(Fig. 15.) A wedge was removed from the fibula; the tibia was divided transversely and opened out, and the gap filled up as far as possible by the piece of fibula and by shavings from the side of the tibia. Union is good, and a useful foot has been obtained. The appearance and function are now excellent. (Figs. 16 and 17.)

FIG. 15.—Pre-operative radiogram.

FIG. 16.—After osteotomies and graft.

FIG. 17.—Present appearance.

In conclusion, I would emphasize that function is the ultimate test of treatment, and I know of no better evidence of restoration of function than a return of the individual to his pre-accident work. I stress "pre-accident" because in some estimates of end-results a return to "work" has been accepted. The 1908 article of Clinton Dent, which has been so often quoted, stated: "I can hardly recall a case of Pott's or Dupuytren's fracture where recovery has been good enough to permit a man to return to police work. Among the Metropolitan Police 30 per cent. of all ankle injuries failed to recover for work."

This was a very serious state of affairs, and I hope that I have shown that some progress has been made since that time. Many causes have brought about this improvement, but none, I think, so much as the treatment of the fractures by one department. If you will permit me, I would like to quote the substance of two paragraphs in an article which I wrote for the *British Medical Journal* in 1921. The article was headed "The hospital treatment of fractures."

In order to ensure the most perfect functional result which is possible it is desirable that the same surgeon should supervise the case from start to finish. He should be entirely responsible for the case whilst in the wards; he should supervise its manipulation and splinting, its early massage and, on discharge, its progress through the out-patient and physio-therapeutic departments. In hospitals which have no special department for orthopaedics a member, or members, of the surgical staff should be selected to take charge of the fractures. The number of beds which should be allotted to them for this purpose will, in an industrial area, be about 10 per cent. of the total surgical beds of the hospital.

This arrangement, by which the continual supervision is under one control, has been responsible more than any other single factor for the improvement which may have been obtained in our fracture results.

CONCLUSIONS.

1. Reduction of all displacements should be carried out as thoroughly and as early as possible.
2. A suitable splint which will permit constant inspection, early massage, and active movements at the earliest time should be applied.
3. Open operation in recent cases should be reserved for those in which the displacement cannot be satisfactorily reduced or maintained.
4. In old cases of mal-union operation is indicated.
5. In compiling statistics of end-results percentages should not be calculated on less than 100 cases, and "return to work" should mean the pre-accident work.
6. In the present series 89 per cent. of ankle fractures, of all types, returned to pre-accident work.
7. A fracture case, from the time of its arrival at hospital to the time of its discharge to work, should be under the supervision of one surgeon.

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THE TREATMENT OF ACUTE DIVERTICULITIS.*

BY

J. P. LOCKHART-MUMMERY, F.R.C.S. Eng.,

SURGEON, ST. MARK'S HOSPITAL.

DIVERTICULITIS, like appendicitis, is, as regards our knowledge of it, a comparatively new disease, and is still considered by many doctors a very rare condition. It was not known to the last generation of surgeons, and cases of the condition were described as "pericolitis sinistra," iliac abscess, etc., and the underlying cause of such states remained unrecognized until recently. It is now a fairly well-recognized condition, although it has hardly found its way yet into the ordinary medical textbooks of the time. Nevertheless, it is by no means an uncommon condition, and is accountable for quite a considerable number of deaths and a still larger number of chronic invalids each year.

SYMPTOMS.

The symptoms are fairly characteristic and, if recognized, are not easily mistaken for anything else. The patient is practically always over 45 years of age, and usually rather stout; for some unexplained reason the condition appears to be quite uncommon in thin persons. The main symptoms are pain in the lower part of the abdomen, tenderness in the left iliac fossa, or just above the pubes, and raised temperature, often accompanied by shivering and mild rigors. The pain is not very acute as a rule, but is distressing, and prevents the patient from moving about freely. The tenderness, if looked for, is very definite and quite characteristic of the condition. Any pressure in the lower left side of the abdomen causes the patient to flinch, and as a rule the area of acute tenderness is very well localized to a certain spot. The temperature is seldom high; usually it ranges from 100° to 101° F. rather than higher. Quite high temperatures are sometimes seen, but are exceptional, except at the onset of an acute perforation, and in this respect it differs from appendicitis, where high temperatures are the rule. Also vomiting, though occasionally a symptom, is a rare one, whereas it is a common symptom at the onset of appendicitis.

