urgently required, without any loss of time, as the technique is simple.

2. The technique involves less labour, and entails no sacrifice of rabbits—that is, it is economical.

3. Human blood can be used for the preparation of this medium when required for special purposes or for general use.

4. The fluid does not alter appreciably by age within the limit of four to six weeks, owing to the small surface exposed to evaporation.

5. A larger quantity of the culture fluid in each culture tube is available, so that when abundant cultures are required—for example, for the preparation of vaccine—this gives a decided advantage.

6. The culture fluid keeps sterile during the working period of six weeks more easily than does the N.N.N. medium in the tropical laboratories.

NOTES ON FOUR CAESAREAN SECTIONS.

BY

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The following four cases requiring Caesarean section fell to my lot within the short period of nine months; in each case, except No. 1, one or more craniotomies had been previously performed, the narrowed pelvis being caused by rickets.

Sterilization was done in each case by the following method: The tubes were clamped with small forceps close to the uterine cornua, the mesosalpinx transfixed by double ligature, one ligature tying off the proximal end of the tube close to the uterus, the other ligature being brought round the fimbriated end, thus removing the whole tube; the serous coat of the uterine end of the tube was stitched over. In two of the cases I was able to deliver the uterus, which I think is an advantage, although it needs a slightly longer incision; but there is less bleeding, as the uterine arteries can be easily controlled by an assistant and cloths are placed behind the uterus, completely shutting off the intestines. The placenta in all four cases was attached to the anterior uterine wall; this occurs in 75 per cent. of cases, but there is no difficulty in recognizing the placental tissue on incising the uterine wall. With regard to sutures for the uterus, I think No. 3 catgut for the main and No. 2 for the intermediate, both specially prepared to resist absorption for twenty-one days, is preferable to silk or Pagenstecher's material. Ligatures should not be tied too tightly, as the tissues of the gravid uterus are soft and easily cut through; a double hitch for the first half of the knot, and completed, followed by a single knot, is, I think, preferable to three single knots.

As to the time of operating, the best, in my opinion, is before the commencement of labour pains, the operation can then be conducted in daylight, adequate assistance secured, and the usual preparation for an abdominal operation can be carried out; the fear of haemorrhage and uterine inertia need not be considered seriously.

Sterilization was done in each case by the patient's desire, and I think the method described above is likely to prove satisfactory. It is stated that excision of a "portion" of the tube has been followed by pregnancy, even when the ovaries have been removed; this I can well understand, if a small portion of ovary has been left behind. This matter is fully referred to and discussed by A. J. Wallace and Sir W. Sinclair, in the *Journal of Obstetrics and Gynaecology of the British Empire*, 1907.

CASE 1.—S. L., aged 31, primipara, had arrived at full term. She was first seen by Dr. Anderson, of Bodmin, May 6th, 1911, who says, "There was no dilatation of the os, or other signs of labour. Next morning the pains were few and infrequent, and the os just admitted the tip of the index finger, but was too high up to make out the presentation; she was a short, rickety woman with a contracted pelvis; the antero-posterior diameter was $2\frac{1}{2}$ inches."

She was brought into the East Cornwall Hospital, Bodmin, and I was asked to see her with a view to Caesarean section. On May 8th I was able to confirm Dr. Anderson's notes, and performed an immediate Caesarean section. The child was dead, but the mother did well, the wound closing by primary

union. On the fourteenth day she developed slight thrombosis of the left femoral vein, which delayed her convalescence. She left hospital in six weeks quite well.

Case.	Age.	Date.	Doctor.	Previous Labours.	Convalescence.
1. S. L.	31	May 8, 1911	Dr. Anderson	Primipara	Good; child stillborn; in hospital six weeks.
2. L. W.	33	Sept. 28, 1911	Dr. Wagner	1 Craniotomy	Good; child strong; in hospital 24 days.
3. M. S.	30	Dec. 21, 1911	Dr. Hodge	3 Craniotomies	Uninterrupted recovery; in hospital 33 days.
4. L. G.	28	Feb. 3, 1912	Dr. Anderson	1 Craniotomy	Uninterrupted recovery; in hospital 30 days.

The Arris and Gale Lectures

ON

THE MECHANISM AND TREATMENT OF SHOCK.

DELIVERED AT THE ROYAL COLLEGE OF SURGEONS, MARCH 22ND, 1912.

BY

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(Concluded from page 1072.)

LECTURE II.—PART II.

THE PELVIC ORGANS AND GENITO-URINARY SYSTEM.

Pelvic Viscera.

PELVIC and perineal structures do not share in the local vascular mechanism referred to in connexion with the abdomen proper. We have shown that all variations from these regions are eliminated by blocking afferent impulses. Blood-pressure variations, therefore, from interference with these structures are a true indication of the shock value. In the human subject sigmoidoscopy, with the accompanying inflation of the bowel, causes a definite rise in blood pressure of the pressor type. A study of the blood pressure, pulse, and respiratory-rate shows: (1) That the vasomotor variations are not due mechanically to increased pressure in the abdominal cavity. For the pulse and respiratory-rate are increased instead of being slowed, as

Diagram 2.—Proportion of afferent and efferent splanchnic nerve fibres mechanically stimulated by traction on mesentery in different situations.

would then be the case. (2) That direct stimulus to the bowel wall is not responsible. For our experiments have repeatedly demonstrated that manipulation of the bowel wall is without any ascertainable effects on the blood pressure. (3) That distension of the intestine induces tension upon the mesentery, and from the stimulus to this

structure, with its contained nerves, arises the impulse to the vasomotor centre; to this the rise in blood pressure is responsible. The stretching and stimulation of the mesenteric nerves during intestinal distension has, we believe, an important bearing on intestinal activity, blood supply, and shock (from afferent impulses) in clinical medicine and surgery. The time at our disposal, however, does not permit detailed reference to this subject. (See Diagram 2.)

