

Original Articles.

A CONSIDERATION OF SOME OF THE INDICATIONS FOR OPERATION IN HEAD INJURIES.¹

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THE subject of the indications and contra-indications for operations in head injuries is so large that it will be possible to cover only a small portion of the ground this evening, and I shall therefore confine myself to presenting to you certain points which seem to me to have not yet been appreciated at their full value by the profession. For the purposes of this paper it will be necessary to limit ourselves to the consideration of some of the indications for *immediate* operation in cases of head injuries in *adults*. By immediate operations we understand such as are performed within at least twelve hours of the time of the injury. As a rule, the operations under consideration are to be performed within as short a time after the injury as possible.

The indications for later operations, that is, more than twelve hours after the injury, differ very considerably from those for immediate operations, and are, on the whole, much easier for determination. These we will not consider to-night.

The symptoms on which we rely in order to decide on the advisability of immediate operation in cases of severe head injury may be roughly divided into four groups: (1) extracranial or cranial local symptoms, (2) intracranial local symptoms, (3) extracranial general symptoms, (4) intracranial general symptoms.

(1) The direct local symptoms, those which are immediately due to the injury and are perceptible to the surgeon. Such are wounds, ecchymoses, evident fractures of the cranium and all other local and evident conditions due to direct injury in any part of the body, which have any bearing on the diagnosis or prognosis. These are *local extracranial* or *cranial* symptoms.

(2) The intracranial or indirect local symptoms. In this group I would include all localized symptoms which are to be referred to intracranial conditions. Such are the various forms of paralysis, partial or total, of local spasms and certain conditions of the pupils. Symptoms of this group point more or less distinctly to affections of special portions of the intracranial organs, and so far as they do so are *localizing*.

(3) The third group is a somewhat indefinite one. It includes such general symptoms and conditions as may exist after any injury without special relation to intracranial conditions. Under this heading I should place the general strength or weakness of the patient and the condition of the pulse. I have called the symptoms of this group the *extracranial general* symptoms.

(4) The intracranial general symptoms. This includes all general conditions presumably due to intracranial conditions. Such are the states of cerebral activity or of cerebral repose; consciousness, semi-consciousness, unconsciousness, coma and delirium. These may be reasonably referred to intracranial conditions caused by the injury and may be fairly considered as the *index* of these conditions.

The direct external symptoms are too well known to every surgeon to render their discussion valuable. The localizing symptoms, though often of great value in combination with other symptoms, in themselves rather determine *where* we shall operate than *when* we shall operate.

I propose this evening to pass over the symptoms which I have placed in the first three groups, and devote myself principally to the consideration of those general symptoms indicative of intracranial conditions which we have mentioned in the last group.

It is largely from the general mental condition of the patient, meaning by this term those states or conditions which we have before enumerated, consciousness or unconsciousness, delirium, quiet or irritation, that the indications or counter-indications for immediate operation in head injuries are in many cases to be drawn. We will now, therefore, consider the indications for and against immediate operation in cases of severe head injury in adults where the external signs and the localizing symptoms do not exist or do not afford sufficient indication.

The *primary* indication for immediate operation in severe head injuries is *increased intracranial pressure*. The question of operation in these cases depends on the degree of this pressure.

Whenever it has reached a certain point operation is imperative, unless otherwise contra-indicated. This point is the condition of deep stupor where the patient cannot be roused by supraorbital pressure. When, however, the unconsciousness is deep, but the patient can still be roused by passive movements or in other ways, the indication is less definite. Yet in all such cases, and they form a large proportion of those entering the large hospitals, we must be largely guided in our action by the depth of this unconsciousness.

Cases of this kind, as a rule, grow more unconscious during the first few hours, so that if we wait we may often be obliged to act rapidly later. On the other hand, the lighter cases of unconsciousness, which do not tend to become more unconscious, should not be operated upon at once. In any doubtful case the most careful watch should be kept for any increase in the degree of unconsciousness, and as soon as this has been thoroughly determined operation should be performed.

