quantity. To the ether which contained the extracted diacetic ether, about five cubic centimetres of a weak solution of ferric chloride were added and the mixture was shaken in the cylinder for several minutes. On standing the solution of ferric chloride was distinctly claret in colour, showing the presence of diacetic ether. Since salicylates were present it was necessary to extract the urine with ether in a slightly alkaline solution or salicylic acid would have been extracted by the ether and a colour given to the ferric chloride by it. In alkaline solution no salicylic acid is extracted.

Diacetic acid in the form of the sodium salt was not tested for, since the presence of salicylates would have masked the reaction with ferric chloride and the quantity of urine available was not sufficient for the application of other tests. Since the sodium salt of diacetic acid almost invariably accompanies diacetic ether in urine and occurs in greater amount than the latter body there is little doubt that it was present.

Oxybutyric acid was not tested for owing to the small quantity of urine available.

Conclusion.—This urine contained large quantities of acetone and also diacetic ether. Sugar was not present. Since the acetone was accompanied by diacetic acid, and probably also by oxybutyric acid, it appears that the condition of the urine as regards these bodies is similar to that which occurs in the so-called acid poisoning of diabetes and in cyclical vomiting and other conditions.

THE NATURE OF SURGICAL SHOCK.¹

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[AFTER some preliminary remarks upon the physiological aspects of the subject, with a demonstration of various instruments used for clinically determining arterial blood pressure and of a number of blood-pressure charts, Mr. Sheen continued:]

Those of us who have held somewhat fairly defined views on the nature of shock must have experienced a considerable confusion of ideas on reading the very conflicting papers of Mr. J. P. Lockhart Mummery² and Mr. J. D. Malcolm³ published last year in *THE LANCET* and the *British Medical Journal*. Mr. Mummery, calling attention to Crile's work,⁴ maintains that the lowered arterial blood pressure which occurs in shock is due to a dilatation of arterioles throughout the body. Accompanying this the veins of the splanchnic area are enlarged so that with a diminished carotid there is a raised portal pressure. Such a condition may be brought about by injury to afferent nerve paths or by exposure or injury of the abdominal viscera. The splanchnic area is the key to the situation. Clamp the vessels supplying it and shock does not occur.

Mr. Malcolm, on the other hand, upholds the view that in shock there is a contraction of arterioles throughout the body. The first effect of stimulation of a sensory nerve is vaso constriction. With prolonged stimulation constriction is prolonged and pronounced. In shock there is no evidence of constriction passing into dilatation, such as flushing of the body surface. The thrady pulse, the coldness and pallor of the surface, and the little bleeding from wounds indicate constriction rather than dilatation of the arterioles. We must not assume that a lowered systemic blood pressure always follows arterial dilatation; the more a tube containing fluid contracts the less the pressure within it and if it contracts to absolute closure such pressure will be *nil*. As the shock becomes greater the arterial contraction spreads to larger and larger vessels, hence the lowered carotid pressure shown by a manometer. Clinically, cases are sometimes observed of patients pulseless at the wrist but with a full beat in their carotids. Lister's experiments on frogs many years ago show how almost impossible it is to exhaust the vaso-constrictor nerves. While all the arterioles, including those of the splanchnic area, are contracted the

veins of the splanchnic area are enlarged to accommodate the blood which is forced rather than drawn there. These veins do not lose their tone, for if so the individual would die.

Each writer comments on the usual modes of treatment of shock. Mr. Mummery says that alcohol is of little or no value; vaso-dilators are obviously of no use with already dilated arterioles. Repeated doses of strychnine produce shock and if shock is already present aggravate it by hastening the exhaustion of the vaso-motor centres. Saline infusion is beneficial indirectly by increasing the volume of the circulating fluid. The Trendelenburg position and a tight abdominal binder are commended as emptying the blood out of the splanchnic veins. Crile's pneumatic suit is of value by creating an artificial peripheral resistance. One of the strongest proofs of the dilatation of the arterioles is the fact that suprarenal extract or ergot invariably causes a rise of the blood pressure in shock. Mr. Mummery's definition of shock is "the condition resulting from a fall in the general blood pressure due to an exhaustion of the vaso-motor centres."

Mr. Malcolm takes the more usual view that such drugs as alcohol, strychnine, and nitro-glycerine are of value in the treatment of shock; by them the pulse volume is improved and the pallor of the surface is lessened. The vaso-dilators dilate the contracted arterioles. Crile's observation that in profound shock it is difficult to get saline fluid into the vessels and that it soon leaves the vessels for the tissues producing oedema is in harmony with a contracted condition of the vessels, whereas when shock is passing off the vessels are dilating and sucking in fluid from wherever they can get it, so that all methods of introducing saline fluid become of value. Suprarenal extract and ergot are useful because they brace up the dilated internal vessels. Mr. Malcolm's definition of shock would appear to be a condition resulting from a fall of systemic blood pressure due to a general contraction of the arterial system from over-stimulation of the vaso-motor centres. Both writers note the rise in the specific gravity of the blood which accompanies shock. Mr. Mummery gives an explanation which I find it difficult to understand. Mr. Malcolm says that the fluid parts of the blood are squeezed out of the contracted vessels.