The Bladder and Urethra.

We have taken tracings during cystoscopy (and distension of the bladder) under chloroform and under spinal anaesthesia. The passage of instruments through the urethra under chloroform induces impulses of a definite pressor type, accentuated in the case of vesical distension. This latter is not due to a mechanical increase of intra-abdominal pressure, for (1) the gradual fall after vesical distension argues a tolerance to the stimulus, and (2) distension under spinal anaesthesia does not produce a similar rise. Postponing deductions for a moment, we present Chart 48,

Chart 48.—C.E. anaesthesia. Suprapubic cystotomy.

representing suprapubic cystotomy in a man of 64 years with malignant disease of the bladder under C.E. anaesthesia. The contracted nature of the bladder rendered considerable amount of traction essential, in order to bring the fundus up to the abdominal wall. Vesical distension in its relation to the blood pressure is well shown from A to B. B to C represents the incision of the integuments and fascia; and at C the blood pressure fell approximately to the original level during the cessation of manipulations and while the patient was placed in the Trendelenburg position. The enormous pressor effect from stimuli to the trigone of the bladder at C is really remarkable; for the original level is never regained during the tracing, and surpasses in degree all the pressor variations hitherto studied, except perhaps in the case of circumcision (Lecture I). Further evidence in the confirmation of this tracing is to be found in a chart representing enucleation of the prostate in an old man of 72 with markedly thickened arteries, under chloroform anaesthesia. The enormous pressor rise of pressure is well marked; and the fall in the interval between manipulations is also to be observed. Attention should further be called to the fact that this pressor effect continues some time after the cessation of the stimulus; for the blood pressure had not sunk to the original level three minutes afterwards. In Lecture I cystectomy in the dog was demonstrated in an original tracing. Consecutive blood pressure readings of this tracing, plotted out in the same manner as in the case of the human tracings we have studied, reproduce these features in a striking manner. At the same time the greater susceptibility of the lower animals to the depressor effects of chloroform makes the interval fall of blood pressure more abrupt than in the human subject (Chart 50).

To sum up: The prepuce, urethra, and the bladder all show marked pressor types of curves, and have, therefore,

a low shock value. The susceptibility of the bladder and urethra to infection is well recognized; and in these structures, therefore, and in the viscera of the abdomen proper (where the reverse condition obtains) the law of

Chart 50.—Partial cystectomy in the dog. CHCl₃.

inverse immunity is again supported. The enormous rise of pressure in prostatic operations may cause sudden death in two ways: (1) Failure of a hypertrophied heart which cannot resist such a sudden enormous increase of pressure; (2) possibly, in some cases, cerebral haemorrhage. The indications for nerve blocking in these operations will be referred to shortly.

The Kidney.

Interrupted readings in an operation for the removal of vesical calculus in a small boy show the following features: Traction on the renal pedicle is accompanied by a marked rise in blood pressure, and this rise gives way to a fall to about the same level. (Chloroform anaesthesia was employed.) There is hardly any increase in the pulse-rate commensurate with this rise of blood pressure. Further, release of traction is not accompanied by a fall, but a marked rise in the pulse-rate. The explanation is as follows: This rise in blood pressure (confirmed by animal experiment) during traction on the renal pedicle is due to a direct stimulus to the solar plexus—that is, direct splanchnic stimulus and vaso-constriction, such as has been observed in previous tracings in the abdomen (see Trace 16a, hh'). The failure of the pulse to rise proportionately to the blood-pressure supports this view, as also does the sudden drop to the original level on the release of traction, with, at the same time, acceleration of the pulse-rate. Clearly, then, pressor manifestations in the case of the kidney are not evident, and we conclude that this organ has a high shock value, while, in accordance with the law of inverse immunity, and in view of the importance of this organ with regard to the functions of excretion, the high immunity of the kidney to bacterial infection cannot be denied. In the case of the kidney, blood-pressure readings are again of no value whatever in the estimation of shock from interference with this organ.

Finally, traction on one kidney, by the direct stimulus to the solar plexus, induces vaso-constriction in the opposite kidney. If this be continued long enough vaso-constrictors are fatigued, and a reversal is established—that is, vaso-dilatation. We believe this alternation of vaso-constriction and vaso-dilatation in operations on the kidney (when both organs are diseased), with consequent physiological disturbances, is of greater importance than the anaesthetic in the production of post-operative urinary suppression.

THE NATURE AND SITUATION OF AFFERENT FATIGUE IN SHOCK.

Experimental.

