The Arris and Gale Lectures

ON

THE MECHANISM AND TREATMENT OF SHOCK.

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LECTURE I.—PART II.

SHOCK VALUES AND SHOCK INDEX.

CLINICAL experience has taught that the shock value attaching to the operation for hernia is infinitely less than that arising from surgical interference with bone. A comparison of the charts taken from the former (Charts 21 to 24) and the latter (Charts 27 and 28) shows quite a different type of curve. In the former a step-ladder rise was remarked; but in the bony operations the mean blood pressure was not so markedly raised, but was characterized by enormous rises followed by equally marked falls. Clearly the actual height to which the pressure rises is not the main factor in controlling the onset of pressor fatigue; although it is, to a certain extent, an indication of the violence of the stimulus, which is one of the factors influencing pressor fatigue. The other main factors influencing the onset of pressor fatigue are: The nature of the stimulus, and the area involved. Clinically these facts are generally accepted. Experimentally—if Chart 27, which necessitated free exposure and prolonged manipulation, be compared with a chart representing a simple osteotomy with an osteotome and mallet, the difference is striking. Here the same region is attacked, but the difference in the type of curve obtained is attributable to the degree of stimulus to the area of operation, and to the duration. Thus, all our observations show that wide and forcible retraction of wounds, crushing, tearing, and traction, without exception induce a more marked rise in blood pressure than clean incision; for the former manipulations stimulate a larger number of nerves, and raise the pressure in proportion to the aggregate number of pressor fibres stimulated. These factors, relating to the exciting cause, have a prominent bearing on pressor fatigue. But, even when they are all more or less constant, experience teaches us that one of the most important factors in the production of shock is the region of the body which is attacked. Thus to each organ, structure, tissue, or operation may be assigned its own *shock value*. Now, in the first group of charts the stimulus of the operation induced a step-ladder rise, consequent on the summation of stimuli in a region where pressor fibres are markedly in excess of the depressor. If an equal stimulus be applied to a region where depressor fibres are in excess, the stimulus may be sufficient to induce a rise of blood pressure from the stimulation of such pressor fibres as are present; but this rise of pressure will clearly not be maintained in sufficient degree to cause the characteristic step-ladder rise. Accordingly, we should expect more evanescent rises in blood pressure, followed by rapid falls in the intervals between successive stimulus, in contrast to the gradual fall observed on a cessation of the stimulus in the former. Now, if we regard a surgical operation as a series of rapidly alternating periods of stimulation and quiescence, it is reasonable to expect that: (1) In operation in regions where depressor elements are in excess, charts of the blood pressure will show large rises and correspondingly marked falls—that is, spikes in the general

curve—corresponding to the steps of the operation; (2) in interference in regions where pressor elements are predominant, the after-stimuli between the steps of the operation will be sufficient to maintain the ascending or step-ladder character of the curve. The operations on bone which we have demonstrated are types of regions where depressor elements are in excess. Consequently, we may conclude that in any particular region the shock value varies directly with the relation of pressor to depressor fibres in the nerve supply. The physiological needs of different organs naturally require varying dispositions of pressor and depressor fibres in their nerve supply, to provide for local vaso-constriction or dilatation under different conditions. For example, stimulation of an afferent nerve induces a rise in the general arterial blood pressure, but a local vaso-dilatation in the organ supplied by that segment, Lowén's reflex.

Now, operations on the tibia induce a smaller degree of shock than similar operations on the femur. The respective vasomotor demands of these bones deserve a moment's consideration. The tibia is an exposed bone, and less protected from injury by direct violence than the femur; consequently we should expect Nature to provide a more adequate mechanism for the defence of the organism against the results of trauma, in the case of an exposed bone like the tibia, than in a bone like the femur, which is well protected by its ensheathing muscles. One of the greatest demands made by a damaged bone is the means of stopping haemorrhage, and this is effected by vaso-constriction. Accordingly it is to be expected that pressor influences would predominate in the case of the tibia on account of its exposure to injury. Again, the mechanism of the early defence of a structure against infection by micro-organisms consists in vaso-dilatation, in order that a greater supply of blood may be brought to the infected area. Such a mechanism involves the predominance of depressor, or active vaso-dilator, fibres in the nerve supply. Acute osteo-myelitis of the femur is of rarer occurrence than similar infections in the tibia. Hence it is reasonable to infer that the predominance of depressor influences renders the femur, which is anatomically protected against injury, less liable to acute infection by micro-organisms; while in the case of the tibia, the predominance of pressor, or vaso-constrictor, fibres (provided against its liability to direct trauma) renders this bone less immune against infection. These considerations are borne out by clinical experience, and support the contention that the shock value of a structure varies directly with the proportionate number of depressor fibres in its nerve supply, and that the higher the shock value the greater the degree of resistance to infective lesions. Accordingly we suggest that the following law, the law of inverse immunity, will be found to be supported (with reservations) by the whole of our research, and it may be stated as follows: That those parts which are physiologically the most active, contain the largest proportion of depressor fibres in their nerve supply, are most immune from bacterial infection, and show the highest shock value; such structures are deep seated, and anatomically protected against injury. The converse is equally true.

