early death. The urine is completely suppressed or is greatly reduced in amount. It is dark red or plum-coloured and contains a large amount of albumin with hyaline and granular casts and leucocytes. Pigment casts are sometimes present. In the first sample the urine urea may be in high concentration; thereafter it quickly falls to low levels. Within two days the blood urea is raised, and may reach a high level between the 5th and 9th days. The patient succumbs about the 7th to 10th day or, alternatively, there is a free diuresis, a rapid drop in the blood urea, and eventual recovery, which may apparently be complete. This is the picture in the severe type; but all grades of severity are found, and in some cases eclamptic convulsions are present. The lesion consists of marked distension of the decidual vessels with thrombosis and free extravasation in the retroplacental area. In some instances there is an accompanying widespread haemorrhagic lesion in the uterine wall ("utero-placental apoplexy"). The area of placenta thus deprived of its blood supply dies and, in minor cases in which continuing pregnancy is possible, undergoes infarction. In severe cases the massive placental involvement results in early foetal death and delivery. But the maternal circulation may continue and the foetus may survive for hours even after as much as half of the placenta is so thrown out of the blood circuit and is undergoing autolysis. A partial circulation may even persist for some time in this affected segment. The cause of this utero-placental lesion is unknown. It is determined by a critical interference with the maternal circulation by those factors (probably generally hormonal in nature) which are commonly responsible for abortion in the early months of pregnancy. In the present instance, however, they are operating in the later months, when it is alone possible for a partial circulation to persist despite the occurrence of massive autolysis in the adjacent segment of the placenta (Young, 1914, 1942).

The Kidney Lesion.—Bratton (1941) in 6 fatal cases of antepartum bleeding with anuria or oliguria found a renal lesion resembling that seen in the crush syndrome. In none had blood transfusion been given. With the collaboration of Dr. Bratton and the medical superintendents of the hospitals concerned we have had the opportunity of studying the records of five of the cases. In three with anuria and the full renal picture the clinical evidence suggested that the patients had suffered from accidental haemorrhage between the 26th and 28th weeks of pregnancy. In one patient, as Bratton indicates in his paper, there was a haemorrhagic and necrotic lesion in the liver. In one of the remaining two cases the patient suffered from concealed accidental haemorrhage and eclampsia at the 30th week of pregnancy.

Through the kindness of Prof. R. W. Johnstone we have obtained for personal study the clinical notes and pathological material from 4 fatal cases of accidental haemorrhage. In none had blood transfusion been given. This material was submitted to Prof. J. H. Dible, who has kindly furnished the description of the microscopical appearances of the organs and the comments included in the following statement.

Case 1

Para 0+1 abortion; aged 28. March 17, 1940, 6 a.m.: Sudden onset of dizziness and dimness of vision. 8 a.m.: Severe abdominal pain and vaginal bleeding; pale, pinched, cold, and clammy; pulse 70; B.P. 130/80; urine—albumin +++; uterus tense and tender; foetal heart not heard. 1.10 p.m.: Spontaneous delivery of stillborn child weighing 4 lb. 2 oz. (36 weeks' maturity), followed by large amount of dark-red blood clot and torn-up and infarcted placenta. 1.30 p.m.: Pulse soft and irregular, but still slow; glucose-saline given intravenously, with strychnine, adrenaline, and nikethamide (coramine). 2.55 p.m. (1st day): Died. The interval between the onset of the acute symptoms and death was thus 8 hours 55 minutes.

