THE USERS OF OPIUM BY INJECTION

In regard to the patients treated who used morphin hypodermically, or opium by mouth, there is very little to say. They were by far harder to treat than the smokers, and their treatment presented no ideas other than those which have been reported in the past. They were gradually reduced in their daily dose and treated symptomatically. Only one matter of interest occurred during the treatment of these patients, and that was the results of our experimenting with the hyoscin substitution treatment. Fifteen patients who used morphin hypodermically were picked out for the experiment, and at 7 o'clock one evening were each given one-quarter of a grain of morphin and one one-hundredth of a grain of hyoscin. This first night the result seemed to be fairly good. The second evening the procedure was repeated, but not so successfully. Some of the patients were very restless all night, others seemed to be dazed, and four were delirious. Despite these facts, however, the dose was again repeated on the third night. Within fifteen minutes after the administration of the hyoscin, one-half of the patients had developed a wild, talkative delirium, wandering about the ward if not held in bed, absolutely oblivious to their surroundings, not recognizing their friends or hearing or heeding what was said to them. Of the remaining patients only two were entirely unaffected; two developed the most violent gastric symptoms, expressed by excruciating pains in the stomach and abdomen and most violent retching and vomiting; the breathing apparatus of one was so disarranged as to require artificial respiration; the rest suffered cardiac collapse with pulse that could hardly be felt at the wrist, one being affected to such an extent that I felt for a time that dissolution was imminent. Whether or not these demonstrations were due to an accumulative action of the hyoscin, to an idiosyncrasy for the drug which the Chinaman may possess, or what it was, it is sufficient to say that no more hyoscin was used on these patients, and I most thoroughly condemn its use as a substitute for morphin in treating this class of patients.

OBSERVATIONS ON CHOKED DISC

WITH ESPECIAL REFERENCE TO DECOMPRESSIVE CRANIAL
OPERATIONS *

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During the past four years we have been occupied with some experimental and clinical studies on the relationship between various intracranial processes and choked disc.¹

* Read in the Joint Meeting of the Section on Nervous and Mental Diseases, and the Section on Ophthalmology of the American Medical Association, at the Fifty-ninth Annual Session, at Chicago, June, 1908.

1. *Nomenclature.* We have chosen Clifford Albutt's term "choked disc" as a designation of all grades of the neuroretinal edema and its consequences which occur in association with states of intracranial tension, realizing that some are still inclined to accept von Graefe's division and to distinguish a condition of "optic neuritis" with its low grade of swelling from the so-called "choked disc" when the edema of the papilla reaches a distinctly measurable height—of 2 D. according to Elschnig. With the feeling that the terms "neuritis" or "papillitis" give an erroneous impression of the process, we have abandoned these designations altogether.

Though in the 3d edition of Gower's classical *Medical Ophthalmoscopy* (1890) the author, in collaboration with Mr. Marcus Gunn, evidently adheres to the inflammatory theories and though Mr. Gunn in his most recent discussion of the process (*Brit. Med. Jour.*, Oct. 26, 1908, p. 1126) has apparently been largely won

Our clinical experience is the outcome of studies made for the most part in the wards of the Johns Hopkins Hospital on about two hundred cases of brain tumor. In addition to these tumor cases there has been an equally large number of examples of what we regard as choked disc occurring in such diverse conditions as apoplexy, cerebral thrombosis and embolism, hemorrhage at birth, meningitis, hydrocephalus, nephritis, acute operative compression, postoperative edema, and the cerebral edemas which have accompanied cerebral contusion in cases of cranial injury. Unquestionably our experiences of greatest value have been acquired through the consecutive study of the often rapid alterations in the eyegrounds which usually follow the diminution of intracranial tension brought about through decompressive measures.

Our experimental investigations, undertaken in the Hunterian Laboratory, though still in progress, nevertheless suffice to justify the statement that through mechanical means alone we can not only produce an acute neuroretinal edema which will rapidly subside, but by a long-continued pressure can bring about a more lasting swelling of the nerve head possessing the histologic features which characterize this lesion in clinical cases.

It is our intention on this occasion, after a brief review of the present theories, to present merely a general statement of our views in regard to the etiology of the process, reserving for a future time a more explicit description of the experimental work; of the retinal changes which occur in tumor cases after decompression; of the relation between so-called albuminuric retinitis and choked disc; and of the effect of choking on the visual fields for form and color.

SUMMARY OF VIEWS CONCERNING THE ETIOLOGY OF
CHOKED DISC

Though the matter had been hinted at by Türck in 1853, Albrecht von Graefe seven years later was the first to fix the attention of the medical world on the relationship between encephalic disease and the remarkable changes in the eyegrounds—called by him *Stauungspapille*—which are disclosed by the ophthalmoscope. He was the first to offer an explanation of the phenomenon, albeit his conjecture that the process is attributable to a venous stasis brought about by compression of the cavernous sinus never received wide acceptance. It was opposed, for example, by Sesemann (1869) on the ground that the free anastomoses between the facial and orbital veins would prevent any such vascular stasis, and, indeed, was abandoned by von Graefe himself before his death.

The first opportunity for a reasonable explanation of the process was offered in 1869 through Schwalbe's important discovery of the intercommunication between the subvaginal space of the optic nerve and the intracranial meninges. Thus, independently of one another, Manz and Schmidt-Rimpler, as the result of the injection experiments, showed that fluid could be made to pass from the intracranial chamber into the sheath of Schwalbe, leading to a hydrops vaginalis with ampulla-

over to the view of its mechanical nature, he nevertheless suggests the term "tumor-papillitis" for the lesion when occasioned by a new growth.

The German *Stauungspapille*—which "choked disc" does not quite translate—is preferable to this, and George E. de Schweinitz has called our attention to Herbert Parsons' suggestion that *Papillædema*, a term introduced by Elschnig, be used, and we agree that it is the best designation as yet offered, provided it is recognized as including all stages of the process.

like distention of its distal end; and though appearances of inflammation might supervene in the course of time they attributed the chief rôle to the mechanical pressure of the distended sheath which led to an incarceration of the retinal end of the nerve with resultant edema of the disc.

This *Transporttheorie* of Schmidt and Manz met with opposition as well as support on the part of subsequent observers. It led in 1873 to von Wecker's suggestion that the sheath of the opticus should be opened in the treatment of choked disc and the strangulated scleral ring divided. Loring, in 1875, opposed the theory, while Panas a year later, as a result of the study of traumatic cases, upheld it. Hughlings Jackson was the first to report cases of tumor without choked disc, and it was difficult to reconcile this finding with the *Transporttheorie*.

Parinaud (1879) regarded an internal hydrocephalus, whether with meningitis or tumor, as essential to the appearance of choked disc. He asserted that an edema of the brain, which extended to the optic nerve, accompanied the ventricular hydrops, and he was unable, in rabbits, by simple intracranial injection of fluid, to produce a *Stauungspapille*. His views of a spreading cerebral edema have been upheld by Ulrich (1885), Siebriicht (1902) and Kampherstein (1904).

In 1881, before the International Congress in London, the inflammatory theory was propounded by von Leber, and the term "papillitis" has since been prevalent in English writings. He thought that in no stage of the process was there a true edema or venous stasis, but that it was due to some toxic irritant which found its way through the cerebrospinal fluid to the nerve-head by way of the vaginal sheath. It was therefore a matter of quality rather than tension or quantity of fluid. This view of a toxic neuroretinitis was strongly upheld in a series of papers by Deutschmann, a pupil of von Leber. Similarly, Galezowski, Brailey, Edmonds, and many others, have expressed the belief that in tumors there is an irritant toxin in the cerebral tissue itself which finds its way to the disc down the optic nerve.

Thus toxic or inflammatory, as opposed to mechanical theories, have up to the present day been the most strongly entrenched views, although other bizarre hypotheses have been offered by a number of writers. Among these may be mentioned Loring (1882), who proposed a view of trigeminal irritation with disturbances of trophic function; Ulrich (1887), who attributed the process to compression of the central vessels of the opticus resulting from an interstitial edema of the brain and nerve (Parinaud), a view supported by Deyl von Grosz, Yamaguchi, and others; Adamkiewicz, who regarded it as a neuroparalytic inflammation of the optic nerve centers and who asserts (1905) that artificial increase of pressure in the skull never leads to a *Stauungspapille*, but only to a stasis of the chorioidal veins; Benedikt (1868), Schweller and Hughlings Jackson, who ascribed the process to a reflex stimulation of the vasomotor nerves of the disc leading to inflammation.

The *mechanical* or Schmidt-Manz theory has received support from the experiments of Schultén, from Bramwell, from Oppenheim, from Hoche,² from Merz

(1900); and as a result of observation³ on the retrocession of choked disc after palliative operations by Sänger, Axenfeld, von Bruns, Paton, and a host of other recent observers.

The *inflammatory* or Leber-Deutschmann view has been upheld by Knies; by Gowers, who believes in a descending neuritis; by Elschmig, whose view (1895) that choked disc arises in consequence of inflammation, never from a pure edema, is upheld in 1901 when he adds that an increase of intracranial pressure can never be the cause: by Jacobsohn (1897); by Fleming (1904), who regards the nuclear increase as a certain evidence of inflammation; and by many others³.

