Blood cultures taken at this time became positive on Sept. 30, and on the same day the spleen became palpable for the first time.

Treatment with 1 g. of streptomycin eight-hourly intramuscularly, and 2 g. of sulphadiazine four-hourly by mouth, was begun on Sept. 24 and continued for fourteen days. On the sixth day of treatment fever began to fall. By the end of treatment the patient was afebrile, but immediately after its cessation occasional pyrexia up to 99° F. (37.2° C.) recurred; but this disappeared without any further treatment, and the fever has not recurred up to the present time over the last two months. There have, however, been during this time several attacks of abdominal discomfort, to which she has been prone for the last few years. The agglutinin titre for *Br. abortus* had fallen to 1 in 1,250 on Oct. 19 and to 1 in 500 on Nov. 26. The splenomegaly had disappeared two weeks after treatment was completed.

Characters of Br. abortus.—This organism was found in only one of many tubes after incubation for twelve days in 10% CO₂. On first isolation it was fully CO₂-sensitive, but after four subcultures growth, poor at first but later fairly profuse, was obtained in a normal atmosphere. The sensitivity to streptomycin was tested by preparing slopes of beef-heart extract peptone agar of pH 7.4 containing falling concentrations of streptomycin and inoculating them with a 2-mm. loopful of an approximate 1 in 500 dilution of a broth culture. The minimum inhibitory concentration was found to vary with the atmosphere as follows:

	Minimum Inhibitory Concentration	Maximum Concentration Permitting Growth
No added CO ₂ ..	0.5 µg. per ml.	0.25 µg. per ml.
10% CO ₂ ..	1.5 " "	1.0 " "
25% CO ₂ ..	2.5 " "	2.0 " "

These differences are evidently due to the alteration in pH of the medium produced by CO₂: it is well recognized that even a slight shift towards the acid side diminishes the activity of streptomycin. The findings indicate that this strain is fully streptomycin-sensitive.

Comment

Of these two cases, one was of several months' duration and the other was recent. Both responded to combined treatment with streptomycin and sulphadiazine, the former with a fall in temperature to normal within three days, the second somewhat more gradually, the temperature reaching normal only at the end of the course. They have now been afebrile for six and two months respectively. These observations confirm American reports of the efficacy of this treatment. We do not claim that the optimum scheme of dosage has been defined, but from the successful results in these two cases and the case described by Eisele and McCullough, all of which were given 12 g. of sulphadiazine daily, contrasted with the relapses in Pulaski and Amspacher's series, it seems likely that the larger dose is necessary.

We are indebted to the Streptomycin Clinical Trials (Non-Tuberculous Conditions) Committee of the Medical Research Council for the streptomycin used in the treatment of these patients, and to Mr. Daniel T. Davies for earlier clinical details of Case 1.

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THE TREATMENT OF ANURIA

BY

D. A. K. BLACK, M.D., M.R.C.P.

AND

S. W. STANBURY, M.B., M.R.C.P.

(From the Department of Medicine, the Royal Infirmary, Manchester)

Although anuria is not a common emergency in medical practice it is an important one, partly because two of the commoner forms of anuria are iatrogenic and partly because persistent anuria is surely followed by death. The causes and treatment of anuria have alike been illuminated in the past few years, the one by important advances in our knowledge of renal function, the other by the painstaking ingenuity of workers like Kolff and Alwall in devising means of maintaining excretion during what is hoped may be a temporary suspension of renal activity.

Suppression of urine has been observed in so many different disease states that it is doubtful whether the same underlying mechanism can be present in all. Sometimes, as in acute nephritis or bilateral urinary obstruction, there is a clear background of morbid anatomy to account for the anuria. Sometimes, as in patients with shock or dehydration, the anuria is just as clearly the result of the patient's general state, and the kidneys cannot be primarily incriminated. The greatest interest, however, attaches to those types of urinary suppression, such as transfusion kidney and the crush syndrome, in which the ultimate cause is extrarenal but the kidneys show definite morbid anatomical changes. Further examples of anuria, not primarily renal, in which there are renal changes of doubtful significance may be found in sulphonamide anuria, blackwater fever, anuria following abortion, and poisoning with uranium or mercury salts.

