very inexpensive and simple to use and the monitoring procedure is easily accomplished. The importance and the cost effectiveness of PST monitoring cannot be overemphasized in performing endoscopic sympathectomy. Intraoperative monitoring of PST should be considered an essential aid in confirming an adequate sympathectomy leading to definite and long-lasting relief of PH.

MING-CHEN KAO, M.D.
National Taiwan University Hospital
Taiwan, China

References


Response

I appreciate Dr. Kao’s kind remarks and comments. At my institution, we also perform surgery for patients with palmar hyperhidrosis using the video endoscopic system. It is quite true that we cannot easily identify the superior intercostal artery in some circumstances (such as in the 15.4% of the ganglia noted in our article) and the distance between this artery and the sympathetic chain is also greatly variable (Table 2 of our article). Moreover, as Dr. Kao had previously noted, in one-third of cases studied the sympathetic trunk is not readily visible intraoperatively.² Obviously, this technique is a challenge for an inexperienced practitioner, especially one unfamiliar with anatomical orientation.

Anatomical localization is not the only controversial issue surrounding the surgical treatment of palmar hyperhidrosis. Other questions that must be considered are: the mechanism of recurrent sweating and its surgical treatment; how to avoid the accessory pathways; how to ensure that denervation is sufficiently extensive; the precise nature of the thermoregulatory pathway; and patient selection. Although some authors³⁴ have claimed that elevation of digital temperature on the surgically treated hand guarantees a definitive and successful T-2 sympathetic block, no statistical proof has been presented for this claim. It is also common sense that anesthesia causes the loss of systemic sympathetic tone, resulting in generalized vasodilation, which in turn elevates digital temperature: this is the reason the digital temperature in Dr. Kao’s patients rose spontaneously by 2°C just after induction of anesthesia and before sympathectomy.²

In our recent case-controlled study of 40 consecutive patients (unpublished data), we measured the dynamic changes in digital temperature simultaneously on surgically treated and nonsurgically treated hands just after cauterization of the T-2 ganglion and again 5 and 10 minutes later. We found that the measurement at 10 minutes after cauterization was the most accurate for prediction. When the temperature was elevated by 2°C, it resulted in predictive values of 27.5% sensitivity and 90% specificity. Its positive predictive value was 73.3% and the odds ratio was 3.4 (p < 0.05). Therefore, correct localization and then adequate ablation of the T-2 ganglion is the prerequisite for effective prediction of this low-sensitivity but highly specific monitoring system.

THOMAS SHANG-MING CHOU, M.D.
Chung Shan Medical and Dental College Hospital
Taiwan, China

References


Peripheral Nerve or Brainstem Glioblastoma

To The Editor: I read with interest the article by Reifenberger, et al. (Reifenberger G, Bostrom J, Bettag M, et al: Primary glioblastoma of the oculomotor nerve. Case report. J Neurosurg 84:1062–1066, June, 1996). I would argue that the title of this article is incorrect and misleading. Malignant tumoral processes may originate from either side of the boundary between the central nervous system (CNS) and the peripheral nervous system (PNS) to infiltrate the opposite side. The oculomotor nerve is formed by axons that originate from motor neurons located in several midbrain subnuclei. Along the course of these axons, from the midbrain subnuclei up to the ocular muscle, the nerve is divided into two parts, one belonging to the CNS and the other belonging to the PNS. The central part of the oculomotor axons is both intraaxial and extraxial for a mean length of 1.88 mm (1–4 mm). In the central part, the oculomotor axons are sheathed by the central myelin produced by oligodendrocytes; the extraxial segment of the central portion is covered by a prolongation of the pia mater.¹ The peripheral portion of the oculomotor axons is extraxial; axons are sheathed by the peripheral myelin produced by Schwann cells and covered by the endoneurium, a connective sheath. The peripheral portion of the oculomotor axons forms the oculomotor nerve in the strict sense. The boundary between the central and the peripheral myelin marks the passage from the CNS to the PNS in cranial nerves three to seven. This boundary is not located exactly at the exit or entry of the axons from or into the brainstem, as the central myelin, glia, and pia mater are found extraxially for some millimeters. Therefore, the oculomotor nerve begins when axons are myelinated by the peripheral myelin and no central glial cells are present. In the article by Reifenberger, et al., Fig. 2h shows part of this phenomenon: the visible neuroglial islands are in the extraxial portion of the cen-