Two things, it seems to us, have added immensely to our knowledge of choked disc: one the electric ophthalmoscope, which makes the routine examination of ill or recumbent patients a matter of ease, and the other the decompression operation, which has of necessity modified the views of many who heretofore have clung to the inflammatory theory of von Leber. Misconceptions as to the nature of the process are lamentable for the very reason that the matter rests in its chief import on a therapeutic basis. This is illustrated by the following quotation from a paper published in 1906 by a leading American neurologist:

The toxic factor in the production of neuritis has long been recognized and it is believed by many to be of importance in the production of headache, mental disturbances and other symptoms. Relief of pressure will, therefore, have little influence on any symptoms of toxic origin.

If our observations will modify, if not completely alter, views such as this, they will serve the purpose for which they are here set down—namely, to accentuate the importance of the mechanical factors. After all, it is possibly a mere quibble, for the physiologic chemist may claim that behind all edema there is some toxic element, and the pathologist, likewise, that the round-celled infiltration which appears sooner or later with new-formed tissue, leading to shrinkage and atrophy, indicates inflammation; but in spite of this it is the mechanical element producing edema which underlies these changes, and hence they demand a mechanical therapy. It is analogous to the effects of a tourniquet loosely applied about an extremity, which leads first to edema but in the course of time to infiltration, thickening and contraction of the tissues: on removal of the tourniquet the process will completely subside unless the induration has advanced to the stage of permanent scar-tissue formation.

EXPERIMENTAL OBSERVATIONS

In agreement with the experiences of Schwalbe, Manz and others, it was noted by one of us some years ago, while engaged in experimental observations on cerebral compression, that when fluid is allowed to enter the subdural space through a canula accurately screwed into a small trephine opening in the skull, a swelling of the optic discs will occur. The degree of tension necessary to induce this acute edema may be measured by inserting a mercurial manometer in the inlet tube between the pressure flask and the canula.

2. Hoche drew the interesting analogy between choked disc and compression myelitis of the cord which, as demonstrated by Schmaus, is not an inflammatory process but the result of a simple *Stauungsödema*.

3. Excellent summaries in regard to the causation of choked disc have been given by Th. Kocher in Nothnagel's *Specielle Pathologie und Therapie*, 1901, ix, 206, and by Alfred Saenger in the *Wiener med. Wchnschr.*, 1904, No. 47. Gower's *Medical Ophthalmoscopy*, of course, remains to this day the best monograph in English on this and allied subjects. References to the more important recent papers accompany Elschmig's chapter in Flatau, Jacobsohn and Minor's *Handbuch der pathologischen Anatomie des Nervensystems*, Berlin, 1904, ii, 1165.

When the fluid is allowed to enter the cranial chamber under a pressure in excess of venous pressure, venous engorgement will be apparent in the retinal veins, and soon a measurable elevation in the optic discs can be observed. If, by elevating the flask, the pressure is increased so as to nearly equal the normal arterial pressure there will result a marked engorgement and tortuosity of the veins with a high grade of swelling in the disc. Under these circumstances, if the optic nerve is exposed by a free opening of the orbit a marked distention of the vaginal sheath with the well-known ampulla-like dilatation of its distal end will be disclosed. If the fluid through which pressure is being made is colored the color rapidly makes its appearance under the sheath of the nerve.

In order to determine whether the retinal edema was a consequence of this fluid in the nerve-sheath under increased tension or whether it was due in part also to a stasis of the retinal veins, in certain experiments the nerve-sheath was ligated distal to the chiasm with sufficient pressure to obstruct the venous return without interrupting the circulation through the retinal arteries. Under these conditions, though the retinal veins became tortuous and engorged, no appreciable swelling took place in the disc. On the other hand, with removal of the ligature and the exertion of sufficient pressure on a portion of the dura exposed by a trephine opening over the hemisphere, the ampullated distention of the subvaginal space would quickly occur, and the disc promptly become edematous.

In brief, therefore, through the backing up of fluid under pressure—and it is indifferent whether this fluid be artificially introduced or be merely the normal fluid which has been crowded from the subarachnoid space into the vaginal sheath—a neuroretinal edema will occur, whereas venous stasis alone will not produce it.

These transient edemas are naturally difficult of histologic demonstration, inasmuch as the fluid is abstracted from the tissues in the early process of fixation. We have, however, been able, by a long continuation of pressure in an animal possessing a large subtemporal, healed, cranial defect, to reproduce the typical permanent lesion of a long-standing choked disc, with hemorrhages, round-celled infiltration and all, which was not only demonstrable with the ophthalmoscope, but which has later shown the usual histologic picture.

Animals may exhibit different changes in the discs, even under the same degree of fluid pressure: indeed, both discs of the same animal are seldom affected in like degree. It has been our experience, furthermore, that when choked disc has been experimentally induced in this way the edema first makes its appearance in the eye contralateral to the hemisphere over which the fluid is being introduced.

Our efforts to produce choked discs through the introduction of foreign bodies, with encroachment on the intracranial space after the manner of a tumor, have so far met with only a certain measure of success, doubtless owing to imperfect methods. Such a degree of swelling of the nerve-head as we have occasioned in this way has been much slower in making its appearance, has been less pronounced than when fluid was used, and rarely holds for any length of time. In these cases the disc in which swelling was first observed corresponds to the hemisphere in which, or over which, the foreign body had been introduced and thus was homolateral to the lesion.

We feel justified in the conclusion that a simple increase of intracranial pressure without any inflammatory or toxic reaction is capable of producing a typical choked disc.

CLINICAL OBSERVATIONS

Operative and Postoperative Edemas.—In the older methods employed for the removal of the Gasserian ganglion some considerable elevation of the temporal lobe, with resultant local cerebral compression, was necessary for a satisfactory exposure of this structure. In consequence of this temporary compression, a low grade of choked disc was not infrequently found after the operation in a considerable number of patients who were made the object of study a few years ago. In one patient there was a marked choking on the side of the operation, with retinal hemorrhages. The changes were often found immediately after the operation, and in a few cases lasted for several days before they disappeared completely. In no case was there any permanent damage either to the nerve or retina.

According to the method which we now use in approaching the ganglion we have failed to find any evidences of these changes, doubtless owing to the fact that with the present operative procedure but little elevation is needed in comparison with the earlier measures and because the pressure, such as it is, is continued for a comparatively short time. It is natural to suppose that the neuroretinal edema occurring in these patients corresponds with the condition induced experimentally by pressure on an area of exposed dura, the normal cerebrospinal fluid being crowded by the pressure into the adjoining optic sheath. Naturally the choking will not occur when there is a considerable escape of fluid during the course of the operation.

A postoperative choked disc has been seen in a number of cases when, after an exploratory osteoplastic craniotomy, there has been a subsequent extravasation of blood leading to an extradural clot. Also in cases of tumor extirpation, when there has been considerable cerebral contusion with resultant edema and swelling of the brain, the height of a pre-existing choked disc may be increased.

Choked Disc in the Newborn.—The fact has long been recorded that after difficult instrumental deliveries a choked disc, often with retinal hemorrhages, may be found in the newborn infant. This we have been able to verify. We have not only observed the condition in infants after prolonged and difficult labor, but also have ample proof that compression of the skull in supposedly uneventful deliveries is at times sufficient to elevate the discs to a height of from 5 to 6 D. In all infants in whom choked discs have been found, tense fontanelles have been the rule.

Cranial Injuries in the Adult.—It has been our good fortune to observe many examples of injury to the skull associated with edema of the papilla, and, indeed, it is the rule for a choked disc sooner or later to accompany at least all bursting (basal) fractures. The choking may appear early, a matter of a few hours, in case there has been extensive contusion or extravasation of blood, or it may appear late, a matter often of a few days, when the edema of concussion is at its height.

It consequently has been possible for us in many cases to study the eyegrounds before there was any evidence of swelling, and it has been interesting in these cases, since decompression has become a routine treatment for them, to compare the ophthalmoscopic

findings with the degree of tension disclosed at operation. Thus, no case in which choked disc was present failed to show increased intracranial pressure: cases in the absence of choked disc, with possibly one or two exceptions, invariably disclosed a brain and cerebrospinal fluid not under unusual tension.⁴ Many illustrative records might be cited—two will suffice.

CASE 1.—Forty hours after a basilar fracture of the skull a patient was brought to the hospital. During the next two days, despite the apparent improvement in his general condition, the choked discs, which were present on admission, gradually increased in height, and for this reason a decompressive exploration was deemed advisable. A large amount of bloody cerebrospinal fluid, holding numerous small clots, spurted from beneath the dura when the tense membrane was opened. A temporary subtemporal drain was placed with escape of fluid for a few days. The patient's choked disc quickly subsided and his recovery was rapid.

CASE 2.—A workman fell from a height on the back of his head. There was profound loss of consciousness with development of marked rigidity in the extremities. The discs were normal and there were no pressure phenomena. Nevertheless an exploratory decompression was performed, exposing dura and brain, which were not tense. The patient died after a few days, with symptoms resembling those of a decerebrate animal. Autopsy disclosed no lesion other than two small symmetrically placed pontine hemorrhages.