In all cases, whether only lightly or deeply unconscious, where there has been a rapid increase in the depth of unconsciousness, or where there is a distinct increase of paralysis of the extremities within the course of a few minutes or hours after the injury, operation should be performed. Gradual increase of paralysis and gradual increase of unconsciousness becoming apparent shortly after a head injury, suggest intracranial hemorrhage, usually middle meningeal, and demand immediate surgical interference in all persons under forty years of age. In persons much above this age, in cases where the external injury is slight or doubtful, the question of the presence of slow hemorrhage from some of the deep vessels of the cerebrum (ingravescent apoplexy) must be considered.

When together with unconsciousness there exists cerebral activity or irritation, as evidenced by delirium, it has usually seemed to me more advisable to wait and not operate at once, although some of these cases have fatal terminations. On the whole, I consider delirium as rather a sign for delay than for immediate action. Where delirium without unconsciousness exists, it con-

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tra-indicates operation or indicates that we should not operate at once.

Localized cerebral irritation, as evidenced by clonic convulsions, either general or local, is not a common sign of head injury (surgical). In cases where there is no history, convulsions render it probable that the case is non-traumatic, or that some ordinarily non-traumatic condition has been set up by the trauma. These are the results of clinical experience.

It would seem scarcely needful to state that in each individual case we must consider the special symptoms and indications. In those cases in which there is no evidence of paralysis, and no pupillary symptoms exist, we rely on the general condition of the patient. The important signs are two: (1) the depth of unconsciousness, (2) the increase of unconsciousness.

(1) It is hard to define exactly the degree of unconsciousness at which—there being no special contraindication—operation is absolutely demanded. In most of the more doubtful cases we have some secondary symptoms (fracture, paralysis, pupillary conditions) to guide us. Where these do not exist, we must rely to a certain extent on the general appearance of the patient, his pulse, apparent strength and respiration. If his strength is failing but he can still bear operation, operate at once. As a rule, we should advise operation in any case where the patient (adult) could not be roused by supraorbital pressure and where the pupils did not react to light.

(2) When the unconsciousness, light at first, rapidly becomes deeper, especially if this be accompanied by any commencement or increase of paralysis, we should at once suspect intracranial hemorrhage, usually middle meningeal.

Where an unconsciousness, deep from the beginning, becomes slowly and very gradually deeper, without an increase of paralysis of the face or limbs, we can only diagnosticate an increase of intracranial pressure, and cannot determine whether such increase be due to hemorrhage or not.

What now are the pathological conditions existing in these cases? I believe that wherever the conditions of lasting unconsciousness, stupor and coma exist, we have to deal with an increase of intracranial pressure. This conclusion is based on the results of many operations and observations.

The cause of this increase of intracranial pressure is not altogether plain. It is *not by any means*, as is sometimes supposed, *always* a pressure from intracranial hemorrhage. In fact, I am inclined to believe that pressure from this cause is much less frequent than is supposed. It takes a considerable quantity of blood to produce from outside the brain substance an active pressure on the brain, even when the pressure is produced rapidly.

In many cases, moreover, on operation we have no evidence suggestive of any severe hemorrhage, and yet the increased pressure is apparent. Again, this increased pressure in all probability occurs in the cases of so-called concussion, and in other mild cases where unconsciousness exists but where there can be no question of any profuse hemorrhage. What seems to occur is this: The brain in some way acts as a sponge, and swells and pushes so hard against the dura as to inhibit or diminish pulsation. If in these cases the dura is incised, the cerebral pulsation again becomes visible, and the relief to the patient is instantaneous and extraordinary.

