infected it is longer in freeing itself from that infection than the bladder. Moreover, the urine enters it in a continuous stream from many points and not in intermittent gushes from two as in the case of the bladder or one as in the case of the urethra. The value of an intermittent flow is well illustrated in automatic flushing tanks and cisterns.

The composition of normal urine is unfavourable to the growth of most of the organisms which produce cystitis. Doubtless the acidity of urine fulfils other functions, such as keeping earthy phosphates in solution, but it is in addition unfavourable to the growth of all the common micro-organisms of cystitis except the bacillus coli. If to a prostatic case with alkaline cystitis we give sufficiently large doses of acid sodium phosphate to change the reaction of the urine the micro-organisms will disappear with the exception of the bacillus coli communis and the cystitis will clear up. Dr. A. E. Wright informs me that the bacillus coli grows by preference in alkaline or just neutral media but in common with all this race of bacilli it can grow in acid urine and does not render it alkaline. There are certain indications, moreover, that normal urine contains some factor inimical to the growth of micro-organisms, and this factor belongs perhaps to the class of opsonins which have been found in the serum and other fluids of the body. Dr. Wright believes that such an antibacterial factor exists in the urine and he has kindly written me that he has found on several occasions "that bacteria will grow infinitely more readily in urine of a coli cystitis case than in normal urine. Whereas in the one case 24 hours' incubated urine will contain, say, 300-400 million per c.c., normal urine 24 hours incubated will contain only the same number of thousands." The conclusion to be drawn from this observation is apparently either that the absence of the antibacterial factor leads to the coli cystitis or that the bacillus coli by its growth had exhausted that factor. In any event it would appear that some factor exists which renders normal urine unfavourable for the growth of micro-organisms and assists in maintaining the asepsis of the urinary tract.

The ways in which these means are circumvented and the ordinary cases of urinary sepsis come about.—We have seen that the three great means of protecting the kidneys from bacterial invasion from below are: (1) the small lumen of the urinary passages which secures an efficient scour; (2) the complete emptying of cavities and tubes at frequent intervals of the putrescible urine; and (3) an intermittent flushing from above by a sterile fluid. All these means are circumvented by an obstruction which dilates the urinary passages and cavities. The best marked instance is that of a stricture which has produced retention with overflow in a case in which the ureters and kidneys are already dilated. Here a continuous stagnant chain of urine extends from the kidney to the septic trousers sodden with incontinence. In lesser degrees and earlier cases of stricture the urethra is dilated behind the stricture and this pouch fails to empty itself after micturition. Leakage slowly occurs along the urethra after urine has been passed and the pouch becomes infected. By this means septic decomposing fluid is held against the mouth of the sphincter vesicæ continuously, and as this sphincter has already been dilated and stretched during efforts at micturition it soon allows the passage of these micro-organisms to the urine in the bladder. The bladder, partly through atony of its muscular wall and partly through pouching, never completely empties its cavity and a continuous culture of micro-organisms is thus maintained at the orifices of the ureters. The urine no longer gushes from these in frequent forceful jets. The orifices are dilated. The tubes themselves are wide, sinuous, and their muscular coat is quite incapable of emptying their cavities. The sepsis spreads from pond to pond in the varicosities of the ureter and with the greatest ease reaches the irregular and often huge cavity of the pelvis of the kidney. For a time the continuous flow of aseptic urine down the discharging tubules will protect the cortex from infection, but as the pyramids are destroyed by pressure and these tubes are consequently shortened, and as interstitial nephritis obstructs their lumen at numerous points, the sepsis spreads up them and finally the vital renal substance becomes infected. The history of urinary infection in a case of prostatic enlargement is very similar but, as has already been pointed out, the sphincter and the urethra are here intact and as a rule preserve the urinary tract above the obstruction from infection until the passage of a catheter pushes septic matter bodily past these protecting structures. Then sepsis often spreads with tragic rapidity to both kidneys. Cases of

obstruction of the ureters as by calculi are even better protected but not infrequently become infected in women, as in a case that I had under my care in which double pyonephrosis was produced by stones obstructing both ureters and was cured by removing these calculi.

Instances in which micro-organisms invade a normal urinary tract.—The commonest example is, of course, the gonococcus but the spread of this micro-organism is often arrested by the sphincter and it seldom passes beyond the bladder. The class of case which we are now discussing appears to me to provide a second example of a micro-organism capable of ascending a normal urinary tract in spite of all safeguards. My reasons for regarding these cases as ascending infections are as follows: 1. The majority of the cases commenced with urethritis and cystitis. 2. The microscopic examination of the wedge-shaped areas of inflammation in the cortex showed that they were in the lobules and not between the lobules. Moreover, these cases can scarcely be hæmatogenous since: (1) no source of infection was found; (2) the other kidney escaped and all other organs; and (3) their immediate recovery after nephrectomy showed that septicæmia was not present. It is difficult to show that they were not lymphatic in origin for, as has already been pointed out, our knowledge of the lymphatics of the kidneys is obscure. A point of infection in the lower urinary tract would, however, appear essential to a lymphatic infection.

