almost cheerful. Respirations 30. The dressing was soaked with black "coffee grounds" material mixed with gastric secretion and mucus. Both tubes were flushed out with saline. The abdomen was exquisitely tender all over; a stitch removed near upper tube to facilitate drainage. There was slight hiccough, the thirst was intense, and the tongue dry.

On November 10th the wound was dressed twice, there being some mucous discharge from the fistula. She had slept well, thirst was less, and the tongue more moist; slight bronchitis and congestion were present at the right base. Temperature, morning, 97.8°, evening, 99.2°; pulse 100; respirations 24. The tube was removed from the pelvis on the following day, and from the upper wound on November 12th there was considerable discharge of saliva, mucus, and gastric juice. The bronchitis was better; several liquid faecal motions had been passed during the previous afternoon and night. Thirst less, tongue moist, and there had been no return of abdominal pain since an opium suppository on November 11th; methylene blue solution $\frac{3}{4}$ j, was given by the mouth, and dressing was stained when changed in the evening.

On November 13th she was improving; much less thirst, but had parotitis on the left side. Pulse 110, temperature 98° at 6 a.m., 100.4° at 10 a.m. She had several loose faecal motions, and during night and on November 14th the parotid was more swollen and acutely tender.

On November 15th the fistula, which was still discharging, was plugged and mouth feeding commenced. There was very little discharge on removing the plug in the evening. Digitalin was stopped; strychnine reduced to $\frac{1}{8}$ gr., six-hourly. The rectal saline injections were stopped; she had had six fluid motions in 24 hours.

On November 16th the right parotid was swollen and tender. Fistula dressed. One ounce clear mucus and curd escaped when the plug was removed. She had had five small fluid motions in the past twenty-four hours.

On November 18th a left parotid abscess was incised, and on the following evening a right parotid abscess, and a quantity of thick pus evacuated. The fistula still discharged when the plug was removed. The general condition was improved. Urine showed traces of albumen.

On November 23rd the purulent discharge from both parotids was profuse. The abdominal wounds were healing and looking cleaner. The fistula still discharged when the plug was removed.

November 28th. The temperature rose yesterday to 103°, and she had pain over the back and postural oedema of the back and lower part of the right side of the chest. About midnight she began to expectorate purulent, fetid, watery material constantly without any effort, to the amount altogether of about $\frac{1}{2}$ pint. When the chest was examined on November 29th it was found dull over the right side posteriorly and breath sounds were absent. Pus having been found on exploration with a syringe, the chest was opened, 2 in. of rib excised in the posterior axillary line, and about 2 pints of foul pus evacuated. A tube left in. She experienced very little shock from the operation, and had a very comfortable night; the quantity of expectoration was small, but the discharge from the chest wound was copious. Temperature 98°; pulse 120, strong and regular.

On December 10th it was noted that she had had some oedema of the left foot and leg for three weeks; it began in the foot and spread up to the thigh. During the past week this had become worse and had extended to the lower part of the back and the left labium majus, causing some difficulty in micturition. The general condition was better to-day; she slept better and the cough, which had been severe, was less troublesome.

On December 11th, after methylene blue solution $\frac{3}{4}$ j, given by mouth, there was no stain on either dressing, but the urine blue. On December 23rd it was noted that no food had come through the gastric fistula for a week, and on January 8th, 1908, the oedema of left thigh had entirely gone. When the chest was examined on January 19th some crepitation was still present over the base of the right lung, with impaired resonance, and there was still slight discharge from the empyema wound. The gastric fistula was practically healed. She was getting up in the afternoon, but complained of pain in the epigastrium on slight exertion; it had apparently no relation to food.

On January 23rd she was sent to a convalescent home at Torquay.

REMARKS.

When I first saw this case I regarded it as quite hopeless and was disinclined to operate. She was well advanced in the final stage of perforative peritonitis, that of septic intoxication. Free from pain and almost void of tenderness on pressure of her meteorized abdomen, apathetic and complaining only of thirst, and with a pulse of 140, it did not seem possible that even a successful operation could lead to recovery, while there was every probability that an operation upon an abdomen so far advanced in a septic adhesive peritonitis would cause her much pain and hasten her death by shock. Supported, however, by the opinions of my colleagues, Dr. Davy and Mr. Harmer, and requested by the patient, I decided to give her what chance there was. Published statistics show that it is of little or no use to operate when 36 hours have elapsed after perforation of the stomach or duodenum, if the perforation is an acute one, and one naturally seeks for the explanation of her recovery after a period of such

unusual length. That she was profoundly toxic is shown by the complications which she suffered; while, although I have not seen her since she went to Torquay, I cannot doubt that her abdominal pains are due to adhesions, which were universal at the operation and which are exceedingly likely to call for surgical treatment in the future.