Kidneys.—Slightly softer than usual, with on section a pale-yellow surface in which the cortex and medulla were fairly well differentiated. In the subcapsular region there were a few small haemorrhages. *Microscopical.*—"The tufts are generally rather bloodless. The capsular spaces are filled with protein material. There is marked hyaline droplet degeneration of the cells of the first convoluted tubules. The cells are swollen and desquamating; their brush borders are lost and their lumina are full of debris. This condition is continued into the descending limb of Henle. At one site in the intermediate zone a focus of necrosis with hyaline casts in the ascending limb is found. The second convoluted tubules do not show the same severe cellular change. Their cells are a little pyknotic and they tend to contain casts of a condensed and hyaline type. No pigment or necrosis is found in these tubules. A considerable number of collecting tubules show long dense hyaline casts, but these are not pigmented. *Comment.*—The lesion is an acute 'toxic' tubular nephritis. It is similar to the early stage of the crush syndrome in this respect, but differs from this in that the damage to the first convoluted tubule is more pronounced. *Other organs.*—The lungs are congested, oedematous, and emphysematous, but the liver, spleen, and heart show no noteworthy changes."

Case 2

Para 7; aged 30. Feb. 4, 1937, 11 a.m.: Slipped and fell heavily on stone stairs at 28th week of pregnancy. In the evening she had fairly severe vaginal bleeding and fainted. Feb. 5, 12.15 a.m.: Admitted to hospital; drowsiness; marked oedema of hands and feet; B.P. 135/80; a "few ounces" of dark-brown urine withdrawn by catheter—albumin +++; membranes ruptured artificially. 2 p.m.: No urine passed; catheter withdrew 60 c.cm. of "dark-brown viscid urine"; R.B.C.s and granular and hyaline casts present; blood urea 54 mg. per 100 c.cm. 7.30 p.m.: 10 drops of "oily black urine" withdrawn; under a short gas–oxygen–ether anaesthesia bipolar version was performed and 1/2-lb. weight attached to ankle of child. Feb. 6, 1 a.m.: Delivered; child 3 lb., stillborn; large amount of dark retroplacental clot. During the next 7 days patient had marked oliguria despite a large intake of glucose-saline solution. The blood urea was not recorded, but on two occasions (8th and 10th days) the urine urea was 300 mg. per 100 c.cm. The minimum blood pressure until the terminal stages was 130/70. Feb. 13 (10th day), 10.25 p.m.: Died.

Kidneys.—Moderately enlarged and fairly firm. The surface in each was diffusely congested and the cortex and medulla were fairly well differentiated. The cortex showed some increase in depth. Each capsule stripped readily, exposing a smooth surface, over which multiple petechiae were scattered. *Microscopical.*—"The glomeruli do not show any noteworthy changes. The tubules are generally dilated. The first convoluted tubules contain debris, desquamated cells, and globular masses; they are, however, fairly well preserved and there is no widespread cellular necrosis. Many of the second convoluted tubules show marked lesions. Their interior contains casts, which are made up of granular material that is lightly pigmented. The presence of this pigmented material is associated with a pronounced cellular reaction—desquamation, degeneration, and regeneration of the epithelium and some polymorph infiltration. Similar changes are seen in the ascending limb of Henle. In the region of the second convoluted tubules and in the boundary zone necrotic tubules and foci of cellular reaction (similar to those described by Bywaters and Dible) can be seen. In the medulla there are oedema and foci of lymphocytic and plasma-cell infiltration. The collecting tubules contain pigmented casts and pigmented necrotic cells and show epithelial proliferation. *Comment.*—The picture is practically indistinguishable from that found in the kidney in cases of the crush syndrome. *Other organs.*—The liver and heart do not show any noteworthy changes."

Case 3

Para 0; aged 24. June 1, 1939: Admitted because of oedema of ankles at term; B.P. 130/100. Remained satisfactory. On June 17, 5 a.m., artificial rupture of membranes. 6 a.m.: Labour started. 2 p.m.: Vomiting. 11 p.m.: Sudden onset of severe abdominal pain with collapse and sweating; foetal heart ceased and concealed accidental haemorrhage diagnosed; 1/4 gr. morphine given, and intravenous drip saline. 12 midnight: Delivered by easy low forceps after craniotomy to diminish size of head. Placenta expelled one hour later: no blood loss. Examination of the placenta showed a portion which seemed to have separated prematurely. There was marked oliguria (the maximum daily output being 140 c.cm. on 4th day), and the blood urea on 7th day was 164 mg. per 100 c.cm. Treatment: glucose-saline, 10% glucose, sodium sulphate, 20% glucose. The lochia became offensive. Coma developed and on June 25 (9th day), at 10.10 a.m., the patient died.

