CORTICAL VISUAL DISTURBANCES FOLLOWING VENTRICULOGRAPHY AND/OR VENTRICULAR DECOMPRESSION

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This paper deals with severe visual disturbances noted after ventriculography or ventricular decompression in 5 patients harboring intracranial tumors. It also attempts to confirm a logical explanation, based on anatomopathological findings, for this unfortunate though rare sequence of events.

Perusing the available literature one is surprised to find but scant reference to the complications that may follow the introduction of air directly into the ventricular system or the drainage of the ventricular cavities. Only occasionally can one find words of caution about the rapidity with which to exchange air or to tap a ventricle in the surgical attack of an intracranial space-occupying lesion.

There can be no doubt about the usefulness of ventriculography and its high degree of accuracy in the detection of intracranial space-taking lesions. Nevertheless, it is felt that insufficient stress has been laid upon the rare but possible risks involved in the performance of such a test or of such a procedure as ventricular decompression, both of which occasionally may produce permanent sequelae.

It is a well known fact that, if an intracranial tumor is present, ventriculography will often aggravate the pre-existing neurological picture, forcing the neurosurgeon to perform immediate surgery. When the intracranial pressure is elevated, a sort of balance exists among various intracranial forces. This balance at times is very precarious and the mere puncture of a ventricle will upset it. It is easy to understand how a sudden shift of a mass of tumor or herniation of edematous brain can occur through the tentorial opening, under the free edge of the falx or through the foramen magnum. The introduction of air into the ventricular cavities will, of course, be even more disturbing as it is known that gases will expand in warmer environments, such as the cranial cavity. If it is remembered that the maintenance of the pressure balance is essential, it becomes obvious that the exchange between ventricular fluid and air must be carried out slowly. In this fashion it is hoped that a slow re-adjustment of intracranial-pressure relations will occur. This caution should apply not only to the diagnostic procedure itself but also at the time of surgery when the decompressive effect of the ventricular drainage is sought. These facts were well known to Lindgren who, discussing the radiological features of cerebral herniations in cases of increased intracranial pressure, cautioned that "... a puncture of the lateral ventricle, performed to decrease the intracranial pressure, may result in an increase in the herniation. ..." Schwarz and Rosner too pointed out that neurological signs and symptoms may be exaggerated not only by lumbar puncture or encephalography but also by ventriculography or even craniotomy.

This paper is concerned primarily with severe cortical visual disturbances which may follow ventriculography or ventricular decompression. Let us, at first, review briefly the pertinent literature.

Masson reported 6 patients, out of a series of 100 ventriculograms, who experienced such visual complications as temporary blindness or amblyopia. The author felt that

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in some cases they were somehow related to
the repeated punctures of the occipital lobe.
In other cases Masson could not offer any
satisfactory explanation for the temporary
blindness. He surmised that in some instances
the evacuation of the ventricular fluid, by
removing the support of the brain, may
disturb the circulation to the visual path-
ways and cortex, "as a result of a profound
change in the pressure conditions within the
cranial cavity even before any air had re-
placed the fluid."

Holmes,\textsuperscript{4} discussing the prognosis in pa-

tients with papilledema, noted that "occas-
ionally there is immediately after operation a
rapid deterioration of sight, which is, how-
ever, often transient; it is probably due to a
sudden fall of pressure in the central artery
of the retina as a result of too rapid relief of
intracranial tension, and is therefore com-
parable to the diminution of vision which some-
times follows a successful operation for
glaucoma." He also noted that "unfortu-
nately vision may also fail permanently after
a decompression operation in patients in
whom neither the ophthalmoscopic changes
nor the state of the visual fields had sug-
gested the risk of such a catastrophe." In
Holmes' experience, this tragic complication
had been most common after either partial
or complete removal of tumor from the
posterior fossa.

Rea,\textsuperscript{14} also discussing the prognosis of
vision in patients with papilledema, stated
that "those cases in which vision has been
lost after a decompression were probably due
to a further rise of intracranial pressure
following severe secondary haemorrhage,"
without elaborating any further.

