four to six centimetres in dimensions, involving the entire glans. The inguinal lymph-nodes were palpable; the Wassermann reaction was negative.

On January 5, 1917, a bilateral inguinal lymphadenectomy, followed by total extirpation of the penis, was performed under ether anæsthesia. The laboratory report rendered by Doctor Kolmer showed a squamous-celled carcinoma, with very early involvement of the lymph-nodes. During his stay in the hospital, the patient was given intensive X-ray treatment, in the hope of preventing recurrence. The patient was discharged from the hospital exactly four weeks after the operation.

The patient was exhibited to show the satisfactory results, inasmuch as he has shown no signs of recurrence to date, over two years. He has complete control of urination, obviously assuming the sitting posture, and is actively engaged in laboring work.

Stated Meeting, held March 7, 1918

The President, DR. EDWARD MARTIN, in the Chair

EPITHELIOMA OF THE FACE FOLLOWING X-RAY TREATMENT OF A KELOID

MR. J. S. RAVDIN presented a woman, aged forty-one years, who was admitted to Dr. Chas. H. Frazier's service at the University Hospital, November 20, 1917, with a "running sore" on the right side of the face and the neck.

At fifteen she had typhoid fever, during which time she developed an abscess in the right mastoid region. This was lanced and pus was evacuated. A few years later a keloid appeared in the scar and this was followed by another keloid below the scar. These enlarged until when she presented herself for treatment at the Howard Hospital in 1902, the original keloid covered the entire mastoid process, while the lower one extended from the tragus of the ear above to below the angle of the jaw below, and from the anterior border of the sternocleidomastoid to the anterior border of the masseter.

Doctor Frazier removed the upper keloid, but it was followed by a recurrence. The patient was then referred to Doctor Pancoast for X-ray treatment. This was in the latter part of June, 1903. X-ray treatment was continued over a period of about four years. The doses were not measured. At the time of her discharge in 1908 the keloids had practically disappeared, there remaining only a scar. She was told to return occasionally for observation, but this she failed to do.

In 1915, after a latent period of eight years, she returned. The skin at the site of the keloids had broken down. X-ray treatment at this time was futile. On November 20, 1917, she was admitted to Doctor Frazier's service. Examination at this time showed at the angle of the right jaw and extending on to the cheek anteriorly and the mastoid process posteriorly a sluggish ulcer. Its edges were indurated and everted. It was not very movable on the underlying tissues. A serum exuded from it, which was odorless. It was painful. There was no glandular involvement.

The patient was operated on November 26th, and the area was excised by a wide circular incision. The growth did not seem to infiltrate the underlying tissues. The edges were seared with the actual cautery, bleeding points controlled, but no attempt was made at closure of the wound.

C. A. Porter and S. B. Wolbach in *The Journal of Medical Research*, of October, 1909, reported a number of X-ray carcinoma. They called attention to the latent period between the last X-ray treatment and the beginning of signs of degeneration, also to the relatively benign character of many of these epidermic carcinomas.

E. P. Cumberbatch, in the October, 1913, number of the *Proceedings of The Royal Society of London*, reported a technic whereby he claims to get most excellent results from the X-ray treatment of keloids. He divides the area into four parts and each area is treated separately. No area receives more than one-half a pastille dose of Sabourand, tint B, and each area is given a fortnight's rest before repeating the dose.

ADDRESS ON "SHOCK "

DR. MILES T. PORTER, having been introduced, said that with regard to shock one finds, especially on the part of those who have not been actually at the front in this war, a failure to recognize certain things which are obvious enough when one is actually in the work. One of these is in regard to the definition of shock. When the shock case comes in, the ambulance driver or stretcher bearer used to come to report that there was a shock case. There never was any question about the diagnosis. The patient was the color of muddy parchment; the eyes were turned up, the whites only showing; utterly relaxed. The heart is feeble; respiration in uncomplicated shock is never deep; always feeble, but more frequent than normal and the blood pressure is very much reduced. The appearance of the patient alone gives the diagnosis in these ordinary cases of shock. It is very important to recognize the condition at the beginning, the importance lying in the fact that it is very much easier to save the patient in the early hours of shock than in the later hours. If the low blood-pressure is maintained certain changes occur. He knew of no other way of recognizing the very early stages than by the measurement of the diastolic bloodpressure. One need not say that every case of low diastolic blood-pressure will die of shock; but when the diastolic pressure falls to a low level, if the patient has not shock one may be certain that he will have the symptoms and difficulties of shock if the blood-pressure remains low for any length of time. We have come to a point in the treatment of this condition in which the systematic taking of the blood-pressure is of as much importance as the systematic taking of temperature to judge of the continuance of fever. This