The bacillus coli communis in these cases probably travels along the mucous membrane as the gonococcus does and not along the urine filling the passages as is usually the case in ordinary obstructive infections. The reaction of the body to the invasion appears to be very acute and efficient, for the micro-organism in any part of the lower urinary tract appears to be completely overcome in three or four days. This compares favourably with gonorrhœal invasion of the urethral mucous membrane or influenzal invasion of the respiratory tract. The spread of the micro-organism is partly by division and multiplication and partly due to its motility, but one cannot help recalling the most interesting experiments of Mr. C. J. Bond of Leicester recorded in his address on Surgery at the meeting of the British Medical Association in July, 1905. He applied sterilised indigo granules to the orifices of various mucous passages and found that the granules were somehow transmitted up these passages in the face of the natural stream of contents and after a day or two could be identified at various distances above. Thus indigo applied to the lips of the uterus was found a day or two later in a pyosalpinx or, in other cases, even in the peritoneum around the ostium abdominale. Indigo placed in the rectum was found in a cæcotomy wound three days later, although the bowels had been opened in the interim. Perhaps micro-organisms are in part conveyed from the meatus to the kidney by similar means, whatever these means may be.

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INTRAVITREOUS HÆMORRHAGES.

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THE occurrence of large hæmorrhages in the retina and into the vitreous in a seemingly spontaneous fashion is frequently met with in ophthalmic practice. That such hæmorrhages occurred in young people who to all outward appearance were in perfect health was a fact to which Mr. H. Eales of Birmingham first called attention in the year 1880. Mr. Eales described the cases as occurring in healthy young men. We have given here two cases in young women as they presented clinically the same features and showed the same changes in their blood. We have recently had the opportunity of seeing four such cases and of examining the blood by methods devised by Dr. A. E. Wright, whom we must thank for his kind advice and coöperation in the work. One of us (L. P.) is responsible for the clinical notes while the examinations of the blood were made by the other of us. It will be convenient to begin by giving a short history of the cases from the point of view of the ophthalmologist.

CASE 1.—On Tuesday, Sept. 6th, 1904, a patient (a medical man) came to see me (L. P.) who on the previous Friday

had caught cold which brought on coryza with some conjunctivitis. On the Sunday evening a sudden dimness had come over the left eye when he was stooping to pick up some music, and on the Monday morning the lower part of the field of vision of his left eye was cut off by a floating opacity. On examination I found a definite limited scotoma in the lower outer quadrant of the left field and in the fundus of the left eye there was a large hæmorrhage extending along the line of the upper nasal vessels, and where these joined the disc to the inner side there was a somewhat globular hæmorrhage seemingly in front of the retina but subhyaloid. Into the vitreous itself there was only a very slight hæmorrhage which seemed to lie along the line of Stilling's canal. The retinal hæmorrhage was of very large extent, stretching for many disc-breadths along the line of the upper nasal vessels. I could not tell from appearances whether it had been arterial or venous in its origin. The right fundus showed nothing. I asked the patient at that time to investigate his family history carefully to find whether there was any hæmophilic tendency in any member, and he did find that a great-great-grandfather had died from bleeding after a tooth extraction, so that could not be regarded as very definite evidence. I told him to rest as much as possible, on no account to take any violent muscular exercise, and to use mercury in some form and iodide of potassium. Unfortunately the exigencies of his profession called on him to ride a long distance one evening and the result was an immediate increase of the hæmorrhage and when I saw him again on Oct. 3rd the vitreous of the left eye was full of blood and only a faint red reflex could be obtained at some parts of the periphery. Still harping on the possibility of this being of a hæmophilic origin, and with a view to raise the coagulation point of the blood by free purging and calcium salts, I asked Dr. Wright to examine his blood, when to my astonishment it was found that the coagulation was three times faster than normal and that the proportion of calcium salts was distinctly above normal. As the patient lived a considerable distance away it was impossible to watch the effects of various drugs in reducing the rate of coagulation.

In connexion with the following cases I have had an opportunity of following their course.

