THE NATURE OF SHOCK, AND ITS MANAGEMENT.

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Those who have had much experience in abdominal surgery have met and learned to dread that condition known as shock, which may occur after an apparently uncomplicated operation.

It is generally accepted that the sympathetic nervous system is the chief factor in its causation, but how it acts, and what is its condition at this time are points of great importance to determine if we will arrive at correct ideas as to rational treatment.

Shock and collapse are generally spoken of as synonymous terms, but very incorrectly, inasmuch as they are related only in this, that severe shock causes a condition greatly resembling collapse. Shock always depends upon a nervous impression (if I may so term it), while collapse may (and frequently does) result from exhausting discharges. The two may be so intimately blended that it will be difficult to dissociate them, but for purposes of study they should be kept distinct.

DEFINITION AND PHENOMENA OF SHOCK.

I may, for the purposes of this paper, define shock as a condition characterized by a certain train of symptoms (to be mentioned later) and caused by some sudden, more or less violent, impression on the sympathetic system of nerves. It may vary in degree from the slightest appreciable disturbance to one so profound as to cause instant death. While the phenomena of shock are always due to disturbed innervation, the causes originating this disturbance may be very varied. They may be mental or physical. The impulse may arise in the brain, spine, cerebro-spinal nerves, or in some portion of the sympathetic system itself. Wherever originating, the

impulse is transmitted to the centres of the ganglionic system, and thence distributed.

These causes may be predisposing or exciting. The predisposing causes are:

1. A weakened condition of the general nervous system, by reason of which it is easily impressed.
2. A depressed condition of the system generally.
3. A despondent, apprehensive mental condition.

It will not be necessary to analyze these conditions, as they suggest themselves to all as naturally predisposing to shock. But the influence of the mental condition of the patient is so important as to merit further remark. The effect of sudden fear in producing increased intestinal peristalsis, relaxation of the sphincters, palpitation of the heart, sudden pallor, etc., is familiar to all, and is nothing more nor less than shock—the impulse being mental in origin and transmitted through the cerebro-spinal system to the ganglionic centres. In the same way a feeling of apprehension or dread of an operation, while it may not be of such a nature nor so severe as to cause actual shock, nevertheless so influences the nerve centres as to greatly lessen their resisting power. It is not necessary to cite instances, as the experience of every operator will supply them, but I simply wish to emphasize the well-known fact, in order that it may not be lost sight of when we consider the treatment of a patient about to be subjected to an abdominal operation.

The exciting cause, when physical, is always some injury to the nervous system. It may be so slight as to cause no organic change, or so severe as to destroy some portion of nerve tissue. Nor is the severity of the shock proportionate to the degree of injury; for instance, a blow on the epigastrium, or the perforation of an intestine, may cause shock so profound as to cause death, while severe injury to tissues in other parts of the body may not be followed by perceptible shock.

PATHOLOGY OF SHOCK.

What then is this shock? It is often described as a paresis of the entire sympathetic system. Again, as a paresis of the respiratory and cardiac ganglia. Again, as a paresis of the circulatory system. Also as an abnormal dilatation of the abdominal veins. But a consideration of the pathology and symptoms will show, I think, that

it is not a paresis; or, if so, that the paresis is secondary. There are several facts which weigh against the theory of paresis, either partial or general. First. It is an established physiological fact that paresis of the sympathetic fibres supplying any part will cause *dilatation* of the arterioles supplying that part. I will here state that I have used the words "sympathetic" and "vasomotor" interchangeably to a certain extent, because if the vasomotor nerves are not technically part of the sympathetic system proper they are so related in function, and so intimately blended anatomically, as to react similarly to stimuli. The vast majority of vasomotor fibres, after leaving the spinal cord, pass to their destination through the corresponding sympathetic ganglia, and in intimate union with other sympathetic fibres distributed to the same parts. Irritation of a ganglion stimulates its efferent vasomotor fibres. An impulse of irritation received by the solar plexus and thence distributed to the various ganglia, is there redistributed as stimulation to all efferent fibres, vasomotor and others.