Minimal Shock Index.

With a given stimulus the greatest extent of the rise in blood pressure observed is some indication of the proportion of pressor fibres responding to that stimulus. The number of "depressor" spikes in the curve, each corresponding to a definite step in the operation, gives some indication of the amount of stimulation of those tissues where depressor fibres predominate. The total rise in blood pressure, therefore, gives some idea of the resistance of a structure to pressor fatigue, and the number of spikes observed gives some idea of the likelihood of depressor stimulation. Since the stimulus of an operation depends largely on the individuality of a surgeon, and is not a measurable quantity, this factor may be represented as x . Further, the duration of the stimulus increases the shock value of an operation. Since the smallest afferent stimulus in an operation is a step in the production of pressor fatigue, there must be at least *one* depressor spike in the curve, even if this only consists of the curve as a whole (that is, there is always some degree of shock in every operation). From these factors it is possible to work out roughly a shock index for any particular opera-

tion. With our methods of record, however, such an index can only express the least possible figure. Thus, x multiplied by number of depressor spikes, multiplied by the duration, go on the multiplying line, while the total rise in blood pressure multiplied by 100 go on the dividing line. Thus, the operation for radical cure of hernia gave an average index of $0.003 x$ in three cases; while operation for excision of the hip-joint gave an index $0.09 x$, or nearly thirty times as great as the preceding one. The relative shock values of different structures in the lower limb are well exemplified by an operation of amputation through the thigh under deep chloroform narcosis. Such an operation is illustrated by Chart 30, where the more important steps are noted. Thus the

Chart 30.—Amputation. Shock values of tissues.

attack on soft parts is seen to be marked by a pressor rise of a step-ladder character, up to the point *a*, *a* to *b*, during the manipulations of the great vessels, shows the most marked pressor rise; while the attack on bone is heralded by a typical depressor spike, *b* to *f*. The remainder of the operation shows clearly a gradual depression interrupted by pressor spikes; the whole curve, from *f* to *n*, showing a slight, though definite, active depression. It is clear that the shock value of bone exceeds that of all the other tissues involved, that of the great vessels showing the next value, then fascial and muscular tissues, while the lowest value falls to the skin proper. These conclusions are supported both by many of our observations on the human subject, all of which it is impossible to produce, and also by experiments on animals. If the *element of pain* is eliminated by general anaesthesia, surgical lesions of the skin proper have practically no shock value. When, however, the corium and subcutaneous tissues are invaded a considerable pressor effect is observed. The importance of this observation is very apparent, for it shows that shock resulting from extensive skin lesions is due almost entirely to afferent impulses to the centres of consciousness for pain. The shock index in Chart 30, worked out in the manner already suggested, gives a figure of 0.015 *x*. In Chart 27 a far more severe operation on approximately the same structures gives a shock index of 0.048 *x*—that is to say, this operation produced three times as great a degree of shock as amputation. Had this operation (Chart 27) extended over an equal time to the amputation (Chart 30), the index would have been 0.018 *x*. And since the osteotomy involved more violent manipulations, the increase of the figure, 0.018 over 0.015 (in the case of the amputation), may be apportioned to the increased stimulus. Consequently in these cases we see that the difference in the index for the same region of the body depends on two factors—(a) The violence of the stimulus, (b) the duration of the stimulus. This deduction is in complete harmony with clinical and experimental observations. Finally, excision of the hip-joint (Chart 28), if taken throughout the whole operation up to the depressor stage of shock, gives an index of 0.032 *x*. If, however, the observations include the depressor stage of shock, the index is 0.09 *x*. Making use of this formula, we have worked out the shock index of many operations of which we have tracings. And our results are in agreement with clinical

experience. We therefore bring this index forward, not as a practical suggestion, but as some confirmation of the deductions we have drawn with respect to pressor and depressor types of chart. Operations on the neck show the same variations as have been observed in other regions, with the exception that the stimulus to the vagus, on deep dissection, appears to induce some fall of blood pressure and slowing of the pulse.