There is abundant evidence to show that in ordinary clinical shock there is no fatigue of the efferent vasomotor fibres. Splanchnic shock can be induced experimentally, but probably does not occur in surgical practice. The

nature of fatigue of the vessels in splanchnic shock seems to be partly associated with exhaustion of the suprarenals (Elliott). Priestley found absence of chromaffin in 84 per cent. of cases of low blood pressure, especially in advanced asæmia; but this fact has probably a bearing only on the tone of the vessels (collapse), and not to their reaction to nerve stimulus (shock). In systemic shock, however, depending as it does on afferent stimulus and efferent response, we have already produced evidence that the centres and the blood vessels are both active. Thus, in Lecture I the depressor stage of shock was shown to be due to active depression. Further, the injection of strychnine in this case converted depressor impulses again into pressor (Chart 28). This chart afforded clinical evidence that the centres are not exhausted in shock and that strychnine did not contribute towards such a conclusion. Again, if strychnine acts on the synapses, as has been stated by Cushny and others, we should infer that in this case the pressor synapses were fatigued, and that this fatigue established the domination of the depressor impulses, whose synapses were more or less intact. Strychnine therefore restored pressor impulses by reopening paths of conduction across these synapses. Now, electrical stimulation of the sciatic nerve in a fresh dog (under chloroform anaesthesia), and the steps of the operation to expose this nerve show a rise of blood pressure proportionate to the degree of stimulus. In an animal shocked from operation on the trunk and lower limbs vasomotor reaction becomes less and less, and finally disappears in proportion to the degree of shock; so that finally electrical stimulation of the cut central end of a

Trace 34.—Dog pithed in last stages of shock.

dog's sciatic induces no variations in blood pressure at all. Further, stimulation of any part of such a nerve at its origin (that is, a mixed spinal nerve within the dura) produces no effect on the blood pressure, even though a convulsion is excited. The block to centripetal impulses probably occurs, therefore, in the central nervous system and not in the peripheral nerves. Now, in a dog shocked to a degree such as we have described, if the spinal cord be divided in the lower dorsal or upper lumbar region, electrical stimulation of its central end produces hardly any effect on the blood pressure. This seems to show that, even if the block occurs in the segments corresponding to the stimulated area, this is not the main situation where afferent impulses are prevented from reaching the vasomotor centre. At the same time electrical stimulation of any of the component parts of the brachial plexus in such an animal induces a slight rise in the blood pressure. While, in profound splanchnic shock, electrical stimulation of a mixed segmental nerve within the dura induces a definite rise of blood pressure, as also does mechanical stimulation of a nerve of the brachial plexus, the variation being more marked in the latter. A study of such tracings as these seems to show that fatigue is most complete in those afferent paths supplying the area of operation; but that these impulses overflow their own synapses and fatigue, to a less extent, afferent paths from other regions. These facts again negative the view that the centres themselves are fatigued or paralysed. If, in an animal, shocked to such a degree that stimulation of the central end of the cut spinal cord does not induce any rise in blood pressure, electrical stimulus is applied to the floor of the fourth ventricle, a marked alteration in the blood pressure results, that is, the blood pressure is raised and the pulse is

markedly quickened. Finally, if at the moment of death from shock of this degree, a stout needle is driven into the centre of the floor of the fourth ventricle, a still more marked rise in blood pressure, with acceleration of the pulse, is observable. (Trace 34.) Experimentally, then, it seems to us that failure of the centres does not take place until the moment of death, a fact which is further supported by the marked Traube-Hering curves observable in profound shock in many of our tracings.

We conclude that the failure of transmission of afferent impulses occurs at the afferent synapses.

The nature of the synaptic block is a matter for speculation; but, bearing in mind the probable nature of fatigue of the end-plates in voluntary muscle by sarcolactic acid, we thought it possible that hyperexcitation of the afferent nerves in shock might be responsible for the formation of a waste product inhibiting the normal conduction through the synapse. Professors Mott and Halliburton have described the formation of choline in nerve degeneration. We performed a few experiments with a view to ascertaining whether some analogous waste product may not be responsible for these phenomena. These investigations were very limited, owing to our insufficient knowledge of physiological chemistry. A tracing from a fresh dog into whom choline was injected showed the typical fall in blood pressure. Professors Mott and Halliburton described this action of the drug as being directly on the vessel wall. But if a small incision in the abdominal wall of a fresh dog is made, and choline injected into a vein, mechanical stimulation of the semilunar ganglion, in the middle of the resulting fall of blood pressure, definitely breaks this fall, and produces a spike in the normally even curve. Immediate stimulation of the central end of the cut sciatic nerve, after this phenomenon, fails to evoke any alteration in the blood pressure, though, immediately previous to this, a marked rise of blood pressure with electrical stimulation was obtained. Finally, it is known that if choline be repeatedly injected into an animal, this characteristic fall of blood pressure is absent, and a small rise is observed in its stead. If, after this phenomenon, amputation of the leg at the hip-joint be performed, characteristic blood-pressure variations are entirely absent (a condition resembling extreme shock). On these incomplete experiments we suggest that choline acts on the afferent vasomotor synapses (as probably all toxins do), blocking the transmission of afferent impulses through them—a view which would support the immunity so quickly established to this drug, with repeated injections at short intervals.

Dr. Rosenheim, of King's College, has kindly examined some clinical specimens for us. In a case of post-operative shock he could find no choline in the cerebro-spinal fluid. In the spinal cord and medulla from two infants who died from shock twelve and twenty-four hours respectively after their injuries, no choline could be found; while a similar negative result obtained in the case of a child who died from whooping-cough. Clearly such a waste product as we suggest is not identical with choline, though it may possibly be a toxic substance which acts in a similar manner on the synapses.

HISTOLOGICAL.

