THE PRESSURE SIGNS OF CERTAIN INTRACRANIAL CONDITIONS OBSERVABLE IN THE FUNDUS OF THE EYE.

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THE fundus of the eye, and particularly the retina, being an offshoot of the brain is most intimately connected with the brain and the intradural cavity so that any lesion within the intracranial cavity which increases its normal content would naturally tend to be shown in the fundus of the eye, especially about the optic nerve head; that is, unless the normally free communication within the vaginal sheath between the intradural cavity and the optic papilla is obstructed by local disease, adhesions, etc., we should expect the signs of increased intracranial pressure to be observable in the fundus of the eye, particularly about the optic disk; again, an increased intracranial pressure sufficient to retard and even prevent the normal return flow of blood in the retinal veins would also tend to cause a dilatation of these retinal veins and the usual condition resulting from their dilatation and congestion.

The effect of an increase of intracranial pressure upon the fundus of the eye can be very easily demonstrated in its various stages by the experimental production of an internal hydrocephalus in dogs. During the past eighteen months, the condition of hydrocephalus was successfully produced in nine puppies of the age of ten days to two weeks; by means of a

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Suboccipital exposure, a small gelatine capsule filled with cotton is inserted into the aqueduct of Sylvius so that the cerebrospinal fluid could not escape from the third and lateral ventricles; a resulting hydrocephalus with dilatation of the ventricles and therefore an increased intracranial pressure occurred so that it was possible with the ophthalmoscope to observe the changes in the fundus of the eye due to this increase of intracranial pressure. Within five to six hours after the insertion of the cotton plug into the aqueduct of Sylvius, in each one of the puppies the retinal veins gradually became dilated; apparently the veins over the nasal half enlarged earlier and possibly more than the veins over the temporal half of the retinal fundus; this congestion and dilatation of the retinal veins was the first sign indicative of an increase in the intracranial pressure. Within two to four hours later, the nasal margin of the optic disk would become blurred, then the temporal margin, then the nasal half and finally the temporal half of the disks would become obscured, so that within a period of twelve to twenty-eight hours following the production of a definite increase of intracranial pressure, the details of the optic disks could no longer be observed; in four dogs the edema of the optic disks was so great within this period of time that a measurable swelling could be observed with the ophthalmoscope and thus the condition of "choked disks" could be applied; in the other five dogs, the measurable papilloedema occurred from twelve to twenty hours later. In one dog this condition of internal hydrocephalus with high intracranial pressure was not relieved by a subsequent drainage operation, and it is interesting to note that definite signs of a secondary optic atrophy began to appear nine weeks later; the remaining eight dogs were all drained through a subtemporal operation with six linen strands being inserted into the ventricle in order to relieve the condition of internal hydrocephalus; in all but three of the dogs, the increased intracranial pressure was relieved and it was most interesting to note that the subsidence of the choked disks was in just the reverse order of their occurrence, that is, the measurable swelling of the papilloedema first disappeared, then the blurring of the temporal half and then of the nasal half of the optic disk; then the temporal margin and later the
nasal margin appeared—though slightly blurred in all of the puppies; that is, the persistent dilatation of the retinal veins and some blurring of the nasal margin of the optic disks indicated that the intracranial pressure had not been entirely relieved by the operation. In the remaining three puppies the optic disks remained entirely obscured—only the measurable swelling disappeared, so that in these dogs the operation of drainage to relieve the condition of internal hydrocephalus was not successful.¹

This experimental work has been most instructive to me regarding the mechanical factor in the production of papilloedema and the condition known as "choked disks." There exists at present much confusion in the terminology of retinal conditions especially in their relation to intracranial pressure; the terms papillitis and papilloedema have been used interchangeably, and "choked disk" most freely and carelessly. Naturally, the terms papillitis, retinitis, and optic neuritis imply a condition of inflammation of the nerve head and the retina. Inflammation (in its modern conception) is due to toxic and infective causes alone, so that in the condition of nephritis, diabetes, and the various forms of meningitis, the term papillitis would indicate a retinal change due to some toxic or infective cause, whereas the blurring and oedema of the details of the optic disk due to an increase of intracranial pressure in purely mechanical conditions, such as an intracranial tumor mass and hemorrhage, could be termed as a papilloedema; and if a measurable oedema, then a swelling of the disk up to the stage of "choked disk." In order that a choked disk occur it must always be preceded by a series of blurring of the details of the optic disk, so that these early oedematous blurrings of the optic disks have been termed the mild or early stages of papilloedema or even a choking of the disks. If it is believed that intracranial pressure alone does cause in the fundus of the eye an inflammation (using the term inflammation in its modern sense as being due to a toxic or infective cause), then the term papillitis is perfectly proper although the etiological factor in its production is pressure, but I do feel that the modern methods of examination of the cerebrospinal fluid, and especially its cell count, whereby any