Duke-Elder\textsuperscript{2} also was well aware of the
possibility of rapid deterioration of vision
after operation or decompression, but never
mentioned any direct relation between the
ventricular punctures and the ensuing blind-
ness.

David \textit{et al.},\textsuperscript{1} discussing the values of air
injections, stressed the point that ventricu-
lography is not entirely devoid of dangers.
They described disturbances in the visual
fields, hemorrhages along the tracts of the
needle, and even coma. In the discussion of
the paper, David mentioned the possibility
of occurrence of cortical blindness but offered
no explanation for it.

Schwarz and Rosner\textsuperscript{17} described signs and
symptoms that occur during herniations of
the hippocampal gyri through the incisura
and aggravated by lumbar punctures, en-
cephalography or ventriculographic examina-
tions or craniotomy, but failed to include
visual disturbances.

Reese\textsuperscript{15} was the first to draw attention to
the possible role of the posterior cerebral
arteries for the blindness that may occur
after ventricular tapping. He was concerned
primarily with the position of the posterior
cerebral arteries at their origin from the
basilar artery "... for here they are par-
ticularly vulnerable to pressure either from
prolapse of the temporal lobe through the
tentorium or elevation upward of the cere-
bellum," thus explaining the blindness by
cortical necrosis secondary to ischemia.

The author stressed the fact that extensive clinical
and experimental investigations have proved
definitely that anoxic anoxia, such as may
occur with pressure upon one or both poste-
rior cerebral arteries by the herniating parts
of the brain, may produce irreversible dam-
age to the central nervous system, resulting
in cortical blindness and even death. It is of
interest to note that the occipital cortex is
very susceptible to anoxia, more so than
other areas of the central nervous system.

Lossius\textsuperscript{8} observed and described 3 pa-
tients with complications following ven-
triculography, from a series of 600. This test
has been used by Lossius for many years as a
diagnostic procedure \textit{per se}, not necessarily
followed by craniotomy, even in the presence
of an intracranial mass lesion. This has made
possible the observation of some transitory
disturbances. Discussing the possible causes
for these visual disturbances, Lossius felt
that it was unlikely for the complications de-
scribed to result from a direct trauma upon
tissues conducting visual stimuli. The only
possibility would be an injury to the optic
radiations which run lateral to the posterior
horns; however, trauma produced in this
area certainly could not explain total amau-
rosis which would require extensive damage
to both optic radiations. The most likely
explanation was thought to be a “derange-
ment of the cerebral vasomotoric control
caused by the ventriculography,” which sets
up a “circumscribed or generalized vaso-
contraction by means of a reflex mechanism.”
The vasoconstriction would in turn cause
ischemia, the transitory nature of which
would explain the reversibility of the visual
phenomena. Why should the calcarine cor-
tex be involved particularly? Lossius justified
this by its very delicate function and its
high sensitivity to any disturbance in vas-
cular supply. Furthermore, he considered
the occipital pole as a peripheral part of
the brain, being supplied merely by the
finest, terminal branches of the posterior
cerebral arteries.

Walsh recognized the possibility that
bilateral homonymous hemianopsia may
result from ventriculography and stated that
in his experience this blindness has good
prognosis. He also remarked: “Temporary
depression or even temporary complete loss
of vision as a complication of ventriculog-
raphy occurs, but it is remarkable that per-
manent visual field defects are extremely
rare.” He also has been aware of the fact that
sometimes immediately after a craniotomy
there may be a transient diminution of vision
which, however, improves rapidly. In some
instances the cause of the visual loss has not
been clear, but he thought that probably
thrombosis or spasm of the vessels supplying
the visual cortices may have been responsible.
In a personal communication to one of the
authors (CET), Walsh agreed with the
thought that, in some cases at least, the
blindness may be cortical and caused by
interference with the vascular supply of the
visual cortices. It also has been theorized
that with sudden reduction of pressure by
ventricular tapping there may be something
in the way of incited spasm which, for un-
clear reasons, involves both posterior cere-
bral arteries only.

