Misleading Review of Residency Training Programs

To The Editor: It is understandable but regrettable that occasional Letters to the Editor are not subject to the same degree of careful evaluation as are other reports submitted to the *Journal of Neurosurgery*. The recent communication by Dr. Long reviewing the data listed in the summaries of training programs published in *Neurosurgery* is a case in point (Long DM: Operative summary in residency training programs. *J Neurosurg* 64:526–527, March, 1986).

The writer stated in the penultimate sentence of his letter, "I recognize these calculations are inexact and based upon assumptions that are unlikely to be absolutely correct." This is a masterpiece of understatement and I question why they "provide a basis for comparison" or "would be of interest to program directors, academicians, and residents in assessing their own activities." Moderately careful scrutiny of the *Neurosurgery* summary would immediately reveal the fact that many who are listed as "faculty members" either are not neurosurgeons, are basic scientists or experts in allied fields who have appointments in neurosurgical departments, or are individuals who have tapered off or ceased actual surgical activities.

I would agree with Dr. Long’s parenthetical statement in the next to last paragraph: "(It is not possible from the data at hand to compare the number of operations actually available to the resident for surgical education in the program.)"

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Reference


Steroid Therapy and Nitrogen Excretion in Head Injury

To The Editor: We have followed with great interest the studies of metabolism and head injury conducted by Robertson, *et al.*, most recently their informative paper regarding the effects of steroids on nitrogen metabolism (Robertson CS, Clifton GL, Goodman JC: Steroid administration and nitrogen excretion in the head-injured patient. *J Neurosurg* 63:714–718, November, 1985). This is a particular interest of our own, relating to an overall concern for the effects of trauma on metabolism and the role of nutritional support in this setting.

Our studies parallel some of those noted in their paper; however, we studied a group of patients with isolated closed head injuries in a longitudinal manner for 1 week in the absence of any exogenous nutritional support. When focusing on sensitive markers for level of stress in patients *not receiving* steroids, we have noted a peak in metabolism on approximately postinjury Day 3, with a decline toward levels consistent with progressive fasting by week's end. This is especially borne out by serial plasma amino acid levels; in particular, the branched-chain amino acids are initially low and rise progressively. In short, the pattern is very similar to that observed in nonseptic diffuse trauma, with a peak in stress occurring at about Day 3 followed by subsequent resolution. When we compared this initial group with a similar group that *did receive* steroids (CS Deutschman, *et al.*, in preparation), no resolution of metabolic markers was evident at 1 week.

We therefore conclude that, in the absence of nutritional support, steroids potentiate the hypermetabolic state and acquired malnutrition associated with head injury.

It is to the question of prolonged nitrogen excretion and elevated resting metabolic expenditure (RME) that we must address ourselves. Following the first 3 post-injury days, the patients presented by Robertson, *et al.*, were maintained on a regimen of intensive metabolic support. These authors conclude that steroids were not the cause of the catabolism noted in their patients. They cite as evidence continued weight loss, failure of nitrogen balance to become positive, and the prolonged elevation in RME. The regimen provided to these patients is appropriate for high-level stress, when large-calorie loads and high-nitrogen intake is of benefit. If the level of stress is resolving, as our data would indicate, the metabolic support should be modified to something more appropriate to the reduced demands of lower-level stress. Failure to alter the nutritional regimen to decrease both glucose and protein can lead to excretion of excess protein in the urine and elevation of RME secondary to increased CO₂ production. Also adding to the nitrogen and weight loss is the demographic nature of the patients in question. Most are young, healthy, relatively active males whose activity is being acutely restricted. Increased muscle wasting as a result of reequilibration to level of activity may in part be responsible for changes in weight and nitrogen excretion.

Finally, it should be pointed out that difference in serum albumin levels is not the best marker for visceral protein status. The half-life (T½) of albumin is about 21 days; therefore, persistently low levels of this protein may reflect an event that occurred at a fairly remote time. Use of transferrin (T½ of 10 days), prealbumin (T½ of 2 days), or retinol-binding protein (T½ of 12 hours) would better reflect the true state of visceral protein balance.

We applaud the work of Robertson, *et al.*, both in this and in previous studies. They point out once again that steroids, like all drugs, have detrimental as well as beneficial effects, and raise serious questions regarding the use of these agents in head-injured patients. Nutritional support solutions, however, are also drugs. If
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they are indeed to be used in this setting, it must be with full knowledge of their possible deleterious effects and with constant modification of regimens to insure appropriate support for levels of stress.  

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References


RESPONSE: We appreciate the comments of Drs. Deutschman and Cerra regarding our paper. They address some important points that were not included in this study, which was intended only to examine the catabolic effects of steroid administration.

The major difference in our data appears to be that we have found that the severely head-injured patient continues to have elevated caloric requirements even beyond the acute phase of injury. We have measured energy expenditure in 75 head-injured patients and found caloric requirements to be consistently elevated for up to 4 weeks after injury. These data are summarized in a more recent paper. During the 1st week after injury, the patients are hypermetabolic, similar to any other traumatized patient. Drs. Deutschman and Cerra have demonstrated the similarity of the metabolic changes to non-neurological trauma in their recent article; however, their data stop at 7 days postinjury. Seven to 10 days after a neurological injury, intracranial hypertension is no longer a problem, and sedating and paralyzing drugs are generally decreased or discontinued. In patients who do not improve neurologically and who continue to have abnormal motor responses, total caloric expenditures can exceed 3000 kcal/day. This caloric expenditure is due to a large extent to motor tone and/or activity, and paralysis with Pavulon (pancuronium bromide) significantly reduces metabolic expenditure. Patients who slowly improve neurologically may have frequent semipurposeful motor activity as they begin to awaken from coma; their caloric expenditures remain high, often between 2500 and 3000 kcal/day. This may not be hypermetabolism in the same sense as during the 1st week after injury, but it nevertheless reflects an increased caloric requirement that must be met.

The level of caloric replacement for our study was chosen so that nitrogen intake could be constant and so that we could be assured of total replacement of energy expended, since inadequate caloric replacement can result in significant nitrogen loss. We do not think that the caloric intake significantly increased metabolic rate in this group of patients since their respiratory quotients averaged 0.9, not > 1.0 as would be expected if they were simply depleted patients receiving an excess glucose load. However, this protocol was not intended to be our recommendation of optimal alimentation of the head-injured patient. As pointed out by Drs. Deutschman and Cerra, it is best to neither overfeed nor underfeed patients, and we would add that actual measurement of individual caloric expenditures is the optimal way to assure appropriate management.

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Nasal Glioma and Encephalocele


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