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Metabolic Basis for the Management of Patients Undergoing Major Surgery

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After reviewing our current knowledge of the metabolic response to major abdominal surgery, we present two new studies in which we have followed the changes in body composition, physiologic function, and psychological function for up to 1 year after major surgery. In the first study, 46 patients had changes in body composition, physiologic function, and psychological function measured perioperatively and again at intervals up to 1 year. There was an average loss of weight of 3000 grams (maximum at 2 weeks), and the tissue composition of this weight loss was composed of 1400 grams fat, 600 grams protein, and 1000 grams water. Normal body composition had returned to normal in most patients by 6 months. In the second study of 84 patients, deficits of skeletal muscle function and fatigue were found to be greatest at postoperative day 7. Most patients were back to preoperative levels of muscle function and fatigue 30 days after surgery, and nearly all had normal values 90 days after surgery. Based on these studies we outline our management program for the metabolic care of patients undergoing major abdominal surgery.

Over many years surgeons have been interested in the early metabolic responses in patients undergoing major surgery particularly while the patients are still in the hospital, but it is only recently that long-term changes in metabolism, body composition, physiology, and psychological function have been studied over the weeks and months following surgery. Here we look at what is known about the metabolic changes brought about by the surgical procedure and show how these changes may persist for weeks and in some cases months before the patient is restored to full health. On this basis we set out a management program for the metabolic care of patients undergoing uncomplicated major elective surgery.

Metabolic Response to Surgery

Cuthbertson [1] divided the metabolic response to injury into an early *ebb phase*, characterized by hypovolemia and subsequent sympathetic and adrenal response, and a later *flow phase*, during which the injured patient loses protein at an accelerated rate. The duration of the flow phase depends on the severity of the injury, and it is gradually replaced by a convalescent *anabolic phase*, during which the protein and energy stores lost in the early postinjury period are repleted (Table 1).

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Ebb Phase

The ebb phase is largely a result of hypovolemia, and it lasts until circulating blood volume is restored. The pale, clammy, tachycardiac patient visited by the surgeon soon after surgery is in the ebb phase. Hume and Egdahl [2] demonstrated the importance of the brain to the early endocrine response to injury. They disconnected an anesthetized animal's limb from its body, leaving only the sciatic nerve and femoral vessels. When they measured the level of corticosteroids in adrenal venous blood in response to a burn to the isolated limb they found an immediate rise; when the sciatic nerve was divided, this response did not occur. These same workers showed subsequently that the early hormone response to injury did not occur unless the central nervous system was intact. Thus pain, hypovolemia, acidosis, and hypoxia initiate the neural afferent signals to the brain; this information is processed by the hypothalamus, which leads to increased activity of the sympathoadrenal system, accompanied by release of adrenocorticotropic hormone (ACTH) and growth hormone (GH) from the anterior pituitary and antidiuretic hormone (ADH) from the posterior pituitary. Plasma cortisol levels rise as a consequence of the release of ACTH, and the renin-angiotensin system is activated. These vasoconstrictor influences decrease renal blood flow, glomerular filtration rate, sodium excretion, and urine flow. Volume retention extends beyond the ebb phase, but diuresis occurs within the first 72 hours in most patients. Plasma insulin levels are variable during the ebb phase, but they are low relative to the prevailing high glucose concentration, probably a result of sympathetic inhibition of insulin release from the pancreas and glucocorticoid-induced insulin resistance in peripheral tissues.

The ebb phase may be prolonged if the patient suffers from postoperative hemorrhage, or it may barely occur if the operative procedure involved little blood loss or tissue damage. Once normovolemia has been restored, this acute ebb phase is replaced by the flow phase.

Table 1. Metabolic response to injury: the ebb and flow phases of Cuthbertson.