Histological investigations (prompted by the results obtained by Crile and Dolley) have been carried out by us to determine whether there is any recognizable change in the central nervous system in shock, and, in particular, in that part of the central nervous system in which the vital centres are situated—that is, in the medulla, between the level of the striae acousticae and the tip of the calamus scriptorius. We have examined serial sections of various segments of the spinal cord, medulla, cerebellum, and cerebral cortex of dogs which have been shocked to death. Our observations are based on the careful examination of many thousands of cells. The brains and cords were in each instance removed directly after the death of the animal, and preserved in 10 per cent. formalin, hardened in graded alcohols, passed through chloroform, and embedded in paraffin. Sections 6 μ in thickness were cut and stained by a modified Nissl's method, $\frac{1}{4}$ per cent. aqueous thionin blue being used. Some sections were counter-stained by eosin, others by erythrosin, others not at all. Differentiation was carried out by means of aniline alcohol. The sections were cleaned in bergamot and

benzole, and mounted in acid-free xylol balsam, benzole balsam, or benzole dammar. In comparison with these specimens we were fortunate to obtain a human brain a few hours after death. The case was that of a healthy fireman, who was run over by a motor engine, and died from shock and hemorrhage. The injuries were very extensive, but the skull was not fractured. The examination was performed by one of us about fourteen hours after the time of the accident. The brain was preserved in the way described; and we examined serial sections from just below the tip of the calamus scriptorius to the level of the eighth nerve. In view of the difficulty of definite results being obtained from such methods of examination, we shall content ourselves by stating generally what, in our opinion, is the interpretation of these changes; reserving to ourselves the right to modify our opinions later if necessary. Too much must not be expected from cytological changes thus demonstrated; for Nissl himself states that we cannot be too sceptical in correlating cortical changes with mental diseases.

In the spinal cords of dogs shocked to death we could find no cytological changes whatever. We lay stress on the fact that these results refer also to segments of the cord which were responsible for the innervation of the operation area. The cerebral cortex showed very little change, and Purkinje cells of the cerebellum showed no striking alterations. Most of our attention was directed to the medulla, which was studied in serial sections. If the vasomotor centre is fatigued in shock, we should expect to find some existence of that centre in the deviation from the normal Nissl pattern of certain groups of cells, even though this centre has, in all probability, never been localized histologically. No such evidence has been found; a few scattered cells, in which chromatolysis was seen, on each side of the median raphe, might possibly represent it. This does not imply that a definite group of cells, forming a centre, does not exist (although this is probable); but rather that in shock there is no fatigue of such a centre to be detected histologically.

We have examined carefully the motor nuclei of the bulb, and found them normal. Only very scattered evidence of mild chromatolysis was detected. In a few cells there was marked chromatolysis of a degree described by Professor Mott as occurring in the cat when all four cerebral arteries have been tied. We lay stress on the fact that these motor nuclei are in a condition to respond and respond well, if they can be activated. Therefore, fatigue of the motor cells does not occur in shock. (In the human medulla the same conclusions are arrived at.)

In the sensory cells of the gracile and cuneate nuclei and in the olivary nucleus (much less so in the sensory nucleus of the vagus) there are more definite changes. Many cells show chromatolysis, but rarely in extreme degree. It seems that the scattered cells near the raphe, together with the sensory cells, are less resistant than the motor cells; and these features are more pronounced in the human medulla we examined.

We conclude that the motor nuclei are more resistant than the sensory nuclei under adverse circumstances.

These observations support our view, arrived at clinically and experimentally, that such cell changes as are noticeable are largely the secondary outcome of the altered vascular conditions existing in shock (for in the human medulla haemorrhage played an important part), and not due to direct fatigue, or indeed fatigue at all, by afferent impulses—that is, they are due to starvation.

Clearly the motor cells are in a position to respond to stimulation, but the activating influence fails. This influence may fail from (1) defective transmission of the synapse round the motor cells. (2) The intermediate sensory neuron system being unable to pass along stimuli received, owing to failure of the cell. (3) A block at the cell of this system (a block round the synapses of the efferent fibres from the cerebral cortex, or functional failure of the pyramidal cortical cell, does not play any direct part in the mechanism of shock; these are only secondarily involved). The second possibility—namely, failure of the cell of the sensory intermediate neurone—has little support, for we lay great stress on the experiments of Professor Mott, Sir Victor Horsley, Ehrlich, and Professor Leonard Hill, as showing that cerebral activity may be maintained in chromatolysis from practically complete anaemia, with

a strong stimulus. A similar activity in the cells we are discussing is to be inferred; and the chromatolysis being most marked here, combined with the remarkable absence of cell changes in the rest of the medulla (particularly the motor nuclei), leads us to conclude that the block occurs at the afferent synapse round the cell of the intermediate sensory neuron system. Therefore, on experimental, clinical, histological, and possibly chemical grounds, we suggest that the failure occurs at these synapses, which are to be regarded as a protection to the medullary centres by their failure.

CONCLUSIONS.

Shock cannot accurately be defined, for the term does not express a state or condition of an animal, but rather a mechanism which establishes, first, a defence of the organism against the harmful effects of injury, and, next, the protection of the vital centres from a degree of exhaustion from which they could not recover. The primary conception of shock should be:

Shock is the reaction of the central nervous system to exaggerated or abnormal afferent impulses.