CASE 2.—The patient, a woman, aged 27 years, went to Mr. R. Marcus Gunn's clinique at the Royal London Ophthalmic Hospital (Moorfields) on March 1st, 1905. Her history was that eight years previously she had had a hæmorrhage into the vitreous of the left eye and was quite blind for a time, but by the end of six months the sight had become completely clear again and had remained so till the middle of January of this year, when she had again become blind in the left eye. On examination the right eye was found to have visual acuity $\frac{5}{6}$ Snellen, and normal fundus and vessels. The pupil of the left eye contracted to direct stimulus—but the contraction was not well maintained; the contraction to consensual stimulus was good and well maintained. The vitreous was quite full of hæmorrhage, no red reflex being obtainable in any part. I had the patient's blood examined that afternoon and it was found that it clotted in 1 minute 35 seconds and that the calcium salts were equivalent to 1:1000 solution of oxalate of ammonium. It will be seen here that the rate of clotting was not very great, though it was distinctly above the average, and it must be remembered that in this case the first test was made seven weeks after the hæmorrhage had occurred, that the hæmorrhage occurred at a menstrual period, and that when the menstrual periods occurred the rate of coagulation was very sensibly increased.

CASE 3.—The patient, a woman, aged 34 years, gave a history which was somewhat similar to that recorded in Case 2. She had first of all had a hæmorrhage into the vitreous of the left eye six years previously when she was quite blind for two months. By the end of two years this hæmorrhage had cleared up fairly completely. A second attack had come on 12 months previously, and the present attack had commenced nine days before I saw her on March 1st, 1905. On that day the vision in the right eye was $\frac{5}{6}$ Snellen and the fundus and vessels were normal. The left eye had no vision but the pupil reactions were normal. The vitreous was completely filled with hæmorrhage. The rate of clotting taken that afternoon was 50 seconds and the calcium salts were reduced by a dilution equivalent to 1:1200 of oxalate of ammonium.

The further history of Cases 2 and 3 runs on very similar lines. For the first two or three months there was very

little improvement in visual acuity, then gradually they began to be able to see fingers, and at the present time the patient in Case 3¹ is able to see $\frac{5}{6}$ Snellen and the patient in Case 2 to count fingers easily at a distance of a few feet and excentrically is able to see $\frac{5}{6}$ Snellen. In both there is a partial restoration of the red reflex from the fundus but no fundus details are visible.

CASE 4.—The patient was a male (and in consequence free from the complications arising from menstruation), aged 23 years. For permission to examine him I am indebted to Mr. S. H. Fisher who had seen him in a previous attack, at which time (March, 1904) he had been treated with calcium chloride. The attack in March, 1904, had been of little severity, though vision was reduced to perception of light and the whole duration to complete restoration of vision was not more than six weeks. Nor was the attack in which I saw him of very great severity. He always retained perception of light, though the vitreous seemed full of hæmorrhage. I saw him first on March 15th. At that time the rate of clotting was 1 minute 10 seconds and the calcium salts were equivalent to 1:1000 solution of oxalate of ammonium. Citric acid very speedily brought down the rate of coagulation to 2 minutes 25 seconds and then to 2 minutes 40 seconds. In the course of a month his vision had improved to $\frac{5}{6}$ in the left eye and the hæmorrhage had disappeared from the vitreous, leaving only a long greyish process which passed forward from the centre of the disc into the vitreous, coming almost up to the back of the lens and seemingly running along the line of the hyaloid canal.

In describing these four cases I have not entered into any details of the variations in the clotting point produced by citric acid, but I think the results are striking enough to warrant further investigation of the blood in such cases and the adoption of a different method of treatment from that usually pursued if a recurrence is to be prevented. Obviously it must be wrong to treat such cases with doses of calcium salts when the clotting point is already too high. Yet there are some cases in which a low rate of coagulation may be found at times. As to what actually causes the outflow of blood from the vessels we are still in the dark, but Dr. Wright's suggestion that it is due to thrombosis in some small vein seems to me at present to be most probable.

EXAMINATION OF THE BLOOD.

In dealing with vitreous hæmorrhages an examination of the blood was made by me (W. E. P.) with regard to the rate of coagulation and the content of calcium salts. The method used was that devised by Dr. Wright and described by him in THE LANCET.²

Table showing the Results of the Preliminary Blood Examination.

Ca.	Date of examination.	Coagulation time.	Dilution of ammonium oxalate made up in physiological salt solution which just sufficed to complete decalcification of the blood.
2	March 1st, 1905.	1 minute, 35 seconds.	1 in 1000.
3	March 1st, 1905.	50 seconds.	1 in 1200.
4	March 15th, 1905.	1 minute, 10 seconds.	1 in 1500.