Meningitis.—Contrary to the statement handed on in many neurologic and ophthalmologic text-books, we have found that in uncomplicated meningitis choked disc is rare—a matter that speaks emphatically against the view of an inflammatory origin of the process. Others have made similar observations in regard to the infrequency of von Graefe's so-called descending neuritis.

When an internal obstructive hydrocephalus complicates the late stages of meningitis, however, choked disc is almost inevitable. We are inclined to regard its appearance as one of the most useful clinical signs of this complication.

Ventricular punctures have been performed in a number of these patients—oftentimes repeated punctures. On occasion the puncture has been made while the eyegrounds have been under continuous observation, and the subsidence of the vascular congestion, and, not infrequently of the swelling itself, is most striking.⁵ With reaccumulation of the fluid the original condition becomes restored.

Choked disc is rare in cases of pyogenic abscess, doubtless because brain is destroyed by the process rather than pressed on. Hence unless there is considerable local edema in consequence of the focus of suppuration there is not likely to be any considerable increase of tension. It is otherwise, however, with tuberculous abscesses, but their symptomatology so closely resembles that of tumors that they will be considered together under the latter heading.

Nephritis.—We are more and more inclined to the view of Bramwell, Russell and others that the headache, vomiting and so-called "albuminuric retinitis"—in other words, the cerebral pressure symptoms of renal disease—are due, in large part at least, to a cerebral edema. Histologic studies of the neuroretinal lesion in nephritis have furthermore served to strengthen the

opinion drawn from clinical observations that the lesion accompanying nephritis is merely a modified form of choked disc.

That a retinal picture typical of that characterizing renal disease may occur in association with tumors, particularly those below the tentorium, has long been recognized and has received the authoritative confirmation of Taylor, Gunn, Paton and de Schweinitz.

Eighteen of our 200 cases of tumor showed the typical so-called stellate figures of albuminuric retinitis. In twelve of them there was no clinical suspicion of any renal involvement, and, furthermore, relief of the intracranial pressure, through decompression, in a number of the remaining cases led to a disappearance from the urine of the suggestive renal elements. For example:

A young girl was under observation, owing to headaches and slight choking of the discs. Albumin and a few casts were present in the urine. This condition remained unchanged for a number of weeks; when a convulsion occurred, and it seemed inexpedient to postpone the contemplated decompression longer. At the operation a marked increase of cerebral tension was found; the choked discs rapidly subsided and the albumin and renal elements disappeared from the urine before the patient's departure from the hospital.

On the other hand, in not a few patients whom we have studied in the medical wards a postmortem examination has disclosed advanced renal disease, with a normal though possibly edematous cerebrum, whereas, clinically, definite choked discs had been present with none of the signs which are supposed to characterize nephritic retinitis.

In a number of patients with nephritis we have performed lumbar punctures while the eyegrounds were being observed. When pressure symptoms are present we have invariably found the fluid to be under increased tension, rising occasionally from 60 to 80 cm. or more in the manometer tube. Its withdrawal leads usually to a temporary amelioration in the patient's subjective symptoms and to an improved appearance in the eyegrounds. Unless the neuroretinal condition is far advanced in new tissue formation, a straightening of the vessels and a measurable lessening of the edema can sometimes be observed within a very short period. With the almost inevitable reaccumulation of fluid, the former appearances usually soon return. That this is not always the case, however, is evidenced by the two following histories:

CASE 1.—Two days after parturition, eclamptic convulsions with all the symptoms of cerebral compression developed in a young woman who had suffered for some years with chronic interstitial nephritis. The fundi showed the typical changes of "albuminuric retinitis." An excess of fluid was evacuated by lumbar puncture; the convulsions ceased, and in the course of a few hours her condition had greatly improved. In less than a week there was a marked change in the fundi. After three weeks not a trace of the "retinitis" could be found, and now, a year after the operation, she continues in good health and her eyes show nothing pathologic.

CASE 2.—In a patient with uremic symptoms who showed the typical, so-called "nephritic retinitis," a subtemporal decompression was performed. This disclosed a very tense dura, on opening which an abundance of fluid was evacuated. Our hope that the cranio-dural defect would obviate the return of the former degree of pressure was well grounded, for the patient's uremic symptoms entirely disappeared and there was a rapid subsidence of the extreme swelling of disc and retina, with a most striking change in the appearance of the fundi. The renal condition, of course, remained unaltered.⁶

4. It is true of these operative and traumatic cases, just as in cases of brain tumor, that the swelling involves the discs first on the nasal side and last on the temporal; whereas in clearing the process takes place in an inverse order, the temporal edge coming into view first, while the edema lingers on the nasal side.

5. One of our earlier experiences of the sort was recorded in an article entitled *The Blood-pressure Reaction of Acute Cerebral Compression*, *Am. Jour. Med. Sc.*, June, 1903.

6. This matter of choked disc and albuminuric retinitis, with the detailed report of this latter case, has been made the subject of another communication (*Am. Jour. Med. Sc.*, October, 1908).

Cerebral Tumor.—In our large series of brain-tumor cases we have been fortunate enough to see a number of patients before any injection or edema of the discs has become apparent, and we have thus been privileged to observe the early stages of the so-called "neuroretinitis" while the patients were awaiting operation, as well as the prompt subsidence of the condition after decompression. Indeed, in doubtful cases (cerebral syphilis, embolism, etc.), the question of operation in the medical wards has come to hinge, not on the patient's subjective improvement or on the change in general symptoms so much as on whether or not there is an alteration for the better in the eyegrounds. It may be said that we have seen no appearances in the discs of these patients that were not manifest in the conditions, clinical or experimental, which we have already cited—in other words, no appearances peculiar to tumor.

We have been unable in our study of the eyegrounds to discover anything of value in determining the size or structure of a tumor. Small benign growths in some situations will, by a mechanical effect, increase the intracranial pressure more than large, malignant, infiltrating ones placed elsewhere. Further, as de Schweinitz has emphasized, the stage of the retinal process is no indication of the duration of the cerebral lesion, nor can we predict in a case of tumor in the absence of choked disc when this is likely to occur. We have seen a sudden choking take place in a patient with a cerebello-pontile tumor known to have been present with no retinal change for eight years; and of course it is a frequent experience to have the first suspicion of a brain tumor arise in consequence of the accidental discovery of a choked disc. The whole question resolves itself into the study of the character of the intracranial pressure and its effect on the cerebrospinal circulation, regardless of the histology, the situation, or the duration of the neoplasm.

A growth may increase the intracranial tension in such fashion as to cause a backing up of cerebrospinal fluid in the optic sheaths in two principal ways, namely, with or without the intermediation of an accompanying internal hydrocephalus.

A tumor so situated as to produce an obstructive filling of the ventricles will cause a general increase in tension, often of high degree and of rapid onset. Under these circumstances the extracerebral fluid in the sub-arachnoid spaces, particularly in the basal cisternæ, will be held everywhere at equally high tension and choked discs which are practically equal on the two sides may rapidly appear. This is more apt to be the case in subtentorial tumors, though an obstructive ventricular hydrops may occur with basal tumors anywhere, and even with tumors of the hemispheres.⁷

Growths about the cerebellum have long had a bad reputation so far as the production of choked disc and resultant blindness is concerned, but it must be recognized that certain tumors, like the endotheliomata of the cerebello-pontile angles, may exist for years with absent or insignificant pressure symptoms, until the flattening of the pons and medulla blocks the iter and leads to an acute ventricular overfilling. We have seen long-standing tumors of the hypophyseal region do the same thing, namely, ultimately, lead to a filling of the lateral ventricles through pressure and deformity of the third ventricle, so that a double choked disc may become

superimposed on the old atrophic nerve-heads. Still, it is the rule for tumors in this situation to cause an optic atrophy through direct pressure, without the intermediation of a choked disc.

Centrally located tumors would naturally be expected to increase pressure equally throughout the cranial chamber and cause an equal choking of the discs, and this usually is the case. Indeed, if all tumors exerted their pressure effects equally in all directions and at all distances, we believe that the two discs would always show practically equal changes.

Tumors which do not cause a general increase of intracranial tension through hydrocephalus are much more apt to lead to an unequal process in the two eyes. This is particularly true of growths in the hemispheres and of those which are situated, roughly speaking, in the neighborhood of the optic tract—temporal lobe tumors, for example, and those within a short radius of the insula. There is sufficient pressure discontinuity between the basilar arachnoid spaces on the two sides, through the interposition of the third ventricle and infundibulum to cause the fluid pressure in the optic sheath to be considerably greater on the side of the growth than on the opposite side.

The pressure effects of a growth, as is well known, diminish inversely with the distance from the growth, and it is easy to understand that subarachnoid fluid may be pent up in the neighborhood of the chiasm by a tumor of one hemisphere, so that the homolateral nerve can be affected more than the other. Still it must be remembered that tumors springing from one side may grow entirely away from their point of origin and strongly indent the opposite hemisphere—a matter which is particularly true of the frontal lobes, owing to the relatively feeble support of the falx in this situation. The ophthalmoscopic findings in cases of this character are of course misleading, and they indeed cast the chief doubt on the value of choked disc as a means of localization.