systematic taking of blood-pressure he did not see practised in any of the French hospitals. It would save many lives. The general tendency of shock cases when once in the grip of shock is to die under the treatment, or the lack of treatment, ordinarily given to them at present; whereas, a treatment which demands no great experience, and requires only the intelligence of systematic attention to a few things, will save about four-fifths of the cases. The literature of shock for the last twenty years or so contains various conclusions, various ideas, which are either entirely erroneous or which are only partially comprehended. One of these ideas is that shock is due to exhaustion of the vasomotor centre. This is not the case, because it is shown that in profound shock the vasomotor centre still gives reflex change to blood-pressure on stimulation of the sciatic nerve far too great to be accounted for if the centre were exhausted. Moreover, it has recently been shown that in profound shock the superficial vessels are constricted, which would not be so if the vasomotor were exhausted. It is also not true that shock is due to stimulation of nerves of sensation. It can be shown that stimulation of any nerve for several hours at a time with strong currents can be made without causing shock. The speaker had stimulated almost every nerve in order to find nerves the stimulation of which would cause shock. In none of the cases in normal nerves was there a fall of blood-pressure; on the contrary, the general effect was to increase bloodpressure. It is, however, true that patients are found in such a state that slight stimulus will produce a condition resembling shock. An example is reported in the ANNALS OF SURGERY of an interesting case cited by him twelve years ago in which a woman with cancer of the breast, with enlargement of the glands in the lower part of the neck and axilla, had suffered for many months and had had morphine and was in a highly nervous state. During the operation it became necessary to put a clamp on one of the cut nerves of the brachial plexus, when the woman fell into a condition thought to be shock. He did not believe that that was shock, but inhibition of the heart. He held to that belief because previously the same clinical picture has been produced by experiments which he did and in which the animal had morphine. The reason for the state was that the patient had become sensitized. That conception of sensitization is new. It is based upon good experimental observations. The first of those observations which he had ever recorded is in the second chapter of the first volume of the Journal of Experimental Medicine, in a paper upon "Ligation of the Coronary Arteries," which was the second paper on this subject, the first having been written by Welsh and Flexner. In it are reported a large number of cases, results of ligation of the coronary artery in dogs. If the artery is large there occurs sudden arrest of the heart with fibrillary contractions apt to be fatal in the dog. The interesting point here is that there were several series of these experiments and in all of the series but one dose was given of morphine and cocaine; that following ligation of the left coronary artery in 50 per cent, of cases there was arrest of fibrillation. A second ligation

caused arrest in 85 per cent. of the cases. In other experiments made with pure ether the morphine and cocaine had so sensitized the heart that a stimulus otherwise innocent caused a fatal arrest of fibrillation. Years later he found in studying the tonus of the heart muscle another state of sensitization. In experiments recently published in the Journal of Experimental Medicine upon pneumonia, tracings from the heart muscle were shown from dogs that had died of pneumonia. In these dogs it is found that at the end of three and one-half hours tonic contraction has taken place to an extent equal to the ordinary contraction of the heart. In experiments to determine the effect on blood-pressure of stimulating the sciatic nerve a rat received morphine and cocaine. When about to stimulate the sciatic nerve to obtain the vasomotor reflex we found upon lifting it that the heart "fell down"; there was inhibition, frequent and feeble beat which lasted three-quarters of an hour. Warm saline solution was given, and the nerve was again lifted when the heart fell into the same state, remaining so for half an hour. With more warm saline solution the rat recovered and the experiment proceeded in the ordinary way.