Sachs mentioned the case of a child who,
following removal of a posterior-fossa tumor,
became and remained blind. He stated that
“Fortunately the vision rarely goes suddenly,
after pressure has been relieved by the re-
moval of a tumor. Such an experience, when
it does happen, is most trying and is difficult
to explain.”

OUR EXPERIENCE

Case 1 (B 28 73 71). J.C., a 21-year-old colored
male, was admitted on May 29, 1958, with a
1-month history of bilateral parieto-occipital
headaches, occasional nausea, vomiting and
diplopia.

Examination. The patient had bilateral papill-
edema without hemorrhages. No evidence of ex-
ternal or internal ophthalmoplegia was noted.
Gross visual acuity was normal. Muscle-stretch
reflexes were active and equal. Roentgenograms
of the chest and skull were within normal limits.
Although no definite history of head injury was
obtained, it was felt that the patient might have
been suffering from a chronic subdural hematoma,
while a mid-line intracranial tumor had to be con-
sidered also.

Operation. On May 30, 1958 bifrontal and right
temporal burr holes were made. No clot was
found on the surface. Three days later ventricu-
lography was performed through the right frontal
burr hole. Roentgenograms suggested a tumor in
the posterior portion of the 3rd ventricle, possibly
a pinealoma. A right ventriculocervical subarach-
chond shunt was done under general anesthesia, us-
ing a No. 10 French catheter. No excessive change
of blood pressure occurred during the entire pro-
cedure, but the pulse rate did go up to 120 at the
time of cannulation of the ventricle and remained
in that range until the end of the operation.

Postoperative Course. The patient was very
restless, uncooperative and confused. On the 2nd
day, he complained of total blindness. The pupils
were equal and reacted to light promptly. The
fundus showed no gross change. On June 5, how-
ever, i.e. 3 days after operation, the patient began
to perceive light and 2 days later he was able to
count fingers held close to his eyes. No further
improvement of vision occurred until a few days
after roentgen-ray therapy was begun. At the
time of discharge, the patient was able to read
large printed letters.

On July 9, 1958 the patient was seen in the
Out-Patient Clinic. He had no headache, but his
vision was still quite poor, especially in the left
eye. Funduscopic examination disclosed early
secondary optic atrophy. There was a coarse, well
sustained horizontal nystagmus, with the quick
component to the left and no evidence of opthal-
moplegia. The patient failed to report again for
follow-up until April 2, 1959, at which time he
was brought to the Emergency Room, deeply unconscious. His wife stated that since February he had begun to lose the use of both arms and legs and that the day before he had become very stuporous. On admission, a ventricular tap was done with a spinal-puncture needle, through the right frontal burr hole, and it yielded clear colorless cerebrospinal fluid under markedly increased pressure. The patient was given supportive therapy. He expired on April 3, 1959.

**Autopsy.** A very large mass of tumor, with indistinct boundaries, was found to occupy most of the 3rd ventricle and to extend into both thalami, hypothalamus and the tegmentum of the brain stem (Figs. 1 and 2). The tumor was partly cystic and showed several areas of necrosis and hemorrhages. The microscopic picture was compatible with an astrocytoma Grade III or IV. Both occipital lobes were normal. The posterior cerebral arteries were patent.

**Comment.** In this case, only one puncture of the right posterior horn was made when the French catheter was introduced for the shunting procedure. This would hardly interfere with the anatomical continuity of the visual pathways or of the visual cortex, and thus explain blindness. The only reasonable explanation would have to be found in a temporary, though pronounced disturbance of the vascular supply to both visual cortices without gross or microscopic evidence of infarct. This could have been produced by interference with the circulation through the posterior cerebral arteries at the incisural level, in two possible ways: (1) upward herniation of cerebellar tissue through the tentorial hiatus, with or without shift of the brain stem, at the time of the ventricular puncture or at the time of introduction of the catheter into the posterior horn; (2) downward herniation of the unci at the time of opening of the cisterna magna and the upper cervical subarachnoid space with rapid release of large amount of cerebrospinal fluid. The persistence of the pupillary light reflex certainly points toward a cortical lesion. Furthermore, no evidence of brain-stem disturbance was noted during the immediate postoperative period. No drop in blood pressure occurred and this would rule out a systemic cause for the blindness. Finally, because of its enormous bulk, mostly situated in the median and paramedian planes, a sudden "movement" of the entire mass of tumor could have occurred. Whether or not this contributed to the production of the visual disturbances, it is hard to say.

