SOME REMARKS ON CHOREA.

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CHOREA is a disease possessing numerous points of professional interest—nay, so unique are its phenomena that we must not even dare to call it a disease, but only a symptom of some "functionally disordered nerve centre." In the milder forms, as generally observed, it readily yields to treatment or ceases of itself, but now and then it is met with as a terrible disease, running a rapid and fatal course, uncontrolled by any treatment at present known; and notwithstanding that much careful investigation has been given to it, its etiology and pathology are still obscure. Then, again, we have the alleged close connexion between chorea and acute rheumatism, or rheumatism generally, and between chorea and endocarditis affecting especially the cardiac valves irrespective of rheumatism, doubtfully admitted by observers; and, lastly, the statement that in a few weeks the disease will pass away without the help of drugs of any kind, which is corroborated by daily experience. These questions, I hold, invest chorea with peculiar interest, and they will receive due attention as I proceed.

Etiology.—Chorea is peculiarly a disease of childhood, is observed much more frequently in girls than in boys, and is seldom met with before the age of six years, and rarely after sixteen. Like other neuroses, it appears to be inherited; at least the disease is found in connexion with families who are the subjects of epilepsy, hysteria, &c. It is said to be more prevalent in large towns than in the country, but this is questionable; at all events the disease is so common in some parts of the country that I am disinclined to believe there is any proof of this statement. On the other hand, it is certainly more prevalent in some localities than in others. A low state of health, in whatever way brought on, as by want of sufficient and proper food, general neglect, any severe disease of any kind, as acute rheumatism, scarlatina, typhoid fever, diarrhœa, and impure air, or bad sanitary conditions; all of which things tend to impoverish the blood and give rise to anæmia and general malaise, may predispose to the disease, especially in children whose nervous system is more or less unstable. Then some ignorant lout of a boy acts the ghost, or puts on a hideous mask, and suddenly starts up before these timid and weakly children, filling them with unutterable terror. In other cases they are chased by a dog or a bull, fear a beating at school, or are ill-treated by a schoolfellow, or by their parents, get into a crowd, or are excited by some sight or catastrophe, or fall from a boat into a river,—in these and similar ways, being already predisposed, the system receives a shock, and the result is often at once the beginning of chorea. Nor is this any exaggerated or imaginary picture, for very many striking instances of fright having caused the disease are on record. It is, indeed, stated that two-thirds of any given number of cases of chorea can be traced either to fright or to something nearly allied to fright. Thus, "pursued by a drunken man," "sent for a policeman to quell a family disturbance," "frightened by a drunken father," "locked up in a dark cellar," "frightened by a dog," "frightened by seeing her brother in flames," "frightened by being caught hold of by a man in the dark," are instances in which the disease immediately followed the assigned cause; and, as has been observed by a recent writer, "such instances afford good ground for supposing that the same cause has really been in operation in other instances when a greater interval has elapsed." The brain-pressure of many studies in delicate and half starved girls in our Board Schools is said to be one potent cause of the disesse. In some cases chorea is said to result from imitation; but in the many cases we have seen in hospital practice no such instance has been observed to occur.

Rheumatic fever is regarded as an important factor in the causation of chorea, and in fatal cases it is stated that endocarditis and its results, as vegetations similar to those observed in fatal cases of acute rheumatism, are universally found on the cardiac valves. It is also an accepted belief that these diseases frequently occur in the same individual,—e.g., a child who has had chorea falls ill with rheumatic fever a few years afterward, and *vice-versâ;* or, again, slight choreic movements appear in the midst of a rheumatic attack, or some rheumatic affection of one or more joints in the course of chorea; thus showing that in some mysterious way there exists a close connexion between these two diseases. This point will be resumed when we deal with the pathology of chorea. Dr. Broadbent observes that "in adults pregnancy divides with rheumatic fever the causation of this affection"; but more especially may it be said, the shame and anxiety of pregnancy in unmarried women and girls. Bad habits and intestinal worms, both in adults and children, have been named as causes of the disease, and certainly cases are on record which got well on the expulsion of the latter from the intestines.

