Continuous Epidural Blockade Arrests the Postoperative Decrease in Muscle Protein Fractional Synthetic Rate in Surgical Patients

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Background: Epidural anesthesia with local anesthetics is associated with postoperative attenuation of nitrogen loss. The protein-sparing effect could be the result of either a decreased protein breakdown or increased protein synthesis. Although the role of epidural local anesthetics in effectively limiting the increase in postoperative protein breakdown is established at the whole-body level, it is necessary to determine whether the muscle protein fractional synthetic rate is directly modulated when nociceptive stimuli are blocked.

Methods: Twelve otherwise healthy patients scheduled for elective colorectal surgery, who were receiving a constant intake of nitrogen (0.1 g kg⁻¹ day⁻¹) and calories (20 kcal kg⁻¹ day⁻¹) before and after surgery, were randomly assigned to receive either general anesthesia (with thiopentone, vecuronium, fentanyl, or enflurane; control group, n = 6) or epidural anesthesia (T₃–S₅ sensory block with 0.75% bupivacaine) and general anesthesia (epidural group, n = 6). In the control group, postoperative analgesia was achieved with papaverine given subcutaneously, whereas a continuous epidural bupivacaine infusion (T₈–L₅ sensory block) was maintained for 48 h in the epidural group. The postabsorptive muscle protein fractional synthetic rate was determined using a 6-h continuous infusion of ¹³C-labeled leucine (1 mg kg⁻¹ h⁻¹), and the ¹³C enrichment in muscle biopsy specimens before surgery and 48 h after surgery was measured.

Results: Plateau ¹³C enrichment of plasma α-ketoisocaproate (taken to represent the intracellular leucine precursor pool enrichment for protein synthesis) was achieved during the 6-h infusion (mean coefficient of variation was 2.8%). Muscle protein synthesis at 48 h after operation compared with preoperative levels decreased significantly in the control group (P = 0.03). In contrast, it increased by 25% in the epidural group. Although this was not significantly different from preoperative levels, it was significantly greater than in the control patients.

Conclusions: Epidural infusion of local anesthetics begun before surgery and continued during the first 48 h after operation significantly attenuates the decrease in the postabsorptive muscle protein synthesis rate associated with surgical injury. Effective block of nociceptive stimuli thus preserves tissue protein synthesis. (Key words: Epidural. Local anesthetics. Protein metabolism. Muscle. Stable isotope.)

Epidural anesthesia with local anesthetics initiated before surgery and maintained during the perioperative period attenuates the postoperative loss of body protein as assessed by urinary excretion of nitrogen¹ and 3-methyl histidine² and the efflux of glutamine from the muscle.³ The obvious shortcoming of these measurements is that the contribution from changes in protein synthesis and in protein degradation cannot be differentiated. For example, an improvement in nitrogen balance only indicates that synthesis is elevated in relation to degradation, and thus synthesis can either increase or decrease, whereas degradation would increase less or decrease more, respectively.

In a recent study using ¹³C-leucine, we found an effective influence of epidural blockade on postoperative whole-body protein breakdown and amino acid oxidation, with minimal changes in protein synthesis.⁴ One criticism of that study was that the measurements were taken two days after the epidural blockade was withdrawn. In a subsequent study, protein metabolism was assessed while the nociceptive block was maintained as well as when the block was withdrawn. The results showed a significant inhibition of epidural local anesthetics on whole-body protein breakdown as long as the epidural block was effective.⁵ Nevertheless these findings do not provide any quantitative information regarding protein metabolism in individual tissues and, in particular, nothing about skeletal muscle. In healthy adults, muscle tissue is approximately 45% of the whole-body weight, is by far the...
largest protein pool in the body, and contributes as much as 25% of the total-body protein synthesis.\(^6\)

Muscle protein synthesis has been shown to decrease by approximately 40% after surgical and accidental trauma. This information has been obtained from studies using the arteriovenous concentration differences between amino acids,\(^7\) ribosome analysis,\(^8\) and intracellular free amino acid concentrations.\(^9\) This fact has now been confirmed in studies of the incorporation of isotopically labeled amino acids into muscle protein,\(^10\) which is the only approach that can give a quantitative estimation of protein fractional synthetic rate.