Ebb phase	Flow phase
Hypometabolic	Hypermetabolic
Decreased energy expenditure	Increased energy expenditure
Extremities cold and clammy	Extremities warm
Cardiac output below normal	Cardiac output increased
Core temperature low	Core temperature elevated
Normal glucose production	Increased glucose production
Blood glucose elevated	Blood glucose normal or slightly elevated
Catecholamines elevated	Catecholamines high normal or elevated
Glucagon elevated	Glucagon elevated
Insulin concentration low	Insulin concentration low or elevated
Mediated by central nervous system	Mediated by central nervous system and cytokines

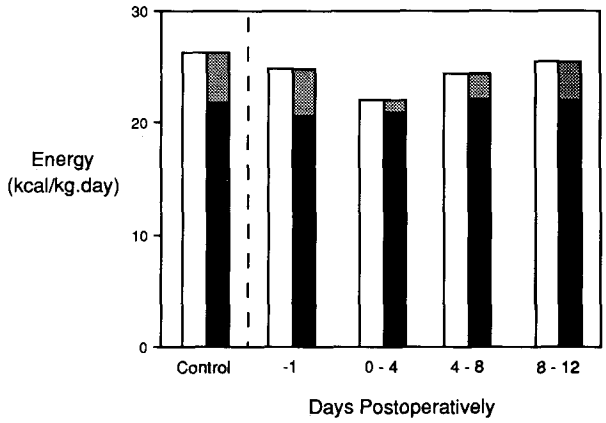

Fig. 1. Components of total energy expenditure in 10 patients undergoing uncomplicated major surgery. It can be seen that resting energy expenditure rises slightly postoperatively. The reduction in total energy expenditure occurred during the period of reduced physical activity. □: total energy expenditure; ■: resting energy expenditure; ▨: activity energy expenditure.

Flow Phase

The flow phase is characterized by oxidation of muscle protein to supply glucose, which is essential fuel for the brain and healing tissues in particular. The accelerated loss of protein results from an increase in the rate of breakdown of muscle protein rather than a reduction in the rate of protein synthesis [3]. In severely injured patients, such as those who have suffered major trauma or serious sepsis, this phase is associated with an accelerated metabolic rate, although as we shall see the resting metabolic expenditure of patients who have undergone elective surgical procedures is only slightly increased. The patient's energy requirements during this phase are mostly met by fat oxidation. The plasma concentrations of the counterregulatory stress hormones decrease during this phase, although the plasma insulin level increases during the flow phase to reach a maximum level paradoxically at the time of the greatest rate of loss of body protein. The reason for the inappropriate rise in the plasma concentration of insulin is little understood, as is the apparent resistance to its usual anabolic effects.

Given that the plasma concentrations of the catecholamines, glucagon, and cortisol are falling during the flow phase, it is difficult to attribute the increased loss of protein observed over this period to their catabolic actions. Although there is little doubt that the central nervous system and neuroendocrine response to surgical injury explain most of the metabolic changes observed during the flow phase after major surgery, the stress hormones are not completely responsible for all of them. Much recent research has focused on the possible role of cytokines released from cells at the site of the wound. Many cell types release *cytokines*, which have not only local effects but systemic effects as well. Interleukin 1 (IL-1), interleukin 6 (IL-6), tumor necrosis factor (TNF), and interferon γ (IFNγ) are considered important mediators of the integrated metabolic response of the host; but with the exception of IL-6, these cytokines have yet to be detected in the plasma of injured patients [4]. Infusion of TNF replicates many of the clinical and metabolic features of sepsis, including fever, hypotension, anorexia, hyperglycemia, and a negative nitrogen balance [5, 6]. Many of the features of the endocrine and metabolic reaction to sepsis, such as insulin resistance and protein loss, are present in injured patients; and it is surprising that to date TNF has not been detected in the serum of patients after major surgery.

Perioperative Energetics

During the first 2 weeks following a major operation the patient accrues a substantial energy deficit. Only half of the energy requirement is met from intravenous dextrose and food; and in order to achieve energy balance, body fat stores and to a lesser extent glycogen and protein stores are burned. In 1968 Kinney and his colleagues conducted a study of energy balance in 10 patients undergoing major surgery [7]. This remarkable detailed study of energy intake and energy expenditure showed that resting energy expenditure—in health around 22 kcal/kg/day—increases slightly early after surgery but from then on, unless there is a complication, remains at normal values (Fig. 1).