¹ A detailed report of this experimental work will be published later.
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Inflammation and infectious condition within the cerebrospinal canal and also its projecting connections, such as the optic vaginal sheath, can easily be demonstrated,—that these methods should disclose the infective character of the cerebrospinal fluid if that is a cause of the so-called papillitis in the purely pressure cases. It has been frequently demonstrated in the body tissues and elsewhere that pressure can and does cause congestion and œdema, but not inflammation (in its modern sense), unless infection is present; pressure upon tissue renders that tissue more susceptible to inflammation by infection,—a predisposing cause,—but pressure in itself does not mean an inflammation. I believe it is rare for retinal hemorrhages to occur in cases of choked disks due to even extreme intracranial pressure alone, whereas in conditions of neuroretinitis, optic neuritis, and papillitis due to toxic causes such as diabetes and nephritis, these hemorrhages occur very frequently. However, if we must take it for granted that the retinal and optic-disk changes in conditions of intracranial pressure are due to both pressure and toxic factors,—possibly the pressure producing the toxic appearance of inflammation,—then the condition of blurring and œdema of the details of the optic disk would precede the stage of papillitis, and if the papillœdema became measurable to two or more diopters then the term “choked disk” should be applied. In discussing the pressure signs observable in the fundus in the following intracranial conditions, I shall use the terms blurring and œdema of the details of the optic disks to indicate the earlier stages of increased intracranial pressure, and its later stages of measurable swelling of the optic disk by papillœdema, and the “choked disks” in the conditions of extreme intracranial pressure where the papillœdema is greater than two or three diopters. These signs of increased intracranial pressure as exhibited upon the fundus of the eye have been checked up and confirmed by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by a spinal mercurial manometer, so that when there is observed a blurring or œdema of the margin on the nasal half of the optic disk in a patient following a head injury, or in a patient in whom a brain tumor or brain abscess is feared, or in a child following a difficult labor and convulsions occur so that an intracranial hemorrhage has occurred, it should be a clue to the presence of increased intracranial pressure.
rhage is suspected, then it is very important to record accurately the pressure of the cerebrospinal fluid at lumbar puncture; if the pressure of the cerebrospinal fluid too is shown to be increased and thus the ophthalmoscopic findings are confirmed, we can then reach a more accurate diagnosis of the intracranial condition. Naturally, in normal fundi blurring and mild obscuration of the details of the optic disk occur and are considered as being within physiological limits; especially is this true in myopia, but if this obscuration of the details of the optic disks is observed and then the measurement of the cerebrospinal fluid is performed at lumbar puncture by means of a spinal mercurial manometer (the most accurate method now known to record the pressure of the cerebrospinal fluid) we are thus enabled to exclude those cases of so-called normal blurring of the optic disks.

There are certain intracranial conditions that frequently produce definite pressure signs observable in the fundus of the eye. I should like to speak briefly regarding these pressure signs of the following intracranial conditions and in this order: brain tumor, brain abscess, intracranial hemorrhage and oedema following fractures of the skull, selected cases of cerebral spastic paralysis due to an intracranial hemorrhage at birth, and lastly the condition of hydrocephalus.

I. We all know the condition of choked disk and, if not relieved, its subsequent secondary optic atrophy in patients having the signs of intracranial tumor. Naturally for a choked disk to occur, there must be high intracranial pressure, and I believe it is rare in cases of brain tumor for a choked disk to result unless the tumor has become of very large size, or it causes a blockage of the ventricles, and thus produces an internal hydrocephalus, such as the posterior mid-brain tumors and the subtentorial tumors and cysts. It is comparatively easy to make the diagnosis of brain tumor at this stage of papilloedema, and I feel that if these patients had been examined ophthalmoscopically early, then the more mild pressure signs observable in the fundus resulting from the smaller tumor mass would have been ascertained and the patient thus spared an impaired vision, if not blindness itself. The stage of choked disk must naturally be preceded by the earlier and milder stages of disk blurring and papilloedema.
and should therefore be recognized as being more significant than being within physiological limits. An interesting syndrome ophthalmoscopically is that of certain frontal tumors which may in their growth produce by direct pressure down upon the ipsilateral optic nerve a primary optic atrophy, and as the result of the increased intracranial pressure there is observed in the opposite fundus a choked disk—the forerunner of a secondary optic atrophy.

II. In brain abscess, there is a replacement and substitution of brain tissue by the purulent detritus, and thus, as in gliomatous tumors which infiltrate and replace brain tissue rather than push it aside, it is rare for definite signs of intracranial pressure to be observed in these cases, unless, as has been stated before, the ventricles are blocked, or a toxic and infective meningitis occurs from the presence of the abscess; this latter condition is of frequent occurrence in subtemporal and cerebellar abscess, but it is rare for temporo-sphenoidal abscess—the most common location of brain abscess following the usual cause—an otitis media—to produce fundal changes even though the abscess may reach the size of an orange and even larger.