If you make a survey of the diseases in which the arteries are affected, in certain migraines in which there is prolonged restriction of the fibrillary vessels, you will see that patients subject to migraine get into a state in which a little extra fatigue or other stimuli which under ordinary circumstances will produce no such effect bring on the prolonged spasm and all indications of the disease. It is obvious that sensitization of the heart leading to prolonged inhibition is a factor in the production of shock.

Then there is the hydrostatic fall, a fall which can be illustrated by an artificial scheme. The best example of a hydrostatic fall is seen in cases in which novocaine has been given carelessly. The sudden fall of blood-pressure does not last long and is rarely fatal.

The question of hemorrhage is not well understood. In the loss of blood there is a critical point below which you can take but a small quantity of blood without dangerous symptoms appearing. Just here is another point deserving of attention: The amount of blood necessary to carry on nutrition of the tissues varies with the state of nutrition in those patients. If the metabolic factor or the chemical operations of the tissues are diminished or impaired, then obviously more blood will be necessary to nourish those impaired tissues than if these elements were at their normal level. Consequently, the blood which can be lost is a variable quantity. Hemorrhage *per se* is not a cause of shock.

These are the principal sources of confusion with regard to the cause of shock. When the speaker first went abroad in 1916 to study at the front the cause of shock, he was informed by the surgeons that shock was seen oftener in fractures of the thigh and in multiple wounds through the subcutaneous tissues. His personal experience bore this out. It has been found that the blood of such patients contains large quantities of fat. It is also known from experiments on animals and examination of tissue of human beings that fat embolism takes place in this condition. Fat emboli have been found both experimentally and after shock in the human subject; after fractures they have been found especially in the lungs, brain and other organs. When he came back from France he tried to find a chemical substance which might be absorbed by bone marrow; it occurred to him that fat embolism might be the cause of shock. He demonstrated this by injecting $3\frac{1}{2}$ c.c. of neutral olive oil into the jugular vein of a cat.

He had taken pains since acidosis was considered in connection with shock to inquire of several persons supposed to know more about the subject than anyone else in this country. The result of that inquiry was that he should not quote them personally but that they believed that while there was no doubt that even in early stages of lowered blood-pressure the alkali reserve is diminished in the blood there is a doubt whether the amount of diminution observed in the ordinary case of complicated shock is of great clinical importance. That doubt is a very complicated matter resting upon matters which he personally is not competent to discuss. The question is whether the reduction shown by the test in the alkali reserve really does mean acidosis in the cases in which it has been said to mean acidosis.

As practical men it is necessary that we should not leave any reasonable chances untried. He, therefore, inquired what it was best to do under the circumstances. It was their opinion that where acidosis was suspected it was advisable to draw the urine from the bladder, then to administer from 3 to 5 grammes of sodium carbonate properly diluted and after an hour to test the urine reaction. If the urine were found to be alkaline no acidosis was present; if it were still acid the probability would be that acidosis was present. It is the opinion of these gentlemen that the sodium bicarbonate is not without certain potential dangers.

In the practical handling of a case of shock you should in the first place take the diastolic blood-pressure. The maximum blood-pressure is of uncertain value in shock because the arteries are well filled, the heart beat frequent and feeble, and therefore the maximum blood-pressure is impaired more than 15 mm. The diastolic pressure is a much more certain sign and it is also very easy to take. The test is of greatest importance because shock is above all a disease in which the understanding of a critical condition is vital to the saving of the patient. In shock there is a level to which the blood-pressure sinks above which level the patient is likely to recover. Below that level there is a vicious circle which results in the patient's death. That level he had called the critical level in shock. This level depends somewhat upon the instruments used. He would say that sixty was this critical level; that with a level of from 55 to 50 the tendency is to die; that with a level of 65 the tendency is to get well with almost any sort of treatment.