Energy expended during physical activity falls to low levels (about 1 kcal/kg/day) over the first four postoperative days; hence during this period total energy requirements are substantially reduced: from the normal 26 kcal/kg/day to 22 kcal/kg/day. By the end of the first postoperative week activity energy expenditure has doubled, and by the end of the second week total energy requirements are near normal values—25 kcal/kg/day—and the patient is eating sufficiently to approach energy balance (Fig. 2). Once home, the appetite improves and positive energy balance is achieved, with slow accumulation of fat and protein and restoration of normal values for fat, protein, and body weight.

Changes in Body Composition After Surgery

We measured the changes in body composition that occurred after major uncomplicated gastrointestinal surgery in 46 patients (23 men, 23 women; average age 47 years). Just before surgery each had measurements of body weight, total body fat,

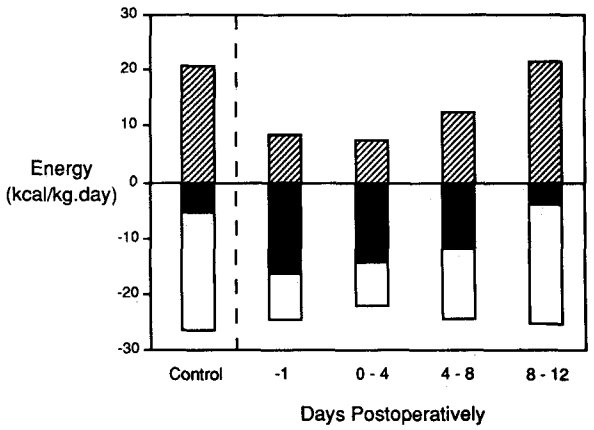

Fig. 2. Energy balance in 10 patients after uncomplicated major surgery. Energy is plotted above the baseline and energy output below it. It can be seen that energy balance was in all cases negative, although at 8 to 12 days just before discharge from hospital energy intake was almost sufficient to achieve energy balance.

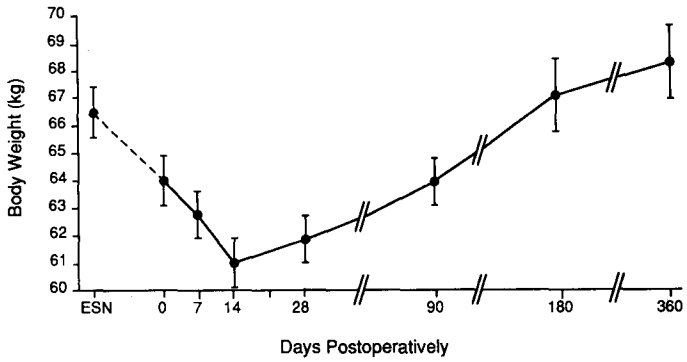

Fig. 3. Study of the postoperative changes in body weight in 46 patients undergoing major surgery of the alimentary tract. ESN: estimated normal weight (mean ± SEM).

protein, and water using our body composition nuclear activation facility [8]. The measurements were repeated 7, 14, 28, 90, 180, and 360 days later. In only 16 patients were measurements performed beyond 90 days. None of the patients in the study received nutritional support perioperatively. Postoperatively, they received 2 to 3 liters of 4.3% dextrose and 0.2 N saline until they were able to take sufficient fluids orally.

Body Weight

After uncomplicated surgery 3 kg of body weight was lost, and the maximum weight loss had occurred by the end of the second postoperative week. From then on, weight was gained slowly and steadily (Fig. 3). At 3 months the weight loss during the postoperative period had been restored, but in those who had had preoperative deficits weight gain continued until normal values were achieved, usually taking about 6 months but sometimes as long as a year. Many patients overshot their preillness or well weight and were as much as 2 to 3 kg overweight at 1 year. The excess weight gain was due to accumulation of fat.

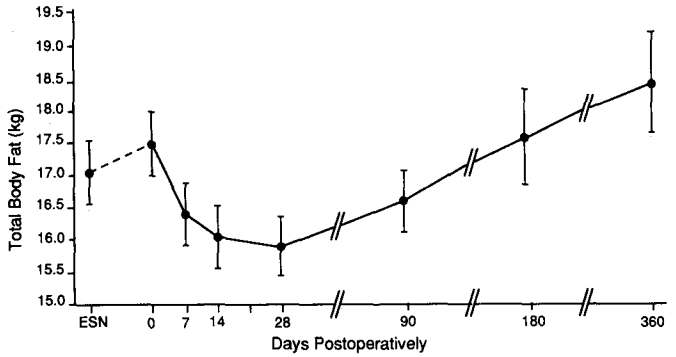

Fig. 4. Study of the postoperative changes in total body fat in 46 patients undergoing major surgery of the alimentary tract. ESN: estimated normal body fat (mean ± SEM).