In other words, a study of shock means the study of pathological vasomotor reflexes regarded as a symptom of the activity of the central nervous system. In their order of importance the centres most concerned are (1) the vasomotor and cardiac centres, (2) the respiratory centre, (3) the centres of consciousness and special senses. Their susceptibility to trauma of any sort is in the inverse order. Clearly shock commences with the first abnormal or exaggerated afferent impulse, and continues until death. Clinically, therefore, surgical shock commences with the skin incision, and it is continued long after the operation, until the abnormal or exaggerated afferent impulses cease and physiological conditions are once again restored. It is necessary to realize that *collapse* is associated with a low blood pressure and its sequelae; but that it is not peculiar to shock, though it is a sequel of the second stage; and it may be induced by many other agencies—for example, haemorrhage, toxæmia, prolonged loss of heat, etc. Collapse is only an end result of, and not identical with, the reaction to abnormal or exaggerated afferent impulses constituting shock.

Finally, we have shown that a low blood pressure is not an essential accompaniment of the clinical picture of shock; for in Lecture I we showed that the classical symptoms of shock occurred when the blood pressure was at the highest recorded point. It is of interest to note the fact recorded by Takaki that in sudden perforations during enteric fever the pulse tension is high, for this phenomenon is accompanied by the classical features of shock. These classical features may be marked in the pressor stage of shock as well as in the depressor, though in the former the high pulse tension and the widely-dilated pupils contrast with the low pulse tension and the intermediate pupils in the stage of collapse.

Mental Shock.

The activity of the higher centres raises the blood pressure proportionately to its intensity, while the individual variations are proportionately increased. In pain the mean blood pressure is not markedly raised but the individual variations are well marked. One cannot help being struck by the approach from pressor to depressor type of chart with the increased violence of afferent impulses from the outside world to the centres of consciousness, as evidenced by their efferent impulses to the vasomotor centre. Fatigue of the pressor paths from the centres of consciousness to the vasomotor centre, establishes the domination of the depressor impulses. And this varies with (1) the duration of the stimulus; this is familiar to all in the effects of prolonged mental strain; and (2) intensity of the stimulus. Sudden, very violent stimuli may fatigue the pressor and initiate immediately the domination of depressor impulses—that is, the extent of the stimulus is carried to a more marked degree than in the charts we have studied. Such a sudden stimulus may cause sudden active depression, leading to collapse and even to death; and this is the mechanism of mental shock (also of shock from blows on the head, etc.). This collapse may be maintained, or rather its persistence may be favoured, by the exhaustion of the suprarenals, which Cannon has demonstrated to take place in the cat, when terrified.

So-called "traumatic shock" is an exaggerated type of mental shock. It is initiated by a slight trauma, which might have been, and by mental anticipation was pictured as, serious and painful. The persistence of the mental picture of the trauma, coupled with the possible suprarenal exhaustion, may initiate a progressive active depressor stage of shock, leading to death in the same way as in delayed surgical shock.

The mechanism of mental shock is identical with that of surgical shock, the stimulus being "manufactured" in size and nature by the hyperactivity of the centres of consciousness.

Toxins.

Toxins (amongst these we class general anaesthetics), by their effect on the synapses, render pressor fatigue more likely; and this is notoriously the case in the toxæmia of hyperthyroidism and exophthalmic goitre, lymphatism, etc. In exophthalmic goitre the prolonged toxæmia, and constantly magnified small stimuli, tend to place the sufferer, figuratively speaking, on the precipice of pressor fatigue. The additional toxæmia of chloroform, the stimulus of the operation, and the pre-existing mental excitement, all tend to initiate pressor fatigue and active depressor impulses. We believe, with Crile, that sudden deaths under operation in these cases are really manifestations of extreme mental shock.

Pain.

The depressor type of chart obtained in pain renders the control of this, before and after operation, of paramount importance, since it appears to be an active agent in initiating active depressor impulses.

(Inhalation anaesthesia, lumbar puncture and spinal anaesthesia, abnormal reflexes in surgical operations, relations of vasomotor changes to the onset of "surgical shock," shock-values and shock-index, and shock-values as applied to the central nervous system, etc., have all been sufficiently considered in Lecture I, and need not detain us further in the present lecture.)

The Abdomen and Genito-Urinary System.

We would emphasize again the importance of the fact that elimination of centripetal impulses by transection or excision of the cord showed that a very definite improvement in the blood pressure, action of the heart, and general condition of the animal, resulted from intestinal manipulations. This is due to direct splanchnic stimulation unhampered by central depressor stimuli. This beneficial effect of blocking afferent impulses, combined with the local splanchnic stimulus of the operation, on the condition of the patient is similarly observed in spinal anaesthesia, and forms one of the strongest recommendations for its employment in suitable cases. It is within the experience of most surgeons that severe intra-abdominal manipulations are "deceptively" well borne by the patient during their continuance, and that such patients often rapidly get worse during the suture of the abdominal wall. This feature is due to the relaxation of the visceral vessels when the local stimulus is stopped. Consequently a sense of false security is likely to be rapidly disillusioned when the splanchnic stimulus ceases. Clinically, however, the raised blood pressure, combined with the slow pulse, gives an indication that the phenomenon is of peripheral origin. The exhaustion of the suprarenals is probably an accessory factor in maintaining the progressive collapse from the depressor stage of shock.

TREATMENT.

The treatment of shock naturally falls into two groups—*preventive* and *curative*. Only the briefest indication of the principles is possible.

PREVENTIVE.

This consists in the maintenance of pressor impulses and the avoidance of the domination of depressor ones. Factors directly concerned are:

- A. Nature and degree of violence.
- B. Duration of the stimulus.
- C. Area involved—that is, number of fibres stimulated.
- D. Shock value of the tissues.

