clot being derived from the heart, and that death was not due in any way to the derangement of the circulation in the lower extremities, or its sudden return. The embolus was approached by the abdomen rather than retrogradely by way of the femoral vessels because the physical condition of the patient was satisfactory for the abdominal route.

Subsequent Progress.—The patient stood the operation well, and pulsation was evident in both popliteal arteries and the posterior tibials the evening after the operation. She seemed very toxicæmic, and the pulse was quick, though it slowed down to about 80 per minute the same evening. She died rather suddenly at 9.20 a.m. the following day, from a simple failure of respiration and heart, without any further incident such as might point to pulmonary emboli or embolic lesions in other organs. A post-mortem examination was performed the following morning.

Externally was seen a recent abdominal incision which appeared to be a healthy abdominal operation incision. The thyroid contained three adenomata, and was moderately enlarged. The tracheal and bronchial glands were small and pigmented. The lungs were healthy. The heart was obviously dilated. There was an increase in subpericardial fat. The right auricle was much dilated and the tricuspid valve admitted three fingers easily. The cusps showed chronic thickening. The coronary arteries were normal. The mitral opening admitted only one finger, and the cusps showed evidence of old sclerotic endocarditis with a superimposed recent acute endocarditis. Multiple small vegetations, with a small blood clot still adherent, were seen. Both cusps were affected. The aortic valve was competent, and the cusps showed very slight patchy thickening; the right posterior cusp showed a tiny vegetation. The abdominal aorta, which had been exposed and incised at operation, was found to be closed efficiently; there was no leak anywhere. On opening the aorta and its iliac prolongations there was found to be a very small adherent clot, and below in the iliac, especially the right external iliac, there was an adherent clot. The internal iliac was clear on this side. The veins appeared to be clear—this includes the popliteal

femoral, and iliac. The right popliteal artery contained a clot, probably post-mortem. The aorta was notably small.

Brain.—Pia-arachnoid: there was a diffuse hæmorrhage over the right occipital pole and the left frontal lobe. On section this was seen to be due to obvious recent infarction. Attached to the dura and tucked under the anterior pole of the right temporal lobe was a circumscribed mass which resembled endothelioma, although it might have been of circulatory origin—? infarction.

The spleen contained a recent large infarction in its upper pole. The upper pole of the left kidney contained a large infarction and evidence of an older one. The liver and other organs were healthy.

Comments.

The diagnosis was comparatively easy, owing to the lack of peripheral pulses in the legs.

This case also shows how it is possible safely to open the abdominal aorta and restore its integrity; and if the patient had lived long enough opportunity might have offered to study whether the wound of the artery would have led to any subsequent aneurysm or blocking of the vessel by later clotting.

The cause of death, in the light of the post-mortem findings, was evidently a sudden embolism which travelled towards the brain. There was no convulsion, but the patient died quite suddenly after appearing to rally from the operation. The loss of sensation very closely corresponded anatomically in its upper limit to the first lumbar nerve root on the surface; there was actual tenderness on pressure over the bifurcation of the abdominal aorta, and the striking feature of the case was the intense pain complained of, chiefly below and to the left of the umbilicus and in the left iliac region.

REFERENCE.

¹ *British Medical Journal*, 1925, ii, 985.

GLUCOSE: WHEN AND HOW TO USE IT.*

BY

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WHEN I was a child I read a book entitled *Madam How and Lady Why*. Its author, in ennobling "Why" and making "How" a commoner, symbolized a profound truth; it is one thing to know the workings of natural phenomena, but quite another to discover the reason which underlies those workings. My old professor of chemistry used to say: "Don't bother too much about *why* things happen. If you know how they happen, can make them happen, and can control them while they are happening, it does not much matter what theory you use to explain your results."

My subject to-day is the use of glucose—chiefly as an antidote to the shock of operation and anaesthesia—and the mechanism of its working according to McDonagh's theory of electrical and other changes in the body protein, which he expounds in his book *The Nature of Disease*.