Kidneys.—Both swollen and moderately firm. Abnormally broad pale cortex and reddish-brown medulla, both areas being oedematous. Each capsule stripped readily, exposing a smooth pale surface. *Microscopical.*—"The glomeruli tend to be bloodless but show no anatomical change. The first convoluted tubules contain a good deal of debris, are generally dilated, and their epithelium is flattened; the cells are not well preserved and there is some widespread necrosis. The second convoluted tubules are pyknotic. Many of them contain lightly pigmented granular cast material. In connexion with this there is epithelial desquamation and necrosis, some of the necrosed epithelial cells absorbing pigment. Most of the nephrons are affected. Changes in Henle's tubules are minimal. The medulla shows lymphocytic and plasma-cell infiltration. A majority of the larger collecting tubules contain pigmented casts, reproducing the picture of 'blockage' seen after intravascular haemolysis—as, for example, in fatal incompatible blood transfusion. *Comment.*—This picture is essentially similar to that found in the kidney in the fatal anuria of the crush syndrome. *Other organs.*—Liver: Typical patchy eclamptic necrosis. Lung: Oedema, emphysema, acute bronchitis, and early bronchopneumonia. Pituitary: Acute necrosis of the anterior lobe. The heart and spleen show no noteworthy changes."

In the fourth case the appearances were those of bilateral cortical necrosis, the rare lesion which has long been associated with cases of accidental haemorrhage resulting in death with uraemic symptoms. The difference in the causal factors responsible for two such striking variants in the renal lesion is a matter for further study. Meanwhile it is interesting and perhaps relevant that cortical necrosis has been described in a case of traumatic anuria (McFarlane, 1941).

Discussion of the Aetiology of the Renal Failure

The following factors, operating singly or in combination, call for consideration in this connexion: (1) shock, (2) haemolysis and tubular blockage, and (3) a toxic material or toxic materials of tissue origin. For this study we have 5 personal cases associated with renal impairment and a significant rise in blood urea out of a total consecutive series of 79 cases of accidental haemorrhage. All 5 patients recovered.

Shock

This may be severe, and a considerable proportion of the mortality from accidental haemorrhage is due to this cause. That the collapse of haemorrhage does not in the present series explain the kidney lesion is suggested by the fact that,

in 59 consecutive cases of obstetrical haemorrhage (post partum, from placenta praevia, accidental haemorrhage) so grave as to require blood transfusion, renal failure is restricted to the accidental-haemorrhage group (Table I).

TABLE I.—*Severe Haemorrhage at Childbirth necessitating Blood Transfusion. Analysis of 59 Consecutive Cases*

	Cases	Renal Impairment
Post partum	37	0
Placenta praevia	12	0
Accidental (retroplacental)	10	5

This table demonstrates the correlation between renal impairment and accidental haemorrhage and suggests that the former is determined by some factor other than the shock of haemorrhage. Further, it can sometimes be clearly established that the renal impairment antedates the shock phenomena.

Case 4.—Mrs. M., aged 27, walked into hospital at 7 p.m. with lower abdominal pain and thinking she was in labour. She was unable to pass urine, and next morning the catheter revealed an empty bladder. The general clinical condition was good; blood pressure 120/80 mm. Hg. During the rest of the day the suppression continued. At 5.30 p.m. she developed acute abdominal pain with severe shock. Diagnosis: Concealed accidental haemorrhage. Bladder empty. The suppression of urine had thus existed for 22½ hours before the first onset of shock.

Case 5.—Mrs. E., aged 37, wakened at 7 a.m., rose, and passed urine as usual. She prepared her husband's breakfast, and later in the morning, in her usual health, went shopping. At 11 a.m., on the way home, was seized with abdominal pain; walked home and went to bed. At 12 noon felt sick and vomited. Sent to hospital at 5.30 p.m. in condition of shock (with pallor, shivering, perspiration, and rapid pulse), but with B.P. 155/98. Diagnosis: Concealed accidental haemorrhage. 3 c.cm. urine withdrawn by catheter. No urine had been passed since 7 a.m. She thus had suppression for 10 hours, and this antedated the shock.

