Letters to the editor

Microcirculation of Peripheral Nerves

TO THE EDITOR: Drs. Nobel and Black (The microcirculation of peripheral nerves. J Neurosurg 41:83-91, 1974) are to be complimented for the thoroughness of their perfusion techniques and the clarity of their macro- and microangiograms of intact peripheral nerves in this as well as in Nobel's earlier article.2,3

We have used similar angiographic techniques to supplement electrophysiological studies in primates.1 Compressed air forced through a pressure regulation valve set at the animal's mean arterial pressure helped to insure an even perfusion. A micropaque (25%) — gelatin (8%) mixture was perfused into distal aorta without prior heparinization. Perfusion was checked by looking for a relatively heavy mixture of contrast in vena cava return and by x-rays of the lower extremity. As pointed out by Nobel, microdissection or radiographs of microvasculature are necessary, however, to insure good perfusion. Specimens were not cleared so that histological studies of the axons as well as studies of connective tissue and perfused vessels could be done.

Electrophysiological techniques can be combined with microangiographic studies by exposing a short segment of nerve proximally and distally for electrode placement and by taking care to preserve large collateral vessels. Evoked nerve and muscle action potentials documented extent and subsequent course of various injuries. Mobilized and injured nerves kept electrical pace with those injured but not mobilized. Patterns of revascularization at injury sites were similar except in the early weeks after injury where a larger quantity of both collateral and intraneural vessels was seen in nonmobilized than in mobilized nerves. Longitudinal intraneural and subepineural vessels and not collateral vessels in mesoneurium were the predominant source for new vessels which reached the injury from both proximal and distal stumps. A photograph of the primate sciatic complex demonstrated mesoneurium in situ. Mesoneurium with collateral vessels could be seen without the aid of perfusion techniques although the extent of the vasculature is more fully appreciated in Nobel's perfused and cleared rabbit nerves and Smith's prior studies of cadaver limbs injected with a silicone elastomer.

Nobel and Black have demonstrated a superior technique for angiography of intact nerves and are to be congratulated.

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References

RESPONSE: This is in response to some of the remarks of Dr. Kline and co-workers, who commented most graciously on our article.7

Kline and co-workers, in animal experiments with Macaca mulatta monkeys, found little difference in the electrophysiology and ultimate revascularization of mobilized and unmobilized injured nerves. They do note that mobilization delayed revascularization during the first few weeks after the injury.

It has been my own (W.N.'s) experience with human surgery, and most surgeons seem to agree, that healthy human nerve can withstand considerable loss of collateral blood supply, as during neurolysis or stripping for transposition or suture. This ability can be explained by the superabundance of neural blood supply and anastomotic microvascular networks, seemingly disproportionate to a healthy nerve's minimal oxygen requirements under normal circumstances and constituting a generous reserve for periods of injury and vascular emergencies.8

In considering the possible relevance of the
Neurosurgical forum

Macaca monkey experiments to humans, one should call attention to two previous papers by Kline and co-workers noting that different species respond differently to nerve injury and that laboratory methods with one species cannot necessarily be applied unconditionally to other animal species or, of course, to humans. In human surgery, especially, caution is indicated with reference to stripping and to early mobilization as the generosity of blood supply holds true only for completely healthy nerves. Subclinical and clinical impairment due to arteriosclerosis of fine vessels, injury, diabetes, gout, clotting tendency in cancer etc., often not recognized before surgery, can make extensive nerve stripping much more hazardous for the already impaired blood supply, and early mobilization may further stress this marginal blood supply of the injured nerve to the point of ischemic degeneration.

I (W.N.) have encountered two cases of radial nerve palsy following extensive stripping for skeletal fixation of pathological fractures of the humerus. In these cases we suspected thrombosis of the vasa nervorum associated with the well-known tendency toward clotting in carcinoma to be the underlying cause.

In chronic compression of the sciatic nerve of rabbits with inflated balloons, we observed that injured nerves were more severely paralyzed by lighter pressures than normal nerves, indicating a greater vulnerability of injured nerves to ischemia. (These cases were excluded from our study and are not published.) This observation is in conformity with E. Gutmann (quoted in Kline and co-workers' article on Macaca monkeys), who observed more atrophy of legs in cats with crushed tibial nerves and ligation of popliteal artery and vein than in those with intact vessels. Increased susceptibility to ischemia of injured nerve fibers has also been described by Gilliat and Wilson and by Fullerton.1,2

Kline and co-workers have done important research on the effects on injured nerves of deprivation of collateral circulation and of mobilization, using modern elegant electrophysiological techniques. Their excellent work should lead to better understanding of the problems involved in nerve surgery.

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References

1. Fullerton PM: The effect of ischemia on nerve conduction in the carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 26:385-397, 1963

Subtemporal Craniectomy for Recurrent Shunt Obstruction


In the introduction the authors state that "very often elevated intracranial pressure makes revision mandatory before there has been a significant increase in ventricular volume. Therefore, adequate replacement of the ventricular catheter may be difficult or even impossible." One wonders about the cause of the elevated intracranial pressure in a patient with presumed hydrocephalus and ventricles too small to be punctured.

The authors' laboratory studies simply emphasize the well-known fact that the absence of osseous and dural continuity

J. Neurosurg. / Volume 42 / January, 1975 115