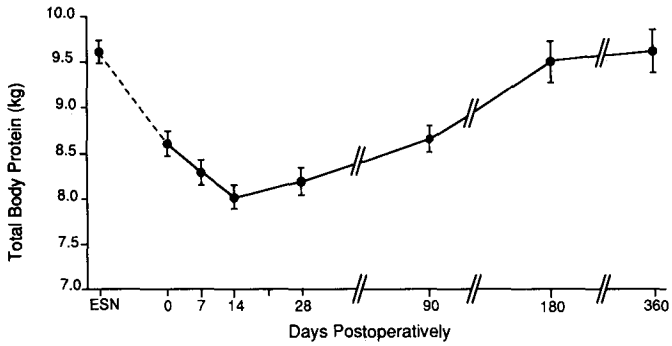

Fig. 5. Study of the postoperative changes in total body protein in 46 patients undergoing major surgery of the alimentary tract. ESN: estimated normal body protein (mean ± SEM).

Tissue Composition of Weight Changes

The postoperative weight loss resulted from oxidation of fat and breakdown of protein to provide energy and amino acids during the first two postoperative weeks. Figures 4, 5, and 6 show how 3000 grams of body weight lost over the first 2 weeks postoperatively was composed of 1400 grams fat, 600 grams protein, and 1000 grams water.

Fat. Most of the fat loss occurred during the first few days postoperatively when the energy deficit was greatest: One kilogram or more was lost during the first week (Fig. 4). After 2 to 3 months, when the patients were well and protein stores had been repleted, fat gain was at its maximum. *Posttraumatic obesity* occurs when this fat gain continues, the result of a continuing positive energy balance. The surgeon should alert patients to this common problem and encourage more physical activity and a tighter control on appetite.

Protein. Protein catabolism occurred over the first two postoperative weeks with losses of total body protein of about 600 grams (6% of body protein) (Fig. 5). Thereafter, with the resumption of normal food intake, protein was slowly and surely accreted, reaching preoperative levels at 3 months and normal values at 6 months to a year. The protein was lost mainly from muscle, but our studies of the loss of potassium and nitrogen in these 46 patients suggest that some noncellular protein was lost as well. There are about 3 mmol of potassium

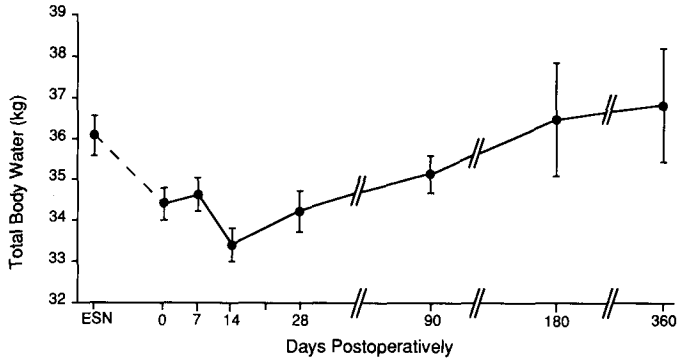

Fig. 6. Study of the postoperative changes in total body water in 46 patients undergoing major surgery of the alimentary tract. ESN: estimated normal total body water (mean ± SEM).