Pathogenesis of Anuria

Leaving aside the primary aetiology of anuria, which concerns prophylaxis, we come to the mechanism or mechanisms by which anuria may be produced; it is here, if anywhere, that we may expect to find clues to the rational treatment of the established condition. We shall exclude from present consideration anuria associated with a definite pre-existing renal lesion or gross urinary tract obstruction and assume that we are dealing with a kidney which is normal so far as the general arrangement of nephrons is concerned. In the normal kidney the raw material of urine is formed in the glomeruli, and the tubules have the function of transmitting glomerular filtrate to the renal pelvis, modifying it on the way so that the dilute ultrafiltrate of plasma becomes the concentrated and highly selected final urine. A failure of the glomeruli to form filtrate, and a failure of the tubules to transmit it to the right place, would alike be followed by anuria. The possibilities for impaired function of glomeruli and tubules can be considered separately.

Normally the glomeruli filter off about one-fifth of the plasma which is presented to them, and so far as is known this process is a purely physical one, influenced only by the amount and pressure of glomerular blood flow and by the physical permeability of the glomerular membrane. A loss of glomerular permeability need not be seriously considered as a cause of complete anuria, but alterations in the supply of blood to the glomeruli are obviously important. Van Slyke (1948) has reviewed extensive experimental work carried out on renal function during shock; when blood volume and cardiac output are diminished the renal

blood flow falls to a much greater extent than does the cardiac output. The dog's kidney can survive experimental deprivation of its blood supply for as long as two hours, a time which causes irreversible renal damage in the rabbit. Badenoch and Darmady (1947) express the opinion that more than six hours' occlusion might be required in man to cause irreversible renal damage. Results obtained in man by clearance techniques show very low renal blood flow after haemorrhage (Black *et al.*, 1941) and during surgical shock (Lauson *et al.*, 1944).

Until recently a partial or complete "renal shutdown" was the only form of circulatory upset that had to be considered; but Trueta, Barclay, and their colleagues (1947) have shown that in rabbits a shunting of the circulation within the kidney may occur so that the glomeruli of the renal cortex are no longer fully perfused. They found anatomical and radiological evidence of an alternative circulatory pathway through large glomeruli in the boundary zone of the kidney and the medullary vasa recta. Although the vessels involved in the Barclay-Trueta shunt can be demonstrated in man, direct proof of the occurrence of the shunt in man has not yet been obtained, and the effect of the shunt on renal function is also not fully established. While it is certain that the shunt will not explain all the unsolved problems of renal disease, the possibility of its presence cannot be neglected in any of them. When a technique has been worked out for determining in the intact patient whether the shunt is in operation or not, then it may be found that some types of anuria, such as reflex anuria, which are difficult to explain in any other way, involve the presence of a shunt.

There are, however, several types of anuria, such as sulphonamide and mercurial anuria, in which it is hard to conceive of the renal damage as being due to suppression of glomerular filtration. Damage to the renal tubules could cause anuria in several ways—by blockage, by excessive reabsorption, or even by direct communication between the distal tubule and the blood stream. Tubular blockage of significant degree may be found in multiple myeloma, in transfusion kidney, and in sulphonamide anuria, though here the blockage may be more of pelvis and ureter than of the tubule proper. It is exceptional, however, for tubular obstruction to be universal, and the general opinion is that simple blockage will not account for complete anuria in such conditions as blackwater fever (Maegraith, 1944).

More important probably than actual blockage of the tubules is damage to the tubule epithelial cells of such a nature as to destroy the selective permeability on which the formation of urine from glomerular filtrate depends. The changes which have been observed in the tubule cells in functional renal failure range from hyaline droplet change to complete necrosis and desquamation, followed later by regeneration. The lack of constancy in the histological picture, and the known liability of the renal tubules to early post-mortem change, have led to relative neglect of the part played by the tubules in the pathogenesis of oliguria and anuria. However, they have been restored to full prominence by the work of Bywaters (1941, 1944) on the crush syndrome, and of Lucké (1946) on the post-traumatic syndrome, which he designates "low nephron nephrosis." Moon (1948) accepts Lucké's views on the importance of tubular lesions in renal damage following "shock," but denies that the lesions are limited to the distal segment of the nephron. Shaw Dunn used the telling metaphor of "a morass" to describe the state of affairs in which fluid leaks freely back from damaged tubules into the peritubular blood vessels; he was the first to demonstrate direct communications between tubules and veins similar to those found in experimental phosphate poisoning (Dunn *et al.*, 1941).