McDonagh's theory seems to me not only to explain much that was obscure in pathology, but also to show many directions in which physicians, surgeons, and anaesthetists can advance in their use of the means they already possess. Its exact truth does not matter so long as it helps us to get better results, and this it undoubtedly does. Its substance is as follows:

If the blood serum of a healthy person be examined under dark ground illumination it shows a large number of little globular refractile particles in colloidal suspension, exhibiting Brownian movement. These are known as protein particles; they exist throughout all the tissues of the body, but those of the blood are the most convenient for examination. Each protein particle can be compared to a solar system; its sun is the protein nucleus, and its planets, in the order of their distance from the nucleus, are fats, amino-acids, urea and uric acid, sugar, inorganic salts, and electrons. The whole system is kept together by water, as is the solar system by gravity. It is always being attacked by hostile substances, whether chemical

products or micro-organisms, which tend to break it up. The most distant "planets," since they are the least closely attached, are the first to be split off. An invader, therefore, will strip off the electrons first; their electrical energy is dissipated in the form of heat and the body temperature rises. This appears clinically as fever. Then the salts are broken off; those which the plasma cannot hold are excreted in the urine. As the attack develops, the sugar, urea and uric acid, amino-acids, and, lastly, the fats, are reft from the protein nucleus and dissolved in the plasma, leaving the nucleus bare. In the end this itself fades away into true solution. Now as these planets are stripped away one by one, the water in which they swim is set free; the process is therefore called "dehydration." Some of the particles, however, undergo dispersion—that is to say, they subdivide to a certain point and there stop; there is a numerical increase and, in places where their volume exceeds that of the liquid present, the liquid and solid colloidal phases of the blood may be reversed. Instead of being a suspension of solid particles in a watery medium, it becomes a suspension of liquid globules in a solid medium; in other words, it "sets" like a jelly. This is called "gelation," and when it happens the patient suffers from intravascular clotting and thrombosis, which cause the clinical signs of cerebral sinusitis, hemiplegia, pulmonary embolism, and thrombosis, hyperemesis gravidarum, phlegmasia alba dolens, and a host of other conditions. Though gelation may occur primarily, it occurs more often secondarily, as a cyclical change of hydration. This explains why venous thrombosis is met with more frequently after operations and in the puerperium.

Now when the protein particles are being subjected to dehydration they do not follow one another like sheep. Only some of them are dehydrated—that is, split up into their constituent planets and driven into true solution, or dispersed; others resist the process, and they have only two means by which they can do this: one is to increase in size, the other is to agglutinate, or cling together. Since in both cases they take up water, they are said to become "hydrated," the opposite of dehydrated. When protein particles hydrate, their planets are at once drawn into a more intimate connexion with the nucleus and resist the more stoutly any attempt to split them off. Protein particles which have been hydrated tend to draw in upon themselves and work out their own salvation. Hydration is for the most part a defence mechanism

* Read in the Section of Anaesthetics at the Annual Meeting of the British Medical Association, Manchester, 1929.

evoked by a slow, insidious chronic invader like cancer, chronic tobacco poisoning, syphilis, tuberculosis, or alcohol, and chronically hydrated protein particles are very difficult to reduce to normal.

Hydration may, however, also be produced suddenly, as by a drug with a large nucleus like insulin, which produces a considerable lowering of surface tension. When this happens, the swollen or agglutinated particles may be precipitated from the blood stream in masses large enough to block the capillaries. This change produces the condition known as shock, which may be cerebral, pulmonary, or splanchnic, according to the viscus in which precipitation chiefly takes place.

ANAESTHESIA AND THE PROTEIN PARTICLES.

The behaviour of the protein particles gives a valuable guide to the anaesthetist, first in preparing the patient and secondly in averting the ill effects of the shock which is inseparable from all anaesthesia. One of the most interesting facts which appear from a study of the protein particles is that anaesthesia and pregnancy affect them in very similar ways. The essential features of any anaesthetic are that it shall produce simultaneously a sudden dehydration of some of the particles and a sudden hydration and precipitation of the rest, in exactly the proportion required to cause unconsciousness. Pregnancy produces the same effect, with the difference that anaesthesia may do in seconds what pregnancy does in months. If the proportion is wrong, the same symptoms occur both in anaesthesia and in pregnancy. Excessive dehydration due to an anaesthetic causes vomiting; that due to pregnancy causes hyperemesis gravidarum. In anaesthesia and pregnancy alike the processes of dehydration and dispersion may continue to the stage of gelation and cause some form of venous thrombosis, such as pulmonary embolism. Excessive sudden hydration, on the other hand, produces the familiar condition known as shock.

The Treatment of Shock.