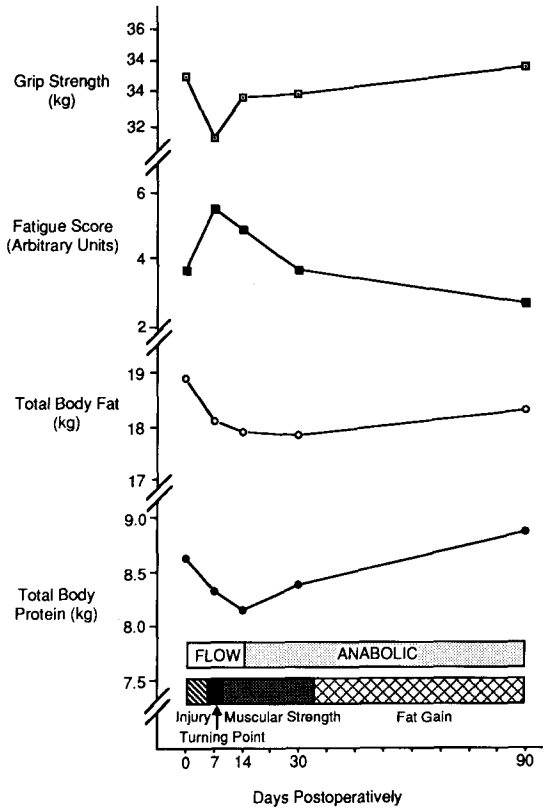

Fig. 7. Changes in total body protein and total body fat together with associated changes in postoperative fatigue and muscle function in 84 patients undergoing major surgery of the alimentary tract. In the lower part of the figure Cuthbertson's flow and anabolic phases are shown (the ebb phase was too short to show) together with F.D. Moore's phases of convalescence.

(K) for each gram of nitrogen (N) in muscle, a K/N ratio of 3:1. Over the first seven postoperative days the K/N was much higher (about 10:1), showing that more potassium than protein was lost from cells, either because glycogen was being mobilized and with it bound potassium-rich water, or an intracellular potassium deficiency was developing. Probably both events were occurring. After 2 weeks, when the patients were in positive energy balance, potassium was replaced at a ratio of 6:1, suggesting that there had been an intracellular deficiency that was now being repaired. Late during convalescence, when protein gains were small, the K/N ratio of the tissue gained was less than 3:1, showing that body protein as a whole, not just cellular protein, was being laid down. Kinetic studies have shown that the loss of total body protein during the early postoperative period is due to an increase in the rate of protein breakdown rather than a reduction in the rate of protein synthesis [3]. The duration and extent of this loss of body protein is a function of body stores of protein (the larger the stores of protein, the greater the loss) and whether the patient is protein-depleted prior to surgery (depleted patients suffer less loss of body protein).

The protein loss would have been greater during the early postoperative period if the fluid regimen used had not contained dextrose. The infusion of 130 to 150 grams of dextrose per day results in a 40% reduction in urine nitrogen loss [9, 10].

Water. During the first postoperative week, when ADH levels were high, water was retained despite close attention to water balance by the attending surgeons (Fig. 6). The patients experienced a fall in plasma sodium levels due to additional sodium-free water, which arose from loss of cell substance and the oxidation of protein and fat. This phenomenon can be understood by reference to Figures 4 and 5. Here it can be seen that 300 grams of protein were oxidized during the first postoperative week. Because 1 kg of wet muscle contains about 200 grams of protein, this loss of 300 grams of protein represents the loss of about 1500 grams of wet muscle, comprising 300 grams of protein and 1200 ml of sodium-free potassium-rich water, which is added to the extracellular space. Furthermore, the oxidized protein itself yields sodium-free water; 300 grams of protein upon full oxidation produces about 120 ml of water.

Over the same period 1100 grams of fat were oxidized. Because each 1000 grams of fat that is completely oxidized yields 1000 ml of sodium-free water, the oxidation of 1100 grams yields another 1100 ml, making a total of nearly 2.5 liters (1200 + 120 + 1100) of endogenous water added over this period. Because ADH levels were high during the postoperative period this endogenous water was not excreted normally, resulting in early positive water balance, increased hydration of the fat-free body, and bodily hypotonicity. Normal tonicity had returned by the end of the first postoperative week, and hydration of the fat-free body had returned to normal within a month of operation. The oliguria and salt and water retention that occurred over the first few days postoperatively were also related to high aldosterone activity and may have been partly due to decreased atrial natriuretic peptide. Alterations in vascular tone that are associated with positive-pressure ventilation decrease venous return to the right side of the heart and as a consequence may decrease secretion of this hormone.