However this may be, in 70 per cent. of our cases the papilla on the side of the tumor was the one most involved; in 80 per cent. of the cases seen before choked discs made their appearance the first swelling ultimately occurred in the eye homolateral to the tumor; and in 72 per cent. the ultimate damage to the nerve was greatest on this side.

We have seen some patients in whom the opposite eye has shown the greater degree of swelling. This possibly can be attributed to anatomic peculiarities of the optic sheath, which may be a factor to be considered in some individuals; to the indentation of, or a possible area of cerebral edema of the hemisphere opposite to the growth; or, finally, to the fact that the patient's examination may have been made when the disc primarily most affected is declining in height. As a matter of fact, it is of greater value in determining the location of a tumor to estimate the damage already done to one or the other nerve than to depend simply on the existing height of a swollen disc. With this in view it is our custom to search for the new white connective-tissue changes in the disc and around the vessels, to look carefully for the signs of atrophy, to make a careful plot of the visual fields and to measure the central vision. These things are the best index of what has already taken place in the disc and may prove to be the best localizing (right or left) signs.

As has been pointed out, subtentorial tumors, owing to their situation and the resultant hydrocephalus,

7. In J. M. Martin's analysis (Lancet, 1897, II, 83) of 601 tumors those of the corpora quadrigemina gave 100 per cent. of choked disc. We have had three of these cases not only showing choked discs but double blindness and deafness.

which first or last is almost inevitable, will often produce at some time in their course a greater swelling of the discs, and one more rapid in its onset, than tumors situated far forward. Only in exceptional cases has this failed to be the rule in our series. The average height of the discs in cases of cerebellar tumors was $5\frac{1}{2}$ D.; in tumors of the cerebral hemispheres $3\frac{1}{2}$ D. The difference is probably greater than would appear from these figures, since many of the patients with cerebellar lesions have been operated on during a period in which the discs were steadily gaining in height.

Tumors may continue to grow and still show a fluctuating degree of neuroretinal edema, often with complete temporary subsidence of the process, even without decompression. This can occur from a variety of causes. There may, for example, be periods of temporary relief to an obstructive hydrocephalus through the occasional escape of fluid. Then, spontaneous hemorrhages into the substance of a vascular tumor may cause a sudden rise of intracranial tension with a resulting elevation in the height of the discs, which subside again as the blood is absorbed. Further, cysts which possess a variable degree of tension not infrequently develop in degenerated areas of a tumor. Finally, an advancing growth may lead to a local cerebral edema in its immediate neighborhood, or indeed in a remote situation, with temporary increase of tension and a swelling of the discs which lessens as this edema passes away.

There is great variability in the length of time a choked disc may persist without leading to infiltration sufficient to produce degeneration with optic atrophy. It depends of course on the degree of severity of the process and this in turn on the degree of increased tension. In one of our patients a marked papillary edema, which had been known to be present for three and a half years, subsided in less than three weeks after a decompressive operation, leaving a central vision of 20/20 in each eye and only a slight constriction of the visual fields for form.

A fresh choking may occur on atrophic discs, whether the atrophy be primary or the consequence of a pre-existing choked disc which has disappeared. Marcus Gunn some years ago recorded an instance of this; Yamaguchi and de Schweinitz have recently called attention to a similar occurrence; and we have had opportunities of confirming these observations.

A young man was operated on for the removal of a large cerebral cyst, which presumably was a degenerated glioma. Atrophy of both optic nerves without swelling was present, doubtless a relic of the active stage of the growth some six years before. A large, postoperative, extradural extravasation formed, and the resulting increase in intracranial pressure led to an acute swelling of the discs, which subsided promptly after the evacuation of the clot.

These occasional postoperative extravasations have often given us information of great interest, and the examination of the eyegrounds has usually been the tell-tale of a process which demanded reopening of the wound.

A young woman was operated on for a focal epilepsy without other symptoms, and a fairly large, infiltrating, cortical glioma was unexpectedly disclosed. There had been no choked disc or other clinical evidences of cerebral pressure, and on reflecting the bone flap no tension of the dura or brain was found. The growth was extirpated and the patient recovered well from the anesthetic, but in the course of a few hours she became unconscious and examination of the fundi revealed an acute choking of both discs. The wound was immediately reopened, disclosing a large subdural clot, which was evacuated.

The surgical experience in the case of this latter patient confirms what Hughlings Jackson was among the first to point out, namely, that a choked disc need not accompany a tumor even of fairly large size. It serves, furthermore, to give the most reasonable explanation for the absence of pressure symptoms, on the ground that an infiltrating tumor need not increase the intracranial tension. We have met with a number of other examples of this same thing.

On the other hand, we believe that a certain low grade of choked disc is not infrequently overlooked. There is one type in particular which is detected with the greatest difficulty, namely, that in which both disc and retina are uniformly edematous and the structures remain clear. For example:

A patient was sent to us whose history and physical condition clearly indicated the presence of a growth in the central region of the right hemisphere. His eyes had been frequently examined by a capable ophthalmologist, who reported that there had never been any observable "retinitis" of either disc. We were rather inclined to believe that the nerves and retinae were not normal, but we could not be positive of this. A palliative exploration with decompression was performed, and in a few days the complete disappearance of an unsuspected edema from the retina, leaving prominent the discs, the swelling of which subsided more slowly, cleared up our uncertainties.

Thus, without decompression, incipient choked discs may be extremely hard to distinguish from simple hyperemia, and the greatest care must be used to avoid error.

On other occasions we have observed, while the process is in its incipency, this diffuse obscuration from swelling of both disc and retina, but as the nerve-head increases in height the retinal edema usually disappears. Conversely it has been seen to return as the swelling of the disc subsides. Thus, with a low grade of pressure or with an early process, edema of the retina may be more extensive than when there is a pronounced choking of the disc.

We find it difficult to reconcile these observations with the view that the low grade of retinal edema is characteristic of a true neuritis, for every link connecting the two processes can be made out. Thus, we have seen cases of so-called "descending neuritis" lose every supposed ophthalmoscopic characteristic of that condition and insensibly pass into choked disc; and conversely not a few choked discs have eventually shown the picture of descending neuritis. Though they are not common, we have seen typical choked discs in cases of meningitis, but only when the intracranial tension was found to be greatly elevated. After operation, despite the continuance of meningeal inflammation, the choked disc or, if you will, the descending neuritis, may entirely clear away.

Subdural hemorrhages from bursting fractures of the skull occasionally produce the picture of descending neuritis in one eye and of choked disc in its fellow, while removal of the extravasation may lead to a complete subsidence of both conditions. It hardly seems credible that one is a true neuritis and the other a choked disc, both caused by the same injury, appearing in the same individual and relieved by the same operation.

When choked disc is of high grade, periods of transient blindness may occur lasting from a few seconds to an hour or more. These periods of temporary amaurosis we find, in agreement with Leslie Paton, to have been most common in association with cerebellar

tumors. It is Oppenheim's conjecture that a distention of the third ventricle due to an obstructive ventricular hydrops may, by direct pressure, so affect the optic tracts near the chiasm as to cause the early amblyopia and blindness, and it is conceivable that such a process can account as well for these temporary losses of sight. One of our patients was able to induce the condition on assuming a certain posture.

A young married woman for some years had suffered from headache, homonymous hemianopsia and hemianesthesia, which were thought to be hysterical, until a double choked disc finally made its appearance. On sitting up and bending her head well over, her pupils would dilate *ad maximum* and she would slowly become blind. When in this condition, an ophthalmoscopic examination would reveal no appreciable change in the condition of either arteries, veins or degree of swelling. A combined exploration and decompression was performed, with amelioration of her subjective symptoms, disappearance of the choked disc and cessation of the attacks of blindness.

Hughlings Jackson was among the first to lay stress on the fact that central vision may be preserved in almost normal acuity until a late stage of "optic neuritis." Owing to this fact the tragedy of a sudden and unexpected total loss of vision is unfortunately not uncommon. We have, on a number of occasions, been persuaded to operate on patients after such an occurrence, in the hope that some vision might be restored. This has never occurred even though two of these patients were decompressed within a day or two after the blindness set in, and though in one of them a cerebellopontile tumor was successfully removed. Indeed, as has been the experience with others, we have seen cases in which a palliative operation has been deferred so long that blindness from complete atrophy followed the operation as the choked disc slowly subsided.

The study of the visual fields will usually give a much better index of the extent of implication of the nerve than will any tests for acuity, and there are certain features of the color fields in particular which we regard as characteristic of an early stage of implication of the nerve—a matter which must be deferred for another occasion.

HISTOLOGIC AND SURGICAL NOTES

The histologic alterations in the nerve and retina we have found to vary with the extent and duration of pressure and to bear little if any other relation to the character of the associated disease or injury. The greater the swelling and the more enduring the process the more marked the histologic changes have been.