I am inclined to think that she owes her recovery to the dilution of the toxins by, and the envelopment of the pathogenic bacteria in, the large quantity of milk which was free in the peritoneal cavity. I am not a bacteriologist, and do not even know whether milk is a good culture medium for the various bacteria which one finds in the stomach, but I can quite imagine that bacteria, or rather their toxins, enveloped in what we in Devon call "junket," would be not readily absorbed by the peritoneum, and that the delayed absorption of toxins thus caused just saved this patient's life. I cut down upon a typical "junket" in the epigastrium, and found another, in a more advanced stage, in the pelvis; and, from the absence of any further post-operative discharge of a similar nature, suppose that my copious irrigation of the kidney pouches and pelvis fortunately removed most, if not all, of this material which was fouling the lower abdomen. Lest I should be misconstrued to advise feeding a perforated stomach, I must state my conviction that the case most likely to do well is one which perforates while the stomach is empty; and further, that, had this patient drunk water or beef tea instead of milk, she would, owing to the greater diffusibility and absorbability of those fluids, have been moribund or dead from septic absorption before 44 hours had elapsed. There was no difficulty in tracing the perforation in this case, inaccessible as it was; because, owing to the presence of the mass of curded milk which prevented the formation of adhesions, elsewhere universal, a track to the ulcer was retained.

THE SCIENCE COMMITTEE OF THE British Medical Association.

REPORT CVIII.

SOME POINTS ON THE EXPERIMENTAL PRODUCTION AND CONTROL OF THE VASCULAR ATONY OF SURGICAL SHOCK.*

BY

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AND

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INTRODUCTION.

THE term "shock" is unfortunately employed to cover conditions due to many causes. For the purposes of this work it is applied to a state characterized, *inter alia*, by low vascular tone, evidenced by sustained fall of blood pressure, and produced by prolonged excitation of various peripheral nerve areas by manipulation or electrical stimulation.

This definition was, in substance, adopted by one of us (P. L. M.) in the Hunterian Lectures¹ at the Royal College of Surgeons in 1905.

The main object of the work was to test the action of some of the drugs which raise blood pressure, with the view of gauging their value for the treatment of shock, and of determining the best mode of administering them. This led secondarily to observations on the comparative effects of certain manipulations in producing shock and on the blood in that condition. The present paper deals with these originally secondary issues, with the mode of administering drugs in shock, with the use of adrenin

* Towards the expenses of this work a grant was made by the Science Committee of the British Medical Association.

in particular, and with a preliminary report on that of pituitary extract, and ergot.

PRODUCTION OF SHOCK.

The whole of the experiments were carried out under complete anaesthesia by ether or chloroform, and all the animals killed without recovery therefrom. The animals used were cats and dogs, and the main modes of procedure were two.

In the first a condition of extreme shock was produced by total destruction of the central nervous system. This was done under anaesthetic, either by decapitation followed by pithing the cord, or by pithing both brain and spinal cord through the orbit. The latter of these alternatives is the more convenient, on account of its greater rapidity and the smaller haemorrhage which it entails. Most of the pithing experiments were done on cats and resulted in a blood pressure of about 30 mm. Hg, which, provided that artificial respiration were efficiently maintained, did not fall much further for some time.

The value of the pithed animal in studying the treatment of the low blood pressure of surgical shock is obvious. A drug that fails to raise the arterial pressure of such an animal will also fail to raise that of the surgically "shocked" animal, or will raise it, if ever, by acting on the central nervous system—that is, by stimulating a depressed organ, the logical treatment of which, as urged by Crile,² is rest. Hence any drug, to be of value in raising blood pressure in surgical shock, must raise blood pressure in the pithed animal. It did not appear necessary at this stage to supplement the observations on the pithed animal by separate perfusions of the coronary and other blood vessels, since no drug that raises and maintains blood pressure when circulation is effected by the heart, will at the same time produce any material depression of that organ. But the pithed animal, though an excellent or even indispensable preface to the surgically "shocked" animal in this study, is no complete substitute for the latter. Drugs which will raise blood pressure after pithing will not necessarily do so in the absence of pithing. Hence those which acted favourably on the pithed animal were tested further on animals in lesser degrees of shock.