Taken together, these (A, B, C, D) constitute the shock value of the operation—that is to say, the onset of shock is influenced by technique and the nature of the opera-

tion. With regard to technique, it only requires to be said that the surgeon's aim is maximum speed combined with, but never at the expense of, gentleness of manipulation and perfect haemostasis. One point in particular which we would refer to is the question of traction on the intestinal mesentery. This is to be avoided whenever possible, but, if necessary, traction should always be exerted upwards—that is, towards the semilunar ganglia (see Diagram 2).

E. Shock value of the individual—that is, resistance of the central nervous system to pressor fatigue. The following factors require consideration:

1. *Age*.—Perhaps the most important consideration—resistance to pressor fatigue—is directly proportionate to the age. Thus, an operation which involves equal stimulus, and is performed under identical circumstances, may induce the depressor stage of shock in a child; whereas, in an adult, the same operation, lasting an equal time, would only evoke the normal pressor response.

2. *Physique*.—This probably depends on the presence or absence of circulatory toxins; we do not refer to muscular development.

3. *Toxæmias*.—These form the most important consideration after that of age:

(a) Autointoxications.

We do not use this term in the general clinical sense, but reserve it for true autointoxications arising from the absorption of excessive or perverted secretions of the ductless glands. Paramount in surgery are hyperthyroidism and exophthalmic goitre, and lymphatism.

Hyperthyroidism.—(1) The toxin, perpetually acting on the pressor synapses, places the patient on the "precipice of pressor fatigue." This is enhanced by depressor stimuli which may be initiated from the outset of operations. (2) The afferent impulses of the operation accentuate this tendency. (3) The manipulations of the thyroid add a powerful dose of toxin to a susceptible subject. (4) The constant small, but magnified, mental stimuli in such patients gradually induce a condition of pressor fatigue, accentuated by previous anxiety and the sudden emotion preceding the operation. (5) General anaesthesia, particularly chloroform, assists in accelerating pressor fatigue. (6) The heart muscle is degenerated, and the rhythm is accelerated by the excessive sympathetic stimuli excited by the least afferent impulse—there is, therefore, liability also to heart failure. On these grounds the following routine is suggested: Absolute rest in bed for at least a fortnight before operation (Kocher); during this period every afferent stimulus to the higher centres is to be eliminated as far as possible. The toxin is to be eliminated by copious administrations of fluids before and after operation. Carbohydrates, diaphoretics, diuretics, and laxatives are to be given appropriately.

The emotion of the operation is to be mitigated by Crile's method of "stealing the thyroid." Morphine is to be given without stint; atropin is to be avoided until after anaesthesia is induced, owing to its stimulation of the higher centres.

During the operation afferent impulses are to be blocked by infiltration anaesthesia, which is to be combined with general anaesthesia; of these ether, to unconsciousness only, is preferable; chloroform is strongly contraindicated. The manipulations of the thyroid are to be reduced to a minimum, and removal of the excess of toxin by temporary drainage is advocated. The condition of the heart is to be treated during the preliminary rest.

Lymphatism.—A totally opposite picture is presented in this condition. (The enlargement of the thymus in both conditions forms the only point in common in these two diseases.) We have shown that in animals and man operations in regions where the vagus stimulus predominates are accompanied by marked cardiac and respiratory inhibition. The fullest doses of atropin, almost to poisoning, at least half an hour beforehand (and continued afterwards) are strongly to be recommended. Direct cardiac inhibition is particularly well seen in operations on the fauces and pharynx, etc.—for example, operations for tonsils and adenoids, retropharyngeal abscess, etc. (We believe the credit for advocating atropine in such cases is due to Scharlieb.)

(b) Infective.

Acute.—Toxæmias of bacterial origin can of themselves initiate pressor paralysis. The rapidity varies with the

virulence and with the age, younger patients being the most susceptible. Operations on the infected area are to be considered in the following light:

The danger of *pressor fatigue* is influenced by the following: (1) The factors already referred to—A, B, C, D, comprising the shock value of the operation. (2) The existing toxæmia is inducing a synaptic block. (3) The steps of the operation accelerate pressor fatigue; and are aided by the after stimulus, pain, etc. (4) Fresh raw surface, for the absorption of additional toxin, is exposed to infection by operation. (5) General anaesthesia adds another toxin to the already poisoned synapses. (The same considerations apply to chronic infections in a lesser degree.) These observations apply, further, to acute and chronic infections where toxæmia is of independent origin from the lesion calling for interference—for example, pneumonia with strangulated hernia.

(c) General Anaesthesia.

General anaesthesia has been shown to precipitate pressor fatigue, chloroform more than ether. We present Chart 52 showing the depressor stage of shock initiated in

Chart 52.—Depressor stage of shock, initiated by excessive CHCl₃ narcosis. Pressor stage restored by strychnine.

a simple operation from an overdose of chloroform in a child. The conversion of the depressor stimulus into the original pressor stimulation by strychnine is also well shown in this chart.

4. *Haemorrhage, loss of heat, and mental shock* all precipitate pressor fatigue.

The control of these factors which have been shown to favour the onset of the depressor stage of shock is to be considered in the following way:

1. Indications for Nerve Blocking.

Local, and intravenous local, anaesthetics are of limited application; with the exception of operations in more or less superficial regions they do not institute a complete nerve block (direct cocainization of large nerve-trunks is included).