These cases further exemplify how the causal agency has a selective action on the kidney.

An added argument against the view that the renal failure is a local expression of general circulatory collapse due to shock is the frequent absence of hypotension. In 4 out of the present series of 5 cases of accidental haemorrhage the blood pressure was recorded at frequent intervals, and the respective minimum readings up to the time when the kidney impairment was firmly established were 100/70, 110/90, 155/98, 145/86. The relatively high pressure levels in some of these women are clearly related to the coexisting hypertensive toxaemia.

Dunn and Montgomery (1941) have suggested that bilateral cortical necrosis, the rare variant of the renal lesion, which is also remarkably selective in its character, may be due to circulatory failure from haemorrhagic "shock", with complete stagnation of the blood flow in glomeruli previously damaged by the eclamptic "poison." Such an explanation, however, does not seem to be valid for the lesion now under discussion. We have seen (1) that the selective lesion may be tubular, with a relative immunity of the glomeruli; (2) that renal failure does not follow the shock from other forms of obstetrical haemorrhage (post partum and from placenta praevia) complicating the eclamptic state; (3) that the renal failure may develop before the "shock" while, in other cases, there may at no time be hypotension.

The data invite some further tentative conclusions on the aetiology of the shock phenomena. In accidental haemorrhage shock may be severe and even fatal, and in some cases the blood loss by itself is clearly a determining factor. That, however, there may be another factor, or other factors, is demonstrated by those cases in which the degree of shock is out of proportion to the amount of bleeding (Whitridge Williams, 1925) and by the frequency with which it fails to respond to the therapeutic influence of blood transfusion (Sheehan, 1942). In the evolution of shock in surgical trauma the fall in blood volume caused by the loss of plasma to the damaged tissues is believed to play an important part. In the case of accidental haemorrhage there is no clear evidence of such a mechanism. Although there may be oedema of the uterine wall (especially when the retroplacental bleeding is associated with a muscular lesion—"utero-placental apoplexy") this local loss of blood fluid to the tissues cannot be regarded as an adequate explanation of the total phenomena. We have not found evidence of haemoconcentration, though this may, of course, be obscured by the haemorrhage, and it is known that the eclamptic state may be accompanied with marked haemoconcentration due to the explosive generalized oedema (Zangemeister, 1903). This haemoconcentration, which may develop rapidly in the crisis of the eclamptic state, may, however, be associated with an equally rapid rise in blood pressure, even to high levels, and with a clinical condition which in this respect is the reverse of shock (Dieckmann, 1942). Further, in many instances, although the shock phenomena may be clinically brisk, the accompanying hypertensive state, as in the 4 surviving cases reported in this communication, would seem to protect the patient against the major risks. It is only when the "shock factor" breaks through this protective hypertensive state and paralyses the vasomotor mechanism that the full shock effect is induced. Whether this action is central or peripheral it is impossible to say. This view of the genesis of the initial shock often seen in such cases resembles the conclusions of Noble and Collip (1942) based upon the study of experimental trauma in rats. They conclude that a poison is generated by the damaged tissues and that this can cause death rapidly from shock with little haemoconcentration. Only where death is delayed is there the development of a severe haemoconcentration due to the loss of blood plasma through the capillaries. Glen (1941) has shown, also, that the shock may develop so rapidly after the restoration of the circulation in an ischaemic limb as to suggest the immediate effect of a circulating poison.

We may summarize the above evidence as follows. The data are opposed to the view that the shock mechanism is essential for the production of the renal lesion. This is dependent upon some other influence whose action on the kidney may be strikingly selective. Neither haemorrhage nor the loss of blood fluid to the tissues (local or general) with haemoconcentration is an adequate explanation of the shock phenomena. To explain the full shock effect we must postulate some other factor (toxic agent?).

