Extra- to Intracranial Arterial Anastomoses in Therapeutic Embolization:
Recognition and Role

Anastomoses from extra- to intracranial vessels have been shown in normal patients
and, more commonly, in patients with arteriosclerotic occlusive disease, arteriovenous
malformation, and postligation of the carotid artery. These channels may be of clinical
importance during therapeutic embolization and probably account for two cases of
complication, posterior fossa strokes that occurred after blockage of occipital arteries.
By identifying these channels and then monitoring the ever-decreasing vessel accept-
ance of contrast agent and emboli, patient safety may be enhanced.

In 1953, Richter [1] angiographically showed anastomoses between the occip-
al artery and the vertebral artery in two patients with internal carotid artery
thrombosis. Subsequent reports by others [2–5] demonstrated frequent ex-
amples of extracranial anastomoses in patients with arteriosclerotic occlusive
disease and also in patients with arteriovenous malformation. In 1974, Seeger et
al. [6] indicated these anastomoses could be seen to varying degrees in patients
without known vascular abnormalities. They also reported [6] that visualization of
these channels was sometimes entirely technique dependent. Channels may fill
by a superselective approach but not by injection into the main arterial trunk.

The wide acceptance of therapeutic embolization to obliterate the vascular
abnormalities of head and neck makes recognition of these channels important.
Our attention was forcefully drawn to this need after we caused two severe
posterior fossa strokes after embolization of the occipital artery and its branches.

This report reviews these extra- to intracranial anastomoses, indicates the
need for careful preembolization selective angiography, and suggests an embol-
ization technique should those channels be present.

Materials and Methods

In the past 5 years, 37 patients at the University of Oregon Health Science Center and
University of Pittsburgh School of Medicine have undergone therapeutic embolization of
extracranial vascular abnormalities.

All patients were evaluated prior to embolization with small catheter (5 French or smaller)
superselective angiography. Primary magnification and subtraction techniques were rou-
tine.

Examinations and embolizations were performed under local anesthesia (except in
infants and young children). Diazepam premedication, medical hypnosis, and Fentanyl
during the procedure allayed the pain and fear.
Fig. 1.—54-year-old woman with large glomus jugulare tumor. A–C. Selective angiograms (0.5 sec interval) of left occipital artery. Vascular mass lesion and main feeder, branch of occipital artery. D. Left common carotid angiogram before embolization. E. Angiogram after embolization. Complete occlusion of occipital artery. Internal carotid artery remains patent.
Fig. 2.—68-year-old man with extracranial arteriovenous malformation. Left common carotid angiograms. Early (A) and late (B) arterial phases. Markedly dilated occipital artery and branches supply malformation.

Fig. 3.—16-year-old boy with occipital scalp arteriovenous malformation. Selective left external carotid angiogram, arterial phase. Dilated occipital artery supplies malformation. Opacification of basilar artery and left vertebral artery via muscular branches (arrow).

Fig. 4.—14-year-old boy with nasopharyngeal angiofibroma. Selective angiogram of right ascending pharyngeal artery. Vascular lesion in nasopharynx. Right vertebral artery filled via muscular branches (arrow).

Findings

Two of the first four patients suffered severe posterior fossa stroke. A 54-year-old woman had a large left glomus tumor. Angiography delineated its blood supply (fig. 1). A 3 French coaxial microcatheter was directed through a 5 French angiography catheter into the main feeder, a branch of the occipital artery, and tantalum-impregnated isobutyl 2-cyanosacrylate was infused (Bucrylate, Ethicon, Inc., Somerville, NJ 08876). An error in mixing the tantalum made radiographic control of the infusion difficult; too much was injected, and the main trunk of the occipital artery was also blocked (fig. 1E). The patient became unresponsive, semicomatose, moved all extremities poorly, and developed within moments a bradycardia with arrhythmia and a left seventh nerve palsy. After supportive treatment, recovery was only partial.

A 68-year-old man had an occipital arteriovenous malformation (fig. 2). The left occipital artery was selectively catheterized, and gelfoam powder was introduced suspended in a contrast agent. Almost immediately he became unresponsive. His blood pressure dropped 30–40 mm, eye movements were random but conjugate, and he became quadriparetic. No follow-up angiography was performed. After supportive measures, recovery was incomplete.

As a result of these strokes, we began searching for extra- to intracranial anastomotic channels. We found these six times in the next 33 patients (18%). Most commonly
these communications were between the vertebral artery and the occipital artery (fig. 3). However, these were also seen between the ascending pharyngeal artery and the vertebral artery (fig. 4), the deep cervical artery and the vertebral artery (fig. 5), and the middle meningeal and internal carotid arteries via the ophthalmic artery (fig. 6). These anastomotic channels were predominantly shown by selective angiography (fig. 7).

Discussion

Treatment of vascular abnormalities of the head and neck by therapeutic embolization is becoming well accepted [7–10]. Although the initial wave of enthusiasm has passed, the complications and risks of these procedures, and more importantly, the factors which increase patient risk, have not yet been stressed in the literature.
Passage of emboli to normal regions of the brain may result in ischemic or toxic damage. To prevent emboli from going astray—from refluxing back along the catheter once the desired occlusion has been accomplished—some have advocated blocking the vessel by wedging the catheter in place or more recently by occluding the artery completely with a balloon-tipped catheter [11]. We suggest a more physiologic approach [12, 13]. Emboli are first suspended in contrast agent. The appropriate artery is catheterized atraumatically to prevent spasm, then the emboli are injected. The angiographer watches the infusion which has become readily visible because of the contrast agent, with fluoroscopy. Monitoring the ever decreasing vessel acceptance of emboli allows slowing of the introduction rate. With this technique the angiographer maintains precise and instantaneous control of the occlusion process. We have successfully used this technique in one patient with patent occipital to vertebral artery anastomosis and in another patient with external carotid–ophthalmic artery patient anastomosis. Anastomotic channels that connect the extra- and intracranial arteries are patent in many normal patients, and are even more common when abnormalities such as arteriosclerosis or arteriovenous malformation are present. If unrecognized, these may be a hazard to those undergoing therapeutic embolization. We have caused two severe strokes, probably by embolizing the posterior circulation through the occipital to vertebral artery anastomoses. We wonder if balloon occlusion of arteries during embolization, advocated to enhance safety, will not in fact open and encourage emboli to pass through these channels, and we suggest that a more physiologic, low pressure injection technique be evaluated.

REFERENCES

13. Kerber CW. Safe end-point determination during therapeutic embolization (or when should I stop?). Presented at the annual meeting of the American Society of Neuroradiology, Toronto, May 1979