Spinal anaesthesia is the most efficient method of inducing a complete afferent block over a large area; and experience has proved that it can be safely, scientifically, and accurately administered to those cases where its employment is called for. The principles on which indications for its employment are based have been referred to. Two broad groups of cases, therefore, will be briefly considered (it being granted that age always predominates as an indication—that is, the younger the child the more urgent is the need for the protection of the synapses by spinal anaesthesia likely to be).

1. *The Shock Value.*—Consideration of the shock value, arising from factors A, B, C, D, in conjunction with the age, form the principles on which a surgeon will act according to his experience.

2. *Toxaemia.*—When toxæmia is paramount, the shock value of the operation, together with the age, will decide the advisability for nerve blocking. In our limited time

detail is impossible, and the line of reasoning to be recommended is best illustrated by two examples:

A child, aged 5 years, suffering from acute osteomyelitis of the femur; toxæmia profound. The dangers are: The existing toxæmia is paralysing the pressor synapses; the age of the child is young; the shock value of the femur is great; the shock value of the operation is very high; general anaesthesia adds another toxin; the opening up of fresh tissues increases the surface for the absorption of the toxin, accordingly toxæmia is increased afterwards. If the pressor synapses are fatigued by the operation (a likely contingency) the increased toxæmia and pain afterwards will induce progressive depression leading to death perhaps a day or two later. A block to afferent impulses by spinal anaesthesia is indicated in such a case.

In a child aged 5 years, suffering from gangrenous appendicitis, with diffuse, spreading peritonitis, toxæmia was profound. Here similar conditions obtain; but another important factor is also to be considered. In order to minimize the violence of manipulations ample exposure is advisable; this, however, would expose the synapses to further poisoning, due to increased septic absorption from the additional raw surface created. On the other hand, if, for this reason, a small exposure is made, the synapses are exposed to increased afferent impulses (from more prolonged and violent manipulation, wide retraction, etc.). In these cases, therefore, the surgeon is situated between the Scylla of pressor fatigue and the Charybdis of increased toxic absorption, both equally dangerous in a child. If spinal anaesthesia be employed, the complete block to afferent impulses permits almost any degree of retraction and manipulation through a quite small exposure, without accelerating pressor fatigue (indeed, we have shown that the patient's condition improves during these manipulations); the duration of the stimulus is lessened, and the increased "after-toxaemia" is minimized. The synapses are then in a better position to withstand such increased subsequent toxæmia as may exist. Further protection of the nervous system is affected by copious rectal and subcutaneous saline infusions, to which the addition of dextrose is recommended. Morphine in full doses is also advisable, in order to minimize all afferent impulses, particularly those of pain. The bowel is to be kept absolutely at rest by complete starvation, in order to diffuse as little toxin as possible (that is, "an inflamed part should be rested"). It is the recognition of these principles which at the present time enables us to say with confidence that the prognosis of acute appendicitis in small children is excellent in all but moribund cases (probably well over 90 per cent. of recoveries may be expected).

Finally, nerve blocking, by spinal anaesthesia, is indicated where there is danger of the failure of a hypertrophied heart against a marked increase of an already high blood pressure—for example, in operations for prostatectomy, where this danger exists.

2. The Control of Mental Shock.

In susceptible persons the following suggestions are offered: (a) Rest in bed previous to operation, as has been advised; (b) the patient should be educated and familiarized with the idea of anaesthesia and operation (see exophthalmic goitre); (c) the administration of morphine and scopolamine before operation; (d) the control of the mental factor during the induction of anaesthesia; (e) continuation of morphine after operation to control impulses to the higher centres, etc.

3. The Control of Collapse.

Collapse due to Haemorrhage.—The first indication is to make good the loss of fluid by mouth, by rectal infusion, by subcutaneous infusion, and by intravenous infusion. Details need not concern us at the present time.

Drugs.

Strychnine.—Strychnine acts by virtue of two properties—(1) by establishing the conduction of impulses across synapses which are blocked; (2) by direct stimulation of the heart. Strychnine is not indicated in collapse from haemorrhage, except, perhaps, as a cardiac stimulant in moments of emergency.

Suprarenin is valuable both as a vaso-constrictor and also because it stimulates the suprarenal glands to activity

(Elliott); and there is probably no exhaustion of these glands in haemorrhage.

Atropin is valuable in checking the loss of fluid and heat by sweating; while brandy and ether are excellent cardiac stimulants, provided the haemorrhage has been stopped and suprarenin has not been given.

Collapse due to Toxaemias.—The elimination of these is strongly indicated. We need not discuss the methods beyond saying that the copious administration of fluids is essential, and that *suprarenin* is contraindicated, since vaso-dilatation is to be desired in order to promote excretion.

Drugs.

Strychnine is exceedingly valuable during and after operations on toxic subjects. For it possesses a power of restoring the paths of conduction across the pressor synapses in such patients. This is well illustrated in Chart 52.

CURATIVE.

With the limited time at our disposal we propose to consider very briefly some sudden deaths under operation. These are to be strictly differentiated from respiratory failure due to the anaesthetic, where the prompt performance of artificial respiration and the administration of cardiac stimulants are practically always successful. We refer in particular to those cases where anaesthesia is not primarily responsible for this contingency, and will confine our remarks to such illustrative instances as exophthalmic goitre, status lymphaticus, prostatectomy, and respiratory failure in increased subtentorial pressure.