Not infrequently a definite resistance or tumour may be felt deep in the pelvis on the left side, and it may be possible to feel this bimanually, either by the rectum or vagina. It need hardly be pointed out that very gentle handling of such a tumour is advisable, or serious consequences may result. Such a swelling can, as a rule, be easily distinguished from a carcinoma of the colon by the fact that it is very definitely tender and that there are obvious symptoms of inflammation present.

The bowels may, or may not, be constipated; on the other hand, there may be a mild diarrhoea from irritation of the colon. In some cases frequency of micturition is a well-marked feature of the case, and when this is present it almost invariably means that the bladder is adherent to the diseased colon and that a perforation from the colon into that organ is threatened. Just as in the case of appendicitis, a dose of castor oil will often precipitate acute symptoms and should on no account be administered.

These symptoms apply, of course, in the main to cases of diverticulitis of the pelvic colon, by far the commonest site, and are considerably modified when the affected portion of the colon is elsewhere. The diagnosis is often extremely difficult when either the ascending or transverse colon is involved, as the symptoms then imitate several other conditions, such as appendicitis, perforated duodenal or gastric ulcer, and cholecystitis.

The acute symptoms may arise from several different causes. Most commonly one or, possibly, two diverticula have become acutely inflamed and caused a small local abscess in the bowel wall, generally just beneath the peritoneum. The inflamed area has become adherent to other structures, such as the bladder, the small intestine, the uterus, or a tube, or sometimes to the abdominal wall. Sometimes a direct perforation into the abdominal cavity is the first symptom, and in such cases a general peritonitis will occur, the exact cause of which may easily be

* Read at a meeting of the North London Medical and Chirurgical Society, February 6th, 1929.

missed. Again, a sequence which is not uncommon is for a local abscess to develop around the diseased bowel and for the abscess to rupture into the peritoneal cavity. This is a disastrous happening, as it results in a very acute general infection of the peritoneal cavity owing to the large amount of septic material suddenly released into the abdomen.

The first symptom recognized may be the rupture of an inflamed diverticulum into the bladder. I have known this happen on several occasions, and the result is most distressing to the patient. An acute cystitis results, with the passage of flatus and faecal material "per urethram," and the patient is generally very ill and in great distress.

CAUSATION.

The exact cause of the formation of diverticula of the colon is not yet completely understood. There is no doubt that in some cases an inflammatory process takes place around the diverticula when they are quite small, almost microscopic, and this inflammatory process may spread to a considerable area of the bowel wall. On the other hand, diverticula may reach a considerable size and be very numerous without there being any signs at all of any inflammation around them. In fact, perforation of a diverticulum may occur and cause a general peritonitis without any inflammatory process in the bowel wall having preceded the perforation.

TREATMENT.

Unfortunately the condition is not so easily amenable to operation as is the case with appendicitis. The infective focus is not so localized, nor can it be so readily remedied by the surgeon. In the acute cases, where perforation has occurred with general peritonitis, the only treatment is to drain the abdomen and perform a colostomy well above the perforation. As a rule the perforation will be in some part of the pelvic colon, and in this event the artificial anus should be made with some portion of the left side of the transverse colon. In a few cases simple drainage of the abdominal cavity has been successful, the opening in the bowel closing up. This can hardly be relied upon, however, and is not likely to occur unless the perforation is very small. Deliberate closure of the opening caused by the ruptured diverticulum would seem a possibility, but this is seldom the case. The parts around the opening are much thickened and indurated, and such tissues do not lend themselves readily to satisfactory closure; omental grafting of the opening would be safer, but unless the diverticulitis is very localized attempts to close the opening will usually be failures and may be disastrous; colostomy is the best procedure. When a perforation has occurred into the bladder, immediate colostomy above the site of perforation is the only rational treatment. I have seen a number of such cases, and in all of them the opening into the bladder subsequently healed up quite satisfactorily.