**Case 2** (B 25 06 19). E.B., a white male, was seen first in 1955 at the age of 47. At that time he was complaining of pain in the right shoulder of about 8 weeks' duration, following a fall from a ladder. He was treated conservatively and apparently did well until September 1956, when he started to notice weakness in all extremities, especially in both legs; he was staggering and had a peculiar feeling of electric shocks running from his neck to his legs. The only positive finding at this time was an ankle clonus on the right and
generalized hyperactivity of all his muscle-stretch reflexes. A cervical myelogram was done and revealed no abnormalities.

The patient was readmitted in July 1958, again with complaints of weakness in both legs. At this time, however, he was found to have palsy of the left 6th nerve and asymmetry of the face with a very mild left facial weakness of central type. The fundi were normal and so were roentgenograms of the skull.

By Dec. 5, 1958, the patient began to complain of failing vision and headaches. The fundi showed bilateral papilledema, more pronounced on the right side, with a few hemorrhages. The right blind spot definitely was enlarged. No field defect was found.

When admitted to the Neurosurgical Department, he was found to have severe spastic paraparesis with bilateral ankle clonus and positive Babinski's sign. He had bilateral palsy of the 6th nerve and a clear-cut Parinaud's syndrome.

On Dec. 23, 1958, about 3 years after the onset of his symptoms, ventriculography was done under local anesthesia. The right posterior horn was punctured lowermost and 110 cc. of cerebrospinal fluid were exchanged with 100 cc. of air. The roentgenograms were compatible with the diagnosis of pinealoma.

Operation. A Torkildsen procedure was done under general anesthesia. No change in the vital signs occurred at any time during the operation except for speeding up of the pulse rate.

Postoperative Course. The 1st day was characterized by severe lethargy and tachycardia. His pupils were very large and reacted poorly to light. It was not clear at this time whether the patient could see or not. On the 2nd day, it was certain that the patient could not see at all. His state of consciousness had cleared up remarkably, but he still would not complain about his loss of vision. From Dec. 28, 1958 on, the patient became less and less responsive. Spikes of temperature would occur every day, although there was no clinical or laboratory evidence of infection. On Jan. 9, 1959, after some temporary improvement, the patient was started on roentgen-ray therapy, following which there appeared to be some slight mental improvement. His vision never returned.

The patient was sent to a City Home from which he was returned on Feb. 27, 1959, when he expired.

Autopsy. A large tumor was found in the pineal region (Fig. 3). It involved the tegmentum of the midbrain. The histology was that of a pinealoma. The posterior cerebral vessels were normal. The calcarine cortices were normal both macro- and microscopically.

Comment. The patient had visual anosognosia, i.e., he was unaware of his blindness (Anton's syndrome). This occurs only with cortical lesions. The sluggish light reflexes would at first seem to disprove the cortical origin of the blindness; however, one must take into account the fact that the tegmentum of the brain had been involved directly by the neoplastic process and indirectly by pressure.

The sequence of events may be postulated to have been similar to that in Case 1.

Case 3 (B 29 78 85). M.D., a 25-year-old white female, was admitted on Oct. 23, 1958, with the chief complaint of feelings of pressure in the head since the preceding April when she began to notice "spinning" of the head and some impairment of balance, most marked on walking, with some clumsiness of her hands. She had no complaints about her vision.