Symptoms.—The symptoms in a fairly typical case of chorea are striking and definitive, and when they have been once seen, can never be forgotten. We observe numerous movements, singular and grotesque, giving rise to contortions of the countenance often absurd and ludicrous, with sudden, rapid, and irregular muscular actions, sometimes unilateral, sometimes bilateral, and sometimes even less general. The arms, legs, and head may be jerked about or twisted, when any action is attempted, in the strangest and most erratic manner—as if, indeed, almost all control of the muscular system was lost. She (it is generally a female) is highly excitable, and when she is spoken to, the symptoms are all exaggerated, the tongue is thrust out suddenly and as suddenly withdrawn, and when told to walk she drags her legs, and hops and jumps, as if at play or making fun. It is almost impossible to feel the pulse at the wrist, nor in many cases is it easy to examine the heart. And the term which has been applied to this state of the muscular (or nervous) system—namely, "insanity of the muscles"—is by no means inappropriate; for as "in delirium there is loss of control over the mental processes with rapid succession of incoherent ideas, so in chorea there is loss of control over the motor apparatus, with movements excessive in point of number and extent, but wanting in vigour and precision" (Dr. Broadbent). She is careless and forgetful, or slovenly and unsteady, in any work she may attempt, low-spirited, and excitable. Dr. Hilton Fagge sums up the symptoms in a terse, though perhaps a too brief, definition, when he observes: "There are chiefly two—inability to keep at rest while awake, and incapacity for performing voluntary movements with precision"

(To be continued.)

SAPRÆMIA IN BURNS.

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IN severe and extensive burns the patient frequently rallies from the immediate shock caused by the burn, but dies on the second or third day after the injury. Death is said to be due to the reactionary, remote effect of the shock. Mr. Erichsen, in his "Surgical Practice," vol. i., page 374, writes: "On the subsidence of the symptoms of depression, there is usually a period of quiescence before reaction comes on"—i.e., the increased sensibility of the nerve terminals is followed by diminished sensibility (exhaustion), during which period the patient suffers little. If the burnt surface is large, or the resisting power is slight, exhaustion soon sets in, and terminates fatally. Usually when death occurs at this stage, either the patient becomes restless and cyanosed, finally sinking, or drowsiness supervenes, gradually deepening into coma and death. It is at this period that the absorption of dead, irritant material seems to cause the fatal issue.

The lesions found after death at this stage are: "Serous effusion into the cerebral ventricles, congestion of the stomach, intestines, as well as the substance of the lungs." The congestive character of the inflammation might perhaps be explained by the nerve exhaustion following the injury, but there are symptoms which seem to be caused by the absorption of dead and damaged tissue. Immediately after the injury there is no obstacle to the absorption of such material. This absorption the granulations prevent at a later stage. In the superficial parts of granulation tissue there are no lymphatics, the current of fluid and the movement of the cells being from within outwards. If there is pressure on the granulating surface, this current may be stopped, or even reversed. The reversing of the natural outward direction of the current and its effects are best seen in

fetid ulcers of the leg. The discharge may be most offensive and septic, but so long as it escapes freely, the temperature is normal, and the patient suffers little. If pressure is now made on the surface of such an ulcer, so that the outward current is interfered with, or the granulating barrier is broken through so that septic matters find their way into the system, a considerable rise of temperature and marked constitutional symptoms follow. After cauterising living tissues absorption takes place. This is best explained by considering what actually occurs when a vascular part is seared by the cautery. The damaged part may be described in three zones—(1) The part most affected by the heat, which, if sufficiently great and long-continued, drives off the water from the tissue, which it disorganises, forming a crust of carbon; (2) more remote from the injury the tissues are in a state of inflammation, the arteries and veins are plugged with clots, the charred inner coat being curled up into the lumen of the tube, while the outer coat forms with the charred mass a cap over the burnt end of the vessel. The lymphatics in this area are less securely occluded. Although the lymph clots, it does so feebly; the lymphatic walls, containing less elastic tissue, retract and contract less than the walls of the bloodvessels. From this it follows that damaged cells readily find their way into the lymph circulation. (3) Outside this area of stasis, or plugging of the bloodvessels, over a considerable area an active hyperæmia or over-fulness of the bloodvessels occurs. This condition allows increased leakage and diapedesis from the distended arterioles and capillaries, while it makes the part feel hotter and look redder than natural. That the flow in the lymphatics from a damaged area is greater than normal is seen by the increased flow from the distal end of a cut lymphatic, which drains the lymph from the injured part. This increased flow depends partly, no doubt, on the increased leakage and diapedesis through the walls of the dilated vessels, but solid particles are also absorbed and swept into the lymph-current. That this is so is shown by the readiness with which solid particles of iodoform and opium are absorbed by a recently burnt surface, producing constitutional effects. To sum up briefly, it may be fairly assumed that before the formation of granulation tissue increased absorption of fluids and solids occurs. After the granulations have formed, a barrier to further ingress is made, the natural direction of the flow of liquid and the diapedesis of cells being from within outwards.