The most difficult cases to deal with are those in which an abscess has formed in association with some part of the colon, and the abscess has subsequently burst or leaked into the peritoneal cavity. The obvious treatment is to drain the abdominal cavity and to drain the abscess cavity separately. Unfortunately what is generally found is that there is a dense mass of gut and adhesions forming the wall of the abscess, which is most difficult to drain properly, and that it is not possible to in any way disentangle or break up the adhesions without the certainty of damaging other parts of the intestine. These are most difficult cases, and even when the patient survives the initial peritonitis, residual abscesses and pocketing are the usual rule, and will call for all the skill and resources of the surgeon to circumvent. I know of few more dangerous lesions to meddle with than a mass of diverticulitis of the colon with adhesions to other organs and parts of the intestine. The lesion is highly septic and the density of tissue is only equalled by carcinoma. The abscess resembles a sponge more than an ordinary abscess cavity, and it is very difficult to establish any kind of satisfactory drainage.

When there is reason to suspect that an abscess has formed from perforation of a diverticulum, but the general peritoneal cavity is not involved, there should be no

attempt to temporize in the hope that the abscess will subside. The condition is too dangerous, and even if a general peritonitis does not result, perforation of the abdominal wall or some hollow viscus will almost certainly take place and make conditions for operation more difficult. Operation in these circumstances is particularly difficult and dangerous, and very nice judgement is necessary to decide what is the best course to take. I prefer, when operating upon such cases, to use an oblique incision on the left side of the abdomen, the rectus muscle being dissected out of its sheath and displaced towards the right side; local anaesthesia of the rectus muscle is a great help. This gives very good access and allows of good draining in the flank if this should be necessary. The pelvic colon, assuming that this is the portion involved, should be freed if possible by very gentle manipulation, while the main abdominal cavity is well protected by large intra-abdominal swabs so placed as to localize as much as possible the contamination of the peritoneal cavity which may occur. The involved part of the pelvic colon should be lifted up, if it can be freed, and drawn well over into the left iliac fossa, and the abscess cavity properly drained with a tube or tubes. If the omentum is large enough it should be drawn down and stitched all round the damaged portion of the colon, so as to seal it off and at the same time prevent the small intestine from subsequently becoming adherent to it. The wrapped-up damaged portion of colon should be fixed with a few catgut sutures well into the left iliac fossa and away from the true pelvis, as if subsequent abscess formation should occur it will do less harm in this situation and will be much more accessible. I have had some very successful results from this method of treatment, and in one or two cases have been able to separate the diseased portion of the colon from the bladder, and thus prevent a perforation into that organ. Patients treated in this way have got perfectly well, and with reasonable care have been able to live normal lives for very many years. I have now quite a number of such cases under observation. They have been saved a colostomy, and the inflammatory mischief has in course of time entirely cleared up in most of them, although some have had temporary relapses. The fact that the inflammation is no longer involving the true pelvis renders treatment much safer.

If at the time of operation the damage to the bowel is too severe to justify this method of procedure a colostomy should be done well above the site of the trouble; this will generally mean a transverse colostomy. When this plan has been adopted the question will subsequently arise as to whether it is possible to close the colostomy. This is always a difficult question and will require careful consideration, as the patient's life is perfectly safe if he is content to keep the colostomy, and its closure will involve a fair amount of risk. It is doubtless worth some risk to get rid of the colostomy, but as this is mainly a question of comfort and convenience it hardly justifies running a serious risk to life.