The second mode of procedure was to produce "shock," either by electrical excitation of the central ends of afferent nerves or by manipulation of abdominal viscera, both under complete anaesthesia. Of these, the second alternative proved the more efficacious, owing largely to the fact that local injury to the nerves from the exciting current diminished the effects of excitation. In these observations dogs were usually employed, as cats proved less susceptible to shock under the conditions of experiment—that is, under complete anaesthesia. We have made no observations in the absence of this condition.

As above stated, it was found that electrical stimulation produced shock but slowly. The method was to excite by induction shocks (faradism), once per minute, the central ends of cut afferent or mixed nerves. The nerves used were the brachial, lingual, anterior crural, sciatics and their branches, and the nerves accompanying the spermatic cord. Weak stimulation produced but little effect, and strong excitation rapidly damaged the nerve, so that the electrodes had to be continually shifted centrewards. It must not be inferred that such excitation is devoid of danger. Sudden death from stimulation of the laryngeal and abdominal branches of the vagus, and to a less extent from that of other afferent nerves, is well known. This has often been imminent, and has happened at least once in the course of our experiments. But electrical excitation did not lend itself well to the production of non-fatal shock, and was therefore given up in favour of manipulation.

This was done on various abdominal viscera and nerves, the peritoneum, and the testes. The immediate result was temporary rise or acute fall in arterial pressure, accompanied by respiratory disturbance, which passed ultimately into sustained fall of arterial pressure with diminished or abolished respiratory response. These are well-known reflex responses to nerve stimulation. Their effect on blood pressure was partly cardiac, partly arterial in origin, and was only regarded as shock when sustained,

and when strong electrical stimulation of the (undamaged) central end of the sciatic nerve produced little (or no) rise or actual fall in blood pressure. This convenient test, which was suggested by Crile's work, does not seem to have been used by him as a criterion of shock. Curare was not used in any of our experiments.

The manipulations employed were largely those liable to occur during abdominal operations, and comprised traction, compression, sponging, scratching, and cutting.

Of these, traction proved uniformly the most effective, and was especially so when tension was intermittently put on the mesentery, hepatic ligaments, stomach, or (exposed) mesenteric ganglia and nerves.

The effect of compression was variable. Clamping the bowel was ineffective, whilst clamping the stomach usually produced a fall of blood pressure. Ligation of or nipping a pedicle of mesentery often produced marked immediate fall in blood pressure.

Scratching or sponging the parietal peritoneum or mesentery usually evoked response, whilst the peritoneum covering the viscera was generally irresponsive. As stated by Crile, swabbing the abdominal cavity sometimes produced marked results.

Cutting was rarely effective, and castration produced less effect than repeated gentle traction on the spermatic cord.

Generally speaking, those procedures which produced the greatest immediate reflex response led most rapidly to the onset of surgical shock defined and tested as above, and conspicuous amongst them were the less destructive manipulations.

The above results will be described by one of us (Mummery) in fuller detail elsewhere, in their bearing on surgical technique.

The anaesthetic was not without influence on the production of shock, although in all cases our anaesthesia was complete. In the earlier observations chloroform was employed, but on account of the frequent occurrence of syncope, from manipulations during its use, it was later displaced by ether. With ether the tendency to syncope was distinctly less, and the onset of shock slower, though ultimately scarcely less certain. But though chloroform during dangerous manipulations facilitates onset of shock, yet, if given in advance and followed by ether before the commencement of such manipulations, it appears to have the opposite effect. In one instance, a dog which had been under chloroform two hours before the production of shock was systematically attempted, proved practically insusceptible to operative shock.

OBSERVATIONS ON THE BLOOD IN SHOCK.