REGIONAL SHOCK VALUES.

The Central Nervous System and its Envelopes.

If the results on the blood pressure of operative interference with the laminae of the vertical column be reviewed, it is evident that, whereas a very marked pressor effect is observed in all manipulations of the soft parts surrounding the bony column, the attack on the bony structures themselves is characterized by a marked change in the character of the tracing.

The type of curve indicates a preponderance of depressor over the pressor fibres in the nerve supply to the laminae. These structures have, therefore, a high shock value.

Moreover the vertebral column is extremely well protected against injury by soft structures, while the rarity of acute infection in this region is well known. Consideration of the previous arguments would lead us to expect a high proportion of depressor fibres in the nerve supply of bony structures, and this conclusion is supported experimentally. Once more, therefore, we find support to the law of inverse immunity.

Spinal Membranes.

The function of these structures is very largely supporting and protective; accordingly we find a well-marked tendency to suppuration and little power of localization when an infective agent is introduced to this neighbourhood.

The results of our observations in cases of lumbar puncture show that injury of the spinal membranes is accompanied by a well-marked pressor effect, for such a small stimulus as the prick of a needle or puncture with a sharp knife causes an appreciable rise in blood pressure both clinically and experimentally—a rise which subsides very gradually. Further, experiments show that rubbing with gauze, cutting, or flushing with hot saline, all induce a well marked rise in blood pressure of the type which is maintained some little time after the withdrawal of the stimulus. Thus, in the case of the spinal membranes, the shock value is a low one and the law of inverse immunity is again supported.

Spinal Cord.

Experimental injury, division, or other form of stimulus to the spinal cord itself always induces an immediate rise of blood pressure. This rise is independent of the muscular spasm that may be induced and is accompanied by a corresponding acceleration of the pulse-rate—that is, is central in origin. It lasts a very short time; in fact, a rise in blood pressure so produced is followed by as rapid a fall to a point usually below the original. Division of the cord is, of course, followed shortly afterwards by a fall of blood pressure, due to the consequent relaxation of the muscular coats of the vessels involved. This phenomenon we are not at present concerned with. In mechanical division of the spinal cord the domination of the pressor group very soon gives way to that of the depressor group of fibres, which are in great preponderance.

Some clinical evidence of this may be found in the prolonged collapse supervening on crushing injuries to the spinal cord.

(That surgical shock in operations on these regions is due entirely to afferent impulses is proved clinically by the complete absence of shock in Forster's operation when this is performed under spinal anaesthesia, followed, if necessary, by direct stavainization of the cord.)

While, therefore, the spinal cord shows a high shock value to surgical interference and injuries, it shows a high immunity to bacterial invasion ; for, whereas infection of the cord may not infrequently occur, sufficient resistance to withstand pus formation is a notable feature. Here again the analogy is complete.

The Head.

Incision and manipulation of the scalp and periosteum always give a step-ladder rise in blood pressure. It is not

necessary to reproduce charts of scalp operations, but tracings show that the pressor effect is particularly well marked.

A tracing was taken from an operation performed for the radical cure of tuberculous disease of the mastoid in a girl, aged 9 years 10 months.

The initial fall of blood pressure is well shown as consciousness is lost from the effects of the chloroform, the rise due to the excitement stage (followed by vomiting), and, finally, the rapid fall as the stage of surgical anaesthesia is reached.

Now the most noticeable feature of the whole chart is that the mean blood pressure hardly varies throughout. The respiratory trace indicates a very constant depth of the anaesthesia, for the respirations hardly vary at all. The stimuli of the operation maintain a slightly higher level of pressure than at the commencement. This raised level gradually falls as the successive steps of the operation become more gentle and rarer. The remainder of the chart shows a steady rise, with larger variations, as anaesthesia becomes lighter.