Good x-ray photographs of barium enemata will be necessary to see if there is a free enough lumen to justify closing the artificial opening, and also to ascertain whether there is sufficient healthy bowel below the lesion to enable a resection to be done. If a closure is to be done it must not be postponed too long, or the bowel will have become too narrow below the colostomy. Six to eight months is probably about long enough, and, of course, in any case, complete subsidence of all inflammatory symptoms should have taken place. In most of the cases coming under my care the colon at the site of the trouble has been too badly damaged, or its lumen has become too restricted, to warrant any attempt at closing the colostomy without resection of the lesion. I have, however, on several occasions resected the damaged bowel and rejoined the ends successfully, but I have considered it safer to leave the closing of the colostomy to a subsequent operation after the newly joined bowel had properly united.

Resection of the damaged bowel is rather a difficult matter, as there are almost invariably quite a number of dense adhesions to be dealt with before it is possible to free the bowel enough for a resection, and it must always be borne in mind that we are dealing with old septic material, which must not be cut into. When the join has been

made, and as a rule only an end-to-end join is possible, the junction should be wrapped up in omentum. Occasionally it is possible to close the colostomy by short-circuiting the affected part of the bowel. In my experience this procedure has not been very successful, as the fact that faecal material can still get into the diverticulum has resulted in subsequent fresh attacks of inflammatory mischief.

TREATMENT OF GLAUCOMA BY ADRENALINE AND HISTAMINE.

(THE SO-CALLED "GLAUCOSANS.")

BY

W. S. DUKE-ELDER, M.A., D.Sc., Ph.D.,
M.D., F.R.C.S.,

AND

FRANK W. LAW, M.A., M.B.
(From the Royal London Ophthalmic Hospital.)

THE importance of any drug which has a beneficial effect on glaucoma, whether acute or chronic, can hardly be over-estimated, and consequently it may be of interest to look into the claims made by several authors—notably by Hamburger (1923-28)—on the beneficial effect of the so-called "glauco-san" group of substances in bringing about a diminution of the intraocular tension. This name, introduced by Hamburger, is unfortunate, since the two substances upon which the "glauco-sans" are based—adrenaline and histamine—are widely different in both their chemical constitution and their physiological properties.

THE ACTION OF ADRENALINE.

Adrenaline, which is the product of the activity of the chromaffine cells of the medulla of the suprarenal glands, is a methyl-amino-pyrocatechol, with the following formula (Takamine, 1901):

Since it contains an asymmetrical carbon atom there are two possible isomeric forms: the laevo-rotatory form occurs naturally, while the dextro-isomer can be made synthetically. The laevo-form is extremely active, being detectable in a dilution of one part in 250 million of saline (Pysemsky and Kravkov, 1912), but the synthetic dextro-isomer is considerably weaker, so that the racemic mixture has only about half the activity of the natural product (Stolz, 1904). Although the rationale of the action of this substance on the blood vessels has been the object of an immense amount of research since the original investigations of Oliver and Schafer in 1895, it cannot yet be said to be completely elucidated. The generally accepted interpretation of its activity at the present time is as follows:

1. In very small doses it dilates the capillaries, probably by direct action upon the capillary walls, thus causing a small drop in general blood pressure (Dale and Richards, 1918) and an increase in the permeability of the capillary walls (Oinuma, 1924).
2. In larger doses, while the first action may to some extent persist, it is completely masked by an active constriction of all the arteries and arterioles which are supplied by the sympathetic nervous system; this is the generally accepted "classical" view of the action of adrenaline (see Anrep, 1912; Elliot, 1912-13; and many others), and it brings about a great increase of the blood pressure.
3. In relatively concentrated doses applied locally, adrenaline causes a direct contraction of the terminal arterioles, the capillaries, and the small venules (Cotton, Slade, and Lewis, 1915), a reaction which can be observed directly with the microscope by injecting a minute quantity into the skin (Carrier, 1922; Heimberger, 1925). Here the drug acts directly on the walls of the vessels, the reaction being equally vivid after denervation (Breslau, 1919; Lewis, Grant, and Harris, 1927).