Paresis of the entire sympathetic system would therefore naturally cause dilatation of the arterioles of the entire body, and the surface would be flushed; but these arterioles are contracted and the surface is pale; therefore it cannot be a paresis of the entire sympathetic system.

But if there could be a paresis of the cardiac ganglia, with normal action of the rest of the system, it would hardly explain the symptoms. Though paresis of these ganglia would weaken the heart and thus limit the amount of blood sent through the arterioles, rendering them partially collapsed, the action of the heart would be slower. The innervation of the heart is from both the sympathetic and cerebro-spinal systems; from the sympathetic through the cardiac branches of the cervical sympathetic, stimulation of which accelerates and strengthens the action of the heart; from the cerebro-spinal through the pneumogastric, stimulation of which slows the action of the heart. Removing normal force through the sympathetic ganglia allows a disproportionate power from the pneumogastric, which is inhibitory and *slows* the heart's action.

But one of the first indications of shock is disproportionate *frequency* of the heart-beats. There is also another point which, while not an argument, is indicative of the true pathology. The cardiac nerves supplying the cardiac ganglia arise from the cervical sym-

pathetic. Now, paresis of these branches would in all probability be accompanied by paresis of the other branches of the cervical sympathetic; or at least they would not be abnormally stimulated while the cardiac branches were paretic. The nerve which controls the pupil of the eye arises from the cervical sympathetic, and *stimulation* of this nerve causes *dilatation* of the pupil. In shock the pupil is dilated.

Again, it has been said to be a paresis of the vasomotor nerves of the abdominal vessels by which these vessels become greatly distended, and the amount of blood in the rest of the body greatly lessened. Most of those who hold this view base their belief on the results obtained by Goltz, of Strassburg, in what is called his percussion experiment. A frog was taken and suspended in a vertical position with the legs downward and the heart exposed. After waiting a short time till the beats were fairly regular and sent the usual amount of blood into the aorta, the frog's intestines (or the surface of the abdomen) were struck with some violence. The heart immediately stopped. The veins of the abdomen were distended and the upper part of the vena cava was empty. Soon the heart began to beat vigorously, but remained empty, because it had no blood supply.

But careful observers, even while admitting the correctness of the experiment and accepting the conclusions, say that there must be something more to account for all the symptoms of shock than paralysis of the abdominal vasomotors. A violent blow on the epigastrium of a frog causes distention of the abdominal vessels and emptiness of the veins in the upper part of the body. In shock, the abdominal veins are distended, but so also are the veins of the entire body, as is shown by the livid pallor, so different from the waxy pallor of hemorrhage, which would be present if the conditions in the man and frog were the same.

Paresis of the vasomotor nerves of the abdomen cannot explain the symptoms. The splanchnic nerve is the great vasomotor nerve of the abdominal viscera. The greater part of the fibres to the renal vessels are given off from the splanchnic. Claude Bernard and others,[1] have found that section (or paralysis) of the renal nerves causes greatly *increased* secretion of urine, because of the increased

[1] Landois and Stirling: Manual of Human Physiology, p. 579.

amount of blood sent to the glomeruli through the dilated arteries. Section (or paralysis) of the splanchnic causes the same condition, but in a lessened degree, because the pressure is lessened by reason of the dilatation of the other abdominal vessels. On the other hand, stimulation or irritation of these nerves, by which the calibre of the arteries is lessened, caused *decreased* secretion of urine. Moreover, any obstacle to the free flow of venous blood away from the kidney, by which backward pressure is increased, causes scanty secretion of urine. In shock there is greatly *diminished* secretion of urine, therefore it *cannot* be paresis of the abdominal vasomotors.