That the charred material and damaged tissue are sufficient, quite independently of any septic element, to cause a rise of temperature, is very probable. Perfectly aseptic blood-clot, introduced into the circulation, causes a rise of temperature. It may be shown, after simple fractures or large contusions, that there is always a rise of temperature, which is proportionate to the amount of damaged tissue and the absorbing power of the lymphatics and bloodvessels. In fractures of bone the rise of temperature is greater than in injury to soft tissue, probably because the bony walls of the vessels prevent their collapse, and thus allow a ready means of absorption. Again, the rise of temperature is greater when a large than when a small bone is broken—e.g., it increases with the number and size of the vessels torn across and the amount of blood extravasated. All this indicates that aseptic dead material in the body acts as an irritant, the amount of irritation, as indicated by temperature, being proportionate to the amount of damaged tissue and the absorbing power of the parts beyond the damaged area. In burns we have a quantity of dead and damaged tissue lying in a position which makes its absorption easy, and this material, even if quite aseptic, is just the kind of substance which would act as an irritant, when circulating in the blood current. That the "congestive symptoms" of burns are due to irritant absorption seems probable from the following considerations. The temperature immediately after the burn falls to 97° or even 95°F. This depression continues for twenty-four to forty-eight hours, after which there is a gradual rise for the next twelve hours, the temperature rising to 104° or even 106° F. This rise of temperature, which is in proportion to the amount of damaged tissue and the absorbing power of the surface, closely resembles the rise due to absorption of aseptic clot, as it follows immediately on the depression, and is gradual, but continuous, in its increase. The temperature having reached a maximum, remains up, subject to slight diurnal variations, until about the eighth day, when the granulations form a barrier. The pulse-beats during the reactionary period increase in frequency and force. In some of the fatal cases the mode of death is very suggestive of pulmonary thrombosis. There is extreme restlessness, dyspnœa, and lividity. A cardiac murmur, basic systolic, is heard; but with a heart beating irregularly and rapidly, a murmur is difficult to detect, and, if present, might be due to irregular or incoördinate action of the heart muscle. In other fatal cases the patient sinks into a comatose state, which gradually deepens, until death supervenes. The mode of death closely resembles narcotic poisoning. The suggested explanation of these different ways of dying is this: In both cases there is absorption of dead and damaged material, which in some cases causes thrombosis of the pulmonary artery, analogous to pulmonary thrombosis, which sometimes occurs after simple fractures; in other cases the irritant paralyses the centres of the circulation and respiration, resembling narcotic poisoning by ptomaines or in koprostasis.

These observations seem to have an important bearing on the treatment of burns. In all cases of severe burns the temperature should be taken frequently, and carefully watched. If it rises above 103° F., it has reached a dangerous point, as death may follow in one or other of the ways described. Measures should be at once adopted to check the absorption by submerging the burnt part in a cool bath of some non-poisonous, antiseptic fluid. By this means all the loosened tissue is washed from the injured surface, and any pressure relieved, while the cooling effect of the bath lowers the temperature. In one case (burns of chest and abdomen) so treated with a bath of weak sanitas, which was lowered from 90° to 70° F., in twelve minutes the child's temperature fell from 105° to 102°. The whole appearance of the patient changed; from being drowsy and stupid he became bright and lively. This change was no doubt partly due to the cooling of the body surface. When the temperature rose a second time, the child was again bathed, with a result that it did not rise again, and the child recovered. How much benefit was due to the general cooling effect of the bath it is difficult to say; but when one considers that the dressing presses the dead tissue over the mouths of the lymphatics, while the movements of a restless patient pump the lymph along the vessels, it cannot be denied that an occasional or constant submersion in water has many obvious advantages.

SUPPRESSION OF URINE IN A CASE OF SINGLE KIDNEY, WITH AN UNUSUAL MALFORMATION OF THE GENERATIVE ORGANS.

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THE following case is, I believe, absolutely unique, and I have therefore reported it somewhat in detail. For permission to publish it I am indebted to Dr. R. T. Smith and to Mr. Reeves, and for the post-mortem notes to Dr. Dalton.

M. A. M——, aged twenty-six, married five years (no children, but two miscarriages, the last five years ago), was admitted to the Hospital for Women, Soho, on Oct. 14th, 1890, complaining of pain in the epigastrium and vomiting. She stated that she had always been perfectly well until shortly after her marriage. Her illness then began with an attack of "inflammation of the womb," which kept her in bed for three months. At that time she had great pain in her lower abdomen, worse on the right side, but she had no trouble with her urine. She had had this pain ever since, and it had been worse during the last two years. For three years she had been subject to pain after solid food, accompanied by vomiting. The pain was in the epigastrium, came on immediately after food was taken, and was followed by vomiting in a few minutes. The vomited matter was often streaked with blood, but the patient had never had an attack of hæmatemesis. Melæna had, however, been noticed on one or two occasions. She had been losing flesh for twelve months.