III. Intracranial hemorrhage and edema following fractures of the skull with or without gross brain injury rarely produce a measurable papilledema to the extent of "choked disks." The reason for this is obvious: unlike brain tumor, hydrocephalus, and the other intracranial conditions which enlarge slowly and thus permit the brain and particularly the medulla to adapt themselves to this increased pressure with little immediate risk, in many cases of traumatic intracranial hemorrhage and edema in the absence of marked shock the intracranial pressure rises most rapidly, so that the compensatory mechanism of the medulla has little time to adjust its vasomotor and respiratory centers to this increased pressure, and the result in these cases is death before the development of "choked disks" is possible; if these patients could survive

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1 Only too frequently is the surgically successful removal of the brain tumor possible, and yet the patient has already been irreparably damaged by the non-recognition of its pressure signs until it is too late for a normal person to be obtained.

2 The Laryngoscope, March, 1914.
this greatly-increased and rapidly-produced intracranial pressure, then a measurable papilledema and "choked disks" would occur. In brain injuries following fractures of the skull, "choked disks" do occur in the cases of large hemorrhage which have formed slowly, usually the extradural hemorrhage following a rupture of the middle meningeal artery. In these cases of intracranial pressure of comparatively slow production, the medulla can adapt itself to the pressure, and thus death does not occur before a measurable papilledema and "choked disks" are possible.

Within the last month, I have operated upon such a case; the "choked disks" of four diopters developed five days after the basal fracture; at operation, both an extradural and a subdural hemorrhage of large amount were removed; the subsidence of the measurable papilledema began immediately after the operative drainage of the intracranial hemorrhage, so that fifteen hours later there was present only an œdema and blurring of all the details of the optic disks but not a measurable swelling of the disks.

It is most rare in these cases of brain injuries with or without skull fracture for an œdema of the optic disks to appear within six hours following the trauma, and particularly is this true of those patients in the various degrees of shock; the greater the shock, the less the blood pressure and naturally even though a large intracranial sinus or vessel was torn yet it would be difficult for any extensive hemorrhage to occur; just as soon as the intracranial pressure equalled this lowered blood pressure, then no more bleeding could occur because the intracranial pressure would now be equal and even higher than the blood pressure; as the patient rallied from the condition of shock, then naturally the blood pressure would become higher and then more bleeding would occur, and thus the signs of intracranial pressure, such as an œdema of the disk outlines, would now be possible. In these cases the marked signs of shock usually last about four to six hours; and again, those patients who cannot survive the condition of shock—they die within six hours after the injury.

In these cases of fracture of the skull\(^1\) it is not so important

in the treatment to know the site and extent of the fracture, particularly if it is a basal fracture, as it is to ascertain the presence of an increased intracranial pressure, both by careful and repeated ophthalmoscopic examinations, and also by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by the spinal mercurial manometer. If there are definite signs of a marked increase of the intracranial pressure, such as an oedematous blurring of the optic disks and twice the normal pressure of the cerebrospinal fluid and even more, and the medullary compression sign of a lowered pulse rate, then I believe an early relief of this increased intracranial pressure by means of a simple decompression operation is advisable before the patient reaches the dangerous stage of extreme medullary compression and even oedema and thus collapse of the medulla itself. If an operation is postponed in these cases until a "choked disk" occurs, then the ideal time for operation will have been lost, and it is then very doubtful whether the patient will recover; besides should the patient, having had high intracranial pressure, recover without an operation or at best a very late operation, then the danger of post-traumatic conditions is very great indeed; these conditions, due to a prolonged increase of the intracranial pressure, are persistent headache, an emotional instability of either the excited or the depressed type, mental and physical lassitude and easily fatigued, and in rare cases even epilepsy in its various forms.

I feel therefore in patients having head injuries that it is most important to establish the presence or absence of an increased intracranial pressure, whether that pressure is due to hemorrhage or cerebral oedema, and that repeated ophthalmoscopic examinations are of the greatest aid in facilitating an accurate diagnosis and the early treatment of the condition.

IV. In selected cases of cerebral spastic paralysis in children due to a hemorrhage at birth, the ophthalmoscopic examination is of very great importance in aiding the differentiation of this type of intracranial lesion from the other causes of cerebral spastic paralysis. It is very interesting to note that Mr. W. J. Little, in his first monograph in 1843 upon spastic paralysis, entitled "Deformities of the Human Frame,"

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or the now so-called Little's Disease, stated that the condition was due to an impairment of nerve tissues resulting from their lack of development and also to an earlier meningitis; a few cases, however, followed difficult labors, and undoubtedly these were in his opinion due to an intracranial hemorrhage at birth. In 1862 (nineteen years later), in his second monograph upon spastic paralysis, entitled "On the Influence of Abnormal Parturition, Difficult Labors, etc., upon the Mental and Physical Condition of the Child," he says that in his opinion almost 75% of these cases are due to intracranial hemorrhage. Recent investigation of this condition also confirms this belief that about three-fourths of these cases are due to intracranial hemorrhage at the time of birth.