Is it possible to reconcile to any extent these opposing views? Any effort will be of value which tends to bring such conflicting ideas into harmony. This must be my apology for the following imperfect attempt. As a result of injury or surgical operation afferent nerve paths are injured. Along these afferent nerve paths stimuli reach the vaso-motor centres, thence efferent impulses arise which reach the walls of the blood-vessels. At first these impulses are vaso-constrictor in nature, the arterioles (which are microscopic arteries) contract and the blood pressure, as indicated by the carotid manometer or by a clinical instrument, such as the Riva-Rocci sphygmometer, rises. At the same time, without losing their tone, the veins of the portal system and possibly other internal veins dilate, the possible factors concerned in this latter process being lessening of vaso-constrictor impulses with vaso-dilator impulses; blood is forced into the veins by the general contraction of the arterioles, and then the veins reflexly dilate because of the increased pressure of the blood within them. However caused, at one and the same moment the systemic arterioles contract and the portal venous system dilates. What occurs in the first stage of shock is comparable to what occurs in fear, in sleep, on exposure to cold, in the later stages of fever, and in other toxic conditions; in fact, to what occurs whenever conditions are present tending to lower vitality. Man is a warm-blooded animal and in such conditions the blood is accommodated in the internal vessels "owing to the physiological necessity for the conservation of heat" (Malcolm). So when injury or anæsthetic or both render the individual motionless with depressed vital powers a similar distribution of blood takes place.

What is the next stage? There are two distinct sets of nerves going to the blood-vessels, vaso-constrictor and vaso-dilator. The first effect of stimulation of any afferent nerve path such as the sciatic nerve is vaso-constriction—a pressor effect. The pressor effect, however, is the more quickly exhausted so that after repeated stimulation we have vaso-dilator impulses reaching the blood-vessels—a depressor effect.⁵ May not this be what happens in shock? The vaso-constrictor impulses pass off and vaso-dilator impulses

¹ A paper read before the Cardiff Medical Society on March 13th, 1906.

² *THE LANCET*, March 18th (p. 696) and 25th (p. 776), and April 1st (p. 846), 1905: Hunterian Lectures, Physiology and Treatment of Surgical Shock and Collapse.

³ *THE LANCET*, August 26th, 1905 (p. 573), Condition of the Blood-vessels during Shock; *Brit. Med. Jour.*, Dec. 9th, 1905, Remarks on Shock.

⁴ Blood Pressure in Surgery, Lippincott and Co., New York.

⁵ Leonard Hill: Schäfer's Text-book of Physiology, vol. ii., p. 134, Part I.

reach the vessels. But they cannot dilate because there is no blood to fill them, the blood being already accommodated in the portal system and elsewhere, so the condition passes from one of contraction to one of *retraction* and the retracted condition remains when with further stimulation the vasodilator nervous mechanism is also exhausted. Retraction is contraction followed by relaxation but not by extension. The use of the term originated with the obstetricians who apply it to the condition of the uterus after the child has been delivered and the uterine muscle relaxes. Similarly, one can contract the biceps and keep it actively contracted; then one can relax it, keeping the elbow as fully flexed as before. The biceps is now in a condition of retraction. I suggest that Mr. Mummery and Mr. Malcolm may find common ground by regarding the arterioles and arteries in shock not as dilated nor as contracted but as retracted. In fatal shock the abdominal veins gradually lose their tone and blood collects in them. This stagnation results in insufficient oxygenation and impure blood reaches the vaso-motor, respiratory, and other centres, still further impairing their efficiency, "vicious circles" being established. Less and less blood reaches the brain. The heart hurries because it has less blood to work on and finally with stoppage of the heart death ensues. When recovery takes place on the cessation of the local irritation the vaso-motor centres gradually return to their normal condition and effects comparable to those of suprarenal extract but more gradual are produced throughout the vascular system—i.e., a general bracing-up which gives the heart its proper amount of blood to work on. This, however, must be only one of the many factors operative in cases of recovery.

To recapitulate, we have three stages leading to fatal surgical shock: 1. Pressor impulses reach the vaso-motor centres and are followed by contraction of arterioles throughout the body. At the same time the portal venous system relaxes. Both the arterial and the portal pressures are raised. 2. Depressor impulses reach the vaso-motor centres, the arterioles throughout the body relax but do not dilate because there is no blood to fill them, remaining in a retracted condition. The arterial blood pressure falls. 3. The veins of the portal system and possibly other internal veins lose their tone. All the blood in the body collects in them and death follows.

