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 pathways and cortex, "as a result of a profound change in the pressure conditions within the cranial cavity even before any air had replaced the fluid."

Holmes, describing the prognosis in patients with papilledema, noted that "occasionally there is immediately after operation a rapid deterioration of sight, which is, however, 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 comparable to the diminution of vision which sometimes follows a successful operation for glaucoma." He also noted that "unfortunately 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 suggested 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, 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 was also 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 blindness.

David et al., discussing the values of air injections, stressed the point that ventriculography 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 described signs and symptoms that occur during herniations of the hippocampal gyr through the incisura and aggravated by lumbar punctures, encephalography or ventriculographic examinations or craniotomy, but failed to include visual disturbances.

Reese 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 particularly vulnerable to pressure either from prolapse of the temporal lobe through the tentorium or elevation upward of the cerebellum," 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 posterior cerebral arteries by the herniating parts of the brain, may produce irreversible damage 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 observed and described 3 patients with complications following ventriculography, from a series of 600. This test has been used by Lossius for many years as a diagnostic procedure 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 described 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