Shock is caused by sudden hydration of the protein particles and their consequent precipitation in the capillaries and pericapillary lymphatic vessels, which they obstruct. If they block those of the brain they produce fits, convulsions, coma, and sometimes death; if they block those of the lungs, they cause pulmonary collapse; if they block those of the abdominal viscera, the result is vomiting and diarrhoea. The lowering of the body temperature in shock is due to the adsorption of heat by the hydrating protein particles. In the worst forms of shock the blood collects in the big veins, in less severe forms in the important viscera. In mild or prolonged shock congestion is selective and confined to one or a few of the important viscera. The congestion due to anaesthetic shock selects first the brain, then the lungs, and finally the liver.

The value of glucose before, during, and after operation lies in its power of dehydrating rapidly those protein particles which have been hydrated by the shock of the anaesthetic and the surgical assault. The process can be watched under the ultra-microscope. When glucose is run into a field containing a number of slow-moving hydrated or waterlogged particles, some of which are agglutinated, the effect is almost miraculous: the agglutination breaks up, and the bulky spheres are shattered in a few seconds into multitudes of tiny globules. The same change takes place in the body, and the clinical effect is to pull a badly shocked patient together in a few minutes. The temperature rises, the colour returns, sweating ceases, and the patient settles down into normal respiration. Transfusion of normal blood produces the same effect, and for the same reason: it dehydrates the particles which the operation and the anaesthetic have hydrated in excessive numbers.

It is obvious that in treating acute shock the first consideration is to give enough glucose at once, and for this reason steps have to be taken to guard against an overdose. If exactly the right amount were given it would all be absorbed by the time the hydrated particles returned to normal, but in practice it is impossible to gauge the correct dose. Glucose is therefore given intravenously in large doses; the particles are dehydrated or dispersed, and the symptoms of shock pass off. But the protein particles, though they are brought back to normal, will not remain so,

because they are unstable. They have lost their electrons, their furthest planets, or else they would never have become hydrated. They therefore proceed past the normal stage into dehydration and dispersion; they are broken up into their component planets and greatly increased in number, and, if the process is not checked, gelation and thrombosis occur. There are two ways of checking the dehydrating action of glucose so as to avoid thrombosis. One is to give fifteen to twenty units of insulin, a powerful hydrator, about two hours afterwards, when the glucose has exerted its maximum effect. This prevents too many particles being sent into true solution by the glucose. The other method is to give a "conductor," or a substance which will supply the particles with the electrons they need and stabilize them. Conductors are of no value when hydration or dehydration have progressed far, but are very valuable in the initial stages, when only electrons are lost, and also when particles are passing through the normal phase on their way from one abnormal phase to the other. One of the most effective conductors is S.U.P. 36, or, to give it its full name, the symmetrical urea of para-benzoyl-para-amino-benzoyl-1-amino-8-naphthol 3:6 sodium sulphonate. S.U.P. 36, like insulin, should be injected on the first sign of recovery. Oxygen acts like glucose when given subcutaneously, but it is not so potent. It is therefore usual to give intravenous glucose with subcutaneous oxygen in severe cases only, but in mild ones to neglect the glucose and merely give 500 to 1,000 c.c.m. of subcutaneous oxygen. Oxygen acts so mildly that there is no fear of dehydrating the protein particles excessively; insulin is therefore unnecessary.

PREPARATION OF PATIENTS FOR ANAESTHESIA.

Chronic alcoholics, late-stage syphilites, and patients with malaria, trypanosomiasis, cancer, and other chronic ailments are notoriously difficult to anaesthetize. There are two reasons for this. The first is that their protein particles have been subjected to a long and increasing process of dehydration, and there are not enough normal particles left to undergo the chemico-physical changes necessary to bring about anaesthesia. The second reason is that many of the protein particles left in the colloid state have undergone secondary hydration, and the first effect of the anaesthetic—namely, dehydration—upon these enlarged particles is to cause them to undergo dispersion. The normal process of anaesthesia cannot take place until the hydrated particles are broken down and then subjected again to hydration by the anaesthetic. These patients therefore need preparation for some days beforehand with intravenous injections of colloidal iodine, and intramuscular injections of sulphur in the form of contramine, the carbon disulphide compound of di-ethyl amine.