The primary pathological condition in shock is a *hyper-irritation* of the sympathetic nervous system. All involuntary muscles tend normally to rhythmical contraction, and any disturbance of this rhythm is abnormal. Witness the action of the heart; when in health the rhythm is the same, though the frequency of its contractions may be varied. So with the uterus, the intestines, etc. That this rhythmical action is not confined to involuntary muscular fibre, I fully believe, though it is not as easy of demonstration. It undoubtedly pervades all organs and glands whose action is under the control of the sympathetic system, and is induced by the normal balance between the stimulating action of the sympathetic nerve-fibres and the inhibitory action of cerebro-spinal nerve fibres, and is disturbed by a preponderance of either. These two nervous systems are entirely distinct in their functions, but are mutually interdependent. They are not only interdependent as to function, but are so intimately blended anatomically as to be almost inseparable. Cerebro-spinal fibres largely enter into the composition of the solar plexus, and generally accompany the fibres sent off therefrom. On the other hand, sympathetic fibres and ganglion cells penetrate the spinal column and traverse the brain.

The function of the sympathetic system is to promote this rhythmical action of glands, involuntary muscular fibres, and cells. Stimulation of a sympathetic centre increases the action of the parts supplied by its fibres. Stimulation of the fibres supplying the heart and the muscular coats of the arteries increases the contractile power of those muscles and lessens the calibre of the arteries and arterioles. Over-stimulation of those fibres tends to cause *tonic* contraction, and may result in death by spasmodic contraction of the heart.

We can see, by studying the action of the uterus, the mode of action of the involuntary muscular fibre. Under normal conditions, at time of labor, the contractions come regularly at even intervals, beginning at the fundus and passing downward with a wave-like motion till lost in the lower segment. This normal contraction is followed by a period of perfect rest and relaxation. But if the normal stimulus be increased by an excessive use of ergot or external irritation, this rhythm is impaired. The contractions become more violent and frequent and lose their normal peristaltic character, tending to become tonic. The period of relaxation is greatly impaired—so much so that often it is represented simply by a little less violent tonic spasm. After delivery, absolutely tonic hour-glass contraction may supervene. The heart acts, under stimulation, in the same way.

Landois and Stirling, in their work on *Human Physiology*, in speaking of electrical stimulation of the cardiac nerves, say: "A constant electrical current of moderate strength increases the number of heart beats."

"If the constant current be very strong . . . . *the cardiac muscle assumes a condition resembling, but not identical with, tetanus* (Ludwig and Hoffa), and of course this results in a *fall of blood pressure* (Sigm. Mayer)."

Brunton and other experimenters have explained this action by the fact that all non-striated muscles, for instance, the muscular coat of the intestines and the uterine muscle, are induced to increased contraction by an excess of carbonic acid in the blood, and *vice versa*, an excess of oxygen decreases such muscular action. However the explanation may be, the fact remains that stimulation of the fibres of the sympathetic system of nerves supplying involuntary muscular fibre tends to cause increased and tonic contraction of those muscles. Thus the heart is not allowed to dilate or relax fully. The rhythm may or may not be destroyed, but the muscle contracts powerfully and relaxes scantily. So with the arterioles; their calibre is diminished by reason of this tonic spasm, if I may so term it. So with the intestines; their peristalsis is increased.

But this contraction of the arterioles introduces another factor into the process of nutrition and life. By it the amount of arterial blood supplied to a part is lessened. By this imperfect relaxation of the heart the venous system fails to unload itself, and every-

where it is gorged with blood saturated with carbonic acid. And this is the true explanation of the unnatural distention of the abdominal veins, noticed by many observers. Not a stimulation of vasodilators, nor a paresis of vasomotors, but a *stimulation* of the entire "vaso-constrictor" system, by which the entire arterial system is contracted, and the blood of necessity distends the veins.

By reason of this contraction of the arteries and consequent distention of the veins, the cerebrum would not get its proper supply of arterial blood, and its action would be dulled; there would be a condition of mental apathy and an indifference to external impressions and sensations. By reason of this hyper-irritation of the sympathetic system, the heart would contract rapidly and would relax very imperfectly. The pulse felt at the wrist would be rapid and small or almost imperceptible. The extremities and surface would be cool, pale, and livid; the finger-nails, purple; the temperature depressed, and respiration slow. By reason of the general impulse of irritation sent out from the solar plexus, intestinal peristalsis might be increased, and the function of the perspiratory and intestinal glands augmented.