We did this study to determine whether the demonstrated protein-sparing effect of epidural local anesthetics would be, under carefully controlled conditions of nociceptive block and nutritional intake, associated with a change in muscle protein fractional synthetic rate.

**Methods**

Twelve patients scheduled to undergo elective resection of localized nonmetastatic adenocarcinoma of the rectosigmoid colon were studied. None of the patients suffered from malnutrition or recent weight loss. Patients with anemia, diabetes, morbid obesity, or severe cardiovascular disorders were excluded. Skinfold thicknesses (biceps, triceps, subscapular, and iliac crest) and mid-arm circumference were measured and the percentage of body fat was calculated.\(^11\) The study was approved by the local hospital ethics committee, and all patients gave written informed consent.

They were randomly assigned to a control group or an epidural group, each containing six patients.

**Nutrition**

A nutrition regimen based on 0.1 g nitrogen kg\(^{-1}\) day\(^{-1}\) and 20 kcal kg\(^{-1}\) day\(^{-1}\) was designed for the period of the study. Nonprotein calories were 60% from lipids and 40% from carbohydrates. The intake was started orally 6 days before surgery, under dietetic supervision, and changed to peripheral parenteral nutrition (500 ml Vamin 14, 11 Intralipid 10%, 11 dextrose 10%, KABIPHARMACIA, Stockholm, Sweden) 2 days before surgery. This was discontinued from midnight before surgery and then restarted 4 h after the end of the operation when cardiovascular and respiratory functions were stable, and it was continued for 2 days after surgery.

Anesthesia and Surgical Care

No premedication was administered. At arrival in the anesthetic room, the patients in the epidural group assumed a sitting position and an epidural catheter was inserted in the T8 lumbar space. Bupivacaine 0.75% (10–15 ml) was injected to produce segmental sensory block to pin prick from T3 to S5. Additional 0.75% bupivacaine (5 ml) was given every 90 min during surgery. General anesthesia in both groups was induced with thiopentone and maintained with vecuronium, nitrous oxide, oxygen, and enflurane. Fentanyl 250 µg was administered in the control group before surgical incision. The lungs were ventilated at normocapnia. Hypotension (arterial systolic blood pressure below 80 mmHg) was treated with incremental doses of methoxamine and intravenous fluids.

At the end of surgery the patients were tracheally extubated and transferred to the recovery room. The epidural group received a continuous infusion of 0.25% bupivacaine at a rate of 8–12 ml/h and maintained for 48 h after surgery. The patients were nursed in a semireclining position (20–30 degrees head up) to facilitate a downward spread of local anesthetic. We believed that an extended sensory block from T8 to L3 would provide adequate analgesia to cover a 10–15 cm paramedial abdominal incision. Sensory block to pin prick and ice was assessed every 4 h. An additional bolus of 5 ml bupivacaine 0.25% was administered every 8 h, if needed, to maintain the spread of the block. Papaveretum given intramuscularly (8–10 mg) was administered to patients in the this group every 8 h to supplement analgesia and provide sedation. In the control group, pain relief was achieved with a continuous subcutaneous infusion of papaveretum set at 3–8 µg/h. Pain at rest and on coughing was assessed during the postoperative study period in both groups every 4 h using a visual analog scale (VAS, 1–10 cm). The degree of motor blockade was assessed every 4 h in the epidural group according to a modified Bromage scale (0 = no motor block; 1 = inability to raise extended legs; 2 = inability to flex knees; 3 = inability to flex ankle joints).

Patients were asked by the ward nurses to rise in bed, stretch the lower limbs, sit on the bed, walk from the bed to a chair, and walk around the room. Time out of bed was recorded.

A crystalloid solution (Hartmann) was infused intravenously at a rate of 6 ml·kg\(^{-1}\)·h\(^{-1}\). Blood loss were measured and replaced with homologous blood transfusion if loss exceeded 20% of the patient’s circulating...
blood volume, but otherwise crystalloids were administered intravenously at twice the volume of blood lost. Parenteral nutrition was administered via an 18-g peripheral line and supplemented in the postoperative period with dextrose saline to provide a volume of 40 ml·kg⁻¹·day⁻¹. Nutritional supplementation was discontinued 8 h before protein metabolism studies were started, and all patients received an intravenous infusion of NaCl 0.9% at the same rate as noted before.