Incision of the integument and periosteum, reflection of the flap, and dragging forward the pinna by means of a plug through the meatus, are all accompanied by a perfectly typical step-ladder rise in blood pressure. It is significant that the attack on bone with the gouge is followed, unlike other cases of bony operations seen, by a rapid sinking of the pressure to about the original level. In fact, at no time does the application of the gouge or bone forceps to the mastoid cause any disturbance of the blood pressure. The respirations at this time were quite constant; and, accepting the blood-pressure variations as an indication of the extent of the shock induced by the operative stimuli, we suggest that the bony tissue of the mastoid is without appreciable shock value. Such a statement, however, in the case of an operation on the mastoid requires qualification. Thus, it is most significant that the application of the hammer is followed by a marked sudden rise of pressure; whereas, directly the hammering ceases, and the use of bone forceps is substituted, the blood pressure sinks again to practically the same level.

The rise can, we think, have only one interpretation: the same area is attacked by the forceps as by the hammer; yet with the forceps there is little or no variation in blood pressure; therefore the rise is due to the direct concussion to the centres produced by the hammering of the skull. These concussions act as a stimulus to the centres, and induce corresponding spikes in the curve. Further, there is seen in the respiratory tracing some slight increase in the rate and amplitude accompanying the application of the hammer; while the change from hammer to forceps is accompanied by a diminution in both rate and amplitude. These alterations, however, are not sufficiently marked to justify stress being laid on them.

The opinion has been expressed that chloroform attacked the synapses of the afferent fibres to the respiratory centre during full anaesthesia, but, except in lethal doses, hardly affected the vasomotor centre. This fact would account for the marked effect of the concussion stimulus to the vasomotor centre, and the slight accompanying effect on the respiratory centre.

To sum up, the mastoid itself has little or no shock value; but concussions, induced by the use of the hammer in operations on this region, have a deleterious effect on the central nervous system in proportion to their violence. The bearing of this inference on practical surgery is obvious.

The pressor effect of surgical interference with the scalp and calvarium is illustrated by a series of charts taken during head and brain operations, though the extent of the variations is subject to the condition for which operation was undertaken. Experimentally, the pressor effect of lesions of the scalp and calvarium is seen in a tracing taken from a dog in a condition of profound shock. An appreciable pressor effect is evident as the result of removal of the skull over the cerebellar region with bone forceps. For not only is there a definite rise in blood pressure, but also a synchronous acceleration of the rate, and diminution of the percussion stroke, of the pulse. It is, of course, difficult, in the human subject, to obtain

consecutive evidence as to the pressor character of the tracings in injuries of, and surgical interference with, the calvarium under physiological conditions; and the charts we produce are necessarily modified by the existence of pathological conditions. Even these charts, however, testify to this pressor effect.

Now experimental work has repeatedly demonstrated the intimate relationship between the cerebro-spinal and the blood pressure. Thus, Professor Leonard Hill has shown that equilibrium is maintained between the pressures in the cranio-vertebral cavity and the large cranial venous sinuses and cerebral veins. Also Cushing has demonstrated, more recently, that an increase of pressure in the cranio-vertebral cavity is accompanied by an increase in the blood pressure, which tends to assume a slightly higher level.

The explanation of such a mechanism would appear to be as follows:

The intracranial pressure is equal to the cerebral venous pressure. Any increase of the former will produce in the first place most evident results on the most collapsible structures—that is, on the veins. When, therefore, the venous return is impeded from the gradual compression of the venous channels (as in increased intracranial pressure), the return of deoxygenated blood from the centres is delayed and the blood pressure rises, as in asphyxia; for the venous blood stimulates the vasomotor centre and induces a general vaso-constriction.

When such abnormal conditions exist, as in the cases we present, it is found that the stimuli of the operative manoeuvres evoke a less marked response from the vasomotor centre, which is already reacting to the stimulus of the imperfectly drained venous blood. When, however, the intracranial pressure is relieved the variations in blood pressure, as the result of the steps of the operation, become more marked; this is due, doubtless, to the renewed supply of arterial blood to the vasomotor centre, and the consequent relaxation of the arteries of the body.

The tracing of Chart 33 was taken from a child of

Chart 33.—Decompression; cerebral oedema.