The pathologic findings usually varied from a slight swelling of the disc with overfilling of the veins to great dilation of the subvagal spaces around the nerve and to complete atrophy and marked degenerative changes in the retina. The microscopic picture corresponds with the gross pathologic changes, consisting in the main of small and large hemorrhages in the nerve-head and retina, cystic and colloid changes, hyaline thrombi, marked separation and tortuosity of the nerve bundles, proliferative changes in the vessels, perivasculitis, round-celled infiltration, fatty deposits in areas where exudates were present, cytoid masses, retinal separation and more or less complete disintegration of the fibers of nerve and retina.

Through the courtesy of Dr. F. H. Verhoeff of Boston we have had the privilege of studying and comparing with him sections of the optic nerves and retinae taken from a number of undoubted cases of brain tumor and of nephritis in his and our own collections. The his-

tologic changes varied but little in the two conditions, and the essential features of each left little doubt as to their close histologic connection. It seems very probable that the two lesions represent only modified forms of a neuroretinal disturbance originating from the same cause. Sclerotic changes are present in the vessels in nephritis more often than in tumor; this, however, is another sign of the primary disease, and, so far as the retinitis is concerned, is a mere coincidence. It possibly gives the retina less power to resist changes resulting from edema and modifies the picture seen ophthalmoscopically. Thus, since the presence of a stellate figure has been recorded in our series in nearly as many cases of undoubted brain tumor as of undoubted nephritis, we naturally incline to the view advanced by others that it can not be regarded as typifying renal disease exclusively.

Our experience encourages us to advocate, not only in many cases of cerebral tumors but in a number of other conditions, an early decompression. Needless to say, a cranial operation is regarded by many people, both lay and professional, as a serious performance—and perhaps justly so. It is necessary, therefore, that an operation proposed for the relief of the retinal lesion in its incipency, at a time when it is difficult to make the patient appreciate the serious nature of his trouble, should be one practically without risk, easy of performance, one which requires the shortest possible hospital residence and leaves the least possible external evidence of its having been carried out. We know of no operation other than the subtemporal decompression described by one of us which in so large measure meets these requirements.

Permanent alterations in the fields and loss of visual acuity invariably take place if choked discs are allowed to run their course, and in a very large proportion of cases complete blindness supervenes before death terminates the story, and indeed only too often in cases in which the primary lesion does not lead to a fatality.

Any permanent alteration in the nerve will cause a corresponding change in sight. This often holds true even if the process is interrupted by an operation, but the important point is that the longer the changes persist the greater will be the loss in vision. In a number of patients the choked discs have subsided after operation with complete restoration of normal visual acuity; in a larger number the condition has been checked without further advance, and the sight has remained as before the operation. Unfortunately we have seen a few patients in whom, in spite of the decompression, vision has become worse, and one, indeed, in whom it was lost. These latter patients all had extensive exudates and marked proliferation of connective tissue in the disc and retina, and the further loss of sight was due solely to the contraction of these masses with resultant degeneration of the vulnerable nerve fibers. These cases offer the strongest argument for early decompression; for if the choking has been allowed to advance to this high degree, unavoidable contracture of the new tissue may shut down on the nerve fibers and serve to produce atrophy regardless of any intervention.⁸

8. The results of our surgical measures, so far as the changes in the eyegrounds are concerned, do not differ greatly from those given by Leslie Paton in his excellent and concise review of a series of cases observed by himself and Marcus Gunn in the National Hospital, Queen Square (Optic Neuritis in Cerebral Tumors and its Subsidence after Operation, Tr. Ophth. Soc. U. Kingdom, 1905, xxv, 129). Our series of cases has been considerably larger than that which forms the basis of his report, and

If we are correct in thinking that increased intracranial pressure—a condition which in its less severe degrees can be relieved by operation—is the cause of choked disc, and with the knowledge that choked disc frequently leads to blindness, the proper course to pursue must seem clear to every one. We do not wish to seem radical in this, for we zealously endeavor to give medicinal therapy and cautious delay their just dues. Still, in many cases it is often the safer course to decompress first and push the drug treatment afterward. One method promises sight, the other possible blindness, and the iodids are much better borne when the patient is free from headache and nausea.

Further, in undoubted tumor cases it is often unwise to wait until the growth can be located before operating. The primary effort should be made to alleviate the patient's physical discomforts and assure the preservation of vision; then one may delay with lessened anxiety. We have had, during the past five years, many patients with unlocalizable tumors, who, as the result of an early decompression, are perfectly comfortable, with either normal sight or vision amply sufficient to allow them to continue with their livelihood. A number of these patients have returned after variable periods with an onset of focal symptoms which have enabled us to determine the situation of the growth; in a few of them the lesion has proved to be accessible and has been successfully removed.

SUMMARY

It is our view that the early injection with stasis of the vessels, the marked edema with projection of the papilla, and the ultimate round-celled infiltration with new tissue formation which leads to atrophy, are merely stages of the same process. We believe that the mechanical views of Schmidt-Rimpler and Manz are largely correct and that the lesion is primarily due to the crowding into and distention of the sheath of Schwalbe by obstructed cerebrospinal fluid, resulting in an edema of the nerve-head, in which toxic elements play an insignificant part.

We believe, with Parinaud and his followers, that an acute internal hydrocephalus in the closed adult skull almost inevitably leads to a choked disc, but we attribute this to the presence of arachnoidal fluid forced into the optic sheath rather than to an edema spreading from the brain to the optic nerve; furthermore, choked disc is often present with tumors which have not led to hydrocephalus and is usually absent in the "essential" hydrocephalus of infancy.

We believe, since a small, benign, slow-growing and remote tumor so situated as to cause hydrocephalus—for example, by pressure on the iter—can lead to a high grade of choked disc, whereas a malignant, rapidly-growing glioma which causes but little pressure, owing to the way in which it infiltrates the brain, may produce no change whatsoever, even though situated near the optic nerve, that toxic products in the cerebrospinal fluid can hardly be held responsible for any stage of choked disc. With others, we have observed cases of sella turcica tumors which have led to optic atrophy from direct pressure without producing choked disc.

contains many more cases of early one-stage decompression for unlocalizable growths, which may account for the fact that there is a considerably higher percentage of complete restorations to the normal. It would be superfluous for anyone to attempt to improve on his vivid description of the ophthalmoscopic appearances to be observed during the advance and subsidence of the process

In these cases, owing to the situation of the lesion, fluid is not crowded into Schwalbe's sheath.

We believe, since choked disc, contrary to many authorities, is rare in meningitis and equally so in abscess unless there is an obstructive hydrocephalus leading to greatly increased pressure, that meningeal inflammation of itself must be looked on as an unlikely source of the lesion. Furthermore, the process is frequently seen after cranial fractures, apoplexies, and not uncommonly after simple operations when the question of an infection can safely be ruled out.

We believe, finally, since there is an almost uniform subsidence of what we consider to be choked disc (barring certain cases complicated by obstructive hydrocephalus) after decompressive operations, whether conducted for the pressure of tumor, cerebral edemas of one sort or another (traumatic, nephritic or vascular), infections, or intracranial hemorrhages, that a mechanical rather than a toxic process must play the chief rôle in the causation of this well-recognized lesion.

ABSTRACT OF DISCUSSION*

ON PAPERS BY DRS. SPILLER† AND CUSHING AND BORDLEY

DR. H. V. WÜRDEMANN, Milwaukee: My experience with the procedure of trephining for this condition is confined to three cases, and I speak mainly from a theoretical standpoint.

As the causes of optic neuritis are so varied, and in the main are general diseases, as syphilis, nephritis, the rheumatic diseases, the grave anemias and diseases of the vascular system and poisons, we are limited in a surgical sense to optic neuritis caused by diseases of the orbit and adjacent sinuses, amenable to the usual surgical treatment, to infectious diseases characterized by spinal and cerebral meningitis (for this lumbar puncture is certainly preferable); and to papillitis occurring in brain tumor, due to increased intracranial pressure, and speedy relief by trephining seems indicated.

Although the pathology of choked disc is usually given as due to increased intracranial pressure, with tension of the optic nerve sheaths, from a theoretical standpoint, I consider that this is true only in the case of intracranial hemorrhage and actual new growths. Certainly, in all cases there is local infiltration of white cells and exudates within the sheaths, but this is only the effect, not the cause, of papillitis, which is in most cases due to general diseases, acting through germs or toxins. The treatment is that of the general disease, together with eliminative measures, more particularly pilocarpin sweats and iodid of potassium in large doses; the same form of treatment is used for papillitis due to drugs or poisons. In diseases of the orbit and adjacent sinuses, certainly local surgery is indicated. In certain infectious diseases, more particularly spinal and cerebral tuberculous meningitis and epidemic cerebrospinal meningitis, lumbar puncture not only relieves the meninges of the excessive amount of toxic cerebrospinal fluid, but likewise the pressure within the optic nerve and the cerebral ventricles, and is here recommended. In intracranial hemorrhages and tumors, optic neuritis is believed to be caused by pressure; the relief of this intracranial pressure has relieved or brought the papillitis to a standstill, preventing the further infiltration of the white cells into the neuroglia and consequent destruction of the optic neurons and preventing the secondary deposition of white elastic tissue, which causes the resultant atrophy of the optic nerve. Therefore, I believe that trephining should be restricted to cases of optic neuritis caused by intracranial hemorrhages and tumors, lumbar punctures being reserved for the other surgical indications, as these operations prevent the patient from becoming blind.