Purging and starvation should be, and rightly have been, relegated to the limbo of barbaric things, as they exhaust the glycogen reserves of the liver, the patient's natural defence against shock, and produce the very condition which the anaesthetist is trying to avoid. It is always desirable, especially in the bad risk case, to examine the patient's protein particles several days beforehand. If hydration is present a course of intravenous colloidal iodine and of intramuscular contramine should be given; the night before the operation glucose should be injected intravenously, together with subcutaneous oxygen. The colloidal iodine and contramine act slowly, and in obstinate cases may require several weeks to produce the desired result. The glucose is given slowly, at the rate of about half a litre an hour. It is stored in the liver, and given out as required to correct excessive hydration due to operation or anaesthesia.

POST-ANAESTHETIC TREATMENT.

If most of the particles are dehydrated after operation (and this is not likely until the third day or later), the patient should be given insulin, but it is important not to give insulin if many particles are hydrated, for the sudden increase in hydration which it produces may cause fatal shock. The clinical signs of post-anaesthetic gelation are a rise in temperature and vomiting, cyanosis, or fits, according to whether thrombosis takes place in the splanchnic vessels, lungs, or brain. If the disturbance is in its initial stages and the temperature is high, S.U.P. 36 should be exhibited, but if two or three days have elapsed

since the operation and the patient is still vomiting, insulin should be given. When post-anaesthetic hydration is feared, intravenous glucose and subcutaneous oxygen should be administered rapidly, any excess of glucose being excreted by the kidneys. Pulmonary hydration is especially likely to occur after prolonged anaesthesia in a bad subject if the patient has recently received an injection of a substance with a large nucleus and high molecular weight, such as intravenous arsenobenzene or S.U.P. 36. Glucose and oxygen should therefore be given to all patients who are under treatment for syphilis.

A rough and ready test for determining whether the vomiting is due to splanchnic shock—caused by the precipitation of hydrated protein particles in the abdominal viscera—or to dehydration of protein particles, is to draw off some blood in a syringe. If the blood flows readily and is bright red the protein particles are probably hydrated, but if it flows with difficulty, clots rapidly, and is dark in colour the particles are probably dehydrated. It is most important to determine which condition is present, as the wrong treatment will only accentuate the disorder it is desired to remedy.

THE ORTHODOX ATTITUDE TO GLUCOSE.

Surgeons have recognized the value of glucose without, for the most part, understanding its action. The general tendency is to focus attention on ketosis, which is one of the by-products of shock, and to counteract it by giving glucose in the belief that it replaces the large quantities of sugar consumed in the state of shock or under anaesthesia. The empirical surgical attitude, which is correct within limits is well represented in Barrington-Ward's book *The Abdominal Surgery of Children*. The author rightly eschews purgatives and enemata, and provides the child on the day before the operation with a full diet and a bottle of barley sugar. A light meal and more barley sugar are given on the morning of the operation. When the operation is severe or the patient's resistance appears to be low, a 5 per cent. solution of glucose is given subcutaneously during the thirty-six hours before operation. Older children receive a 10 per cent. solution of glucose in water per rectum. In very anxious cases transfusion is performed and salines are injected during the operation. For the treatment of post-operative ketosis Barrington-Ward gives 10 per cent. glucose per rectum every four hours, or 5 per cent. glucose intravenously, preferably by Matas's drip method. His remarks on alkaline treatment are very interesting from McDonagh's point of view. Quoting the work of Brown and Graham, he says that sodium bicarbonate cannot be depended upon to reduce the acetone in the blood and urine, but is beneficial in producing a rapid return to normal of the low alkali reserve. Glucose, on the other hand, he remarks, causes a rapid diminution of the blood and urine acetone as well as a rise in the alkali reserve, and arrests the formation of ketone bodies. While advocating large doses of sodium bicarbonate as an adjuvant, he definitely gives it second place. The reason for the success of glucose is, of course, not that it substitutes missing sugar, but that it goes straight to the root of the trouble and dehydrates the protein particles. This is proved by the fact that other chemicals which cannot replace sugar, such as di-hydroxy-acetone and adrenaline, produce the same result.

THE TECHNIQUE OF GLUCOSE ADMINISTRATION.

Glucose solution, to exert its full effect, must be used within twenty-four hours of its preparation. I always use and recommend a 10 per cent. solution at a temperature of 100° F. Before an operation it must be given slowly, or it will rapidly be excreted by the kidneys. When it is administered slowly any excess is absorbed by the liver as glycogen and let loose into the blood stream in the form of glucose as required. If the patient's protein particles are already hydrated, the glucose can be given faster than when it is merely desired to increase the glycogen reserves.

