1037-1042, December, 1983), the authors report five cases in which the patients were affected by expansive processes in the cavernous sinus. One of their patients (Case 1) had a clinical picture characterized by sixth nerve palsy and Horner’s syndrome on the right side, with pathological demonstration of a postganglionic sympathetic nerve lesion.

We have described a similar syndrome occurring in a patient who developed a traumatic intracavernous carotid aneurysm. The photographs of our patient’s eyes are almost identical to those in their Fig. 1. We have since had the opportunity to study another case of this syndrome, which has also been reported.²

I believe that this syndrome is a definite clinical entity and may be caused by lesions located in the distal portion of the petrous carotid canal or in the posterior part of the cavernous sinus. The anatomical basis of this syndrome has been described by Parkinson.³ I would like to call the attention of neurosurgeons, ophthalmologists, and neurologists to this entity, which may not be so very rare in clinical practice.

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References


Response: Dr. Abad's letter emphasizes an important point pertinent to cavernous sinus lesions. There is a sound anatomical basis in such cases for the possible simultaneous occurrence of an ipsilateral sixth nerve palsy and postganglionic Horner’s syndrome, without the concomitant involvement of any other cranial nerves. Parkinson³ has commented on this, and has provided a nice illustration⁴ of the likely site for such lesions to occur — along the intracavernous portion of the sixth cranial nerve where it is joined by the sympathetic fibers.

While theoretically possible, such a lesion is seemingly rare clinically: Parkinson⁴ has not seen it, and apparently neither has Thompson.² I have reviewed my records and have not found a single example of it either. Dr. Abad has of course reported two such cases,¹² both occurring after trauma. I agree that the photographs in our Fig. 1 strongly suggest such a syndrome. However, the concomitant involvement of the ipsilateral divisions of the trigeminal nerve invalidates the diagnosis — that is, there is more than just a combined ipsilateral postganglionic Horner’s syndrome and sixth nerve palsy.

Nevertheless, Dr. Abad’s point is important. If clinicians look carefully for such cases, more reports may appear demonstrating, as do Dr. Abad’s patients, the existence of this entity.

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Metabolic Reaction to Head Injury

To THE EDITOR: The recent article by Clifton, et al. (Clifton GL, Robertson CS, Grossman RG, et al: The metabolic response to severe head injury. J Neurosurg 60:687-696, April, 1983) is an excellent and detailed study of the metabolic reaction to head injury. Our studies, while less sophisticated, also demonstrated a striking and prolonged negative nitrogen balance in patients who sustained head injuries.⁴ Since these latter studies were performed before the use of exogenous steroids or mannitol in neurological surgery, they seem to rule out these agents as possible factors in the causation of a negative nitrogen balance.

The disparity between the vigorous response of nitrogen metabolism and the mild response of other portions of the metabolic reaction to trauma (such as sodium and water) was noteworthy when compared to the metabolic effect of craniotomies² or general surgical procedures.² Decreased spontaneous movement has been shown to be a cause of negative nitrogen balance,¹² and may be a factor in head-injured patients.

I agree with the authors’ statement that hyperalimentation has not been demonstrated to be of benefit in patients with head trauma or other injuries. Certainly, in non-depleted patients who remain comatose for relatively short periods, hyperalimentation probably is unnecessary.

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References

RESPONSE: As Dr. Wise has pointed out, the metabolic response to head injury differs from the response to systemic injury and to cranial surgery. The increase in calorie consumption and nitrogen excretion resulting from elective cranial and noncranial surgery is mild. In systemic trauma the intensity of these indices of catabolism increases as the severity of injury increases, with major burns and sepsis producing the most extreme response. Kinney and Long, et al., have found that nitrogen excretion increases in parallel with calorie consumption, with a catabolic peak occurring about 10 days after injury and a predictable anabolic phase following several weeks after injury.

Our present data indicate that in head injury the catabolic phase is long-lasting and has its onset immediately after injury rather than a week later. A close relationship between nitrogen excretion and calorie consumption has not been found in nonparalyzed patients, and in barbiturate coma there is a marked increase in nitrogen loss with a decrease in calorie consumption. Therefore, I would agree with Dr. Wise's statement that decreased spontaneous movement is likely to be a major factor in the nitrogen wasting that has been found. The question of steroid effect is being examined. The most curious aspect of this phenomenon is the basic question of whether in some way the damaged brain benefits from the increased catabolism of protein and increased systemic oxygen delivery and oxygen consumption resulting from isolated head injury. An alternative explanation is that this is a maladaptive response resulting from injury to areas of the brain that in other circumstances would mediate a similar neurohumoral response to a systemic insult, such as a burn.

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References

Adequacy of Intravenous Digital Subtraction Angiography: Erratum
TO THE EDITOR: In reviewing my reply to the Letter to the Editor by Diaz and Ausman (Diaz FG, Ausman JI: Adequacy of intravenous digital subtraction angiography. J Neurosurg 60:1331, June, 1984, Letter: Little JR, Response), I noted that there is a portion of a sentence missing at the beginning of the last paragraph. This phrase must have accidentally been deleted at the time of printing. Unfortunately, its omission substantially changed the overall meaning of the letter. The sentence in its entirety should read: “It must be realized that there are limitations with IV DSA in that 15% of the studies do not adequately visualize the extracranial carotid arteries and 60% do not adequately visualize the major intracranial arteries.”

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BCNU Treatment of Astrocytomas: Erratum
TO THE EDITOR: In our recent article (Layton PB, Greenberg HS, Stetson PL, et al: BCNU solubility and toxicity in the treatment of malignant astrocytomas. J Neurosurg 60:1134–1137, June, 1984), an error appears in the text on page 1135, in the fourth paragraph. The next to last sentence should correctly read, “Once dissolved, each vial of BCNU was transferred by needle . . . .”

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