Unlike the kidneys, in which no special secretory nerves have been demonstrated, the sweat glands are supplied with sympathetic fibres whose special office it is to preside over the secretory function. *Stimulation* of these fibres produces free diaphoresis of the parts supplied by those fibres without regard to the vascular conditions. In shock the surface is often bathed in perspiration; nor is the area limited, but the entire body is equally affected, showing irritation of the sympathetic nerve supply to the entire perspiratory system. These secretory fibres almost invariably accompany the vasomotor fibres, often lying in the same nerve trunk.

It is therefore a fair inference that the *vasomotor* nerves of the entire system participate in the *stimulation* which certainly is demonstrated as existing in the *secretory* nerves of the entire *perspiratory* system. This aggregation of conditions, when induced by an impression on the nervous system, is shock, and that impression is of the nature of hyper-irritation.

It is stated in the *Text-book of American Surgery*, edited by Drs. Keene and White, that in cases of sudden death from shock the heart has been found *contracted* and *empty*, a condition that we

should expect, according to this explanation of the pathology. Whether in death from prolonged shock this condition would be found we cannot determine, because it may be that, while the irritant action of the sympathetic continues, and the ganglia are continuously stimulated, the heart muscle becomes gradually weaker, and increasingly unable to propel the scanty supply of blood allowed by the imperfect relaxation, or to overcome the resistance of the persistently contracted arterioles, and the heart might finally stop in a condition of more or less imperfect relaxation.

It has been objected that the condition cannot be one of "tonic spasm" of the heart and muscular coat of the arterioles, as, in that case, the blood pressure in the arterial system would be increased. This objection does not take into account the *imperfect relaxation* of the heart, whereby it can receive only a limited amount of blood. The large arteries have no muscular coat, and they therefore would not be contracted as are the small arteries and arterioles, which have a well-marked muscular coat supplied with a network of sympathetic ganglia. Therefore the small amount of blood received by the heart and sent into the large arteries would not be sufficient to distend them perceptibly, consequently their elasticity would not be sufficient to send a current with force enough to appreciably distend the contracted arterioles, and the blood-pressure would not be high nor abnormally increased. This is explained by Landois and Stirling in estimating the tension of the pulse; that "a small pulse occurs when a small amount of blood is forced into the aorta, as from mitral regurgitation, etc." Again: "Blood pressure is increased with greater filling of the arteries, and *vice versa*." Again, in speaking of electrical stimulation of sympathetic cardiac nerves, as I have quoted above: "If the constant current be very strong the cardiac muscle assumes a condition resembling but not identical with tetanus (Ludwig and Hoffa), and of course this results in a fall of blood-pressure (Sigm. Mayer).

THE MANAGEMENT OF SHOCK.

How, then, shall we treat shock? It will not be necessary, nor shall I attempt, to discuss in this paper the action of various remedies. I shall merely endeavor to indicate a certain line of treatment that appears to me to be desirable, and the remedies naturally to be selected are those sedative to the sympathetic nervous system.