Edward Stieren says that the skull and dura mater should be opened as soon as the diagnosis of increased intracranial

* The discussion appeared in full in the Transactions of the Section on Ophthalmology and in the Transactions of the Section on Nervous and Mental Diseases, 1908.

† The paper of Dr. Spiller appeared in THE JOURNAL last week.

pressure is established, medical treatment being instituted afterward.

Horsley advises opening the skull and dura mater in optic neuritis of anemic or toxemic origin. He places considerable responsibility on the medical adviser if blindness results, after the process has been diagnosed, due to failure to arrest it by the simple palliative remedy of opening the dura. The ophthalmoscopic appearance of the disc must be carefully interpreted before the degree of vision after operation is predicted. The greater the secondary changes in the disc and retina as shown by the yellowish-white stippling patches of exudation or opal white atrophic changes, especially if associated with muscular figures, the more will vision be impaired.

Babinski and Chaillous say that the therapeutic value of lumbar puncture in ocular pathology has not been thoroughly studied. A few authors only have incidentally noted its favorable effect in certain affections of the optic nerve. According to the observations of Druault, papillary edema, as well as other symptoms of the cerebral compression, may disappear after evacuation of the cerebrospinal fluid, but the result is far from being constant. Quinke joined a vertical glass tube to the needle used to make the puncture to enable him to measure the pressure of the cerebrospinal fluid and establish the relation between intracranial hypertension and the symptoms that are attributed to it. In this way, in case of "choked disc," lumbar puncture may aid the diagnosis, and it may be repeated if it causes any improvement.

The relief, sometimes so immediate, of visual trouble following lumbar puncture, favors the theory of Dupuy-Dutemps, who asserts that papillary edema is due to compression of the central vein in its passage through the dural sheath.

Babinski and Chaillous conclude that lumbar puncture should be considered a therapeutic measure in cases of a papillary edema due to traumatism or inflammation while in intracranial tumors it can be only palliative; that it is well to repeat the punctures as often as may seem necessary to avoid the effects of compression, particularly atrophy of the optic nerve.

Lumbar puncture appears to be particularly effective in the clinical type described by Quinke under the name of "serous meningitis." In many cases of meningeal hemorrhage, particularly the traumatic, the method may be indicated when there is no urgent necessity to evacuate the blood. Palliative results are obtained in hydrocephalus. In optic neuritis from cerebral tumor prudence should be used in treatment by lumbar puncture, as in some cases sudden death has followed it, and in others there has been an aggravation of the nervous symptoms, particularly of headache and vomiting. In the uremia of Bright's disease there may be real relief, though only of symptoms. In labyrinthine diseases of the ear lumbar puncture diminishes the vertigo and tinnitus and even the deafness.

In all three cases the effect is obtained by diminution of the tension produced by the excess of cerebrospinal fluid. There is a very reliable clinical sign of this hypertension in optic neuritis of stasis, and the diagnostic and therapeutic value of lumbar puncture in optic neuritis has attracted the attention of neurologists and ophthalmologists.

DR. CHARLES H. FRAZIER, Philadelphia: I shall base my remarks chiefly on an experience with between fifty and sixty decompressive operations. There is no therapeutic measure, physical, medicinal or surgical, which gives greater relief to so apparently hopeless and intensely suffering a class or group of patients as the decompressive operation. This operation should include among its indications traumatism, perhaps epilepsy, meningitis, acute and chronic, with hydrocephalus. As for tumors, we should not be content with purposeful decompression except in those cases in which we have assured ourselves that the tumor is inoperable, inoperable signifying either that the tumor can not be localized or can not be removed. The ease with which the simple decompressive operation can be performed and the immediate relief it affords, tempt surgeons and neurologists to be content with palliation. If the habit becomes prevalent, many operable tumors will be overlooked, and the percentage of operable growths will not increase as fast as it should.

The influence of decompression on beginning or fully established optic neuritis is naturally uppermost in the minds of the members of the ophthalmologic section. With but few exceptions the results in our work in the University Hospital have been good. The prognosis seems to be better in those cases in which the choked disc subsides comparatively slowly. In fact, we have never observed as rapid a disappearance of the choked disc as has been recorded by others.

Surgeons have been accustomed to relieve intracranial tension due to the result of traumatism, and particularly fractures and hemorrhage. There should be a limited number of cases of cerebral edema, resulting from cerebral contusion, in which decompression might relieve intracranial tension sufficiently to tide the patient over; but certain factors may render operation useless, and especially the rapid and diffuse character of the process. Tumors are more localized, grow more slowly, and do not, as a rule, involve the medullary centers. The edema of contusion is more or less widespread, and even though decompression is established, the process continues and eventually through direct or indirect involvement of the circulation of the vital centers, causes death.

In the fifty or more operations which we have had, excluding those for the relief of traumatic conditions, we have lost one patient in twenty-eight of those in whom the lesion was in the cerebrum. In this case the autopsy proved that the patient had multiple sarcomata, involving the medulla and pons as well as the cerebrum. Often we did more than a simple decompressive operation. In more than half the cases this was preceded by an exploratory operation, which, of course, increased the operative risks. In 25 operations for lesions of the cerebellum, we have had 6 deaths; 1 from pneumonia, 1 a typical cerebellar death, and 4 from shock. In the first 8 of the series there were 3 deaths, and in the last 14 only 3 deaths.

As to the general effect of the operation, 70 per cent. of the patients may be said to be considerably relieved, and 10 per cent. have been entirely relieved of the symptoms for the relief of which the operation was performed.

As to the effect on individual symptoms, we find in our histories that as to choked disc only three are recorded as uninfluenced at the time the patients were discharged from the hospital; and some of these already had optic atrophy. As to headache, two are recorded as uninfluenced by the operation; as to nausea and vomiting one; and as to vertigo one. Some of those cases which were referred to as being uninfluenced at the time the patients were discharged, we know from subsequent observations, have improved. I am referring now particularly to the choked disc.

As to the time in which operation should be performed, I believe that in an increasing optic neuritis we should not wait for the development of localizing symptoms or other signs of brain tumor or intracranial lesion. I believe also that we should not hesitate to operate in cases which are apparently hopeless, the patients being in a grave condition at the time they are brought to the surgeon.

In one case, particularly, in which I recall having operated during the past winter, the patient was almost unconscious, delirious for a number of days, and had violent headache in addition to optic neuritis; the situation seemed hopeless. A rapid decompression, however, was followed by almost as rapid a restoration to consciousness and the subsidence of all symptoms except an aphasia, which is gradually disappearing.

DR. G. E. DE SCHWEINITZ, Philadelphia: I endorse all that has been said on the value of decompressing operations for choked disc and join in all the protests made against unreasonable delay in performing these operations. I would say a word, first, in regard to the relation of the refraction of the eye to the development of choked disc, or, to use another and perhaps a better term, "brain-tumor neuritis," or Mr. Marcus Gunn's recently delivered term, "brain-tumor papillitis." All those who have had the opportunity of observing the effect of the

*Since these remarks were made, the last volume of Mr. J. Herbert Parsons' "Pathology of the Eye" has appeared. In this he urges the adoption of the term "papilledema" as a substitute for the word "choked disc," that is, the *Stauungspapille* of the Germans.—G. E. DES.

relief of intracranial tension on engorgement of the nerve-head must readily agree that any theory which omits this element is not worthy of serious consideration. Not only is the important relationship of increased intracranial tension to the development of choked disc proved by recent work in brain surgery, but it has received strong experimental proof, notably from Merz in 1901; and his work is confirmed by the researches of Drs. Cushing and Bordley. Therefore, it is difficult to be divorced from the term "choked disc," which is so expressive. It is a well-known fact that so-called brain-tumor papillitis, or choked disc, develops either slowly or not at all in myopic eyes. This, as Mr. Marcus Gunn points out, in all probability depends on the difference in the termination of the optic nerve sheaths in the sclerotic. In hyperopia the termination is more abrupt and in a more conical form than in myopia, in which the sheath spreads itself out more gradually into the sclera, and therefore allows an easier filtration of the fluid which is the cause of the edema. Be this as it may, in the examination of all eyes in the subjects of suspected increased intracranial tension from any cause there should be a full consideration of the refraction of the eye.

In making record of a choked disc, or tumor papillitis, there should be a definite statement, not merely of its height, but of its height above the level of the cycloground. Doubtless this is always perfectly clear to the observer, but it has not always been made clear in the records. This is a point of some importance in noting the results of operative interference on the height of the swelling. Take the case of a patient in Dr. Frazier's ward: Although the surface of the disc is + 5 D., since the trephining there is little or no swelling, or, more accurately, edema elevation, because the patient has a hyperopia of 5 D. at the macula, and the original height of the disc was 3 D. above that level.