Summarizing present views on the pathogenesis of anuria, it may be said that emphasis in this country has generally been laid on circulatory changes; Maegraith *et al.* (1945) put forward the unifying hypothesis of "renal anoxia" to account for many types of functional renal failure, and this view has derived support from the discovery of unsuspected possibilities of disturbance in the renal circulation. On the other hand, in a recent American symposium on "renal insufficiency in the shock syndrome" Moon (1947) and others emphasized the tubular damage, so much more impressive anatomically than anything that can be seen in the glomeruli of such kidneys. These two views represent a difference in emphasis rather than a complete contradiction, for any serious impairment of the circulation to the glomeruli is likely to cause damage to the tubules, whose blood supply is dependent on glomerular integrity; and we might even hazard a guess that in the long run both circulatory and tubular factors will be proved important, though in varying degree from patient to patient. The past history of renal physiology should warn us against the "either-or" type of mental approach to this fascinating and versatile organ.

Methods of Treatment

The sudden occurrence of anuria after operation or transfusion may produce a mood of therapeutic desperation in which all sorts of procedures have been carried out, from dry cupping to decapsulation. Since urine flow may be re-established quite spontaneously at any time it is impossible to assess the various possible modes of therapy by the usual method of considering their results, for no single centre is likely to encounter enough anuric patients to do a controlled trial of even one method of therapy. Since reliable conclusions cannot be drawn from the apparent results of therapy, we can only consider the proposed ways of treatment in relation to our present information on the pathogenesis of anuria.

In a general way the objectives of treatment are to restore urine flow, and in default of this to prevent such distortion of the patient's internal environment that he will succumb before urine formation has been effectively restored. Believers in circulatory changes as the usual cause of anuria will approach the task of restoring urine flow with greater optimism than those who lay stress on tubular degeneration, for it is less likely that tubular lesions will be much influenced by any form of active therapy. The vasoconstrictor nerves to the kidney are sympathetic, and Barclay and Trueta found that splanchnic section would prevent the development of the renal shunt in rabbits. On this basis, anuria has been treated by paravertebral block, or by high spinal analgesia as a more certain way of blocking the entire sympathetic supply to the renal vessels. Successes and also failures have been reported with both methods, but it is never possible to exclude an opportune spontaneous recovery of urine flow. Nevertheless, it would seem justifiable to attempt splanchnic block, and the use of sympatholytic drugs such as tetraethylammonium bromide or dibenamine might be considered as an alternative to the use of local or spinal analgesia. Although such procedures may lower blood pressure for a short time they can safely be applied in patients without peripheral circulatory failure, and in a proportion of cases so treated there has been apparent success.

The use of alkalis and of diuretics, also designed to restore urine flow, is much more questionable. Mercurial diuretics would be ineffective on the "circulatory theory" and harmful on the "tubular damage" theory. Osmotic diuretics such as sodium sulphate are commonly used in surgical anuria, and Olson and Necheles (1947) report favourably on the use of sodium sulphate in dogs with

anuria secondary to experimental burns. Sodium sulphate acts osmotically by diminishing the proportion of glomerular filtrate which is reabsorbed, and it is difficult to see how it can be effective unless some degree of glomerular filtration is already established. The cessation of urine flow after experimental burns is obviously different in mechanism from other types of anuria, and the results of Olson and Necheles cannot be generally applied. Sodium sulphate may have a place in the treatment of extreme oliguria, but in complete anuria any addition of osmotically active substances to the body fluids is risky. Treatment with alkalis, although it may be effective prophylactically in preventing haemoglobin precipitation, cannot destroy casts already present in the tubules, and in any event tubular blockage is not considered an important cause of anuria. In the presence of anuria even small doses of alkalis are retained, and alkalosis is a very real danger. The only indication for giving alkalis in anuria is the presence of acidosis, and in such cases correction of the acidosis may be followed by the restoration of urine flow (van Slyke, 1948).