Let us see how these views fit in with the clinical and experimental evidence. In an operation of little severity, such as the radical cure of an inguinal hernia or the removal of the appendix in an uncomplicated case, there is no shock clinically speaking, any slight fall of blood pressure which takes place being attributable to the general lowering of the activity of the nervous centres by the anæsthetic. After severe injuries it is a matter of frequent experience that for a short but variable period there are none of the ordinary clinical evidences of shock. One sees a patient—say, one with a severe injury to both lower limbs—conscious, rational, with good pulse, smoking and talking immediately after his injury, although in one or more hours he may manifest the most severe symptoms of shock. In operations this preliminary stage is shortened or obliterated by the anæsthetic. Chloroform always lowers the blood pressure. Ether does not appear to raise it. Still in most operations the initial effect is a rise of blood pressure and this is most marked when sensory nerves are vigorously stimulated as in stretching the sphincter ani. Experimental evidence agrees with this.

The evidence of the next stage is found in the lowered arterial pressure which we observe clinically with the sphygmometer. In abdominal operations there are various factors which may be productive of shock. Not only is there the irritation of afferent nerve paths, but also the removal of the support of the abdominal wall and the direct irritation of the abdominal nerves. The observations, I think, of the majority of surgeons, with which my own coincide, are that in abdominal operations there is no evidence of dilatation or engorgement of arteries or arterioles but that the veins in the abdomen are full of blood. Clinically we note the remarkable action of intravenous injection of suprarenal extract in bringing the blood pressure of a pulseless individual up to 180 or 190 millimetres of mercury—i.e., considerably above the normal—with correspondingly full pulse. This is explained by the tightening action of the drug on the vessels throughout the body, arteries and veins alike. In fatal shock, unless higher nervous structures are involved, consciousness persists almost to the last and the heart, of course, beats to the last.

Before concluding may I plead with the physiologist to

come to the aid of the clinician in this very difficult problem? Physiological treatises do not neglect "shock" but their interpretation of the term differs from the clinician's. In a leading text-book⁶ we find—"Shock"..... a term more used by the physician than the physiologist..... a term somewhat ill-defined in scope. In some forms of the clinical condition circulatory disturbance and inspissation of the blood play a part, but as understood by the physiologist 'shock' is primarily a nervous condition." After this somewhat contemptuous dismissal of the clinical interpretation of the term rather more than two pages follow dealing with "shock" as the result of various modes of transection of the spinal cord. I can imagine the confusion of the medical student who passes from the "shock" of the physiologist to the "shock" of the surgeon. Here, as in other matters, considering the needs of the great majority of those they instruct, physiologist and clinical teacher should strive to be in accord. Through his special opportunities and his special experience the physiologist is in a most favourable position to undertake the solution of the many problems connected with "shock" and practising members of the profession naturally look to him for the help they need.

Many other factors—cardiac, respiratory, nervous, and toxic—doubtless play their part in the production of the complex condition known as surgical shock and some of these aid in recovery from it. On this occasion I have not attempted to deal with them or with many other points connected with the subject; nor do I pretend that in any way I have given a complete explanation of the condition. My only wish has been to try to throw a little more light upon the matter and particularly to suggest a means of reconciling the divergent views of the two gentlemen who have recently pronounced so ably and so fully on the subject and to whose writings I have been so greatly indebted in the preparation of this paper.

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A CASE OF CANCER OF THE ŒSOPHAGUS COMMUNICATING WITH THE RIGHT BRONCHUS IN WHICH THERE WAS A COMPLETE ABSENCE OF THE USUAL SYMPTOMS.

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IN THE LANCET of Oct. 18th, 1902, p. 1039, Dr. J. G. Emanuel of Birmingham contributed an article on Cancer of the Œsophagus without obstruction, the article being based on a description of six remarkable cases which were treated at the City of London Hospital for Diseases of the Chest, Victoria Park, E., in the years 1897 to 1899 inclusive. In all these cases dysphagia was either altogether absent or else it was obscured by other more prominent symptoms. The following case is remarkable because, not only were there no symptoms of œsophageal obstruction, but there was also a complete absence of the symptoms of a broncho-œsophageal fistula, though the communication between the tubes must have existed for some time before death. It was only at the post-mortem examination that the true nature of the disease was revealed.

A man, aged 55 years, was admitted to the Westminster Hospital on Oct. 30th, 1905, complaining of cough, shortness of breath, and pains in the joints. The only point of note in the family history was that one brother had died from pulmonary tuberculosis. The patient gave a history of scarlet fever at the age of 21 years and for the last four or five years he had suffered from a cough in the winter. In the year of admission the cough had commenced in June—that is, earlier than usual—since when it had been continuously present. There was no history of hæmoptysis, but for some time prior to admission the patient had been losing flesh considerably. He had been confined to bed since July with what he called "pneumonia and pleurisy" and had complained of pains in most of the joints. The cough became very severe and occasionally caused slight vomiting.

⁶ Schäfer's Text-book of Physiology, vol. ii., p. 845.