It is desirable to make record of the corrected vision of the patient, both before and after operation. Unfortunately, sometimes the acuteness of vision has apparently been recorded with the aid of the patient's correcting glasses, but later records of vision have been made without this aid, or sometimes the procedure has been exactly the reverse. I could give a number of examples showing how failure to note the refraction, both before and after operation, has introduced inaccuracies in the ultimate records of the visual results.

It has been my own experience that the subsidence of choked disc after decompressing trephining is not a very rapid one in many instances, although there have been some notable exceptions, one of which I believe has been reported in the Section on Nervous and Mental Diseases by Dr. Sinkler during this meeting. It seems to me that there should be a sharp distinction between the primary subsidence of the engorgement of the retinal vessels, the size of which sometimes markedly diminishes in the first twenty-four or thirty-six hours after trephining, and the subsidence of the height of the edematous disc, which is usually apparent at a later period. This point, I believe, has been made by von Kruedener in his recent article. My own experience has been that the first decided change in the engorged nerve-head is usually about the seventh day (certain exceptions have been referred to), and from then on the subsidence is quite rapid, so that most of the edema disappears by the end of the fourth or fifth week. This experience agrees with that of Mr. Leslie Paton, recorded in his recent admirable papers on this subject. Moreover, it has seemed to me that the ultimate visual results were better if there was a comparatively slow subsidence of the engorgement edema than if this disappeared very rapidly.

Naturally, the prognosis as to sight is better the earlier the operation is performed. If operation can be performed in one of the first three stages of tumor papillitis (with which stages all of us are familiar), the prognosis *quoad visum* is extremely favorable. If, however, the operation must be delayed until the fourth or fifth stage of choked disc, the prognosis is much less favorable, and, indeed, if it is much delayed, the operation can be of little service in restoring sight. In this connection, however, it should be remembered that sight does not always return immediately after the operation, and the records will show that sometimes it is nec-

essary to wait for a considerable period of time, even five or six weeks, before the best results are attained. Therefore, if possible, the surgeon should always operate during the first, second or third stage of tumor neuritis, but should not hesitate to operate, no matter what the stage is, not only for the relief of intracranial pressure, but for the sake of good visual result occasionally possible, even in apparently most unfavorable cases.

A difficult differential diagnosis is between beginning tumor neuritis and the so-called spurious optic neuritis not uncommonly seen in hyperopic eyes. I suspect that in some cases of reported engorgement edema of the papilla the observer may have been deceived by the presence of a hyperopic neuritis which he mistook for a beginning choked disc due to increased intracranial tension.

Occasionally there is rapid loss of sight after trephining, when one would expect just the contrary result. In two notable instances, after perfectly correct technical operations, both of them on cerebellar tumors, I have seen rapid blindness follow in spite of the prompt relief of intracranial tension. It has seemed to me that a possible explanation of some of these cases was great loss of blood at the time of the operation. The hemorrhage may have caused some change in the ganglion cells of the retina, exactly as such changes may be produced by free loss of blood after hemorrhage from the stomach or bowels. If so, the mechanism of blindness under these circumstances is the same as that which occurs in quinine, methyl-alcohol poisoning and other toxins, as has been so well shown in the researches of Ward Holden and others. I know that there are other explanations of this blindness.

DR. PETER BASSOE, Chicago (presenting a patient): This patient, a clergyman, 42 years of age, in the fall of 1904 developed symptoms of brain tumor, severe headache, mostly in the morning, associated with vomiting and dizziness, diminution of hearing on the left side, slight impairment of vision and a tendency to fall to the left in walking. I saw him first in November, 1906, and then he presented the following symptoms: Staggering gait, tendency to fall to the left, slight left-sided ataxia, diplopia due to paresis of the left external rectus, nystagmus, which was coarse and slow in looking to the left, fine and rapid in looking to the right; bilateral optic neuritis, very slight on the right side, but marked on the left side, the swelling as measured by Dr. Wilder being 3.5 diopters. The vision in the left eye was 20/30, in the right eye 20/20. There was total nerve deafness on the left side with no evidence of middle ear disease past or present. A diagnosis of left-sided cerebellar tumor was made and operation advised. The operation was performed by Dr. Arthur Dean Bevan, Nov. 22, 1906. A suboccipital cranial resection was made and a tumor was felt in the left lobe of the cerebellum half an inch below the surface. Just how large it was and whether infiltrating or not could not be easily determined. The patient took the anesthetic very badly, and removal of the tumor was not attempted at that time. The dura was not sutured and the bone not replaced, so as to allow a hernia to form. It was not known at that time whether this operation was to be the first step in removal or a merely palliative operation. Three hours later, as soon as the patient came out of the anesthetic, I examined his eyegrounds and I am certain that there was a distinct change, as I then for the first time could see the outline of the left disc. Unfortunately, he was not examined by an ophthalmologist until three days later. Then Dr. Wilder noted that the disc outlines were clearer, but the swelling remained the same, so the change observed by me must have been chiefly in the amount of blood in the veins. On December 13, nearly three weeks after the operation, Dr. E. V. L. Brown measured the highest point of the disc to be a little less than two diopters; so there had been a considerable recession in the course of the three weeks. The left-sided ataxia decidedly increased after the operation. On December 14 a second operation was made and the tumor removed. It was a cystic glioma of the size of a walnut and shelled out very easily. Since then the patient has made slow but steady progress. The vision was measured a couple of times after the operation and in the left eye one measured 20/40, once 20/30, and is now 20/40; the right eye each time

20/20. The ataxia has gradually diminished. The patient returned to his work, which he formerly had to discontinue on account of severe headaches, and during the past year he has been able to preach twice every Sunday. The severe headaches have entirely disappeared. The chief trouble now is the ataxia on the left side, which I consider to be due to the cerebellar hernia, which can be readily felt. Dr. Brown yesterday examined the patient's eyegrounds and finds that the left disc is best seen with a +1 lens. From what I understand, there is now no sign of any atrophy in the disc, and an abundance of small vessels can be seen. The left external rectus paresis, the left nerve deafness and the nystagmus still exist.

DR. CHARLES K. MILLS, Philadelphia: Trephining for cerebral symptoms dates far back even of the history of modern localization and cranial surgery. During my experience of twenty-five or thirty years, in not a few instances palliative operations were suggested for symptoms, like headache. I recall Dr. Weir in this connection. With regard to what are now called decompressive operations for the relief of optic neuritis, headache, vertigo and nausea, our recent experience, I think, dates distinctly to 1901, when at the Philadelphia Neurological Society I presented a paper on brain tumors, especially with reference to the parietal and prefrontal lobes, followed by a paper by Dr. de Schweinitz on optic neuritis. This was followed by the suggestion of trephining for the relief of optic neuritis by Dr. Spiller, who asked Dr. de Schweinitz whether he considered it justifiable to trephine when choked disc and certain symptoms suggesting brain tumor existed, although the growth could not be localized; that is, whether he considered it justifiable to trephine in the hope of preserving vision and modifying some of the symptoms of brain tumor. In some cases the mere opening of the skull seemed to have been of benefit. An account of this meeting and the discussion is to be found in the *Journal of Nervous and Mental Disease* for May, 1901. Since that time we have had an increasing number of cases in which both radical and palliative operations have been performed.

If optic neuritis is present and increasing, there should be no dallying with sodium iodid or potassium iodid and mercury, no waiting for the development of further symptoms, in many cases no waiting even for focal symptoms, so important is it that these operations should be done promptly.

The rule which we follow at the University of Pennsylvania is, whenever there are any focal indications, however slight, to make the decompressive or palliative operation in a region that would allow of a possible radical operation. It makes some difference, perhaps, but not very much, as to the part of the skull which opened. Over the right temporal lobe or the right prefrontal lobe are favorable positions. We have a habit at the University of Pennsylvania of working in collaboration—Dr. Spiller, Dr. Frazier, Dr. de Schweinitz and others—and in every case in which we have a tumor with optic neuritis we agree on operation as quickly as possible.

DR. CHARLES E. BEEVOR, London, Eng.: When I first began to study neurology the theory held was that neuritis was caused purely mechanically; the choked disc idea was the theory that was advocated; then later on some papers were read, I think at the meeting of the Ophthalmological Society in London, which argued that it was rather due to descending changes in the optic nerve; but I have always felt that the old theory was probably the correct one, and that the cause of the neuritis was really purely mechanical. I was, therefore, greatly interested to learn that Dr. Cushing and Dr. Bordley had come to the conclusion from the experiments that the neuritis is really mechanical.

How soon after the injection of the fluid in dogs do symptoms occur in the optic disc? Is it a question of hours or of days? I understood that this injection of paraffin did not produce the choked disc so easily as injection of fluids. I suppose that the reason of this is that the brain becomes used to, and accommodates itself to pressure, and, therefore, the effects are not so marked.

It has been a mooted question, especially in England, whether the optic neuritis occurs in the eye on the same side as the tumor or on the opposite side. Sir Victor Horsley

holds that the neuritis occurs first of all, or is more intense, on the side of the tumor; but in a paper by Leslie Paton, read at the Ophthalmological Society of London recently, he comes to the conclusion that one can not lay much stress on that. My own point of view is that the question of on which side the tumor occurs on is of least importance. For instance, if all the symptoms pointed, say to the left side and the neuritis was more marked on the right side, I should disregard it as a localizing symptom. In a percentage of cases it is more frequently, I think, on the side of the tumor; but not so with sufficient frequency to be useful in the question of diagnosis.