And here let me state that after a tolerably large experience in these operations I have never seen a case in which the incision of the dura has caused the slightest injury to the patient, or has apparently caused any untoward symptoms whatever. The cause of this brain swelling I will not discuss to-night, because it is a difficult subject, not well understood, and, moreover, has immensely broad bearings in various directions. It occurs undoubtedly in certain cases of apoplexy and, in a chronic form, in many intracranial diseases. This or something analogous, the so-called acute edema of the brain, is the immediate cause of death in cases of acute alcoholism, of sunstroke and perhaps (in its chronic form) in uremia.

Let us now pass to certain surgical considerations in operations such as we are discussing. It is evident that unless the operation be of a suitable and efficient character, its value is likely to be much impaired and the result not conformable to our expectations. It is as important to know *how* to operate as it is *when* to operate or *where* to operate. It may seem a little presumptuous for one who is not a practical surgeon to attempt to speak on this subject, but I have for years followed these operations with care, and feel justified in laying down certain rules. These are:

(1) Be sure that the opening in the cranium is sufficiently large. In the early days of these operations, and to a certain degree even to the present time, there has been a tendency on the part of the surgeon in exploratory trephining or opening of the cranium to make his aperture too small.

This is a serious matter, both because it prevents the surgeon from seeing and from working inside the cranium at his ease, and still more because it prevents him from opening the dura sufficiently, and efficiently relieving the intradural pressure. I believe that in the ordinary case nothing smaller than a one-inch trephine should be used, and then the opening enlarged, with bone forceps, if possible. An opening, as a rule, to be efficient should be at least two inches by one. Of course it makes no difference how this opening is made providing that it is made as *quickly* as possible, with as little jar as may be to the patient and without laceration of the dura.

(2) The second point to be emphasized in these operations is always to open the dura where there is evidence of intradural pressure. There is no more danger in opening the dura under proper antiseptic or aseptic precautions than there is in opening any other serous cavity. The superstition that the dura was a structure which it was exceedingly dangerous to meddle with dies hard, and still lingers consciously or unconsciously in the minds of many surgeons and physicians. I can only repeat that having closely observed for a number of years a very considerable number of patients in whom the dura has been opened, either intentionally or otherwise, I cannot recall a single case *in an adult* where any essential harm was produced. In a very large proportion of cases there is neither any perceptible shock at the time, nor are any future harmful effects (of no matter how slight a character) perceptible.

(3) Remember that in these operations time is an important factor. Do what has to be done as rapidly as possible. In children many deaths are caused in these operations by loss of time. In adults the consequences are not, as a rule, so serious, nevertheless next to asepsis time is probably the most important

factor in the success of an exploratory cranial operation.

(4) In an exploratory operation, where it is not certain that a large extradural clot exists, look out for the middle meningeal artery. Remember that it runs in a deep groove on the inner surface of the cranium, and that in trephining directly over it, unless the dura be separated in some way from the cranium, the artery is likely to be cut by the trephine before the bone has been completely sawn through. Again, remember how closely the dura is or may be attached to the inner surface of the cranium, and that in cutting with bone forceps in the course of the artery or its branches, great care must be taken not to tear or cut the blood-vessel.

(5) It is perhaps scarcely necessary to mention that all cases of intracranial hemorrhage with which the surgeon has to deal (that is, all cases from superficial vessels, from sinuses, etc.) can be stopped by pressure. Ligating the vessel when possible is preferable, but sometimes it is not possible.

(6) The last point I wish to speak of is a somewhat doubtful one in my mind, and I only desire to call your attention to it. This is the length of time during which packing should be left in the intracranial cavity. I am inclined to think that the present tendency is to remove it too early.

Gentlemen: I thank you for your attention, and I hope that you will pardon my presumption in speaking on surgical subjects before a surgical society.

