Alimentation of Head-Injured Patients

To THE EDITOR: I read with interest the article by Dr. Robertson and colleagues (Robertson CS, Clifton GL, Goodman JC: Steroid administration and nitrogen excretion in the head-injured patient. J Neurosurg 63:714-718, November, 1985). Although their chief aim was to study the influence of steroid administration on nitrogen excretion in head-injured patients, I wish to comment on the calorie and protein intake in the study group.

In our opinion, the administration of 600 to 650 kcal/day from Days 1 to 3 after trauma without any protein via a gastric tube is far too low. As Wesemann has demonstrated, the mean calorie requirements of most neurosurgical patients range from 1500 to 2500 kcal/day. Gamble has shown that sufficient energy intake alone can reduce nitrogen loss. Dölp has demonstrated that administration of less than 0.6 gm of amino acids/kg/day leads to further nitrogen loss. As demonstrated in numerous studies using stable isotopes, gastric feeding does not necessarily mean intestinal resorption of nutritional components. From these and numerous other studies it can be concluded that one result of this concept of nutrition is a highly negative nitrogen balance as found by Robertson, et al. Short-lived serum proteins and free plasma amino acids were not measured in the study group. Far better than determination of nitrogen balances and serum albumin levels would have been a demonstration that the nutritional regimen used in their study did not meet the caloric and protein requirements of severely head-injured patients in the posttraumatic period.

In a similar group of patients we have demonstrated that early parenteral feeding with 2500 to 3000 kcal/day and administration of 1.5 mg/kg/day of amino acids can reduce negative nitrogen balances (77.8 gm cumulative reduction on Day 8 after trauma) and can normalize values of short-lived serum proteins as well as of free plasma amino acids. From our study we concluded that gastric feeding in neurosurgical patients with head injuries is indicated only in a later period, and early parenteral feeding is the method of choice during the first posttraumatic days.

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RESPONSE: We appreciate Dr. Piek's letter emphasizing that the caloric and protein intake in our study patients was not optimal during the early postinjury days. Our major concern in designing the study was to compare nitrogen losses in fasted and fed patients in the steroid-treated and the control groups. The protocol, which used both parenteral and enteral feedings, depending on the individual patient's needs, was not intended to be our recommendation for alimentation of the head-injured patient. We agree with Dr. Piek that attention should be directed to the patient's metabolic needs as soon after the injury as is possible.

Dr. Piek's data also support our findings. The average cumulative nitrogen loss on Day 8 in his patients, who were treated with steroids but received alimentation from Day 2, was higher than in our group without steroids (78 gm vs 70 gm, respectively) even though our patients were fasted for the first 3 days after injury. Steroid administration increases the early catabolic response to head injury, and it is difficult to compensate for the protein losses with hyperalimentation.

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Tremor Following Peripheral Nerve Injury

To THE EDITOR: We were interested in the recent paper by Little, et al., describing the development of tremor involving ulnar-innervated muscles following an entrapment neuropathy (Little JW, Burchiel K, Nutter P: Tremor and peripheral nerve entrapment. Case report. J Neurosurg 64:145-147, January, 1986). Although the neurophysiological mechanism underlying peripheral nerve injury-induced tremor is unknown, it may be of interest to the readers that one of us has reported three cases of a unilateral parkinsonian tremor following radial nerve injury. In all three patients, administration of 80 mg/day of propranolol abolished the tremor, suggesting that peripheral adrenergic mech-

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anisms may have been involved in the pathogenesis of
the tremor. Moreover, at the 20th Annual American
Society of Hospital Pharmacists Mid Year Clinical
Meeting, we described finding abnormal autonomous
movements in patients following amputation, indicat-
ing a common mechanism of deafferentation in this
class of involuntary movements (J Linford, et al., un-
published data, 1985).

Deafferentation after peripheral nerve injury has
been reported to initiate abnormal discharges as well as
morphological and neurotransmitter changes in the
dorsal horn. This denervation hypersensitivity may
also be exacerbated by loss of central (reticular and
raphe nuclei) centrifugal inhibition resulting from re-
duced segmental sensory input. Furthermore, the clin-
ical concomitant of deafferentation, which may include
chronic pain, could by itself lead to depletion of central
serotonin and opioid reserves (opioids are known to
exert an inhibitory effect on spinal reflex pathways). Thus,
the factors that may be responsible for activation of
spinal neural circuits in nerve injury may also operate
in triggering involuntary movements.

The beneficial effects of propranolol in the cases
reported by Sandyk may be related in part to peripheral
adrenergic receptor blockade or to the known serotonin-
enhancing effects of propranolol. Our experience with
deafferentation-induced movements in amputees has
demonstrated that other serotonergic agonists such as
tricyclic antidepressant agents may be useful not only
for the pain component but also for the control of
peripheral autonomous movements following nerve in-
juries.

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tremor induced by peripheral trauma. S Afr Med J 68:
898, 1982 (Letter)

“High-Dose” Methylprednisolone and CNS Injury

TO THE EDITOR: The recent article by Bracken, et
al., that reported the 1-year follow-up results of ad-
ministration of methylprednisolone sodium succini-
rate (MPSS) for spinal cord injury deserves comment

(Reuvn M.B, Shepard MJ, Hellenbrand KG, et al: Methylprednisolone and neurological function 1 year
after spinal cord injury. Results of the National Acute
November, 1985). Continued reference to the 1000-mg
bolus (with 1000 mg/day maintenance) as “high-dose
MPSS" is no longer appropriate and is, in fact, clinically
misleading. We have repeatedly indicated that this was
not a “high dose" as far as the treatment of central
nervous system (CNS) injuries with MPSS is concerned.
This has been discussed in a recent review published in
the Journal of Neurosurgery: Indeed, the failure of a
similar “high-dose" regimen to promote functional re-
covery in experimentally injured animals has been re-
ported. Continued reference to the dosing protocol
used in the study by Bracken, et al., as “high dose" implies
that high-dose MPSS is ineffective and should not
be used to treat CNS injuries. This is far from the
case.

No reference is made by Bracken, et al., to the second
National Acute Spinal Cord Injury Study. This new
study compares placebo to treatment with either nal-
oxone or a regimen of MPSS that is far more intense
than was used in the first study. The new MPSS dosing
regimen has been designed based upon careful experi-
mental pharmacokinetic and pharmacodynamic studies
of MPSS in the treatment of the injured CNS. The
intensive steroid dosing regimen being tested entails an
initial 30-mg/kg (2000 mg) intravenous bolus of MPSS
followed by a constant intravenous infusion of 5 gm
over the remaining 24-hour period aimed at main-

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