In cerebellar tumor I agree that the neuritis is more marked than in tumor situated further forward; in some lectures before the Medical Society of London I had the opportunity of bringing together three or four cases of tumors in the parietal region, and the extraordinary point there was that the neuritis developed very late. In one of these cases a man had symptoms of a tumor for over a year with hemianopsia and local fits, before optic neuritis developed, whereas in cerebellar tumor optic neuritis develops earlier and is more intense.

In a case of optic neuritis with no other symptoms it is difficult to say whether one ought to operate or not. I am inclined to wait in those cases, because one sometimes does find optic neuritis even in cases of anemia, and then it would be a great mistake to operate. But if the patient has agonizing headache and vomiting and optic neuritis, I think the sooner one operates the better, and one should not wait for localizing symptoms.

I have seen a case of loss of sight after the operation. Before the operation the sight was fairly good and no evidence of any optic atrophy was present; but, of course, intense optic neuritis, as was shown by Jackson some years ago, is perfectly compatible with good sight. It was formerly supposed to be impossible, but it is now recognized that intense optic neuritis can exist without affecting vision. In this case after operation the vision rapidly deteriorated, the patient became completely blind, so that after decompression the vision became worse, instead of improving.

I understood Dr. Spiller to say that in cases of cerebellar tumor occurring in the ponto-cerebellar angle he would prefer decompression to operation. I should not quite agree with that. I think that in those cases, as already shown by Dr. Bassoe's case, one can operate and remove the tumors.

Lumbar puncture has no effect on optic neuritis, so far as I have seen. When the patient is comatose, respiration has ceased, but the heart has not stopped beating, I think we ought to operate, because, as Sir Victor Horsley has shown in the case of gunshot wound experiments, when the intracranial pressure is increased the respiration fails before the action of the heart does; and I have seen cases in which the respiration had failed in cerebral tumors and on operating and relieving the pressure, the patient has recovered consciousness.

DR. HARVEY CUSHING, Baltimore: We have frequently seen a decided subsidence of choked disc, within forty-eight hours, and in not a few cases soon after the operation. Of course, the rapidity of subsidence depends entirely on two things: 1, the character and success of the decompression, and, 2, the stage of the process, since in a long-standing edema which has led to infiltration the swelling can hardly be expected to subside as rapidly as in the cases in which there is little more than edema alone. An increase of the neuroretinal edema, which Dr. Spiller has seen in a few cases after trephining, is not improbably due to the increase of cerebral tension due to traumatic edema. We do not entirely agree with him on the question of decompression versus extirpation for lesions in the posterior cranial fossa, for a number of our most successful cases of tumor extirpation have been those in which the lesion was subtentorial. The excellent result in the patient with the cerebello-pontile tumor presented a few moments ago by Dr. Bassoe is an illustration of what may be expected in a large percentage of properly conducted operations in this situation. I wish to warn against promiscuous lumbar puncture in cases of brain tumor, particularly those situated under the tentorium, and also to express an opinion

as to the uselessness of ventricular puncture in tumor cases except as a diagnostic measure.

Marcus Gunn's views that papillitis is associated chiefly with hypermetropic eyes and is relatively rare in myopia does not entirely correspond with our own experience. We have had no case of myopia in which there has been demonstrable increase of tension without choking of the discs and have had two cases of hypermetropia with tumors of the hemisphere in which no choking occurred.

Dr. Beevor has asked how soon after injection we have seen a choked disc appear in our experiments. It is a matter of moments rather than of hours or days, and by "choking" we do not mean necessarily merely the signs of injection with venous stasis, but an actual swelling of the disc of three or four or more diopters. In a patient in the Johns Hopkins Hospital we have had, furthermore, a clinical illustration of the rapidity with which a choked disc may appear. An operation had been deferred owing to the absence of a choked disc, when there occurred a sudden loss of consciousness with swelling of the disc up to 7 D. in the course of a few hours. Death followed, and at autopsy a large hemorrhage was found in a glioma of the hemisphere.

I can not agree that it is immaterial where a decompression be done, and have given elsewhere reasons for regarding a subtemporal route as preferable to Saenger's parietal exposure. It is often dangerous to decompress directly over the situation of a tumor, for the dislocation which occurs in consequence of protrusion will not infrequently lead to a rupture of blood vessels entering a vascular growth with marked augmentation of the degree of pressure. I regard as an important measure, the primary subtemporal decompression in a patient who actually has a localizable tumor and one which may possibly be removable; for when patients are in a more or less critical condition, emaciated, with headaches, failing vision, etc., even though the case may be looked on as a favorable one for ultimate possible tumor extirpation, a preliminary decompression, which is the more simple measure, is often a helpful thing in so relieving the general physical condition that the more serious subsequent operation for extirpation can be done much more safely.

A still more important matter which Dr. Bordley and I have tried to bring out in our paper is the prompt decompression in cases of intracranial growth when the lesion is not localizable, for, as all those who have taken part in this discussion have said, the preservation of the eyesight is a matter of chief importance, and with a suitable decompression the tumor in certain cases, after months or perhaps years, may through gradual increase in its size, finally give evidence of its location, and, on exposure, may prove to be removable.

I hope that Dr. de Schweinitz will not give up the term "choked disc," at least in exchange for the term "tumor neuritis." It possibly is only a quibble of nomenclature, but I think that we ought to have some one term which will describe all grades of a condition which we recognize to be one, rather than to attempt to distinguish, as did von Graefe, the two stages, namely, those before and after marked swelling. Believing as we do, that the inflammatory condition is entirely a secondary process, we think that the term "neuritis" and "papillitis" are misleading and that the term which lays stress on the primary factor, namely, edema, is the proper one.

DR. WILLIAM G. SPILLER, Philadelphia: At the beginning of the winter I asked Dr. Sweet to have one of the students of the third year class of the University of Pennsylvania undertake some experiments on decompressive operations, and a certain amount of swelling of the disc by the introduction of foreign bodies under the dura in dogs was produced. Dr. Shumway made the examinations of the eyes. We were impressed by the amount of endurance exhibited by the dog under pressure of a considerable degree. A large piece of bone in many cases failed to produce choked disc or neuritis; and in other cases produced it only for a short time. These experiments will be reported later. I am convinced that tumor of the motor region—and probably the same is true of tumor of the parietal lobe—often causes choking of the disc, only late. I do not expect in tumor of the motor region of the

cortex to find optic neuritis until the tumor has grown to considerable size.

I am somewhat in doubt as to the propriety of decompressive operation when the patient is almost dead. It is probable that it will do no good, and the surgeon may be subjected to unfair and serious criticism after an unfavorable result. We do not, as a rule, operate under such circumstances at the University Hospital.

A STUDY OF THE RESPIRATORY SIGNS OF CHOREA MINOR *

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St. Vitus' dance, Sydenham's chorea, chorea minor, is a distinct clinical entity and while the specific organism or toxic agent has not been definitely isolated, the work in this direction and the natural history of the disease has been and is such as to warrant the deduction now generally admitted, that it is a disease of bacterial or toxic origin. Clinically, chorea minor may be readily differentiated from other conditions resembling it but the differentiation is not always made and hence other conditions are often erroneously diagnosed chorea. This is notably true of the tic conditions. Again the usually observed motor manifestations of chorea may be and frequently are so slight that the true nature of the trouble remains unrecognized. The reason for such errors in diagnosis and for the non-recognition of the disease in its milder forms is to be found in the habit of basing the diagnosis on the presence of abnormal movements about the head and extremities which are usually so prominent in this disease.

"Never found a diagnosis in any disease or condition on the presence of a single sign or symptom," is a working axiom never to be forgotten in diagnosis. There are to-day no pathognomonic signs and symptoms. We are able to recognize prominent signs and symptoms in every disease, but they are always associated with other signs and symptoms if we will but search for them and our diagnosis in any case is most secure when founded on the greatest number of these. No one should be content to diagnose measles by the rash; a brain tumor by an optic neuritis; typhoid fever by the rose spots or tabes dorsalis by the presence of ataxia. Neither should he be content to diagnose chorea minor by the presence of abnormal movements. Abnormal movements about the head and extremities are usually prominent signs of chorea minor but they are not pathognomonic of the disease and they may be so slight as to be overlooked. In chorea minor, the abnormal movements have certain characteristics and when they possess these, we call them choreic or choreiform movements and while they are not pathognomonic of the disease when we really recognize them as such in conjunction with other symptoms just as characteristic, the diagnosis of chorea minor is readily made.

It will help us in the study of the chief respiratory signs of chorea if we consider the characteristics of the true choreic movements. Choreic movements in chorea minor are never confined to one part of the body throughout the disease, they may be one-sided in the beginning but they usually become general; they are never confined to synergicly associated muscle groups, they are indefinite, vary in intensity and frequently

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908.

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