7½ years suffering from spreading oedema of the brain, the result of whooping-cough with convulsions, in whom a decompression operation was performed. The patient was quite unconscious, and little anaesthetic was required. It will be seen that the rises *a*, *b*, *c*, following incision and reflection of the flap, are quite typical, while the separation of the periosteum (*c*, *d*, *e*) forms part of the same general rise of pressure. The application of the trephine at *f* is accompanied by an appreciably marked pressor effect. The application of the forceps to the skull at *g* is again followed by a rise in pressure. But it should be noted that, after this point, the excursions of blood pressure, consequent on the prolongation of successive stimuli, became progressively less marked, so that up to the point *β* the mean curve is about level. The removal of bone with the forceps from *h* to *j*, is accompanied by a very rapid fall in pressure of 32 mm. Hg in 1 minute 20 seconds. The nature of this fall is open to three constructions.

1. *The Onset of the Depressor Stage of Shock.*—In favour of this view are the falls *a*, *β*, *γ*, *δ*, *h*, *j*. This view, however, seems to be negated by the character of

the blood-pressure, subsequent to *j*, on attacking the dura mater.

2. *It may be an anaesthetic effect*; for the character of the respirations, from the point *R*, indicates that anaesthesia is lighter, and shortly after the child coughed. The depth of anaesthesia from this point was increased, as shown by the diminution in amplitude and the increase in regularity of the respirations. The fall *h j*, therefore, may be due to deeper chloroform narcosis. But subsequently the respiratory trace assumes a similar character to that previous to *R*, therefore it is probable that the narcosis was only pushed to a similar degree, and is insufficient to account entirely for so marked a drop in pressure.

3. We believe the fall *h j* is due, to a large extent, to the partial relief of pressure by the removal of a large area of the calvarium. This is calculated to lower the arterial blood pressure in the following way: The removal of pressure permits the expansion of the collapsed cerebral veins, and so allows the removal of venous blood from, and the access of arterial blood to, the vasomotor centre. Whether the stimulation of the vasomotor centre in intracranial pressure is attributable to anaemia (as stated by Professor Leonard Hill) or to excess of CO₂, the stimulus is thus removed, the vaso-constriction is relaxed, the force of the cardiac impulse is diminished, and the arterial blood pressure falls.

At *j* the manipulations and incision of the dura mater begin, and the marked pressor effect from *j* to *k* is noticeable. After the point *k*, following upon the escape of cerebro-spinal fluid, a further very marked fall of pressure is seen at *k l*. At this time the child's condition was one of profound collapse, for no readings could be obtained until near the conclusion of the operation, when the alarmingly low figure of 22 mm. Hg was obtained. In spite of intravenous infusion, the child died an hour after the conclusion of the operation.

The fall of blood pressure consequent on the relief of intracranial pressure appears to be entirely a passive vascular phenomenon, independent of any true shock from the operation; for in this case the fall occurred in both instances during a pause in the steps of the operation.

The vascular collapse, from *k* to *l*, was so profound that marked anaemia resulted; this is shown by the fact that manipulations of the cerebral cortex induced no ascertainable effect on the blood pressure. It will be shown later that such manipulations usually produce marked "spikes" in the blood pressure, and the absence of these indicates a loss of excitability due to the profound anaemia from sudden relief of pressure.

This chart, then, illustrates the pressor effects on the vasomotor centre of the manipulations of the scalp, calvarium, and dura mater; at the same time, this phenomenon is not extreme, owing to the increased intracranial pressure (already causing a constant stimulus to the vasomotor centre) at the time of the operation. In favour of this explanation is the very well-marked pressor effect at *j*, on incising the dura mater, after the partial relief of pressure at *h j*.

This chart assumes considerable importance when we consider the practical bearings of these deductions. The two following charts are of interest in that they represent the first and second stages of the removal of a left intracerebellar tumour in a boy of 7 years.

They are also of importance from the point of view of the comparison of the results of tracings in supratentorial and in subtentorial pressure. The operation area was exposed by Cushing's method. (The chart of the first stage is not reproduced.)

The well-marked pressor effect on incising the scalp is again emphasized by the fall on the cessation of manipulations.

The succeeding excursions of the blood-pressure chart illustrate the attack on periosteum quite well; but the most notable feature is the marked rise in blood pressure on removing the calvarium with bone forceps. A gradual subsidence of blood pressure follows the cessation of the operative manœuvres (during the ligature of bleeding points), interrupted only by well-marked oscillations during the palpation of the cerebellum through the dura mater. A renewal of the stimuli with the closure of the wound (and perhaps a lighter degree of anaesthesia, as evidenced by the variability of the respiratory curve) is responsible for the terminal rise in blood pressure. In interference

with the scalp and calvarium in these two cases there is a marked approach to the depressor type of curve; this is probably due to the nearness of pressor fatigue, due to the continued stimulation of the bulbar centres by the raised intracranial pressure.