All operations were carried out by the same surgical team and at the same time of day (from 2 PM to 5 PM).

Muscle Protein Metabolism

Muscle protein metabolism was studied using steady-state leucine kinetics on the day of surgery and on day 2 after surgery using plasma $^{13}$C α-ketoisocaproate ($^{13}$C α-KIC) specific activity or enrichment as the basis for calculating whole-body leucine flux and muscle protein fractional synthetic rate. All patients were fasted for 8 h before the isotope studies were done, and all studies were started at 8 AM. A superficial vein in the dorsum of the hand was cannulated to provide access for infusion of L-[1-$^{13}$C] leucine. Blood was sampled from a cannula placed in the contralateral hand vein to measure basal $^{13}$C enrichment.

L-[1-$^{13}$C] leucine (99% $^{13}$C) was obtained from Cambridge Isotope Laboratories (Woburn, MA). The tracer was prepared in normal saline by the hospital pharmacy, and the solutions were shown to be sterile and free of pyrogens. After a priming dose of L-[1-$^{13}$C] 1 mg/kg leucine, a continuous infusion of labeled leucine (1 mg·kg⁻¹·h⁻¹) was started and continued for 6 h. Blood samples were collected after 3 h into the infusion, when an isotope steady state had been achieved, and at intervals of 30 min for the rest of the study. Each blood sample was transferred to a tube prepared with heparin and centrifuged at 4°C. The plasma was separated and stored at -70°C to await measurement of $^{13}$C-KIC enrichment.

Percutaneous muscle biopsy was obtained from the quadriceps muscle using a Bergstrom needle (size 5 or 6) as described by Edwards. After local infiltration with 1% lidocaine of the skin and muscle fascia, a 0.5-cm incision was made in the skin and in the fascia and muscle tissue (100–150 mg) was removed, blotted, weighed, and rapidly frozen in liquid nitrogen. One muscle biopsy specimen was taken at the end of the L-[1-$^{13}$C] leucine infusion and before surgery. Two muscle biopsy specimens were taken from the opposite thigh 2 days after surgery—one before the start of the isotope infusion and the other at the end. The muscle specimens were freeze-dried to a constant weight and rendered free of fat by extraction in petroleum ether. The protein precipitate was dissolved in 0.5 M NaOH. The alkali-soluble muscle protein was decanted and hydrolyzed with 6 M HCl at 110°C for 24 h under nitrogen. The hydrolysate containing the amino acid mixture was purified through a cation exchange resin (Dowex AG-8X column, Bio-Rad, Hemel Hempstead, England) eluted with 3 M NH₄OH and dried under a stream of nitrogen gas. It was then esterified with 4 M dry HCl in isobutyl alcohol at 120°C for 1 h. The mixture of amino acid esters thus prepared was run through a preparative GC column and leucyl isobutyl HCl ester collected in a special narrow U-shaped glass tube cooled in liquid nitrogen. It was then necessary to hydrolyze this ester and the resulting leucine was decarboxylated in vacuum with ninhydrin at pH 2.2. Carbon dioxide liberated from this reaction was carefully and cryogenically distilled into a glass finger tube. The pure and dry carbon dioxide from the muscle protein leucine containing the label was thus ready to be analyzed in an isotope ratio mass spectrometer (delta S Finningan MAT, Bremen, Germany).

Plasma $^{13}$C α-KIC enrichment was taken to closely represent that of muscle intracellular leucine labeling from which protein synthesis occurred (the precursor pool). It was also assumed that the incorporation of $^{13}$C into muscle protein is essentially linear with time. Work carried out by the Heyes et al. has shown that baseline $^{13}$C enrichment of leucine in plasma protein (before infusion of any label) accurately reflects the baseline $^{13}$C enrichment of muscle protein, thus obviating the need for multiple muscle biopsies from the same person. This was practiced in the preoperative study. However, in the postoperative period, two biopsies were obtained; one before the start of the $^{13}$C leucine infusion (acting as a new zero as this muscle biopsy will still have some of the incorporated label from the preoperative study) and another 6 h into the infusion. The difference in enrichment between muscle protein leucine of the biopsy samples was used to calculate the muscle protein fractional synthetic rate.