Postoperative Fatigue and Skeletal Muscle Function

One of the most unpleasant effects of surgery is the long period of mental and physical tiredness that follows it. In a study of 84 patients [37 men, 47 women; average age 54 ± 18 SD) years] undergoing major surgery of the alimentary tract [11, 12], we found that many of the patients, because of their illness, had a subjective feeling of fatigue prior to the operation, and it

became worse postoperatively, being greatest at the end of the first week (Fig. 7). At 1 month postoperatively most patients were back to preoperative levels of fatigue, and by 3 months the fatigue had all but disappeared. A few patients, however, particularly those in whom postoperative weight loss was most marked, had more pronounced fatigue that was also prolonged, a situation described previously [13]. We found that fatigue was most troublesome in those patients who came to surgery already tired. There was evidence that patients who had few reserves of body protein (those with small muscle mass), the elderly, and those who knew they had cancer, even though a curative operative operation had been performed, most often suffered from prolonged fatigue.

Voluntary muscle function also changes after surgery, and we found that the pattern of deterioration that occurred was in some ways similar to that which occurs for fatigue (Fig. 7). It might be thought, then, that postoperative fatigue was a simple matter of loss of muscle protein affecting muscle function adversely, and the whole effect therefore was that of physical tiredness. Unfortunately, it is much more complicated. For instance, postoperative enteral nutrition, which preserves body protein stores postoperatively, does not prevent postoperative fatigue [14]. Also, involuntary skeletal muscle function, unlike voluntary muscle function, has been shown not to deteriorate postoperatively [11]. Thus postoperative fatigue, which is an exclusively human problem (it does not occur in animals), has a psychological basis as well as a physiologic one.

In summary, then, whereas postoperative fatigue is experienced by all patients to a greater or lesser extent, it is not much of a problem in patients who feel well prior to surgery or those who are physically robust. Those patients presenting for surgery already feeling tired, however, especially if elderly and with a diagnosis of cancer, are likely to suffer from prolonged fatigue. The cause of this debilitating condition is not yet known, but psychological factors are predominant. As yet, no therapeutic manipulation has been found that prevents postoperative fatigue.

Phases of Convalescence

Moore [15] described surgical convalescence in terms of four sequential phases starting immediately with the operation itself and stretching out for 3 or even 12 months until the patient was functionally rehabilitated and returned to normal activity. Moore's four phases are the *injury phase*, the *turning point phase*, the *gain in muscular strength phase*, and the *phase of fat gain*.

In Figure 7 it can be seen how these four phases adequately describe the process of operative injury and convalescence when they are considered in terms of body composition, physiologic function, and postoperative fatigue. Cuthbertson's flow and anabolic phases are also shown in Figure 7; note that with major elective surgery the ebb phase is too short to be properly depicted here.

Injury Phase

The injury phase of Moore comprises not only the ebb phase described earlier but also part of the flow phase. This phase extends over the first four postoperative days. It begins as a phase of high catecholamine and adrenocorticoid activity. It is the time of maximal energy deficit and maximal oxidation of fat and protein. Protein synthesis stays the same or is increased, and protein breakdown increases. Potassium is lost in excess of nitrogen due to mobilization of glycogen from liver and muscle. In the beginning the patient is cold and clammy, pale, and tachycardic; throughout he or she is tired, takes little interest in food and visitors, and likes to be left undisturbed. The duration of this phase depends on the magnitude of the operation and the disturbance in physiology brought about by it. Hypovolemia, atelectasis, and acid-base imbalance prolong it; and the development of a postoperative complication such as sepsis, peritonitis, embolus, or necrosis extends this relatively minor alteration in metabolism into another order of magnitude.

Turning Point Phase

Around the middle to the end of the first postoperative week patients start to take an interest in their surroundings and wonder at their disheveled appearance. They want to get up; men wish to shave and comb their hair, and female patients may be seen to be looking in the mirror and applying lipstick. This phase normally lasts only 1 to 2 days; and if a septic complication is present or developing, the change is incomplete and appears later.