Haemolysis and Tubular Blockage

The eclamptic phenomenon is apt to be associated with haemoglobinaemia and haemoglobinuria. Many references to these conditions are to be found, especially in German literature. Schmorl (1893), in 2 out of the 17 cases on which his classical monograph on the pathology of eclampsia was based, described the presence of yellow pigment in the collecting tubules of the kidney. In one case, in which the urine was plum-coloured, the pigment was also present in the epithelial cells. Brütt and Schumm (1918) reported 6 cases of eclampsia with haemoglobinaemia in which there were haematin casts in the urine. In a fatal case with marked oliguria there were abundant haematin casts in the straight and Henle tubules. Fahr (1924) found haemoglobin casts in the kidney tubules in 18 out of 33 fatal cases of eclampsia, and regards this appearance as next only in importance to the glomerular changes in the total renal lesion of eclampsia. He points out that this incidence is higher than one would expect from the frequency of haemoglobinuria as clinically recorded in eclampsia by Meyer-Wirz and Geipel (6 in 44), Konstantinowitsch (6 in 30), and Zangemeister (5 in 14). At the same time he points out that the haemoglobin casts are often few in number. In general these German writers have regarded this haemolytic process as one of the side-effects of the eclamptic "poison"—an effect which it shares with other poisons, such as arsenic preparations, nitrobenzol, quinine, etc. The haemoglobinaemia and haemoglobinuria are clearly related to the acme of the eclamptic attack, and subside quickly thereafter. The Continental investigators were also aware of the association between haemolysis and the azotaemic type of renal failure dependent upon tubular degeneration (Brütt and Schumm, 1918; Seitz, 1927; Spitzer, 1934). In this country Dunn and Baird (1933) found haemoglobin casts in the kidneys in 3 out of 10 cases of eclampsia.

These investigations have acquired a new significance by their revelation of the close resemblance between the haemolytic phenomenon in eclampsia and its accompanying renal lesion and the changes found by Bywaters and Beall (1941) to follow a massive ischaemic lesion of muscle and by Bratton (1941) and ourselves in the accidental-haemorrhage syndrome. It has been suggested that in the crush syndrome the anuria may be due to blockage by the pigmented casts. But, as Bywaters and Beall (1941) have pointed out, any urine excreted in such cases may differ little from a glomerular filtrate, thus indicating the existence of severe damage even in non-obstructed tubules. Further, in an early case there may be tubular lesions in the absence of casts.

The same considerations apply to the kidney in accidental haemorrhage. In a woman who died 9 hours after the onset of symptoms there were no pigmented casts, but the tubular degeneration was evident; whilst in another case, with 22½ hours' anuria, pigmented casts were absent. We have had the opportunity of examining the kidneys in two women who died with anuria, 6 days and 9 days respectively after an eclamptic attack. In the former case there was a typical tubular lesion with blood casts and a blood-urea figure of 173 mg. per 100 c.cm.; while in the latter, in which the tubular degeneration was characteristic and the blood-urea reading 222 mg. per 100 c.cm., there were no pigmented casts. These findings suggest that the blood casts are necessary neither for the initiation of the renal failure nor for the development of the renal lesion.

Toxic Material or Materials of Tissue Origin

The elimination of competing factors in the aetiological field serves to direct attention to the immediate influence of the massive tissue damage, which establishes a basic link between the obstetrical and the crush syndromes. This brings us to the correlation between the "concealed" type of accidental haemorrhage and renal impairment (Table II).

TABLE II.—*Relation of Type of Accidental Haemorrhage to Renal Impairment. Analysis of 79 Consecutive Cases*

Haemorrhage	Cases	Albuminuric Toxaemia	Toxaemia + Renal Impairment
Revealed ..	59	7 (11·8%)	0
Concealed ..	20	13 (65·0%)	5 (25%)
Totals ..	79	20	5

These results conform to those of other observers. The table shows that the albuminuric type of toxaemia is much more frequent in "concealed" (65.0%) than in "revealed" (11.8%) cases, and that the renal insufficiency is associated especially with the "concealed" type.