To summarize: the patient in shock must not be washed. He must not be put into a soft bed; he must not be given normal saline solution indiscriminately without any record of the blood-pressure; the blood-pressure must not be guessed at, but must be measured every fifteen minutes. It

takes one and three-fourths minutes to make a reading of the diastolic pressure. The patient must not be warmed up in bed and then taken out of the bed without a blanket on an ordinary stretcher, carried 500 feet uncovered and given chloroform. If he does not die it is because he is young and vigorous and not because he has not had ample opportunity at the hands of the surgeon. All of these things he had seen done repeatedly. At the head of one of the hospitals was a distinguished professor of surgery. He said he was glad to see him, that all the shock cases were dying. After he had been there ten days five of the cases recovered. The only difference in treatment was that the things enumerated were corrected. The method finally worked out in France has proved to be a good one. When a case comes in it is taken to a little room canvased off from the part of the tent in which the operations were done. The patient is put upon a slanting table so that the feet are 30 cm. higher than the head. The blood-pressure is taken every fifteen minutes and if at the critical level normal saline is given with adrenalin. If this does not keep the pressure up carbon dioxide is given. The clothes are not removed but cut away round the wound. Chloroform is never used. Whenever possible local anæsthesia is employed. Operation is performed as rapidly as possible and if advisable carbon dioxide is given during the surgical procedure. The patient is wheeled back fifteen or twenty feet and the expert in charge of the case must remain with him for an hour. These are the principal points in the care of the shocked case.

There are a good many cases in which none of the efforts heretofore employed in shock will raise the blood-pressure. In those cases something must be done. It occurred to him that it might be of value to use the respiratory pump. In the use of carbon dioxide we get any type and rate of pressure we like. The result of the increased action of the respiratory pump is to carry much more blood from the veins into the slightly emptied arteries and to the heart which is not properly filled. The inevitable result is that in two or three minutes from the beginning of treatment the pulse is much stronger and the blood-pressure is up 10 to 12 mm. It can be raised 30 mm. in animals and fifteen in man. If the blood-pressure fall to the critical level it makes a great difference in the prognosis. He would suggest to utilize this extremely simple method or improve upon it. He did not maintain that fat embolism is the general cause of shock, nor did he maintain that carbon dioxide respiration is a heaven-sent remedy which will remove all danger from shock. It is of advantage in shock, but the real point in regard to treatment is to make systematic effort based upon repeated measurements instead of risking the patient's life by pure guess work.

DR. H. A. HARE said that the air hunger referred to by Doctor Porter has apparently been taken by many persons as an evidence of toxæmia, meaning that the respiratory centre has been stimulated by the B. oxybutyric acid or that there is an effort of Nature, by deep inspirations, to oxidize the B. oxybutyric and diacetic acids into harmless acetones. He would not be surprised if it was found that this view is entirely erroneous. He was inclined to think that part of this deep respiratory effort which occurs under these circumstances is Nature endeavoring by the respiratory apparatus to help the heart pump; and Doctor Porter in his remarks dropped a word or two which seemed to give some emphasis to this point. He had repeatedly seen cases of pneumonia in which he had been convinced that the cough of the patient which was thought to be annoying, and which the physician tried to stop by the administration of opiates, was really an effort on the part of the respiratory mechanism to assist the heart in pumping venous blood. Deep inspirations and forcible expirations alter venous pressure, and venous pressure is one of the things which has gone wrong in this extraordinarily interesting condition.

In regard to vascular dilatation, the fundamental conception of shock held during the last thirty odd years is that taught by Horatio C. Wood that shock was due to vascular palsy, is due to an accumulation of blood in the great veins. It is only within the last few years that surgeons have employed atropine to prevent the accumulation of blood in the great veins, although those of us engaged in clinical medicine have long regarded it as one of our great aids.

When Doctor Porter referred to the exposure of the patient to cold in the transfer from ward to operating room he was reminded of an incident observed many years ago in London in experimental work upon the brain of monkeys. The monkeys were brought in clad in little red dressing gowns and kept warm by hot water bags. Inquiring the reason for this he was told that if this were not done the monkey died of shock during the operation. In the afternoon of the same day he saw Victor Horsley remove a large brain tumor from a woman clothed only in a nightgown and lying on a one inch glass-topped operating table. He wondered whether the vital resistance of the monkey and the difficulty of its maintaining heat had any bearing on human surgery. The maintenance of heat on the one hand and the presence of excessive heat on the other are points all too frequently ignored. We often see patients brought into the operating room with the blankets rolled up and hanging across the base of the neck; the belly exposed, with no recognition of the fact that it is the heat citadel of the body. As soon as the belly is opened the cold is felt there. The chairman will remember that many years ago we carried on a series of experiments upon animals in which we showed that the introduction of fluids of various temperatures into the peritoneal cavity produced a most extraordinary variation in blood-pressure. Fluids a little below the normal temperature of the animal caused profound drop in blood-pressure. These same fluids applied to the wall of the abdomen or introduced high up in the bowel, where they circulated in the same area except that the bowel wall was between them and the peritoneal cavity, caused no variation in blood-pressure. Heat, therefore, according to these experiments is of considerable importance.