It is at this time that the desire to get up and get moving is tempered by extraordinary tiredness, and the patient quickly returns to bed. Most patients now take an interest in food; and a few sips of water is soon regarded as insufficient, and a soft diet is begun (Fig. 8). The stage of maximum fatigue and loss of skeletal muscle strength is coming to an end. Endocrine function has returned toward normal, although protein catabolism is continuing. Because endocrine activity has now decreased, at somewhere between 3 and 5 days a diuresis occurs. Potassium loss in the urine is less as intracellular deficits in potassium are made up. The transient hypotonicity (from hyponatremia) returns to normal, and the expanded extracellular fluid volume returns toward normal. Active energy expenditure is increased during the turning point; but because energy intake is still substantially less than normal (Fig. 8) an energy deficit persists that is balanced by catabolism of fat and protein. The key to success at this stage is the increasing provision of a good diet.

Muscular Strength Phase

The patient now passes into a period of 2 to 8 weeks where there is rapid psychological and physiologic improvement. The excitement and reality of going home is experienced, voluntary food intake is near normal, and the wound is less painful. This condition together with normal bowel function and diminishing fatigue lead to encouragement. At the end of 8 weeks body composition, physiologic function, and the feeling of tiredness have been restored to near the state in which they were just prior to the operation.

Fat Gain Phase

The final period of convalescence is a gain in body weight due to accumulation of body fat and its supporting structure. It occurs because there is continuing positive energy balance,

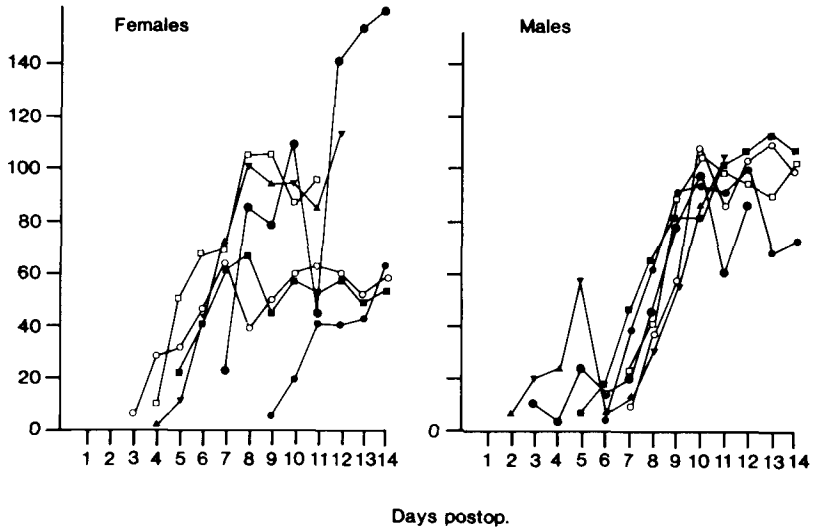

Fig. 8. Daily voluntary food intake (energy) that occurred over the first two postoperative weeks in 12 patients after colorectal surgery. On the vertical axis the results are expressed as a percentage of the estimated previous home voluntary food intake. (Redrawn with permission of the publisher.)

with intake of energy being greater than that used for work and heat. The protein that is gained together with this fat is thought to be that of the supporting structure in adipose tissue, as there is little change in total body water even up to 1 year. Somewhere during the fat gain phase the patient returns to normal daily activity.

Metabolic Management of the Patient Undergoing Major Surgery

Our management recommendations are based on the measured changes in metabolism, body composition, physiology, and psychology we have described. It begins before the patient enters the operating room and ends when the patient is functioning again in his or her normal environment.

Preoperative Management

If the surgeon takes time to give a careful and relevant account of what is to take place in the hospital, describing the sensations that will be experienced and how one copes with them, there is evidence that the hospital stay will be shorter [16]. The surgeon may choose to explain to those who are likely to experience excessive postoperative fatigue that it will be a long convalescence so disappointment and discouragement can be minimized [12]. The physiotherapist may help by teaching the patient exercises that will be used postoperatively and how to turn in bed and move so pain is minimal. If investigations have gone on for several days and particularly if little food has been consumed over the previous week, the wound healing response will be impaired, and special care must be taken [17]. If there is any question of deficiency, vitamin C is given. The prescription of prophylactic antibiotics [18] and proper precautions against venous thrombosis add to a smooth postoperative course.