Muscle protein fractional synthetic rate (%/h) =

$$\frac{^{13}\text{C leucine enrichment in muscle protein at } t_2 - ^{13}\text{C leucine enrichment in muscle protein at } t_1}{\text{mean plasma } ^{13}\text{C } \alpha\text{-KIC enrichment at plateau}} \times 100$$

where $t$ = time when biopsies are performed (h).
Table 1. Physical Details and Clinical Data of the Two Groups Studied

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Epidural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>68 (5)</td>
<td>70 (4)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>63 (4)</td>
<td>66 (7)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>161 (11)</td>
<td>158 (9)</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>25 (3)</td>
<td>27 (4)</td>
</tr>
<tr>
<td>Plasma albumin (g/l)</td>
<td>41 (4)</td>
<td>39 (3)</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>167 (26)</td>
<td>154 (33)</td>
</tr>
<tr>
<td>Blood loss (ml)</td>
<td>675 (118)</td>
<td>759 (106)</td>
</tr>
<tr>
<td>Crystalloids (ml)</td>
<td>3678 (787)</td>
<td>4537 (689)</td>
</tr>
</tbody>
</table>

Values are mean (standard deviation).

Plasma $^{13}$C α-KIC enrichment was determined by electron impact selected-ion gas chromatography–mass spectrometry using ketovaleric acid as an internal standard. In each $^{13}$C α-KIC analysis run, duplicate injections were always performed and their means were taken to represent enrichment and concentration. Calibration curves were included with each analytical run, and these values were used to correct $^{13}$C α-KIC enrichment. The tracer infusion rate was determined directly by weighing the tracer initially and at the end of the study. In addition, the weight of the infusate discarded or not infused was measured. The accuracy of the isotopic enrichment at plateau (2–6 h into the label infusion) of plasma KIC was tested by evaluating the scatter of values above their mean, expressed as the coefficient of variation, standard deviation/mean. A coefficient of variation less than 5% was used as confirmation of a valid plateau.

**Statistics**

The data are expressed as mean values and standard deviation. The Student’s *t* test and Wilcoxon’s two-tailed signed rank test (for paired evaluation) were used as appropriate. Analysis of variation was applied to assess VAS scores. Statistical significance was accepted at *P* < 0.05.

**Results**

The characteristics of the patients studied and the clinical data regarding preoperative plasma albumin concentration, duration of surgery, volume of blood loss, volume of blood replaced, and volume of crystalloids administered during the perioperative period were comparable (table 1).

The mean dose of bupivacaine used in the epidural group during surgery was 154 mg (range, 137–186 mg). During the first and second 24-h postoperative periods, the average doses were 685 mg (range, 618–772) and 560 mg (range, 496–635 mg), respectively. This group also received 58 mg (range, 420–68 mg) of papaveretum given intramuscularly during the 48-h period after surgery. Patients in the control group received a subcutaneous infusion of papaveretum, and the mean (range) dose during the 48-h period after surgery was 106 mg (range, 88–137 mg).

The segmental extent of sensory block to ice (upper and lower dermatomes) and the degree of motor block assessed every 4 h in the epidural group, together with the VAS at rest and on coughing in both groups, are presented in table 2. Sensory and motor blockade during the first 8 h after operation were extensive as a result of greater concentration of local anaesthetics used during surgery. During the postoperative period, the segmental sensory block ranged between T7 and L5, thus covering the entire surgical area. The Bromage scale for motor block ranged from 1 to 3 (mean, 1.8) in the first 24 h and decreased to a mean value of 0.5 (range, 0 and 1) in the second 24 h. The postoperative VAS in the epidural group ranged from 0.6 to 1.6 (at rest) and 2.3 to 4.1 (on coughing), respectively. The VAS in the control group ranged from 0.8 to 2.1 (at rest) and from 2.4 to 6.5 (on coughing). No significant difference in the VAS at rest was observed between the two groups. In contrast, VAS on coughing was significantly lower in the epidural group compared with the control group (*P* = 0.021).

