
Intraoperative Angiography

To The Editor: I read with interest the article by Martin, et al., on intraoperative digital subtraction angiography (Martin NA, Bentzon J, Viñuela F, et al: Intraoperative digital subtraction angiography and the surgical treatment of intracranial aneurysms and vascular malformations. J Neurol Surg 73:526–533, October, 1990). It is encouraging to read their advocacy of intraoperative angiography for the various vascular lesions. There can be no doubt that it is a tremendous advantage to be able to see immediately whether a clip is satisfactorily placed or if an arteriovenous malformation (AVM) is completely obliterated and, if not, just where the residual lies. It is also very helpful to see what is being embolized and to embolize immediately before surgical attack rather than hours or even days before.

I would take exception to one small consideration. Unlike their findings, we used the intraoperative cassette holder and regular film, and found that our quality of film was every bit as good as those taken in the angiography suite. Most assuredly, the more surgeons follow Dr. Martin’s tenets, the less we will hear of the advisability of staging AVM surgery and the “normal-pressure postoperative breakthrough.”

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Neuronal Concussive Injuries

To The Editor: Zwimpfer and Bernstein are to be congratulated on their most worthy article (Zwimpfer TJ, Bernstein MA: Spinal cord concussion. J Neurosurg 72:894–900, June, 1990). There are abundant articles in the literature indicating that, with sufficient force, neuronal function may be interrupted and permanent damage occur. Unfortunately, the process is often termed “concussion.” This very thoughtful article demonstrates that there is a process whereby spinal cord function is interrupted by trauma (acceleration) and neuronal recovery is clinically complete. As the authors quite properly emphasize, there is no way of knowing from their observations whether repeat episodes would lead to permanent damage.

In reference to our work,1,2 they correctly note that we did not make any microscopic studies but did demonstrate that the cord of frogs could be concussed repeatedly with complete recovery. We have since concussed rats up to 20 times in one day and again over several days and been unable to demonstrate any deficiency in maze performance.3 Light and transmission electron microscopic pictures of multiple brain regions were taken subsequently and shown to various neurohistologists who could not differentiate between the concussed and the control rats. This has not been published but was given as part of a presentation to the Pan-Pacific Surgical Association in Hawaii, in February, 1990.

Thus, as a profession we still do not know all that happens when neurons lose function following acceleration short of permanent damage. It is obviously similar to depolarization or hyperpolarization in that billions of neurons lose and rapidly regain function. We know that there are many completely reversible metabolite changes (D Parkinson, J Vriend: unpublished data); we do not know whether complete recovery can ever take place or whether every loss of function is accompanied with some permanent damage, and we do not know what the limits of the force might be within which function is lost and structure preserved (although our work on rats and frogs and observations on human volunteers would suggest that the lower limit is about 35 G and the upper limit—the limit at which permanent damage always occurs in spite of survival—is over 200 G and below 1000 G^24). The answers to these questions should be in our possession in order that we as a profession can better offer advice, when requested, regarding legal settlements and the conduct of some sports.

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References