ACUTE GONORRHEAL RHEUMATISM.¹

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GONORRHEA with its various complications has, of late years, been the subject of much research by investigators all over the world. Much has been learned of the pathology and etiology of the various forms of the disease and of the life history of its determining organism, the gonococcus. We have been taught how to recognize the germs with the aid of the microscope, how to cultivate them artificially in all their virulence, and how to prove them to be gonococci by showing that inoculations of the urethra of dogs with the culture is followed by gonorrhea (Turró). Cultures of the germs have also been obtained from the fluids and tissues of various parts of the body afflicted by inflammation coincident with gonorrhea elsewhere.

The subject of this paper is one of the complications of gonorrhea which, though unfortunately far from rare, has until very lately been so misunderstood that many have even gone so far as to doubt its existence. Some believe that the disease is usually due to germs other than those of gonorrhea, and they are inclined thus to regard it as a pyemia. Doubtless there are instances of mixed infection; and the examination of a few such cases will go far to form an individual opinion. Writers and observers are not wanting who take the ailment to be of nervous origin, vaso-motor in character (Lewin). It is hard to believe that any one educated in the modern way can reach such a conclusion. If the disease were a pyemia, we should more often see other signs of pyemia, such as multiple abscesses. This complication is, however, a very rare one; and when it does occur, it seems to be the result

of an infection grafted upon the gonorrheal rheumatism, and not its cause or a coincident effect from a common cause. It is not in every instance practicable to give the "ocular proof" in this disease, but a good clinical diagnosis may nevertheless usually be made; and often in just such cases treatment is most needed, and the patients are most anxious for an opinion and prognosis.

My belief is that such a disease as gonorrheal rheumatism exists; that it is invariably caused by the gonococcus; and that in the most severe cases there is a group of symptoms so characteristic that one may repeatedly make a working diagnosis without even the corroborative evidence of coexistent gonorrheal urethritis. The discovery of gonococci in secretion or tissue is proof of gonorrhea; but the failure to discover them does not prove absence of the disease, especially if clinical symptoms be present. It has lately been learned that the germs may undergo changes of involution which result in forms of the organism quite unlike gonococci in appearance and in staining properties, and yet on culture and physiological test the usual forms are again evolved (see Tautou).

The part played by the male urethra in the production of gonorrheal rheumatism has, I believe, been overestimated, and the frequency of the disease in females, underestimated. In these days of accurate investigation cases are not wanting where other mucous membranes have been the source of the rheumatic metastasis, for example, the conjunctiva (Kammerer).

Gonorrheal rheumatism has been classified according to the tissue involved, whether joint, tendon-sheath or bursa; and it may be acute or chronic. It is not, however, my intention to enter here upon an exhaustive description of the malady in its various forms, but rather to confine myself to its more acute and serious aspect from a purely clinical point of view.

Acute gonorrheal rheumatism is usually described as a mono-articular inflammation, with considerable pain, but not so much pain nor constitutional disturbance as is noted in ordinary acute rheumatism (Keyes). It is my belief that no inflammation causes more exquisite and demoralizing pain than the one under discussion. I have in several cases been able to guess correctly the nature of the trouble from this one symptom, and the history of its persistence for more than a few days. This, too, in spite of the wilful misrepresentations of the patients. Not long ago I saw a young man of nineteen who had been for about four weeks confined to his bed with an arthritis of the right knee. He had lost much flesh and strength. His temperature had been high (104° F.), but for some days had been below 101° maximum. There was great swelling of the knee, with evident disintegration of the joint. The entire neighborhood was boggy and semi-fluctuating. The gentleman in charge was an orthopedic surgeon, and I was called to give an opinion as to the advisability of excision of the knee. On approaching the patient I saw that he was completely demoralized. He shrieked with pain at the lightest touch, and had to be anesthetized to be properly examined. His physician had made up his mind that bone disease existed, and suggested that it had perhaps gone so far as to demand amputation. My diagnosis was gonorrheal arthritis, with destruction of the joint. This opinion I held principally on account of the peculiar character of the pain. The patient insisted that he had no gonorrhea, and this in spite of a suspicious

¹ Read before the "Warren Club," December 4, 1894.