Examination. The pupils were equal and reacted to light and convergence. There was no ophthalmoplegia. The fundi showed mild secondary optic atrophy with some blurring of both nasal margins. The muscle-stretch reflexes were hyperactive. There was some questionable weakness of the left side of the face and of the left extremities. Speech was slow and bursting in character.

A spinal tap showed a pressure of over 300 mm. of water. No cerebrospinal fluid was removed except for the fluid that entered the manometer. Roentgenograms of the skull showed thinning of the posterior clinoid processes. Electroencephalogram revealed a mild generalized slowing.

Operation. On Oct. 27, 1958 posterior parietal burr holes were made under local anesthesia. The right posterior horn was tapped at first attempt and cerebrospinal fluid escaped under very high pressure; 147 cc. of fluid were exchanged with 140
cc. of air. Films revealed a constant filling defect of the posterior part of the 3rd ventricle. No upward effect followed the test. A right ventriculocisternal shunt then was performed under general anesthesia, using a No. 10 French catheter.

Postoperative Course. Upon completion of the procedure, the patient failed to respond except to very painful stimuli. The eyes were conjugated forcibly to the left and downward. The pupils were very small and fixed. A left ventricular puncture was done (the right posterior horn had been used for the shunt). No air came out and only 1 cc. of slightly bloody fluid escaped. The first postoperative day was characterized by severe mental confusion and incoherent talking. The vision could not be checked because of very poor cooperation. After a stormy course she began to respond better and for the first time it became possible to detect a total blindness. It also was noticed that she could hardly move her limbs. The pupils were now mydriatic and fixed.

On Nov. 13, 1958 roentgen-ray therapy was begun. On November 17 the pupils were still very large, but a sluggish light reflex was present. At the time of discharge on Nov. 22, 1958 the patient was able to sit up in a wheel chair and spontaneous movements of her limbs were possible, though very weak and limited. At no time did the patient complain about her blindness. Extensive physical rehabilitation was continued on an out-patient basis. Another course of roentgen-ray therapy was given in January and another one in April 1959.

The patient was last seen in the Clinic on May 22, 1960. The pupils were still large, but reacted to light very promptly, both directly and consensually. No gross perception of light was present. The family told us that at times the patient stated that she was able to see her son and her husband for short periods of time!

Comment. In this patient, pronounced brain-stem disturbance followed the ventriculography and/or the ventriculocisternal shunt. The blindness was immediate and, at first, the pupillary reaction was lost, probably because of mid-brain damage. When the latter cleared up, there remained a typical picture of cortical blindness, ignored by the patient, with pupils reacting promptly to light.

Case 4 (B 22 14 87). T.M., a 4-year-old white female, was admitted on Jan. 26, 1960, at which time she appeared somewhat lethargic and disoriented.

Examination. The fundi showed marked bilateral papilledema and pronounced venous distention. A few small retinal hemorrhages also were present. Roentgenograms of the skull showed digitate impressions. On January 30, ventriculography was carried out under general anesthesia. The left posterior horn was tapped, at first attempt, lowermost. The ventricular pressure was very elevated and several cc. of ventricular fluid gushed out of the ventricular cannula. After an exchange of 50 cc. of fluid with air, roentgen-ray films showed symmetrical dilation of the entire ventricular system compatible with a subtentorial tumor.

Operation. A posterior-fossa craniectomy was carried out at once. The left posterior horn was tapped and fluid and air escaped rapidly under pressure. The dura mater then was opened and a large mass of tumor was found to fill the entire 4th ventricle and to extend upward in the direction of the aqueduct, and downward into the upper cervical canal. Subtotal removal of the tumor was accomplished. It proved to be a Grade I astrocytoma.

Postoperative Course. The child did very well except for blindness. The pupils were mydriatic but reacted very promptly to light, both directly and consensually. The fundi showed no change from the preoperative status. On Feb. 2, 1960, i.e. 3 days after operation, she began to perceive a strong flashlight. The next day she was able to perceive large moving objects without recognizing their shape. From then on, rapid recovery occurred. On Feb. 19, 1960 she was started on roentgen-ray therapy. No further visual disturbances were noted.