Exophthalmic Goitre.—We believe that sudden death in these cases is due to rapid pressor fatigue, followed by an equally powerful depressor stimulus, and consequent inhibition of the heart. The indications are cessation of the stimulus and injection of strychnine to restore the pressor paths and stimulate the heart; after the restoration of the pressor paths by strychnine, stimulation of the skin by cloths wrung out in hot water, etc.; the immediate adoption of artificial respiration combined with indirect cardiac massage is advised, etc. We would emphasize the importance of not applying any stimulus until after the administration of strychnine. These recommendations are based on theoretical deductions only.

Status Lymphaticus.—The characteristic clinical features of sudden death in these cases are sudden pallor, wide dilatation of the pupils, and cessation of respiration in inspiration. In unfavourable cases the pupils assume an intermediate size and remain thus fixed. These phenomena are due in the first instance to a powerful sympathetic stimulus, dominated subsequently by the direct vagus inhibition of the heart. The indications are—cessation of the stimulus; artificial respiration combined with indirect cardiac massage at once*; strychnine, to stimulate the heart and reopen the pressor synapses; atropin, to inhibit the vagus and stimulate the higher centres; powerful peripheral stimuli—for example, hot cloths, dilatation of the sphincter ani—after the injection of strychnine. We have had many opportunities to put these principles into practice, and have never had any failures with this routine.

Enucleation of the Prostate.—Sudden deaths in these cases have been already referred to. The improbability of recovery accentuates the importance of prevention by nerve blocking.

In *respiratory failure*, from increased subtentorial pressure, we should not hesitate (on the strength of the views expressed in Lecture I) to practise lumbar puncture combined with venesection as a temporary measure until operation could be performed.

The treatment of *progressive collapse from the depressor stage of shock* is twofold: (1) To treat the cause. Here the administration of strychnine during and after operation is of paramount importance. We are quite unable to agree with those who condemn this drug; such a condemnation we feel to be based on an inaccurate recognition of the pathology of shock. Morphine after operation is indicated for reasons we have given. (2) Further treatment is directed towards the alleviation of symptoms due to collapse and its sequelae, and does not come into the scope of the present lecture. We may state, however, that adrenalin is contraindicated in our opinion. For Elliott

* The value of cardiac massage is demonstrated by tracings in animals.

has shown injections of this drug to excite secretion from the adrenals; and these are probably already exhausted in shock. Thus their recovery by rest, rather than their further activity, is to be desired.

In conclusion, Mr. President, Fellows, and Members of the College, we both thank you heartily for your kind attention, and ask your indulgence for what may appear a summary dismissal of some of the important points in this study. But time has been our enemy. However, we hope to have convinced you that surgeons should cease to rely on a high blood pressure as an indication of the absence of shock, or on a low one as necessarily symptomatic of its presence. And if you hold that we have in any degree contributed towards the elucidation of the mechanism of shock, or that we have adduced strong evidence in support of the plea for the adoption of nerve blocking as a means of controlling shock (particularly in toxæmic children), we shall be amply repaid.

Finally, should this research incur the accusation of a strong advocacy for the use of spinal anaesthesia in suitable cases, this accusation we will gladly accept.

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

DEATH AFTER SALVARSAN.

AN athletic man, aged 23, developed a non-ulcerating chancre for which he entered a nursing home, where two intravenous injections of 0.6 salvarsan were given with an interval of a week. There was no pyrexia after the first injection nor any ill effect. After the second, within forty-eight hours there were pyrexia and aching of legs. He returned home. At 7.30 p.m., when in bed, the temperature was 101.6° F. in the axilla; there was no perspiration. The next morning he was seen walking in his room in a dazed state, muttering gibberish, and dribbling from the mouth. An hour later he could talk distinctly though with difficulty, yet was unable to account for his earlier state. The temperature was 97.6° F. There was a punctate red rash on the chest and abdomen and he was sweating. At 6 p.m. he was still sweating; the temperature was 98° F.; he was seen in an epileptiform fit. The following morning there was another fit, and more followed; he was unable to swallow; respiration was rapid; fit followed fit till only the eyes and depressors of the jaw retained power to contract, and he died from respiratory failure. The breath became so faecal, though there had been no constipation, that it was thought an involuntary action of the bowels had occurred. There was no involuntary action. Urine was retained, about 10 oz. of urine being withdrawn by catheter. There was profuse secretion of mucus in the upper airway. He was unconscious for twenty hours.

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ETHYL CHLORIDE SPRAYING IN VENEREAL SORES.

THE following experiences of the curative action of spraying with ethyl chloride on venereal sores may prove of interest. It is more especially valuable in the services, as it accelerates considerably healing of the chancre, thus limiting bubo formation. The time spent in hospital is thus reduced by about 50 per cent. in average cases.

The method employed is as follows: The ulcer is well dried and cleansed with dry cotton-wool and well frozen three times in succession with the ethyl chloride spray, until the surface of the ulcer is quite white, like hoar frost. It is more effectual to warm the ethyl chloride glass tube in a tumbler of warm water before applying it, as a better spray results. Dry iodoform and dry gauze are then applied. This is repeated daily until cure results.

This method is superior to the local use of pure carbolic acid. I have not found it valuable in commencing phagedaena in a chancre, but when active phagedaena has been prevented by pure nitric acid, free exposure and baths, it will then accelerate healing. It is not painful, and requires little experience.

It is superior in my experience to x-rays, which I have utilized and have published the results in the BRITISH