While attempts to restore urine flow are being made, attention has also to be directed to the prevention so far as possible of clinical and biochemical deterioration. On the "tubular damage" theory, conservative measures are the sensible ones to adopt, and this is far from being therapeutic nihilism, for the kidneys in fatal anuria commonly show extremely active regeneration of tubular cells in injured areas (Corcoran and Page, 1947), giving hope that artificial maintenance of excretory and homeostatic function for a longer period might have allowed recovery. Moreover, correct maintenance therapy demands an attention to important detail for which "doing nothing" is the least suitable epithet imaginable.

Protein Metabolism

Although no one now believes that urea itself is responsible for the symptoms of uraemia, the estimation of urea or of non-protein nitrogen can still be used as a convenient measure of the extent to which the body is failing to get rid of harmful end-products of protein metabolism. Maintenance therapy in anuria should aim both at reducing the amount of protein which is being broken down and at eliminating nitrogenous and other waste products by extra-renal channels.

Nitrogen catabolism is at its lowest when the intake of food protein is minimized and the calorie needs of the body are fully supplied by carbohydrate and fat. Borst (1948) has used a butter-sugar diet which has a high calorie value and a negligible protein content; the urinary nitrogen falls to very low levels in normal people on this type of diet. Such a diet is, however, remarkably unpalatable, and would not seem to offer any corresponding advantage over a more varied diet containing about 30 g. of protein per day and 2,500 calories, made up mostly in fat and carbohydrate. So long as the amount is small it does not seem greatly to matter whether the irreducible minimum of protein for catabolism comes from the food or from the tissues; but it has to be remembered that infection may increase the rate of tissue breakdown, and Borst (1948) records a case in which infection seemed clearly responsible for the very rapid development of uraemia. There is therefore a clear indication for the active treatment of any infective process in an anuric patient as part of the programme for cutting down the breakdown of protein. Another likely cause of accelerated protein catabolism is disturbance of salt-and-water metabolism, and the fluid balance demands careful attention for this reason, as well as in its own right. As a general approximation, in a temperate climate the urine accounts for about half of our total fluid loss; suppression of urine

therefore calls for some restriction of the fluid intake, and so long as no urine is being passed a litre of fluid a day is sufficient for any patient who is not visibly sweating and has no fever. Restriction of salt intake is even more important, for in the absence of urine formation the only significant remaining channel of salt loss is the sweat, and sweat has a salt content much lower than plasma or extracellular fluid. When the kidneys fail the body has no means of excreting salt in hypertonic solution, and so cannot protect itself from being pickled by excessive salt in the diet. A "salt-free" diet, in the conventional sense, should accordingly be used. The essential value of such conservative treatment is to prolong the period of survival in total anuria and so allow more time for repair of kidney tubular structure and function.

Elimination of Waste Products

As means towards the same end many methods have been tried for getting rid of waste products by routes other than the kidneys. Sweating and purging are inefficient ways of lowering the blood urea, because the strain imposed on the patient and the amount of fluid lost are out of proportion to any decrease in the urea content of the body which can be achieved. A more helpful approach is the continuous dialysis of blood in a "cellophane" tube against some form of Ringer-Locke solution; as alternatives to a cellophane tube the peritoneum and the mucosa of isolated loops of bowel have been used as dialysing membranes. Kolff (1947) gives an extended account of the use of methods of this type, and it is quite clear that in special clinics these methods are safe and can be useful in getting rid of significant amounts of urea and presumably of other waste products. On the debit side, these methods are far from being generally applicable with safety, for they involve the use of heparin when blood is the perfusing substance, or of large amounts of aseptic solutions when the body cavities are used for perfusion. An isolated loop of bowel avoids some of these troubles, but it is likely to become atonic and even to rupture. An ingenious method described by Maluf (1948) avoids the necessity for heparin or any operative procedure, or the use of aseptic irrigating fluid.