Comment. The presence of the light reflex and the total blindness again point toward a bilateral cortical involvement. It may be assumed that the lesion responsible for the transitory but severe visual disturbance may have been produced either at the time of the ventriculography or at the time of the ventricular decompression prior to the opening of the cisterna magna. The sudden release of intraventricular pressure conceivably encouraged an upward cerebellar herniation through the tentorial hiatus, disturbing the flow of blood through the posterior cerebral arteries sufficiently enough to produce hypoxia, which in turn caused the blindness. This also could be secondary to a downward movement of cerebral tissue, i.e. temporal uncal herniation, at the time of release of the infratentorial pressure. The former mechanism, however, seems more likely as, before the dura mater was opened, the supratentorial pressure had already been relieved by the cannulation of the left posterior horn.
Case 5. I.R., a 32-year-old white female, was admitted on Dec. 20, 1959 with the complaint of nausea and vomiting and weakness of her left extremities. In 1957 this patient had had a malignant melanoma removed from the skin of the midthoracic area with a wide excision.

Examination. Her left hemiparesis was spastic, with brachiofacial predominance. No gross sensory deficit was present. The visual fields were normal on gross confrontation. There was mild bilateral papilledema. The patient was lethargic but easily arousable.

Right carotid angiography was done under local anesthesia. The films were diagnostic of a malignant tumor, the size of a golf ball, in the low posterior-frontal area.

Operation. A right frontotemporoparietal osteoplastic flap was elevated. Immediately overlying the cortex of the motor area a mass of tumor was seen, brownish in color, and very soft in consistency. The entire mass was removed. Before opening the dura mater, the right anterior horn was tapped, cerebrospinal fluid draining out freely.

Postoperative Course. The immediate period was characterized by some drowsiness and by persisting left hemiparesis, most marked in the face and arm. The patient was coherent and offered no visual complaint. Her funduscopic picture was not changed. On Dec. 23, 1959, i.e. 2 days after the craniotomy, she became comatose. The right pupil became large and fixed and a mild decerebrate reaction developed. The right anterior horn was tapped again. A few mm. of cerebrospinal fluid escaped under very high pressure. Intravenous urea was given. The pupils became equal and the patient began to respond to stimuli. She was taken to the operating room, the bone flap was removed and the dura mater was left wide open. On December 29, she became coherent enough to be tested. She was found to be completely blind. The papilledema had not changed. The pupils were equal and reacting. The blindness persisted until discharge on Jan. 14, 1960, at which time the decompression was soft and the motor defect was very minimal.

The patient was readmitted on Feb. 19, 1960 in a comatose state and expired shortly thereafter.

Autopsy. There were extensive visceral metastases of malignant melanoma. The cerebral cortex contained several small subpial and subcortical metastatic nodules. A large hemorrhagic mass of tumor was found in the left thalamus, extending into the internal capsule and into the ventricular system (Figs. 4 and 5). Another nodule was present in the inferior lip of the left calcarine fissure. Bilateral cortical infarction of the occipital lobes was present. The posterior cerebral arteries were normal. There was no evidence of herniations or pressure cone.

Comment. In this case, the ventricular punctures may not be held responsible for the infarctions of the occipital lobes, but could have contributed to a temporary herniation compressing both posterior cerebral arteries. The craniotomy also may have been another factor. The most important reason may have been postoperative cerebral edema, which was at least temporarily controlled by the ventricular punctures and the administration of urea. The anatomical specimen, however, shows very clearly how infarctions may occur in the calcarine and paracalcarine cortices, in the distribution
of the posterior cerebral arteries, without visible alteration of these vessels.