In this state we have found a marked fall in the specific gravity of the blood.³ This fall gradually follows that of arterial pressure, and is greatest when the fall in pressure is greatest, that is, when the animal is pithed. It also follows the smaller fall in arterial pressure produced by anaesthetics, slight shock, and by nitrite of amyl. It is turned when arterial pressure is raised. Hence the fall in arterial pressure appears to be the chief cause of the fall in specific gravity. These points are illustrated by the following tables:

TABLE I.
Cat, 2.6 Kilograms.

Time.	Arterial Pressure.	Specific Gravity of Blood.
2.30 p.m.	200 mm. Hg.	
2.45	172	1055
*3.0	60	
3.10	34	1045
3.50	35	1040
†4.15	25	1035
4.40	110	1040
4.50	100	1045
5.0	130	1045
5.45	70	1035

* Pithed.

† From 4.15 to 5.45 continuous injection of 1 in 25,000 suprarenin, about 2 c.cm. per minute.

This shows considerable fall in specific gravity associated with great fall in arterial pressure, followed by rise in specific gravity when arterial pressure was raised.

* Conspicuous amongst such are preparations of ergot.

TABLE II.
Cat, 3.8 kilograms.

Time.	Arterial Pressure.	Specific Gravity of Blood.
3.10 p.m.		1068
3.23	160 mm. Hg	
3.40	120	1063
4.10	125	1063
4.40	120	1062
4.45	amyl nitrite inhaled	
4.55	"	1060
5.20	"	1060
5.35	"	1055
6.0	"	1055

This shows fall in specific gravity from anaesthetic alone in the first instance, from nitrite of amyl, during anaesthesia, later.

Fall in specific gravity of the blood is well known to follow severe haemorrhage. But in our hands the haemorrhagic fall has been small. For instance, in a cat (2.5 kg.) from which 40 c.cm. of blood were drawn, the specific gravity, which was 1050 immediately before the bleeding, did not fall as low as 1045 after it. Hence, since even in our decapitation experiments the external haemorrhage rarely amounted to 20 c.cm. (by measure), we do not attribute to this factor (bleeding) any great share in the large falls of specific gravity that we have observed.

Fall in specific gravity of the blood implies transudation of tissue fluid into the blood stream. This is confirmed by the accompanying diminution in percentage of the haemoglobin. In a cat (2.7 kg.) the haemoglobin, on the Gowers-Haldane scale, fell from 70 to 58, whilst the specific gravity of the blood dropped from 1048 to 1043. This percentage diminution of the haemoglobin shows that each 100 volumes of blood was diluted (in round numbers) to 120, which, in conjunction with the accompanying fall in specific gravity of the blood, indicates that the transudate had a specific gravity of 1018, that is that of average lymph.

The reaction of the organism, then, to fall of blood pressure, is increase in volume of the blood. That such an increase will tend to restore the lost pressure is obvious. It is, therefore, not unreasonable to regard the reaction as protective, and to infer that transfusion is the logical treatment of the condition.

But a suitable transfusate has yet to be found. In common with Crile, we have found the effect of transfusion of saline solutions, on the blood pressure, to be but transitory, the transfusate leaving the blood, and, if given in excessive quantity, producing marked oedema. Perfusion of "surviving" organs, with so-called "normal" fluids leads to the same result (cf. Locke and Rosenheim), and that rapidly, unless the perfusion pressure is kept far below the arterial pressure of normal life.⁴ Hence, in spite of its theoretical value, there is little to be hoped for from transfusion, until we have a fluid that the blood vessels are able to retain.

Sherrington and Copeman found, in 1893,⁵ that on opening the abdomen in anaesthetized rabbits, there was an immediate slight rise in specific gravity of the blood. We have noticed a like increase once or twice in the cat, and attribute it to the rise in blood pressure which usually immediately follows abdominal section in these animals. That we did not notice it more frequently is possibly due to the fact that our scale of specific gravity was usually coarser than that of these writers, only the larger of the increments described by them being recognizable in it.

ADMINISTRATION OF DRUGS IN SHOCK.

That a drug may produce any immediate effect on a pithed or severely shocked animal, it is essential that it be injected into the actual blood stream—that is, into a large vein.

We have found absorption from subcutaneous tissue and from muscle to be almost zero in such cases. In the pithed animal intravenous injections of some ergot preparations produced well-marked and sustained increase in blood pressure, whilst hypodermic and intramuscular injections of the same samples were almost without effect. In the severely shocked (not pithed) animal hypodermic and intramuscular injections of strychnine produced no obvious effect on the pulse-rate or blood pressure, whilst intravenous injections led to a noticeable though slight and transitory elevation, followed by steady fall, of the latter.