Now, one point to which we wish to call attention is the absence of any precipitate fall of pressure following the removal of the skull below the tentorium.

In order to consider the significance of this fact it is necessary to revert for a moment to the subject of lumbar puncture.

We have already shown that the net result of lumbar puncture is to raise the blood pressure for at least twenty minutes afterwards; and it appears that this rise may be greater and more sustained in subtentorial cases.

Now it is essential to realize that the increase of tension of cerebro-spinal fluid is circulatory in origin; for, in cases of intracranial pressure, lumbar puncture during life releases the cerebro-spinal fluid *under pressure*, a phenomenon which is never observed after death.

Further, it is reasonable to infer that the ventricles share in this increased tension and become proportionately distended.

With the increase in pressure we have already stated that the veins are the first structures to be affected.

Hence, the removal of venous blood becomes impeded, and the volume of the cerebellum decreases proportionately as the volume of the cerebro-spinal fluid increases. At the same time the cerebrum shares in this obstruction to the venous return.

Lumbar puncture under such conditions must produce a twofold effect:

1. The sudden engorgement of the partly collapsed veins with venous blood, with the consequent increased stimulus to the vasomotor centre; a rise in arterial blood pressure from general vaso-constriction, and an increase in bulk of the cerebellum.

2. The synchronous engorgement of the cerebrum, whose bulk will also tend to increase and shut off the communication between the upper and lower chamber.

The second phenomenon prevents the escape of more than a certain amount of cerebro-spinal fluid, increases the venous engorgement of the cerebellum and medulla, which is responsible for the raised arterial pressure, and thus assists in the production of those factors which favour the respiratory failure from sudden excessive stimulation.

The failure of respiration in such cases has been attributed to the formation of "pressure cones" (Cushing). Against this view may be urged the following considerations:

1. At first the blood pressure should fall, consequent on the sudden diminution of intracranial tension from the descent of the cerebellum into the foramen magnum. The blood pressure has been shown, however, to rise, owing to the immediate replacement of the loss of cerebro-spinal fluid by venous engorgement, and the stimulation of the centres.

2. Sudden cessation of respiration during lumbar puncture may occur in cerebellar cases without any evidence, either at operation or *post mortem*, of the existence of "pressure cones."

(We may quote a case of raised intracranial pressure in which sudden respiratory failure occurred. Artificial respiration was maintained by the battery for some hours before, during, and after operation. At the operation no evidence could be found of the existence of "pressure cones," and at the autopsy there were no indications that these had ever been present.)

3. Pressure cones have been found *post mortem* in cases which showed no sudden terminal respiratory failure.

4. It is difficult to believe that a comparatively soft structure such as a cerebellum (which is probably oedematous) can exert sufficient direct pressure on a comparatively firm and fixed structure like the bulb to make any appreciable effect on the respiratory centre.

5. In lumbar puncture performed on cases of supratentorial pressure the first brunt of the venous engorgement falls on a far larger vascular area more remote from the bulb—a fact which is sufficient to account for the difference in behaviour in the two classes of lesion.

Thus, though the existence of "pressure cones" is recognized and admitted, we believe that these must be regarded as frequent, but by no means constant, accompaniments of increased subtentorial pressure, and of lumbar puncture performed on such cases.

We believe that the respiratory failure referred to is independent of this phenomenon and due to the sudden

blood pressure swinging back to an intermediate level (f^1) during the pause following the removal of the tumour; while the application of hot saline (f^1) produces a further active rise of pressure, which subsequently falls below the original level—the total curve $f^1 g$ being of the depressor type, but less pronounced than the curve $d e f$.

Perhaps the curve f^1g is an indication of the near approach of pressor fatigue—a view which subsequent readings appear to confirm. Thus the suture of the dura mater, usually accompanied by pressor manifestations (see curve $a\ b$) are signalized by marked depressor “spikes” ($g\ h, i\ j$); while at the end of the second of these ($i\ j$) the pressure fell to 34 mm. Hg.

Once more it seems evident that a pressor type of blood-pressure curve accompanies injuries to, and surgical interference with, the protective coverings of the brain—that is, the scalp, skull, and membranes—while the resistance of the latter two structures to infective agents is low.