J. Neurosurg. / Volume 86 / April, 1997
central part of the oculomotor axons, which is a prolongation of the brainstem, and not in the peripheral part of the oculomotor axons or the oculomotor nerve in the strict sense. In the same manner, if a tumor originates, for example, along the intraaxial course of the oculomotor axons, this tumor will be a brainstem tumor and not a tumor of the oculomotor nerve. Therefore, although “the areas of the CNS located within the proximal nerve stump” cited by the authors are characteristic of any area of a boundary, they are in fact areas of the CNS and not the oculomotor nerve. The authors stated that computerized tomography and magnetic resonance imaging did not reveal signs of penetration of the tumor into the midbrain or the upper pons, but at surgery the same authors treated the glioblastoma multiforme by partial resection “because the tumor appeared to infiltrate the surface of, and possibly penetrate, the upper brainstem.” In this instance, the opposite may be more true, that is, that the tumor originated from the superficial part of the upper brainstem, infiltrated, and grew extraaxially along the oculomotor nerve. The photomicrographs of neuropathological findings in biopsy specimens do not justify the title of this article. Considering that Fig. 2e shows both the normal side of the brainstem and the side with the tumor, I would think that another figure, such as Fig. 2f, should demonstrate a different picture, perhaps of an interruption of leptomeninges caused by the brainstem tumor. In conclusion, I think that this case of glioblastoma is unusual for location, but is no more than a small glioblastoma of the brainstem growing especially extraaxially along the oculomotor nerve.

EDUARDO FERNANDEZ, M.D.
Catholic University School of Medicine
Rome, Italy

Reference
1. Lang J: [Anatomy, length and blood vessel relations of “central” and “peripheral” paths of intracisternal cranial nerves.] Zentralbl Neurochir 43:217–258, 1982 (Ger)

RESPONSE: In the article to which Dr. Fernandez refers, we reported a 70-year-old woman who presented clinically with an incomplete left-sided oculomotor nerve palsy as the only neurological symptom. On neuroimaging, a large extraaxial tumor of the oculomotor nerve without obvious penetration into the midbrain or upper pons was seen. The tumor was operated on under the working diagnosis of an oculomotor nerve tumor, most likely a schwannoma. Histopathological evaluation of the operative specimen, however, revealed a glioblastoma multiforme. The patient unfortunately died 6 weeks after surgery. Postmortem examination confirmed the diagnosis of an extraaxial glioblastoma at the left pontomesencephalic junction with complete destruction of the oculomotor nerve. We reported this unusual tumor as the only glioblastoma of the oculomotor nerve. We do not agree with Dr. Fernandez’ criticism of our title for the following reasons.

Dr. Fernandez correctly states that the third cranial nerve may extend extraaxially for a few millimeters. However, Dr. Fernandez’ definitions that “the peripheral part of the oculomotor axons forms the oculomotor nerve in the strict sense” and that the “nerve begins when axons are myelinated by the peripheral myelin and no central glial cells are present” are in contrast to standard anatomical textbooks and also to the article by Lang. These references all clearly designate both parts together, that is, the central and peripheral segments, as the oculomotor nerve. We never claimed that the glioblastoma in our patient arose from the “nerve portion in the strict sense;” obviously, it came from glial cells. However, the precise site of its origin is difficult to trace, and we have carefully discussed the various possibilities in our paper.

In another cranial nerve, that is, the acoustic nerve, the central portion can extend even farther from the brainstem (up to 13 mm) than in the oculomotor nerve. Again, the entire structure is referred to as “the acoustic nerve,” not only the segment that has the Schwann cells and no glial cells. Kasantikul, et al., have reported a tumor that can be considered analogous to our case, that is, an astrocytoma growing in the acoustic nerve. The title of this contribution was “Glioma of the acoustic nerve.” Similarly, Panse reported on “Ein Gliom des Akustikus.”

Finally, we would like to emphasize that the tumor in our patient differed from ordinary brainstem gliomas not only with respect to its extraaxial location but also with respect to patient age and clinical presentation. Primary brainstem gliomas mostly occur in young children and adolescents, frequently cause multiple cranial nerve palsies, and may give rise to cerebellar signs early in the course of the disease. In contrast, our patient was 70 years of age and the only neurological symptom was a left oculomotor nerve palsy.

In conclusion, we think that the title of our article is correct, both from the neuroanatomical point of view and as it relates to the clinical, neuroradiological, and neuropathological findings in our patient.

GUIDO REIFFENBERGER, M.D.
JAN BOSTROM, M.D.
MARTIN BETTAG, M.D.
WOLFGANG J. BOCK, M.D.
WOLFGANG WECHSLER, M.D.
Heinrich-Heine-Universität
Düsseldorf, Germany
JOHN J. KEPIES, M.D.
The University of Kansas Medical Center
Kansas City, Kansas

References
3. Lang J: [Anatomy, length and blood vessel relations of “central” and “peripheral” paths of intracisternal cranial nerves.] Zentralbl Neurochir 43:217–258, 1982 (Ger)