Patients in the control group could sit on a bed and move to a chair on the first day for approximately 3 h, and they could walk in the room on the second day. Patients in the epidural group were helped to sit on a bed could not move out of bed on the first postoperative day due to a motor block of moderate intensity. On the second day, the patients could sit on a bed and move to a chair for a total period of 4 h.

**Muscle Protein Synthetic Rate**

Plateau enrichment of plasma $^{13}$C α-KIC was achieved in all infusions (table 3), and the mean coefficient of variation was 2.7% (standard deviation, 1.7–3.2), indicating steady state. Muscle protein fractional synthetic rate (table 4) in the control group decreased significantly by approximately 40% (*P* = 0.03). Conversely, in the epidural group, muscle protein synthesis increased.
Table 2. Clinical Data of Sensory and Motor Block Assessment in the Epidural Group and VAS in Both Groups

<table>
<thead>
<tr>
<th>Hours after Surgery</th>
<th>Estimated SEM</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>T4</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>T6</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>T8</td>
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<td>16</td>
<td>T9</td>
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<td>20</td>
<td>T9</td>
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<td>24</td>
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<td>T8</td>
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<td>36</td>
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<td>40</td>
<td>T7</td>
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<tr>
<td>44</td>
<td>T8</td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>T8</td>
<td></td>
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</tbody>
</table>

Motor block (modified Bromage scale); VAS = visual analog scale; Estimated SE = Average estimate of standard error for the best fitting model.

from a mean value of 0.063%/h to a postoperative mean value of 0.081%/h (25% increase), although this increase was not significant (P = 0.15) due to large variability. Individual data of both groups are presented in table 4.

Discussion

In contrast to the decrease in muscle protein fractional synthetic rate after surgery observed in the control group, the present findings indicate that epidural anesthesia and analgesia with local anesthetics arrest the decline of tissue protein synthesis.

Whole-body protein metabolism can be assessed using essential amino acids labeled with stable isotopes. The basic concept of most of the available techniques is that the whole body may be simplified to a two-compartment model consisting of a free-amino acid pool and amino acid bound in body proteins. In patients having surgery, use of stable isotopes to measure whole-body protein metabolism indicates that in qualitative terms protein synthesis increases and is accompanied by an even greater increase in whole-body protein breakdown. The estimation of muscle protein metabolism in humans can be executed by assessing the flux of free amino acids across muscle tissue or incubation of biopsy samples of muscle tissue in vitro as well as incubation of ribosome fraction isolated from human muscle tissue. The concentration and size distribution of ribosomes may be used to indicate changes in the rate of protein synthesis. A decrease in these variables has been reported after surgery. Protein synthesis also may be determined at the level of transcription by determining the mRNA for a specific muscle protein such as myosin. All these techniques give indirect and qualitative information about protein synthesis.

The first attempt to estimate muscle protein synthesis quantitatively in humans was by using 15N-lysine as a

Table 3. 13C Enrichment of Plasma-free α-KIC in Two Groups Studied before and 48 h after Surgery (atom % excess)

<table>
<thead>
<tr>
<th>Control</th>
<th>Epidural</th>
</tr>
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<tbody>
<tr>
<td>Preoperative</td>
<td>Postoperative</td>
</tr>
<tr>
<td>7.3</td>
<td>7.6</td>
</tr>
<tr>
<td>6.7</td>
<td>7.2</td>
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<tr>
<td>5.5</td>
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<td>6.2</td>
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<tr>
<td>6.4</td>
<td>7.3</td>
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<tr>
<td>5.9</td>
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constant infusion. In the present study, the duration of the $^{13}$C leucine infusion, and the amount of labeled isotope administered, were greater than that conventionally used for whole-body $^{13}$C leucine kinetics because it was intended to enrich labeled amino acid, in this case $^{13}$C $\alpha$-KIC, in the immediate precursor pool for protein synthesis, the amino-acyl tRNA pool. The amino acids in this pool seem to be recruited from a subcompartment of the intracellular amino acid pool. Very few estimates of tissue precursor pool have been reported, and the technique of measuring amino-acyl tRNA is technically difficult. The problem of identifying the correct precursor pool is shared by all techniques for measuring protein turnover. When measurements of muscle amino-acyl tRNA labeling were made using $^{13}$C leucine as a tracer, $\alpha$-KIC labeling was shown to be a reasonable surrogate for labeling of the amino-acyl tRNA.

