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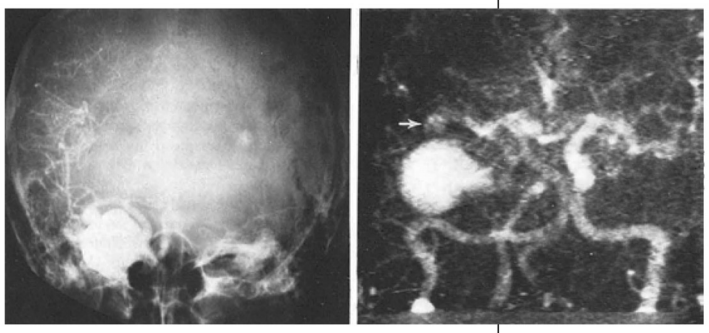

Intravenous Video Arteriography of the Intracranial Vasculature:

Early Experience

A computerized fluoroscopic apparatus developed by members of the University of Wisconsin Medical Physics Section was used for 12 months to perform intravenous video arteriography. In previous papers, the apparatus was described and its use was illustrated for performing time subtraction intravenous video arteriography of the extracranial carotid arteries, the arteries of the abdomen and extremities, as well as angiocardiography. In this report, the use and current limitations of this technique for evaluation of the intracranial vasculature are described and illustrated.

Until recently, it has been impossible to achieve satisfactory visualization of the intracranial vasculature following intravenous administration of contrast medium [1–3]. New digital electronic techniques permit processing of the video signal from a conventional image-intensified fluoroscopic system for isolation of the small iodine signal produced after the intravenous injection of contrast medium. Logarithmic amplification combined with subtraction can increase the ratio of the iodine signal to the signal variation of normal anatomic structures by a factor of greater than 100. This type of image processing ensures uniform visualization of the iodine as it passes from the heart to the arteries of the neck and into the cranial vault. Our experience in demonstrating the intracranial arteries with this new method is described.

Materials and Methods

For examination of intracranial vascular structures, mask mode radiography is employed. An image is obtained before arrival of the contrast medium and is stored in one of two memories. As the iodinated contrast medium passes through the vasculature of the skull and brain, serial images are collected in a second memory, subtracted from the mask, and are then stored on a video disk and video tape. For examination of the intracranial vasculature, the exposure factors are 300 mA, 1/4 sec at 65–85 kVp. Images are generated at the rate of 1/sec. Further details of this imaging sequence have been reported previously [4–6].

Since our initial clinical reports [7, 8], our technique for intravenous injection of contrast medium has been modified. Previously, we injected 40–60 ml of contrast medium at the rate of 12–14 ml/sec through a 16-gauge, 5-cm-long Angiocath inserted into a basilic vein. While using this technique for over 300 intravenous video arteriograms, we encountered three instances of extravasation of the contrast medium at the injection site. To eliminate this potential complication and to obtain a more consistent contrast bolus, we now use a percutaneously introduced 5 French catheter with an end hole and four side holes, positioning the tip in the central innominate vein or superior vena cava.

For examination of the petrous, cavernous, and supraclinoid parts of the internal carotid artery, a projection which positions the top of the petrous bones in the center of the orbit seems to provide the best results. Such a projection is also satisfactory for visualization of the distal segments of the vertebral arteries and of the major part of the basilar artery. Because our resolution is currently limited by focal spot size (over 1.1 mm during the exposures), and a large magnification factor (greater than 1.5) caused by an under-the-

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Computed Tomography in 75 Clinical Cases of Syringomyelia

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D. Bar

Seventy-five patients with a clinical diagnosis of syringomyelia computed tomography after intrathecal injection of metrizamide. A demonstrated in 67 patients. Tilting the patient head down did not i cavity opacification. This evidence favors transneural migration of m cavity. The spinal cord was measurably enlarged in only a minority some, the cavity appeared to have clefts or wall defects. These res according to the etiopathogenic theories advanced by Gardner, Abou

The anatomic basis for the clinical syndrome of syringomyelia the spinal cord. This cavity may be a dilated central canal (hyd within the parenchyma (syringomyelia), or both (syringohydro theories have been proposed to explain the phenomenon According to Gardner et al. [1–3] (fig. 1A) failure of the foram ventricle to open at the 29th week of fetal life with continuing between the fourth ventricle and the central canal allows inc within the ventricles to be transmitted to the central canal, whic same time, the hydrocephalus causes inferior displacement of thus creating an Arnold-Chiari malformation.

In the Williams [4] theory (fig. 1B), the resulting anatomi approximately the same, except for the often observed lack o According to this theory, efforts such as the Valsalva maneu creased venous pressure, which raises the cerebrospinal fluid the ventricles, which in turn is transmitted to the central canal, w efforts induce venous hypertension in the medullary veins and in the cavity.

Aboulker [5] believes that the Gardner et al. [1–3] theory is only valid for children who have hydrocephalus frequently associated with meningocele. According to Aboulker (fig. 1C), the pathogenesis of the clinical syndrome of the syringomyelia in adults is different: a cavity develops within the parenchyma without any relation to the central canal which, in adults (according to pathologists mentioned by Aboulker [6–8]) is often collapsed, closed, or absent. The abnormality is caused by stenosis at the level of the foramen magnum due to Arnold-Chiari malformation, arachnoiditis, or tumors.

This theory is supported by canine experiments performed by Sato et al. [9] that demonstrated that 30% of cerebrospinal fluid is produced in the spinal canal. Due to the stenosis at the foramen magnum, the cerebrospinal fluid finds its way with difficulty towards the intracranial areas of resorption. The pressure of the cerebrospinal fluid within the spinal canal is higher than that of intracranial cerebrospinal fluid, resulting in edema within the cord. The last phase of this edema is a cavity. This mechanism is potentiated by increased venous pressure in the azygos system, which may be congenital or acquired.

According to Aboulker [5], the cerebrospinal fluid filters through the parenchyma or via a pathway along the posterior roots. Ball and Dayan [10] thought

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