The bearing of this on the treatment of severe shock is

obvious and important. In such a condition drugs administered hypodermically enter slowly (if at all) into the blood stream, until the condition which they are designed to treat is passing off. Such administration is, therefore, largely valueless. Moreover, if repeated injections are given of drugs such as strychnine, which are not destroyed in the tissues, recovery of blood pressure may lead to rapid absorption of an undesirably large dose. Unless otherwise stated, all our injections have been intravenous, and to ensure early admixture of injection with blood—that is, to avoid temporarily flooding the heart with a concentrated dose—the femoral vein was invariably employed.

ACTION OF ADRENIN* (= ADRENALIN, SUPRARENIN, ETC.)

As is well known, this body produces intense vaso-constriction by direct action on the arterioles, together with cardiac inhibition of bulbar origin, which latter gives place to acceleration when the vagi are cut or the animal atropinized (Oliver⁶ and Schäfer).

In agreement with this, we have found its action on the pithed animal to be much more intense than on an animal with normal, intact nervous system.

Shock, like pithing, intensifies its action, and we have found that during shock division of the vagi does not always increase its effect on blood pressure. But, however severe the shock, the response to adrenin is never so great as in the animal with nerve centres completely destroyed.

In all cases its effect is transitory (a minute or two), but is somewhat prolonged when body temperature has become slightly subnormal (cf. Langlois⁷ and Elliott⁸). We have never met the fatal syncope imputed to it by other workers, but have, after high blood pressures, seen haemorrhages in the heart (ventricular wall) and lungs on *post-mortem* examination. Paton,⁹ Elliott,⁸ Drummond,¹⁰ and others have found similar lesions after high pressures, and, in addition, other toxic effects from subcutaneous injections which were not attributable to high pressure. Hence the drug should be used as sparingly as possible.

We fully confirm the statements that subcutaneous and intramuscular injections of adrenin, even when large, produce little or no effect on the blood pressure. This is true in the presence as well as in the absence of shock. Such a mode of administering it is therefore useless for the treatment of shock, and gratuitously exposes the patient to the risk of the toxic effects above referred to.

Hence it should be given, when at all, by intravenous injection.

Since its action is transitory, injection, to be effective, must be continuous or frequently repeated. The latter alternative has the disadvantage that for a given average effect it necessitates greater concentration of the drug, thereby incurring greater risk of heart trouble in the absence of atropine, and of temporarily excessive blood pressure if atropine be also given. Continuous injection of a dilute solution (for example, 1 in 50,000), as described by Crile,³ is preferable. This, especially with the aid of atropine, enables production and maintenance of practically any desired blood pressure, with a minimum of dose.

ACTION OF EXTRACT OF THE POSTERIOR LOBE OF THE PITUITARY BODY.

That pituitary extracts produce vaso-constriction of greater duration than that due to adrenin was shown by Oliver¹¹ and Schäfer. Howell¹² located the active principle in the posterior lobe, and noted that it produced depression of the heart, and a certain degree of "immunity" to the second and subsequent injections—results confirmed by Schäfer¹³ and Vincent. Schäfer¹⁴ and Herring showed that it increased the secretion of urine, whilst adrenin had the opposite effect.

We have worked with two preparations, the effects of which are similar, namely, a dried extract prepared by Messrs. Willows Francis and Butler, and a liquid extract manufactured by Messrs. Parke, Davis and Co. As with adrenin, the effect of these on blood pressure is more certain and more intense on the pithed animal. The subjoined table illustrates the effect on the pithed animal. The complete tracing is too long for reproduction, the major portion of the record is therefore given in tabular form.

* A name recently suggested by Professor Schäfer for the active principle of suprarenal medulla, which does not imply any particular proprietary product.

Tracing of the carotid blood pressure of the cat of Table III, showing the effects of three equal injections of liquid extract of the posterior lobe of the pituitary body. The first produced considerable and sustained increase in blood pressure (see Table); the second and third, much less increase, preceded by a fall. The time record shows minutes.

TABLE III.
Cat, 3.6 Kilograms. Ether.