In this method a triple Miller-Abbott tube is introduced until the balloon lodges in the caecum; slightly hypertonic sodium sulphate solution is run into the duodenum and withdrawn from the terminal ileum, the rate of flow aimed at being 20-30 ml. per minute. Early reports of the use of this type of method are now appearing (Oppenheimer and Rosenak, 1948; Odel and Ferris, 1948), and we have ourselves had the opportunity of using it in one patient, whose case history is appended. Like other users of the method we have encountered two main practical difficulties. First, it is difficult to get the tube into the right place without provoking nausea. Secondly, we found it quite impossible to get back the solution at anything like the recommended rate, for any tube which can reasonably be passed is much too fine to allow adequate suction of large amounts of fluid. The first difficulty can be overcome only by patience, and by letting the tube stay down once it is down, avoiding the need for frequent manipulation. The second difficulty resolves itself, with some discomfort to the patient, by the passage of copious watery stools. It would seem reasonable, instead of trying to suck the fluid all the way back to the mouth, to let it flow on through a simple duodenal tube and withdraw it by a wide rectal tube. Although we had these initial difficulties it was possible to rid the body of 15 g. of urea; this amount is much larger than that recorded by Odel and Ferris, using the same principle. This may be partly because they employed only a modified Ringer solution, while we followed

Maluf (1948) in using a slightly hypertonic sodium sulphate solution (in our case 4.5 g. per 100 ml.). The use of the method leads to loss of sodium chloride from the body as well as of urea, and the most convenient way of dealing with this is to carry out dialysis during the day and run in normal saline or Ringer's solution by slow drip through the tube during the night, the amount of saline used depending on the amount found to be lost in the dialysate.

Restoration of urine flow, spontaneous or otherwise, is not synchronous with full recovery of renal function, and an apparently satisfactory diuresis may coexist with low values for renal function tests. The patient described here, with post-transfusion anuria, had inulin and "diodrast" clearances of only about half the normal some weeks after urine flow was re-established. Moreover, we have observed inulin-diodone clearance of only a few millilitres per minute in two patients recovering from extreme oliguria following abortion, at a time when both of them were passing large volumes of urine. Patients who have had an episode of anuria should therefore be followed up in much the same way as those who have had an attack of acute nephritis.

Summarizing our present views on the treatment of anuria, we would say that an attempt should be made as early as possible to induce a splanchnic block by medical or surgical means. When this measure fails, as it often will, the patient should be maintained on a diet with about 30 g. of protein, a fluid intake limited to one litre a day, and a low salt intake. If spontaneous restoration of urine flow is delayed for more than a few days intestinal dialysis should be carried out in order to lengthen the time available for repair of the damaged kidneys.

Case Report

A housewife aged 48 was admitted to another hospital for hysterectomy on account of severe menorrhagia and anaemia. On March 30, 1948, she was given a pre-operative blood transfusion of 1 pint (570 ml.) of compatible group O packed red cells followed by 1 pint of incompatible stored group A whole blood. She developed lumbar pain and tightness in the chest during the second infusion of blood, and from that time there was virtual anuria. We first saw her on April 2, when the daily output of urine was probably less than 50 ml. The patient appeared well apart from anaemia (60% Haldane) and there was no jaundice (serum bilirubin 0.3 mg. per 100 ml.). There was a constant slight uterine haemorrhage, making accurate urine collection impossible, but no urine was obtained by catheterization. The blood pressure was 155/100 and the blood urea was 120 mg. per 100 ml.

The following measures were carried out in an attempt to establish urine flow. On April 2 she was given 300 mg. of tetra-ethyl ammonium bromide intravenously.

This produced a slight fall in blood pressure. On April 3, in an attempt to produce intrarenal vasodilatation (Smith, 1943), a pyrexial reaction was induced by intravenous T.A.B. vaccine (25 million organisms). On the 4th, spinal analgesia to D2 produced a small fall in blood pressure, but, like the other two procedures, failed to induce diuresis. Urine production was still either absent or minimal. The blood urea had risen to 154 mg. per 100 ml. on April 5, and it was decided to attempt intestinal perfusion. A double-lumen intestinal tube without balloon was passed and its entry into the jejunum confirmed by fluoroscopy.