In the case of the scalp, however, the relationship of resistance to infection and shock value do not seem to hold good. For, though the scalp has a low shock value, it also has a notoriously high resistance to infection. The explanation of this apparent anomaly would seem to rest in the fact that the surface of the scalp is naturally very septic, and this

Chart 35.—Second stage, cerebellar tumour.

venous engorgement, over-stimulation, and so fatigue of the synapses (as will be shown in Lecture II).

We also think that "pressure cones" may not infrequently be a *post-mortem* occurrence; for, when the vascular pressure falls with death, the cerebro-spinal pressure falls correspondingly, and it is quite possible that the descent of the cerebellum may then take place under a negative pressure from below.

Chart 35 affords additional evidence that the rapid fall of blood pressure accompanying the opening of the dura mater is of the nature of a mechanical relaxation of the continued "pressure-stimulus" to the vasomotor centre, rather than an example of the negative stage of shock.

This chart represents the second stage of the removal of the cerebellar tumour (enucleation of the mass). There is nothing of special note until the point *a*, at which the reflection of the dura mater is commenced on the left side.

Now in this case the mass was situated in the left lobe of the cerebellum, and was intimately adherent to the dura mater; consequently the reflection of this membrane was followed by a very marked pressor effect (*a* to *b*); and no sudden fall in blood pressure occurred, for there was little, if any, escape of cerebro-spinal fluid.

As a contrast, we note the effect of opening the dura mater on the right side, at *c*. The free flow of cerebrospinal fluid under pressure causes a marked vascular collapse, *c*, *d*. It might be argued that this fall of pressure is incompatible with the explanation offered of the phenomena observed during lumbar puncture; but it must be remembered that in this case we are no longer dealing with a closed cavity, but an open one. Thus, there is no further obstruction to the return of venous blood, and further, the operation is responsible for a considerable depletion of venous blood. Again, the manipulations of the cerebellum, necessitated by the enucleation of the tumour, are accompanied by a very marked excursion of the manometer.

This excursion ($d\ e\ f$), however, is of the depressor type—that is, the removal of the stimulus is followed by a drop to the original level or slightly below it. The rise of pressure, $d\ e\ f$, is open to another interpretation—namely, that the pressure involved in enucleating the tumour exerts direct compression on the bulb, inducing anaemia of the centres, and so a rise in blood pressure (Hill). Against this may be urged that the cerebellar chamber is no longer a closed cavity, and that, in enucleating a mass from the lateral lobe, the pressure is largely directed *away* from the bulb.

Further evidence in favour of the depressor effect of the actual manipulations of the cerebellum is shown by the

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

EPIDEMIC OPHTHALMIA.

In an epidemic recently under my notice, oedema of the conjunctiva, with signs of acute inflammation and oedema of the lids, shutting light out entirely, made me expect serious results. Even in children with the lids absolutely closed, the absence of pain observed in epidemics I had seen a generation ago struck me. There was as a rule little discharge, and when purulent the pus was lighter in colour than in the epidemic Australian (sandy blight, so-called) and our herpetic and gonorrhoeal ophthalmias. The contagious character was evident, as well as its persistence, when not actively treated. In a case of chorea with ichthyosis there was more pus than in most of the other cases, but treatment was equally effective. In no case did I observe corneal ulcers. I may say that I was quite surprised to find the pneumococcus on microscopical examination of the discharge, and no cocci. Albanese found moderate secretion with "*pneumocoque et bacille massé*" (*Annales d'oculistique*, October, 1911, page 249). Métafuné (loc. cit.) found *pneumococcus*.

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ON THE GENESIS OF THE VENOUS PULSE.

DR. SAMWAYS states in the BRITISH MEDICAL JOURNAL of April 13th that the genesis of the *a* wave in the jugular pulse may be entirely explained by supposing it to be due to the inertia of the column of blood in the jugular vein. If this be so, how does he account for the following facts? In the normal healthy patient the *a* wave is much more prominent in the horizontal position; surely if Dr. Samways's explanation were true the wave ought to be greater in the vertical position, for then the inertia of the column of blood in the vein is augmented by the effects of gravity.

Again, in cases in which auricle and ventricle are dissociated, the *a* waves, wherever they occur, are of the same shape, but where the auricular systole has occurred during ventricular systole a much greater *a* wave results. The inference is that the *a* wave is due (in part if not entirely) to regurgitation of blood from the auricle during