The use of glucose at operations can be summarized as follows:

A. Always give glucose before a severe operation: (1) when the liver efficiency is suspected—for example, where there is a history of jaundice; (2) when the metabolic rate

is high—for example, in Graves's disease; (3) when the patient is undernourished or emaciated.

B. Always give glucose after a severe operation when a blood transfusion is impossible.

C. Give glucose after any anaesthetic: (1) where loss of blood has been heavy and blood transfusion is not practicable; (2) where the patient shows signs of shock—cerebral, pulmonary, or splanchnic; (3) where glucose has not been given before the anaesthetic; (4) where a rough surgeon has operated, or where it has been necessary to use more than the usual amount of anaesthetic; (5) where there is a history of epilepsy.

TRAUMATIC RUPTURE OF THE STOMACH.

BY

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In the *British Medical Journal* of July 27th Mr. F. Hector Scotson published two very interesting cases of rupture of the stomach alone, from external non-penetrating violence, which, owing to absence, I have only just seen. That this condition is very rare is evidenced by the literature on the subject from British sources which he has so ably collected.

It may be of interest to quote some extracts from foreign literature on the subject before recording a case under my care in 1927.

Rehn¹ in 1896 collected 13 cases, of which only 2 were uncomplicated. Only 2 recovered, including Rehn's own case.

A. Schwarz² in 1909 reported one fatal case in which both stomach and duodenum were completely torn through.

A. Piancastrelli³ in 1922 stated that only 22 cases of traumatic subcutaneous rupture of the stomach could be found in literature. In his case, the twenty-third, which proved fatal, the abdominal wall under the skin was ruptured and the stomach almost divided in half.

G. Ipsen⁴ quotes a case of his own in the same year, in which the rupture was in the pylorus, and the patient recovered.

H. Neuberger⁵ quotes a fatal case of his own in which there was also injury to the pancreas. He is of opinion that the grave prognosis in these cases is due to concomitant injury to the ganglion coeliacum, which lies dorsally to the stomach.

From this it will be seen that only 9 cases were of rupture of the stomach as the only viscus, and that only 3 recovered.

My own case, like those of Mr. Scotson, showed no signs of external injury, and was not complicated by injury to any other viscus.

A seaman, aged 29, was admitted to the Royal Naval Hospital, Malta, on January 27th, 1927, with a history of having fallen from a ladder and been struck in the lower abdomen by a piece of iron projecting about 12 feet below. He had had a full meal about one and a half hours previously. The patient, a very small, spare, and well-developed man, was groaning and doubled up. There were signs of shock, and the pulse was 92. There was no sign of any injury to the abdominal wall. The abdomen generally was held rigid, but was not board-like, and the patient sat up to take off a tight garment. There was no dullness in the flanks. The liver dullness was present, and there was no hyperaesthesia to pin reflex. Pain was referred to the lower abdomen and radiated down to the testicles. Clear urine had been passed twenty minutes before examination. He was suffering from marked shock, and groaning, with intervals of quiet.

In the absence of any marked physical signs I decided, wrongly as it turned out, to wait for further signs; after five and a half hours the pulse rose to 120, there were distinct signs of tympanites, and the leucocyte count was 15,300. He was therefore given a general anaesthetic, and the abdomen was opened by a right central paramedian incision. The peritoneal cavity was found to be full of turbid fluid. On swabbing this out it was found to be coming from the right upper quadrant. The incision was therefore extended upwards. A hole with everted mucous membrane, and easily admitting the forefinger, was found on the anterior surface and near the greater curvature in the pyloric end of the stomach. Its appearance was almost identical with the picture of this condition in Walton's *Surgical Dyspepsias*. There were no signs of any ulcer present. The gall-bladder was covered with a lymphatic exudate. The liver edge was bruised but not torn. There were no signs of injury to any other viscus. The burst was sewn with Lembert over primary deep sutures, peritoneal toilet performed, and a drainage tube was placed in the under surface of the liver.

The patient was almost pulseless at the end, and nearly died of shock, although the operation did not take long, owing to the patient's favourable build. Severe shock lasted for twenty-four hours, but was overcome by intravenous glucose saline. He