In order to differentiate the three chief causes of cerebral spastic paralysis in children,—that is, first, a lack of development of the cerebral cortex or its pyramidal tracts; secondly, a meningitis and meningo-encephalitis following infectious diseases such as cerebrospinal meningitis, measles, scarlet fever, and whooping-cough; and lastly an intracranial hemorrhage, it is very important that a careful ophthalmoscopic examination should be made as early as possible; the measurement of the pressure of the cerebrospinal fluid at lumbar puncture with a spinal mercurial manometer should also be used to confirm the fundal findings of increased intracranial pressure or not. Naturally in cases of lack of development of cerebral tissues, there can be no increase of the intracranial pressure, and this is also true of those cases which have survived a meningo-encephalitis—a destruction and atrophy of cortical nerve tissue; on the contrary, if an intracranial hemorrhage has occurred, then there should be signs of an increased intracranial pressure as a result of the hemorrhage as shown by a dilatation of the retinal veins and an edematous blurring of varying degree of the optic disks; naturally, the earlier this examination is made, the more definite are the fundal signs of intracranial pressure, whereas in the older children the ophthalmoscopic examination may reveal only a dilatation of the retinal veins with thickened walls from new tissue formation and a shallow disk cup; while the disk itself

1 Obstetrical Transactions, vol. iii., p. 293, 1862.
is blurred in its details, particularly along the nasal margin, the temporal margin and even the nasal half of the disk may be obscured; in no case over one year of age have I found a measurable swelling of the disk to the degree of choked disk. If upon examining a patient having cerebral spastic paralysis the above fundal changes are noted, then the pressure of the cerebrospinal fluid should be measured at lumbar puncture in order to ascertain whether the fundal changes are due to a local condition within the orbit or are possibly within physiological limits; in the children within one week after birth there is usually blood in the cerebrospinal fluid at lumbar puncture, as was demonstrated in seventeen children upon whom I operated within three days after birth.

In my last report of 954 cases of cerebral spastic paralysis up to April 1, 1916, only 26% of them—that is, only one out of every four patients examined—showed these definite signs of an increased intracranial pressure, and the spastic condition was therefore due to a hemorrhage, and in these selected cases, by a cranial operation to relieve this increased intracranial pressure upon the brain, an improvement was to be obtained; at that time I had operated upon 219 children with a mortality of 16—that is, 7%. The history of these children is very suggestive: of the 219 operated patients, only 26 were not first children; only 8 were not full-term babies; only 21 were not born after a difficult labor, with or without instruments; only 49 did not have convulsive twitchings immediately after birth; and in only 21 children was the spasticity noticed before the eighth month after birth. A permission for autopsy is obtained before operation in every patient, both private and ward, and it is by this valuable means as well as by the operative findings, that the diagnosis is verified and other data ascertained.

V. The ophthalmoscopic findings in cases of hydrocephalus depend largely upon the type of hydrocephalus—whether it is of the internal type due to a blockage of the cerebrospinal fluid in the ventricles and thus producing ventricular dilatation and its resulting high intracranial pressure, or of the external type of hydrocephalus which is due to a blockage of

1 New York State Journal of Medicine, October, 1916.
the escape of cerebrospinal fluid from the general cerebrospinal canal; this latter condition rarely causes a measurable papilloedema, whereas the internal type of hydrocephalus can produce choked disks of extreme degree and its subsequent secondary optic atrophy. Fortunately, in little babies before the sutures have firmly united, the skull itself can enlarge and thus a natural compensatory “decompression” takes place so that the peripheral vision may not be impaired; a drainage operation, however, offers these children the best chance of approximating normality.¹

Besides the conditions already mentioned there are still other intracranial lesions in which an ophthalmoscopic examination is of the greatest importance; in cases of the various forms of meningitis, as an aid in differentiating the types of apoplexy, and a most important function in so many conditions—the presence of a negative fundus.

In conclusion, I may say that careful ophthalmoscopic examinations of the fundus are of the greatest importance in the differentiation of many intracranial lesions; that the signs of moderate intracranial pressure should be recognized, and that it should be realized that choked disks occur only as the result of high intracranial pressure; that the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer is the most accurate means of determining the intradural pressure; and lastly the intelligent use of the ophthalmoscope, especially the direct method, should be much more intensively studied in the medical schools and in the hospitals than it is at present.

¹ American Journal of Medical Sciences, April, 1917.