VASCULAR MALFORMATIONS IN THE REGION OF
THE GREAT VEIN OF GALEN

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This report concerns arteriovenous malformations in the region of the pineal gland. The 5 cases reported here and similar cases reported in
the literature have several factors in common. All are of congenital
origin, are arteriovenous shunts with venous drainage into the great vein
of Galen, and appeared to be amenable to surgery. They often are associated
with hydrocephalus. In 2 of the 5 cases the communication was between the
anterior cerebral artery group and the great vein of Galen, in 2 it was be-
tween the posterior cerebral artery group of vessels and the great vein of
Galen, and in 1 it was between the superior cerebellar artery and the great
vein of Galen. In each of the cases the diagnosis was established by angiogra-
phy. When therapy was instituted it was ligation of the vessels on the arterial
side and in these instances postoperative angiography revealed failure of
filling of the vascular malformations.

The first report found in the literature of arteriovenous malformations
leading into the great vein of Galen was by Jaeger, Forbes and Dandy7 in
1937. They described a communication between the circle of Willis and the
great vein of Galen in a 4-year-old child whose symptoms began at approxi-
mately the age of 8 months. The child had an internal hydrocephalus. This
they attributed to the greatly dilated great vein of Galen pressing on the
aqueduct. Because the patient had no history of trauma, they concluded
the aneurysm was congenital.

In 1940 Russell and Nevin11 reported 2 cases in children. In one the aneu-
rysm was a complex sacculated structure communicating between the left
posterior cerebral artery and the great vein of Galen. There was an associ-
ated absence of the sigmoid portions of both lateral sinuses. In their second
case the arteries coming over the corpus callosum connected with the great
vein of Galen. In both cases there was an internal hydrocephalus.

In 1945 Alpers and Forster1 reported on an 18-year-old boy with bilateral
hydrocephalus secondary to a calcified arteriovenous malformation in the
pineal region. The arterial supply came through the posterior cerebral or
the choroidal arteries and the venous drainage was through the great vein
of Galen. Alpers and Forster doubted that involvement of the great cerebral
vein is capable of producing internal hydrocephalus. They mentioned the
investigations of Bedford2,4 indicating that occlusion of this vein in the
dog is incapable of producing internal hydrocephalus despite scattered re-
ports to the contrary. They believed that the hydrocephalus more probably was caused by pressure and occlusion of the aqueduct of Sylvius.

In 1947 Oscherwitz and Davidoff\(^5\) reported on a 27-year-old woman with a calcified aneurysm situated in the midline between the occipital lobes. The arterial supply was from the branches of the circle of Willis and the venous drainage was into the great vein of Galen. There was no hydrocephalus.

In 1949 Boldrey and Miller\(^6\) reported 2 cases of arteriovenous fistulas connecting the arteries of the circle of Willis and the great cerebral vein of Galen. They treated their first patient with ligation of the external and internal carotid arteries in the neck and considered this therapy of distinct value. It is difficult to understand how this would improve the patient unless the temporary diminution of blood going through the fistula would permit some thrombosis within the lesion. However, no mention was made of such thrombosis.

Olivecrona and Riives\(^9\) in 1948 reported on various types of arteriovenous aneurysm of the brain, but it could not, from their report, be ascertained if any of them were between the anterior or posterior cerebral arteries and the great vein of Galen.

Gillingham\(^8\) in 1953 reported 1 patient with an arteriovenous malformation between the cerebral vessels and the great vein of Galen. He emphasized that unless vertebral angiography is carried out as a routine in addition to carotid angiography, in the more posteriorly placed malformations the feeding vessels from the posterior cerebral artery often are not demonstrated. He also stated that it is helpful to perform vertebral angiography with simultaneous carotid compression on the same side as the lesion. By this maneuver, minor implications of the posterior cerebral artery can be more easily demonstrated. Carotid compression, he stated, having robbed the malformation of its principal supply from the middle cerebral artery permits filling of the lesion from the posterior cerebral flow.

Also in 1953 Askenasy, Herzberger, and Wijsenbeek\(^3\) reported on the association of hydrocephalus with vascular malformations of the brain. The purpose of their report was to emphasize the fact that hydrocephalus may be caused by congenital malformation coincident with the cerebral vascular abnormality, or by recurrent small hemorrhages occurring in the subarachnoid space with the production of an adhesive leptomeningitis and subsequent closure of the subarachnoid spaces, or by a faulty distribution of blood and subsequent oxygenation of the brain. Lemmen and Schneider\(^5\) emphasized that obstructive hydrocephalus may occur secondary to vascular abnormalities in the third ventricle.

**EMBRYOLOGY**

The internal carotid artery appears first at approximately the 3 mm. stage of the embryo. At 4 mm. two divisions are apparent. The anterior division gives rise to the anterior and middle cerebral and the anterior chorio-