With these data it seemed expedient, instead of following the course which is often taken of administering calcium, that in view of the presence already in the blood of a quantum of calcium which was in excess of the normal the opposite direction should be aimed at by the exhibition of a decalcifying agent such as citric acid. We were the more inclined to follow this course by the details already published by Dr. Wright, that in typhoid fever when the content of the blood in lime salts was great and the coagulation time of the blood therefore rapid thrombosis was common and was to a certain extent prevented by the exhibition of a decalcifying agent.

The data with regard to the examinations of the blood during treatment are set forth below. In the cases recorded

¹ Since writing the above this patient has had another slight hæmorrhage into the vitreous and on investigating the coagulation time within a day or two of its occurrence it was found to be 30 seconds.

² THE LANCET, Oct. 14th, 1905, p. 1096.

we were unable to prolong the decalcifying effect of the citric acid until such time as the hæmorrhage cleared up.

CASE 2.—The patient was suffering from hæmorrhage into the left vitreous. March 1st, 1905: Coagulation time, 1 minute 35 seconds. Complete decalcification with a dilution of 1 in 1000 of oxalate of ammonium. Two drachms of citric acid were administered three times a day. 15th: Coagulation time, 1 minute 7 seconds. Complete decalcification with a dilution of 1 in 1800 of oxalate of ammonium. 29th: Coagulation time, 2 minutes. Complete decalcification with a dilution of 1 in 1400 of oxalate of ammonium. April 12th: Coagulation time, 2 minutes 40 seconds. Complete decalcification with a dilution of 1 in 1500 of oxalate of ammonium. 26th: Coagulation time, 55 seconds. Complete decalcification with a dilution of 1 in 1050 of oxalate of ammonium. May 10th: Coagulation time, 1 minute 50 seconds. Complete decalcification with a dilution of 1 in 750 of oxalate of ammonium. 24th: Coagulation time, 1 minute 27 seconds. Complete decalcification with a dilution of 1 in 1200 of oxalate of ammonium.

CASE 3.—The patient was suffering from hæmorrhage into the left vitreous. March 1st, 1905: Coagulation time, 50 seconds. Complete decalcification with a dilution of 1 in 1200 of oxalate of ammonium. Two drachms of citric acid were administered three times a day. 15th: Coagulation time, 50 seconds. Complete decalcification with a dilution of 1 in 2000 of oxalate of ammonium. 29th: Coagulation time, 2 minutes 5 seconds. Complete decalcification with a dilution of 1 in 1400 of oxalate of ammonium. April 12th: Coagulation time, 2 minutes 25 seconds. Complete decalcification with a dilution of 1 in 1500 of oxalate of ammonium. 26th: Coagulation time, 2 minutes 5 seconds. Complete decalcification with a dilution of 1 in 1200 of oxalate of ammonium. May 10th: Coagulation time, 1 minute 45 seconds. Complete decalcification with a dilution of 1 in 1050 of oxalate of ammonium. 24th: Coagulation time, 55 seconds. Complete decalcification with a dilution of 1 in 700 of oxalate of ammonium.

CASE 4.—The patient was suffering from hæmorrhage into the vitreous. March 15th, 1905: Coagulation time, 1 minute 10 seconds. Complete decalcification with a dilution of 1 in 1500 of oxalate of ammonium. One drachm of citric acid was administered three times a day. 22nd: Coagulation time, 2 minutes 25 seconds. Complete decalcification with a dilution of 1 in 1600 of oxalate of ammonium. April 5th: Coagulation time, 2 minutes 40 seconds. Complete decalcification with a dilution of 1 in 1200 of oxalate of ammonium. 19th: Coagulation time, 2 minutes 10 seconds. Complete decalcification with a dilution of 1 in 1000 of oxalate of ammonium. May 17th: Coagulation time, 1 minute 37 seconds. Complete decalcification with a dilution of 1 in 1050 of oxalate of ammonium.

THE USE OF PLASTER-OF-PARIS SPLINTS IN THE TREATMENT OF FRACTURES OF THE LEG.

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THE use of plaster of Paris in the treatment of fractures of the leg is a great convenience to the surgeon, particularly in hospital practice. The pressure on the beds of most general hospitals is often so great that it is necessary to discharge the patients from the wards as soon as it can be done with safety. Once in plaster the fracture is immobilised and may be expected to unite in the course of time in the position in which it was put up. The patient, too, is usually anxious to be released from confinement to bed and to be going about on crutches with his leg in plaster. On the score of convenience we cannot dispense with plaster of Paris in the treatment of these cases unless it be shown that the disadvantages attending its use are serious and unavoidable.