A 4.5% solution of $\text{Na}_2\text{SO}_4 \cdot 10\text{H}_2\text{O}$ was led by gravity into the duodenum at the rate of 1 pint in half an hour. Considerable difficulty was experienced in aspirating from the distal end of the tube, and vomiting terminated the attempt. Seven pints (4 litres) of solution had been infused and a watery diarrhoea induced; 2,520 ml. of watery stools was recovered with a urea content of 150 mg. per 100 ml. A total of 3.78 g. of urea was recovered in this way, and the urea content of the watery faeces reached 97% of the blood level.

The tube was left in the stomach and slowly advanced the next day, glucose-saline solution being administered. On April 7, the ninth day of oliguria, the patient was drowsy and apathetic and the blood urea was 186 mg. per 100 ml. Perfusion was restarted and 10 pints (5.7 litres) was infused in about six hours. Aspiration was now more successfully accomplished, but in spite of this a profuse diarrhoea was produced and a not inconsiderable volume of fluid was lost by incontinence. The fluid, urea, and chloride are shown in the accompanying Table. Towards the end of this perfusion the patient

Date	Na_2SO_4 Infused	Aspirated Dialysate			Watery Faeces		
		Volume (ml.)	Cl (as NaCl) mg./100 ml.	Urea mg./100 ml.	Volume (ml.)	Cl (as NaCl) mg./100 ml.	Urea mg./100 ml.
April 5 ..	7 pints	5,880	70	34	2,520	140	150
" 7 ..	10 "				5,460	59	184
" 8 ..	3 " +	Not collected					

experienced scalding and was aware of passing urine for the first time since admission to hospital (see Chart). The following day she was brighter, and since urine flow was established no further perfusion was carried out although the tube remained indwelling. The blood urea was now 166 mg. per 100 ml. Taking into account the steady daily rate at which the blood urea had been rising, a level of approximately 200 mg. per 100 ml. might have been anticipated. Reckoning a body fluid volume of 50 litres, this represents an extrarenal excretion of approximately 17 g. Actually 14.7 g. of urea was recovered and the deficit might be accounted for by incontinence. On subsequent days the urine volume steadily increased, but it is noteworthy that it was the ninth day after establishment of diuresis before the kidney excreted more than 10 g. of urea in

the 24 hours, and even this was at a time when the blood urea level was 238 mg. per 100 ml. and the urine flow 2,200 ml.

On June 5, four weeks after urine flow was re-established, the inulin clearance was still only 54 ml. per minute, and diodone clearance 288 ml. per minute, giving a filtration fraction of 0.19. At this time the blood urea was only 30 mg. per 100 ml., and the haemoglobin was 46% (Haldane). This degree of anaemia would account for some

depression of renal function, but simple anaemia of this degree does not lead to such low figures for inulin clearance, and would, moreover, be associated with a change in the filtration fraction (Bradley and Bradley, 1947). The patient made a good clinical recovery.

Comment

It will be noted that the total fluid volume recovered after perfusion was greater than that of the sulphate solution introduced. This may indicate that the procedure interferes

with the reabsorption of the water of intestinal secretions. Peritoneal dialysis is often complicated by overhydration, which is one of its many disadvantages. This first experience with hypertonic sulphate dialysis suggests that dehydration and salt loss may be a more likely complication. Since, however, the duodenum is intubated, it is possible, by means of saline or other solution during the night, to make good the losses produced by dialysis during the day.

Summary

Anuria may be caused by a failure either of the glomeruli to form filtrate or of the tubules to transmit the filtrate in the usual way. Although a circulatory upset would primarily affect the glomeruli the morphological changes in anuria are predominantly tubular, and it is still an open question whether glomerular or tubular functional impairment is the more important. This uncertainty is reflected in the confused opinion about the treatment of anuria. Some methods of treatment (paravertebral block, spinal analgesia) assume a circulatory disturbance to be the cause of anuria; other methods (low-protein diet, the artificial kidney, peritoneal and intestinal lavage) are designed to prolong life until reparative processes can take place in the tubules. At present it seems justifiable to employ suitable methods of the first type as soon as possible, and, if need be, to resort to the second type of method as well. A patient is described whose treatment illustrates some of the points discussed, in particular the use of intestinal lavage with hypertonic sodium sulphate solution.