In regard to the saline question; he was interested many years ago in

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having a demonstrator of physiology standing beside him who, when he injected a considerable quantity of fluid into the animal, expressed great surprise that the blood-pressure did not rise. There is nothing more fictitious than the idea that such injection of fluid is followed by rise of blood-pressure. You can inject three and four times the equivalent of the total amount of the blood of an animal without causing a rise in blood-pressure.

Doctor Porter did not refer to the hypertonic solutions or to the use of the viscous fluids. If there is one function of the body about which it is most jealous and most cautious it is the maintenance of the normal chemical constitution of the blood. If you give a man with healthy kidneys 30 grains of any potash salt three times a day he will have no symptoms, the kidney will eliminate the potash and maintain the normal condition of the blood; but if the man has renal disease the potash becomes a poison. The moment you put in a pint or quart of strong bicarbonate of sodium solution, unless you are going on the principle that the symptoms are due to acidosis and that you are able to neutralize the acidosis as in a test tube by adding alkali, you are putting into the blood stream a preparation of sodium which Nature never intended should be there. Personally, I believe that little will come of this new acidosis treatment in shock. I do not believe that acidosis has anything to do with shock. Doctor Porter said that the degree of acidosis was so small that it did not produce symptoms. Doctor Hare's conception is that the individual is bleeding to death in his own vessels and that there is an accompaniment of loss of nervous equilibrium. He believed that in the problem of shock there is a condition exceedingly complex in Nature in which certain factors in certain cases are dominant and in other cases other factors are dominant, and that it is not a single pathological state or a proposition as simple as we see in the consolidated lung of croupous pneumonia.

DR. JOHN B. ROBERTS said that about thirty-five years ago he wrote in the first volume of "Holmes's System of Surgery" (which Doctor Packard edited in this country) on shock, and showed the similarity of symptoms of fat embolism and those of surgical shock. He had not, however, seen much reference to this fact in surgical literature, until he read what Doctor Porter has recently written. Surgeons knew that shocked patients seemed to revive considerably under the effect of ether anæsthesia, when we formerly amputated limbs as soon as reaction seemed moderately well established. Indeed, in those earlier days hypodermic use of ether was employed to some extent as a remedy for shock. See "Holmes's System of Surgery," American edition, 1881, vol. i, p. 145. The distinction made then was that socalled "delayed" shock might be fat embolism. May not Doctor Porter's cases have been examined by him after shock had been replaced by fat embolism?

DOCTOR PORTER closed the discussion, saying that with the history of blood-pressure, he had noted that several times in the history of fat embolism a condition simulating shock had been seen. If, however, these citations are examined it will be found that in no case is there anything like the surgical shock as seen on the field of battle. His contribution consists of a method of measuring low blood-pressure in conditions resembling shock and he had presented certain facts of which he was sure. He did not personally know about the use of gum acacia mixed with saline solution. It must be tried with caution. It is not necessary to have the blood-pressure remain up for a long time; these patients are just on the edge between life and death; if you can bring them back to life for ten minutes at a time you will probably get them through. If, therefore, you have a remedy by which you can raise the blood-pressure again and again, try it, and nurse the patient past this critical point.

With regard to opium, the surgeons in France who had seen a good deal of this condition of shock were in the habit of giving opium whenever the patient was restless. He himself believed that it was a good thing, but his experience in connection with the circulation is that it is never safe to speculate. The blood-pressure can be raised even in a normal individual by carbon dioxide inhalation. So, when he is asked to explain how it is that fat embolism produces shock he frankly says that at the present moment he had no definite information regarding it and would be incapable of offering a hypothesis.