Plaster is used in two forms; as a closed case made with bandages impregnated with plaster and as splints made from some shaped material soaked in plaster cream (Croft's and Bavarian splints). The special disadvantages attaching to the plaster case are that it is difficult to remove and that it loses much of its rigidity when it is "cut up." Removal of the case may be necessitated by swelling of the limb if the plaster has been applied soon after the occurrence of the fracture, or at a later period for shrinkage of the limb and consequent loosening of the case. In either case the application of a fresh plaster will probably be required. To facilitate the cutting up of the case a string may be placed underneath it between the plaster and the flannel bandage surrounding the limb, by means of which a wire or Gigli's saw may be pulled through, with which the case may be divided down the front. Another method is to place a strip of block tin along the front of the leg beneath the plaster, on to which one can cut with a penknife without danger of damaging the patient's skin. In any case, the plaster is apt to be spoiled by being cut up.

Bavarian or Croft's splints get over the difficulty of

removal to a certain extent. Their inefficiency in many cases is due to the material of which they are usually made (house flannel soaked in plaster cream). This makes a poor splint, deficient in rigidity, and apt to crumble. Another great disadvantage of the forms of plaster in common use is the uncertainty as to the position of the fragments when the case has been applied. Owing to the manipulation of the limb attending the application of the bandages and the frequent shifting of the hands of the assistant holding the limb it is very easy for the fragments to become displaced and the displacement will only be discovered when it is too late to rectify it—i.e., after the plaster is removed. Another objection to plaster to which much attention has been directed recently is the amount of matting of the soft parts and stiffness of the joints following rigid confinement of the limb in plaster. What is required, therefore, is some form of plaster (1) made of sufficiently rigid and durable material; (2) applied without shifting the limb about; (3) readily removeable for inspection or treatment by massage and passive movement of the limb; and (4) which can be opened up without disturbing the fragments or spoiling the case.

I have been using a form of plaster case for the last two years at the Sheffield Royal Hospital which I consider fulfils all these requirements.

Material.—Gauze such as is used for making gauze bandages is employed. The splints consist of from eight to 12 folds of this gauze, between the layers of which dry plaster is well rubbed in. Before applying they are immersed in warm water, squeezed out, and then more dry plaster is rubbed into their inner surfaces. No plaster cream should be employed. If the plaster is well baked splints made in this way set very quickly and firmly and are very durable.

Reduction of swelling.—If the limb is much swollen elastic compression is applied to reduce the swelling as a preliminary measure. The limb is wrapped from the toes to above the fracture in layers of non-absorbent wool of a thickness of at least one inch and over this an elastic bandage is applied in simple spirals. The limb is then bandaged on to a back-splint with foot-piece. The swelling will usually have subsided in three or four days, though in some cases a week is required. The stiffness of the limb subsequently to the removal of the plaster will be much less if care is taken to get rid of the effusion of blood accompanying the fracture in this manner before applying the case.

Blisters.—After being clipped and the serum squeezed out blisters are dressed with two or three layers of lint wrung out of a saturated watery solution of picric acid which dries the surface, preventing any further discharge of serum.

Method of applying the plaster splints.—Supposing the case to be one of fracture of one or both bones of the leg in the lower third, the plaster splints need only be carried as high as the knee. A pattern of the posterior splint is obtained by laying the sound limb on a sheet of paper and cutting round it with scissors. The splint extends from the root of the toes, under the heel, and up the back of the leg to just below the knee. It should be wide enough to embrace about two-thirds of the circumference of the limb. A good width should be allowed at the ankle to accommodate the projection of the heel. The pattern must be turned over so as to reverse the sides in cutting out the splint for the fractured limb (Fig. 1). From 10 to 12 folds of ordinary gauze bandage material should be laid out in a length equal to, and rather wider than, the pattern and cut out to form the posterior splint. This should be laid upon a considerably wider piece of gauze and the whole thickness stitched together down the middle line. Dry plaster should be rubbed in plentifully between the layers of the gauze. The extra width of the outside layer of gauze should now be split into tails on either side of the splint and the tails rolled up so that they may not become "taffled." The posterior splint should now be rolled up from either end on rollers. Two pieces of broomstick answer the purpose. The anterior splint is simply a strip, from three to four inches wide, long enough to reach from the root of the toes to the knee. From eight to ten folds of gauze suffice and the ends should be secured by stitching them together. Plaster is rubbed in as in the case of the posterior splint and the splint is rolled up on a piece of broomstick. The leg should then be bandaged evenly with a single thickness of flannel, a pad of wool being placed on either side above the heel, this being a point on which the splint is likely to cause pressure. The knee should be bent to a right angle and the leg laid upon a support, such