We are grateful to Mr. H. Varley for the estimations on the dialysate and for the clearance results.

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ADDENDUM

Marquis and Schnell (*Amer. J. med. Sci.*, 1948, **215**, 686) report sudden death in a patient after 10 days of intestinal perfusion; the serum potassium just before death was only 1.2 m.Eq./litre. The danger of death from potassium depletion, once recognized, can easily be avoided by using Ringer's solution rather than normal saline to replace fluid loss overnight.

SPLENIC ANEURYSM AND SPLENIC ENLARGEMENT IN PREGNANCY

BY

H. L. SHEEHAN, M.D., D.Sc., F.R.C.P.

Professor of Pathology, University of Liverpool

AND

NINIAN M. FALKINER, M.D., Sc.D., F.R.C.P.I.
F.R.C.O.G.

Late Master, Rotunda Hospital, Dublin

Rupture of an aneurysm of the splenic artery is a well-recognized complication of pregnancy. In a previous paper (Lennie and Sheehan, 1942) four cases of rupture of splenic and renal aneurysms in pregnancy were described and a review was given of 20 previous cases in the literature. More recently Ogden (1948) recorded a ruptured splenic aneurysm in a woman 35 weeks pregnant and gave details from the literature of three additional cases (one splenic and two renal) in pregnant women. The following is a further example of rupture of a splenic aneurysm in the course of pregnancy.

Case Report

The patient, aged 32, was a 3-para at 36 weeks' gestation. One day, at 1 p.m., she had a sudden onset of abdominal pain, felt faint, and went to bed. Foetal movements ceased at this time. She vomited in the evening and was sent into the Rotunda Hospital with the diagnosis of concealed accidental haemorrhage. There was no vaginal haemorrhage or albuminuria, but foetal heart sounds could not be heard and the abdomen was resistant and slightly tender. Her general condition was poor: pulse 80, B.P. 105/80. At 6 a.m. next day she suddenly developed severe shock, which was treated with saline and blood transfusion. The os was dilated two finger-breadths, so the membranes were punctured and "pitocin" was given; the liquor amnii was not blood-stained. Labour did not begin, but at 8 a.m. she suddenly became very collapsed and a further blood transfusion was necessary. Soon afterwards a classical section was carried out; this showed no uterine haemorrhage, but a large retroperitoneal haemorrhage in the region of the pancreas and left kidney was found. This was diagnosed as a ruptured aneurysm of the splenic or renal artery, but the patient was considered to be too ill for a surgical exploration of these vessels. At 10 p.m. the patient suddenly collapsed again and in a few minutes she died. The time from the first symptoms to death was thus 33 hours. Post-mortem examination showed a rupture of a small aneurysm of the splenic artery; the blood had finally burst through into the peritoneal cavity.

Discussion

The case is fairly typical—the diagnosis of some intra-abdominal catastrophe, the operative discovery of the haematoma in the region of the pancreas, the latent period (which varies in different cases between one and seven days), and the terminal haemorrhage into the peritoneal cavity. The only hope for such a patient is heroic surgery by the operator who has opened the abdomen, usually the obstetrician who has performed caesarean section. The details have been discussed by Lennie and Sheehan (1942).

The aetiology of these aneurysms is not known. One peculiar aspect of the problem is that, of the cases so far recorded in patients below the age of 45, splenic aneurysms are much commoner in women (41 female, 12 male), whereas renal aneurysms are commoner in men (9 female, 21 male). What is of more direct interest here, 23 of the splenic aneurysms ruptured in connexion with pregnancy, nearly always at seven to nine months' gestation. This raises the question whether the rupture may possibly be related in any way to alterations of the blood supply to the spleen during pregnancy. No direct evidence is available on this subject, but some facts which may have a

The American Medical Association sponsored a Public Relations Conference in St. Louis recently. The theme was "Common Targets in Medical Public Relations." About 125 State and metropolitan county medical society leaders participated in the public relations meetings. The problems under discussion included: "Selling the Need for Public Relations to the Profession," "Using Public Relations to Aid Medical Prepayment Plans," "Handling Emergency and Night Calls," "The Rebate Problem," "Co-operating with Special Publics," and "Co-operating with Health Agencies."