THE ACTION OF HISTAMINE.

There is a considerable amount of evidence that histamine, or histamine-like substances, are produced by all organs of the body. Like adrenaline it is an extremely reactive

substance, an injection of one-hundred-millionth part of a milligram producing a well-marked effect. Its chemical name is imin-azoly-ethyl-amine, with the formula:

The general action of histamine is to constrict plain muscle (Dale and Laidlaw, 1910); this includes the musculature of the arterioles, of the larger veins, and also, incidentally, of the pupil. This is accompanied by a dilatation of the capillaries (Dale and Richards, 1918), caused by a direct action on the walls of the vessels comparable to the effect of small doses of adrenaline. The first of these effects tends to produce a rise of blood pressure, and the second produces a profound fall. In such animals as the cat both of these reactions are evident; in dogs, monkeys, and man the constrictor effect on the arterioles is completely masked (Burn and Dale, 1926; Harmer and Harris, 1926), but the dilator effect on the capillaries seems to be universal throughout the mammalia (Lewis and Marvin, 1926; Lewis, 1927). So far as the blood vessels are concerned, therefore, the generally accepted action of histamine in man appears to be:

1. A dilatation of the capillaries by local direct action upon their walls.
2. A widespread dilatation of the arterioles by a local reflex nervous mechanism.
3. An increased permeability of the capillary walls leading to widespread oedema, a result of the direct action of the drug on the capillary walls (Drury and Jones, 1927).

It would seem, therefore, that in their general action the two substances, adrenaline and histamine, are antagonistic; and there is a large amount of evidence that they play such a reciprocal part throughout the body generally, histamine, or histamine-like substances, tending to dilate the capillaries, this influence being kept in check by the constrictor effect of adrenaline (and pituitrin) (see Dale, 1926).

ACTION ON THE EYE.

The action of adrenaline on the intraocular pressure was first adequately studied by Wessely (1900), Parsons (1903), and Henderson and Starling (1904), and later work has merely served to substantiate their conclusions (Thiel, 1924; Samojloff, 1925; and others). There are two main opposing influences at work: the local constriction of the vessels tends to bring about a lowering of the intraocular pressure, while the general rise of blood pressure tends to bring about a rise. In experimental animals either of these may be evident, or they both may occur, the second following the first; in man there is usually a period of hypotension which is followed by a slight rise. It would appear that in cases of simple glaucoma this reaction is retained, the fall of tension being more evident, but that in the acute or absolute forms of the disease the opposite condition may develop. When injected into the blood stream adrenaline dilates the pupil widely, an action which is evident on subconjunctival injection, but which is retained only with difficulty on instillation into the conjunctival sac except in specially sensitive persons.

After the work of the early experimenters several clinicians used adrenaline clinically for the relief of tension in glaucoma, with, in general, indeterminate results (see summary by Rupert, 1909). Erdmann (1914) and Köllner (1918) revived its use, and recently the extensive writings of Hamburger (1923-25) have attracted considerable attention to the matter. He used the drug as a subconjunctival injection of 0.2 to 0.3 c.cm. of 1 in 1,000 solution after the instillation of holocaine, and his technique has been extensively followed (Hagen, 1923; Gradle, 1924; Römer and Krebs, 1924; Heimann, 1924; Jaensch, 1924; Kayser, 1924; Kafka, 1924; Thiel, 1924-25; Safar, 1925; Dominguez, 1925; Vannas, 1927). Fromaget (1921) employed it as a retrobulbar injection, and he has been followed by Aubaret (1923), Bailliart and Bollack (1921), Liebermann (1924), and Bonnefon (1925); Knapp (1921) relied on drops instilled into the conjunctival sac; Thiel (1925) applied it as an ointment; and Gradle (1925) on a pledget of cotton-wool. While many of these observers reported good results, they were by no means invariably successful;