Time.	Mean Carotid Pressure.
3.3 p.m. ...	135 mm. Hg.
3.11 pitthed (brain and cord)	...
3.15 ...	45*
3.17 liquid extract injected through femoral vein†	...
3.18 ...	180†
3.20 ...	140†
3.30 ...	120
3.40 ...	112
3.50 ...	100
4.0 ...	80
4.15 ...	59†

* This pressure is unusually high. Care was taken to verify that pithing was complete.
† See diagram.

The resulting rise in blood pressure, after its preliminary partial subsidence, remained comparatively stationary for nearly half an hour, and had not fully disappeared an hour after the injection. The effect of two subsequent injections of the same amount is shown in the figure, which also illustrates the degree of the "immunity" described by Howell, that we have generally met. The slowing of the heart, also noted by Howell, though often present, is only obvious in our records when sufficient to produce a preliminary fall of blood pressure. No such fall preceded the rise due to the first injection in the present case, though the phenomenon is recognizable at the second and third doses.

In the intact animal we have observed an analogous effect on the respiration.

Injection of pituitary extract, before pithing, gives the arterioles a tone which prevents the collapse of blood pressure incidental to destruction of the vasomotor centres.¹⁵ This is shown by the following experiment, in which three intravenous injections were given prior to pithing, all of which produced slight temporary cardiac depression, and neither of which was followed by any great rise in arterial pressure.

TABLE IV.
Cat, 3.1 Kilograms. Ether.

Time.	Mean Carotid Pressure.
4.0 p.m. ...	156 mm. Hg.
4.1, pitthed (brain and cord)	...
4.5 ...	110*
4.10 ...	88
4.30 ...	78
4.50 ...	74
5.50 ...	66

* Pithing usually lowers mean carotid pressure to about 30 mm. of mercury.

Here, almost two hours after the pithing, the mean arterial pressure was 66 mm. Hg—that is, about double that which normally immediately results from destruction of the central nervous system, and 3 to 4 times as great as that which would have existed at the same time had no injection been made.

On surgically "shocked" animals the effect is greater than on animals not in a state of shock. The following is one of our larger responses in this condition. Shock had been produced under chloroform in a cat weighing 3.3 kilograms. The induction of shock lasted three hours, during which three acute attacks of syncope had occurred, and at the end of which excitation of the sciatic nerve led to fall of blood pressure. The shock was therefore severe.

TABLE V.

Time.	Mean Arterial Pressure.
1.54 p.m., induction of shock commenced	150 mm. Hg.
4.54 ...	66
4.55, injection of pituitary extract	...
4.56, clot in cannula	...
5.3 ...	146
5.30 ...	120
5.58 ...	90

The reading of the initial rise in blood pressure was masked by the formation of a clot in the arterial cannula. On clearing this, there was found an arterial pressure almost equal to that at the commencement of the

experiment—that is, allowing for some subsidence, the injection had led to a rise of pressure of more than 80 mm. of mercury. An hour after the injection more than 30 per cent. of this rise was still in evidence, and, excitation of the sciatic nerve again produced the normal increase in blood pressure as before induction of shock, thereby indicating that some recovery of the vasomotor centres had taken place.

In many cases the first pituitary injection is followed by temporary depression in amplitude of respiration. This is usually slight, and rarely amounts to complete arrest. In all the cases in which we met it recovery was apparently complete. We have never known it follow a second or subsequent injection. It is more pronounced in the dog than in the cat.

In short, the increase in blood pressure produced by this body lasts thirty to sixty times as long as that (increase) of the same amplitude from adrenin. Simple repetition of dose is sometimes useless for raising blood pressure, but the first dose confers no "immunity" against adrenin. Given after the pituitary effect has almost, or quite, passed off, adrenin increases arterial pressure certainly to no less an extent, and seemingly for a longer time, than it does in the absence of a previous pituitary injection. It appears, then, that extract of the posterior lobe of the pituitary body promises to be of some value in the treatment of shock when severe enough to call for intravenous injection. A single injection of it suffices to permit of much recovery, and does not interfere with the subsequent use of adrenin if employment of that drug is indicated.

An additional point in its favour is the above-quoted observation of Schäfer and Herring, that it does not diminish, but increases the activity of the kidney.