The flooding dose technique was proposed for use in large mammals (humans) to overcome the problem of achieving isotopic steady state rapidly in all precursor pools for protein synthesis when using stable isotope labeled amino acids as metabolic probes. However the fractional synthetic rate reported with the flooding dose technique was almost double that reported with constant infusion. We used the constant infusion technique as the most accepted method adopted by many research groups and also for comparison with our previous studies. In the absorptive state, estimates of fractional synthetic rate vary from 0.04% to 0.07%/h. Our average preoperative values, expressed as a percentage per hour, are within the normal range, although the patients in the study reflected an elderly, but healthy, population. In addition, the decrease in the control group on the third postoperative day corresponds closely with that reported by Tjader et al., regardless of whether conventional intravenous postoperative nutrition was given.

From the studies performed so far using a reproducible lower abdominal surgical model, and under strict conditions of constant nutritional intake and with epidural local anesthetics, we can confidently propose a role of noxious block in modulating postabsorptive whole-body and tissue protein breakdown and synthesis. The findings cannot be extrapolated to other types of surgical stress, and more work needs to be done to determine whether a correlation exists between the extent of neural blockade and the beneficial effects on protein economy.

Postoperative muscle protein synthesis in the epidural group did not increase significantly as we might have expected. Possible reasons are the moderate intake of nitrogen used in our protocol, some degree of immobilization during the 2 days after operation, and finally the study design. Muscle protein synthesis has been shown to decrease in animals and humans who were immobilized for a moderate period. Although passive leg movements of the lower limbs were encouraged in both groups of patients, we did not record accurately the time patients were standing, moving from bed to chair, and walking. The large dose of local anesthetics used to ensure adequate nociceptive block from the surgical area inevitably caused some degree of motor block, thus forcing our patients to remain in bed, especially during the first day. We do not know whether this could have counteracted the sparing protein effect of epidural block. Some recent work seems to indicate that prolonged bed rest for a period of 14 days causes a significant decrease in whole-body and muscle protein synthesis, but it is unlikely that the decrease in muscle protein synthetic rate due to a 48-h bed rest could approach the magnitude of that caused by surgery.

Another aspect to be addressed is whether the observed changes in muscle protein synthesis were directly related to the partially deafferented site. There are no data in the literature to support this contention, and it remains to be seen whether epidural block modifies the protein synthetic rate in all body muscles.

Measurement of protein metabolism during the postabsorptive or fasted state has been the preferred method of several workers, but it is subjected to hormonal changes (such as a decrease in circulating levels of insulin) and does not necessarily reflect the diurnal cycling of feeding. A recent investigation using a fasting/feeding method in a group of surgical patients elucidated the acute effect of nutrients on aspects of whole-body protein metabolism, particularly the direct modulation of feeding on protein breakdown during the immediate period after surgery. The technical and ethical constraints of subjecting patients to several muscle biopsies are the major impediments, although considerable progress in the development of new techniques has been made recently. Determinations of the rate of muscle protein synthesis during fed and fasted states could give valuable information about the economy of body proteins because of the large size of the tissue and the recognition that skeletal muscle is an important

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suppliers of substrates to other vital organs in injured patients.

The mechanism for such metabolic modulation is probably hormonal. Epidural block prevents the increase in circulating levels of cortisol, glucagon, and catecholamines, thereby facilitating the antitrophic effect of insulin. The latter inhibits protein breakdown and amino acid oxidation, thereby facilitating the incorporation of amino acids into proteins.

In conclusion, our findings contribute to a further understanding of how nociceptive block with local anesthetics minimizes the degradation of body proteins and preserves muscle protein synthesis.

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