These are principally—

1. Nitrite of amyl for its immediate and temporary effect.

2. Morphine (or preferably codeine) for its slower and more lasting effect.

3. The application of moist heat.

The action of nitrite of amyl in dilating the arterioles is familiar to all, as most of us have seen patients on the operating-table suffering from severe shock, with an almost imperceptible pulse, rally promptly after the inhalation of nitrite of amyl, the pulse becoming fuller and slower. As to morphine, it must be given in sedative doses in order to get the fullest benefit from its use. It is known to be a true sedative to the nerve centres, ganglionic as well as cerebro-spinal, and in this way tends to relax spasm and allow the arterioles to dilate normally, thus restoring the vascular equilibrium and relieving shock and its consequences. In heat we have a most powerful and reliable agent in influencing the circulatory apparatus. I cannot, in the short space of this paper, discuss in any degree its mode of action, but can simply offer some conclusions arrived at through the researches of others. The skin is, as you all know, a network of arterioles and capillaries, and accompanying these vessels are corresponding fibres of sympathetic nerves regulating their action. In health the application of moist heat to the surface produces a sudden stimulation of the nerve fibres, with corresponding contraction of the arterioles, to be quickly followed by a prolonged period of sedation, whereby the arterioles are relaxed, or dilated, and the skin becomes suffused with blood. Neumann, by a series of elaborate experiments, demonstrated that intense stimulation of the surface by hot water diminishes cardiac force and activity, dilates the vessels and slows the current. And this is exactly what is desired in severe shock. He also showed that cooling or heating the blood current itself affects the cardiac ganglia.

That this is practically true has been shown in two cases recently reported as occurring in the practice of Dr. Mundé, of New York. Both cases were suffering from profound shock, threatening immediate death. One case was complicated by excessive hemorrhage; in the other there had been no appreciable loss of blood. In both cases transfusion into a vein with a large amount of water, heated to 118° or 120°, was practised with immediate improvement in all the symptoms and ultimate recovery of the patients.

Thus it seems to be proven that the general application of moist heat to the surface acts: first, locally on the nerve fibres, supplying the arterioles of the skin itself, relaxing them; and second, reflexly through the solar plexus on the entire sympathetic system. The injection of hot water into a vein accomplishes the same purpose more directly, by acting as a sedative to the ganglia of the heart and arterioles and allowing their muscular fibres to relax.

In accordance with this view of the pathology of shock, I venture to offer the following suggestions:

The physical preparation of a patient about to be subjected to an abdominal operation must be as thorough as possible. It is well to begin the treatment of the skin and bowels two or three days previously. When a patient is much prostrated physically the diet must be as nutritious as possible, and yet such as to leave little residue up to twenty-four hours before the operation. Any feeling of apprehension that may exist must be quieted as thoroughly as possible. When the operation is begun such haste as is compatible with safety must be made, inasmuch as prolonged anesthesia adds to the element of exhaustion, and the more manipulation there is the greater the irritation, and hence the more pronounced the shock. All manipulation must be as gentle, rapid, and sure as possible, and the operation completed with the least possible violence. The operating-room must be warm, the patient's surface not chilled, and the patient finally put into a warm bed with dry warm clothing.

As for medication, nothing in the way of prophylaxis is better than the administration, hypodermatically, just before the anesthesia, of a half-grain or more of codeine. Dr. Stephen Smith, in an article published in the *Medical News* of October 11, 1890, says that for eight or ten years he has prepared his patients for operation by rendering them partially intoxicated. They then have suffused eyes, flushed face and skin, and a slow and full pulse (all indications of a sedative or paretic condition of the sympathetic system), and that he has never in all that time had a case of severe shock. Therefore, for prophylaxis quiet the mind of the patient and produce sedation of the ganglionic centres.

Shock occurring on the operating-table must be met principally by lowering the head of the patient and the inhalation of nitrite of amyl. Mild shock following operation is to be treated mildly—keeping the patient as quiet as possible, warm and free from all

annoying or disturbing influences. Severe shock is to be combated actively. The most favorable surroundings possible must be secured, with freedom from all annoyance. The mental condition must be kept as quiet and serene as possible. A free dose of morphine or codeine, administered hypodermatically (all medicines should be given in this way), with nitroglycerin and possibly strychnine. This last drug stimulates the sympathetic system, but it stimulates much more actively the cerebro-spinal system, and may thus, by tending to increase the power of the inhibitory nerves, regulate or control the heart and arteries.