Further study; however, of its dosage and after-effects is desirable before it can be advocated for general administration to the human subject.

ACTION OF ERGOT.

The sustained arterial tonus evoked in pithed cats without unduly high arterial pressure by intravenous injection of some samples of ergot preparations, led us to hope that it would be reproducible by them in less shocked animals. But in no case have we succeeded in reproducing the desired result. The normal effect of our samples of ergot preparations in surgical shock was zero or further fall of blood pressure. We cannot, therefore, point to any preparation of this drug as of value in the treatment of shock.

SUMMARY.

In fully anaesthetized animals non-destructive manipulations of abdominal viscera are more productive of shock than are gross injuries.

Such manipulations produce shock most rapidly when they implicate the parietal peritoneum, the peritoneal ligaments, or the mesenteries. Shock is more readily produced under chloroform than under ether.

In severe shock hypodermic injections are but slowly, if at all, absorbed into the blood stream.

In severe shock intravenous injections of adrenin, and of extract of the posterior lobe of the pituitary body, raise arterial pressure to a greater extent than in the normal state.

A single injection of the latter influences arterial tone for upwards of an hour without producing abnormally high arterial pressure.

This influence is sufficient to enable considerable recovery from shock, and does not interfere with subsequent injection of adrenin, if that be desirable.

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- ¹² *Journ. of Exper. Med.*, vol. iii.
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AT a meeting of the Medical Society of Italian Switzerland, held on August 15th, it was unanimously decided that all the Poor-law medical officers belonging to the society should resign. This decision was come to in consequence of the failure of the society to obtain for them from the Government and the Grand Council any redress of their grievances.

SEVENTY-SIXTH ANNUAL MEETING

OF THE

British Medical Association.

Held at Sheffield on July 24th, 25th, 27th, 28th, 29th, 30th, and 31st.

PROCEEDINGS OF SECTIONS.

SECTION OF OBSTETRICS AND GYNAECOLOGY.

RICHARD FAVELL, M.R.C.S., President.

By way of opening the work of this Section the PRESIDENT, Mr. Richard Favell, contented himself by bidding a few words of welcome to its many foreign guests, and then called on Dr. Herman to open the first discussion.

DISCUSSION ON THE TREATMENT OF UTERINE DISPLACEMENTS.

OPENING PAPER.

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THE TREATMENT OF UTERINE DISPLACEMENTS.

I HAVE been asked to open a discussion on "The Treatment of Uterine Displacements." The question which first arises is—What are uterine displacements? I would divide them into two classes: the common and the rare. The rare displacements are inversion, and the cases in which the uterus has been found dragged into hernial sacs. I shall not discuss these, because as to the principles on which they should be treated there is no difference of opinion; and as to details, no individual has treated so many of these rare cases as to enable him to lay down the law about the comparative merits of different methods.

The common displacements, those which every man in family practice must treat, are those which I think it is intended we should discuss to-day; and because they are so common I hope the discussion may be interesting and instructive to us all.

The conditions meant by the words "displacements of the uterus" are nothing but descent of the uterus and its effects. The Obstetrical and Gynaecological Section of the Royal Society of Medicine has recently been discussing the means by which the uterus is supported. One writer says that the uterus is held up by the fibrous tissue surrounding the vessels—in other words, by the pelvic fascia; another says that the main support of the uterus is the levator ani. I think that these authors are both right. The uterus is supported by the pelvic fascia, the connective tissue in which the vessels run, and the levator ani muscle.

In most of the common cases of displacement there is nothing the matter with the uterus; the disease is that the pelvic floor is weakened or injured, and the change in the place of the uterus is only the palpable sign of defect in the pelvic floor. Thirty years ago books were full of descriptions of various versions, flexions, flexures, torsions, etc., of the uterus; but it is now indisputable that a curve in the canal of the healthy uterus is not disease. Change in its position may disturb the circulation through the uterus. When the uterus sinks, it generally leans back. In about one case in ten the folds which bound the pouch of Douglas on each side are unusually firm, and are made more tense when the cervix sinks or is moved forwards by the falling back of the uterine body. If this is the condition of the utero-sacral ligaments, when the body of the uterus sinks into Douglas's pouch the veins which return the blood from the uterus are compressed against these folds, and the body of the uterus in consequence becomes congested, swollen, and tender.