The distinctive features of the utero-placental lesion of "concealed" accidental haemorrhage have been discussed elsewhere (Young, 1914, 1942). One feature of immediate relevance is that after the production of the lesion, which in the cases we are discussing generally involves as much as half and sometimes even more of the organ, the placenta is retained for some time within the uterus. Where the circulation is maintained in the undamaged portion, with resulting foetal survival, the conditions are such as to favour the escape into the systemic blood stream of any toxic materials which may be generated in the dying area. The circumstances are then similar to those which have been postulated in the case of surgical trauma after the circulation has been re-established in the crushed limb. To this analogy we referred many years ago (Young and Miller, 1921). Elsewhere we (Young, 1942) have given evidence that the "toxaemic" state is immediately related to the massive placental lesion. Thus there is a recognizable quantitative relation between the severity of the toxaemia, on the one hand, and the mass of placenta involved and the period of foetal survival, on the other. When the lesion is so fulminating as to cause complete initial detachment there is no toxaemia, and where, after the onset of toxaemia, the lesion progresses to the stage of causing foetal death there may be an abrupt decline of the toxaemia. This evidence, and evidence of a like nature in ordinary cases of eclampsia and pre-eclampsia, in which, however, there is generally no history of accidental haemorrhage, have formed the basis of the placental hypothesis of the eclamptic phenomenon (Young, 1914, *et seq.*). Evidence has been given that the interference with the uterine circulation which causes the placental lesion is the expression of an antecedent abortion taint (Young, 1927, 1942).

It is firmly established that renal failure with azotaemia is rare in eclampsia and pre-eclampsia. The relatively high incidence (25% in the present series) of renal impairment in concealed accidental haemorrhage would seem, therefore, to be determined by some factor introduced into the eclamptic and pre-eclamptic state by the conditions obtaining in concealed haemorrhage. What this factor is we do not know. It does not seem to connote a quantitative relation to toxaemia, for renal failure is rare even in severe eclampsia not associated with concealed haemorrhage. The massive muscular lesion in the uterus, to which reference has already been made, is especially identified with cases of concealed haemorrhage, and it provides a striking analogy to the conditions in the crush syndrome. But there is some evidence that this lesion may be present in a marked degree without renal failure (Whitridge Williams, 1925; Dieckmann, 1936). Nevertheless it merits fuller consideration. Its existence helps to explain the very high incidence of puerperal sepsis in such cases (Whitridge Williams, 1925; Gibberd, 1936; Bratton, 1941).

The immediate cause of the bleeding in the decidua and muscularis is not clear. There would seem to be no doubt that it is an expression of the "abortion taint," to which reference has already been made and which has been discussed more fully elsewhere (Young, 1942). Reasons have been given for the view that it precedes the placental autolysis (Young, 1914, 1942). This evidence is, however, not unequivocal, and it remains possible that the placental degeneration antedates and determines the retroplacental and muscular lesions. It cannot be claimed that the haemorrhagic lesions are due to a systemic "toxaemia," because out of 79 cases of accidental haemorrhage albuminuric toxaemia was absent in 59, including 7 of the 20 cases of the concealed type (Table II). It is possible, however, that the massive blood effusions in these sites are due to vascular damage by a toxic agent locally produced, and that the purpuric and haemophilia-like haemorrhages occasionally found in accidental haemorrhage in such distant sites as the nose, stomach, and subcutaneous tissues (McGoogan, 1935; Dieckmann, 1936) may be a more generalized manifestation of the same factor. On this question further information is desirable.

Summary

The foregoing clinico-pathological study of the syndrome in concealed accidental haemorrhage suggests the following proximate causes: (1) The syndrome is determined by a massive utero-placental lesion of ischaemic origin. (2) The renal failure characterized by tubular degeneration and azotaemia is determined by a toxic material derived from tissue autolysis. (3) The shock element and the haemolysis which are frequently present may have a similar origin.

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