But I think the main reliance must be placed on moist heat. Externally, it may be employed in the form of the hot wet pack, or the hot bath; internally, by the injection, through the long tube, into the colon, of large quantities of very hot water, thus bringing the heat into very close proximity to the solar plexus. Or, in cases where there has been free hemorrhage, or where the patient was, before operation, anemic and exhausted, transfusion of as large quantity as possible of *hot* saline solution as practised in Dr. Mundé's cases. In this way the temperature of the entire blood current is raised, and the ganglia of the heart and arterioles are subjected to the direct sedative influence of moist heat. This outline of treatment is simply indicative of that plan which, in my opinion, offers the greatest hope of success in the treatment of severe shock.

CONCLUSIONS.

To sum up, therefore, shock is not a general paresis of the sympathetic nervous system, because:

1. The entire arterial system is contracted rather than dilated.

2. The skin is moist, or bathed in perspiration, by reason of the irritation of the secretory fibres of the sweat-glands, instead of dry, as it would be in pallor of the skin, low temperature and *paresis* of sympathetic fibres.

It is not a paresis of the cardiac nerves and ganglia, because:

1. The heart's action would be slow, by reason of the preponderance of the inhibitory influence of the pneumogastric.

2. It is not probable that cardiac branches of the cervical sympathetic would be paretic, while other branches were abnormally

stimulated; and in shock the pupils are *widely dilated* by reason of the *stimulation* of the pupillary branch of the cervical sympathetic.

It is not a paresis of the vasomotor nerves of the abdominal vessels, because:

1. Paresis of these nerves would cause *dilatation* of the abdominal arteries.

Dilatation of the renal artery would cause polyuria, even if accompanied by dilatation of the other abdominal arteries. In shock the secretion of urine is scanty or even suppressed.

2. Dilatation of abdominal arterioles would cause *decreased* intestinal peristalsis. In shock peristalsis is often increased.

On the other hand, it *is hyper-irritation* of the *entire sympathetic system*, because:

1. The skin is pale and livid by reason of *contraction* of the arterioles, because of *stimulation* of their vasomotor nerves.

2. The heart's action is rapid by reason of *stimulation* of its sympathetic nerve supply.

3. There is scanty secretion of urine by reason of contraction of the renal arteries, the result of *stimulation* of their nerve supply.

4. The skin, though pale and livid, is bathed in perspiration by reason of *stimulation* of the secretory nerves of the glands.

5. The pupils are dilated by reason of *stimulation* of their sympathetic nerve supply.

6. The pulse at the wrist, while rapid and small, as would be expected in vasomotor stimulation, is soft and *very compressible*, by reason of the very scanty relaxation or dilatation of the heart.

7. The condition of the heart may not have been actually demonstrated, but may justly be inferred by analogy, reasoning from the action of the uterus under similar conditions. Each contraction of the uterus is normally followed by a period of perfect relaxation, as is the heart. Over-irritation or stimulation of the uterine ganglia or sympathetic nerve supply causes *rapid* contractions, with *very imperfect relaxation*. It is fair to infer the same condition in the heart under similar causation. Thus the supply of blood thrown into the arteries is scanty, and arterial blood pressure is *low*.

8. The first five of these conclusions are justified by well-known experimental demonstration. The sixth and seventh are fair conclusions by reasoning from analogy.

9. That the condition of the heart is one of stimulation rather

than paresis, may be considered demonstrated by the fact that in cases of sudden death from severe shock the heart has been found *contracted and empty.*

Finally, admitting the correctness of this pathology, it follows that our treatment should be on the line of *sedation* to the sympathetic system—as by nitrite of amyl, nitroglycerin, morphine, and the application of moist heat—

1. To the surface.
2. Through the long tube into the colon.
3. Transfusion of saline solution at a comparatively high temperature.

DISCUSSION.

DR. J. HENRY CARSTENS, of Detroit.—MR. PRESIDENT: I wish to congratulate Dr. Boise on bringing before us one of the most important subjects we can consider. There are some questions in regard to shock that are still *in status quo*, and taking the whole question in all of its bearings, as outlined by the essayist, it is one that we can study and look up at our leisure. We are under great obligations to the author for bringing the subject before the Association; and it is to be regretted that owing to the lateness of the hour it is not practicable to enter into a full discussion of the paper this evening.