DISCUSSION

Case 5 confirmed an explanation for the cortical visual disturbances that may follow ventriculography or ventricular decompression in cases of increased intracranial pressure, i.e.: impaired circulation within the area supplied by one or both posterior cerebral arteries, as postulated by Reese. Perhaps some other extraneous factor, such as lowering of the blood pressure at the time of operation, may contribute to the death or grave functional disturbance of the neurons of the visual cortices and explain the extreme rarity of such complications. If the light reflexes remain present or return after some time, in the presence of blindness, one must assume that the lesion responsible for the visual loss is at a cortical level or in the visual pathways distal to the lateral geniculate bodies, which are the stations through which the fibers for the light reflex relay before entering the brain-stem centers. In one case (Case 3) typical visual agnosia was present, with denial of blindness. It is felt, therefore, that in all the cases described above, except perhaps Case 5, a definite time relation had existed between the ventricular puncture and the onset of the ensuing visual loss. In all these cases, except Case 5, in which the anterior horn was tapped, the posterior horns had been entered without difficulty. The anatomical specimens in Case 1 and Case 2 failed to show any evidence of injury to the visual pathways by the ventricular cannula or by the French catheter inserted at the time of the Torkildsen procedure. Excluding therefore direct injury to the visual pathways, the explanation must be found in the sudden change in pressure relations within the cranial cavity brought about by the decompression of the ventricular system and sudden release of cerebrospinal fluid. The disturbance of visual function of cortical type can be explained only by sudden herniations, either of the temporal lobes or of the cerebellar hemispheres, through the tentorial incisura as first described by LeBeau and later by Ecker. These herniations in turn strangulate, at least temporarily, the posterior cerebral arteries as they sweep around the cerebral peduncles, causing interference with the vascular supply to the visual cortices which are situated peripherally and contain cells extremely sensitive to hypoxia.

The possibility of the above-described anatomopathologic sequence had been demonstrated (although not attributed, as in these cases, to ventricular puncture) for the first time by Meyer who described tentorial herniations accompanied by changes in the distribution of the posterior cerebral arteries. Jefferson too had stated that "No other intracranial vessel is vulnerable in this manner in anything approaching the same degree" referring to the posterior cerebral arteries. Confirmation of this pathological occurrence is to be found in a very detailed paper by Moore and Stern who presented 5 cases of calcarine infarctions. In all cases the extra- and intracerebral vessels, feeding these areas, were found to be normal. They stressed the point that localized cortical softenings, such as they described, can occur without the accompaniment of a vascular lesion in the brain stem and, therefore, do not necessarily produce alarming clinical pictures. The authors felt that whether the vascular lesions are produced by mechanical obstruction or by functional anomalies of the vessels, the increased pressure acting as the sole factor cannot be the cause of the lesions. They believed, and it is felt that this may be true indeed, that "acute" changes in intracranial-pressure relations, caused either by acute swelling of the brain or by a sudden release of pressure in the supra- or subtentorial space, produce the train of events that lead to the infarction of the occipital lobes. Lyle also very clearly demonstrated the different types of tentorial herniations but failed to include, among the different structures that may herniate through the hiatus, the cerebellum, in an upward type of mass movement. The author described the Cone-Reid phenomenon, which consists in a dilated pupil and occipital-lobe infarction with resulting visual-field changes, as a complication of
these herniations. He felt that "... the visual field loss at first is reversible and is produced by anoxia and ischemia resulting in functional impairment which may be transient if the pressure is relieved."

This may explain in part the extreme rarity with which this complication is encountered. Why were infarcts not found in Cases 1 and 2? An explanation may be found in the fact that death of nerve cells or loss of neural function can occur without visible changes in the neurons. Furthermore, it is to be noted that cases of cortical blindness following circulatory arrest, with recovery in ensuing weeks or months, are many and well documented thus making recovery, though delayed, quite possible in these instances.

SUMMARY

Five cases are presented in which blindness followed ventricular decompression. The literature is reviewed in this respect. It is stressed that such occurrences are extremely rare. A possible explanation is offered: ischemia of the visual cortices secondary to compression of the posterior cerebral arteries against the tentorium cerebelli following sudden release of intraventricular pressure and subsequent cerebral or cerebellar herniation. The clinical course and the pathological findings of these cases are given in support of this assumption.

REFERENCES

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