At the Time of Surgery

That the magnitude of cell injury, the amount of necrotic tissue created, and the presence or absence of infection influence the release of cytokines helps the surgeon set goals and plan strategies for the operative task. *The goal is to keep cytokines out of the circulation.* The least damaging, least contaminating, and most expeditious procedure is the way to do it. The strategies include excellent exposure, sharp dissection along anatomic planes, careful hemostasis, accurate apposition of tissues, and careful suturing without strangulation. These strategies, so essential to a smooth postoperative course, are the result of a mind set developed some days or hours before the operation. The wise surgeon, no matter how familiar the procedure, goes through it all silently step by step in relation to the particular patient. It is this sort of approach that characterizes surgeons whose patients seem to "fly through."

At the conclusion of the operation, thorough peritoneal toilet minimizes the risk of leaving necrotic debris or clot behind. Careful hemostasis throughout ensures that large hematomas do not interfere with postoperative recovery.

Anesthetist

Although spinal or epidural anesthesia prevents the greater part of the classic endocrine response to surgery, the effect is maximal during pelvic surgery (e.g., prostate and gynecologic procedures) and lower limb orthopedic procedures; it is much less during abdominal and chest procedures probably because of insufficient afferent neural blockade [19]. In any event, most major surgical procedures require general anesthesia, but the hemodynamic effects induced by it as well as the operative procedure can be minimized by the anesthetist. Hypovolemia must be prevented, and fluid deficits due to radiologic procedures or mechanical bowel preparation should be replaced soon after sleep is induced. During the operation blood pressure should be manipulated to within 10% to 20% of the preoperative level and urine output within the range of 0.5 to 1.0 ml/kg/hr by addition of a balanced salt solution given in amounts over and above maintenance requirements. The amount of this extra "third space requirement" is dependent on the nature, extent, and duration of the operation; and it may extend for 12 hours or so into the postoperative period. Proper attention to fluid volume and control of blood pressure, pulse rate, and urine output lessens the endocrine response to the operation [20],

reduces the time of the injury phase, and leads to a shorter hospital stay.

Blood (mainly given as packed red blood cells) requirements during operation are a controversial topic, and the decision to transfuse requires expert clinical judgment [21]. The combination of hypovolemia and anemia, though, may lead to severe morbidity and possibly mortality. There is a minimum hemoglobin value for each patient below which inadequate oxygen delivery is likely to occur. The decision to transfuse takes into account the presence or absence of perioperative anemia, the intravascular volume, extent of the operation, probability of continuing massive blood loss, and presence of coexisting conditions such as impaired pulmonary function, inadequate cardiac output, myocardial ischemia, or cerebrovascular or peripheral arterial disease.

Management During the Postoperative Phase

Pain Relief. Postoperative pain serves no useful function and if inadequately controlled may adversely affect respiratory function and intracardiac demands, decrease intestinal motility, and induce skeletal muscle spasm, which itself impairs mobilization. If regional anesthesia has been used, it should carry through the operation and probably go on for 24 to 48 hours.

Systemic administration of opioids, given according to either a fixed administration regimen or a demand-based regimen, has unfortunately no important modifying effect on the endocrine response to the operation. Nevertheless, opioid effects are vital to reducing or eliminating postoperative pain, improving respiratory function, decreasing cardiac demands, and controlling muscle spasm.

Intravenous Fluids. If on return to the ward the patient is tachycardiac and peripherally shut down (ebb phase), more crystalloid is required. In most situations it is provided by isotonic crystalloid solutions (Ringer's lactate) in sufficient quantities to keep blood pressure and pulse rate near normal preoperative levels and urine output at 0.5 to 1.0 ml/kg/hr. In many patients losses continue from the intravascular compartment for 12 hours or so, and during this time isotonic fluid replacement is required.

After some hours the capillary leak closes, and fluid is slowly mobilized from the periphery into the vascular space; increased urine output results. At this point fluids should be changed to maintenance rates or lower and from isotonic saline to hypotonic saline.

In adequately hydrated patients who have undergone a straightforward procedure with minimal blood loss, maintenance fluid alone is all that is required. For maintenance a 70-kg patient is normally given dextrose in saline (100 ml/hr) to which 20 mmol of potassium has been added. The provision of 130 to 150 grams of dextrose in the postoperative fluid regimen has, by suppressing endogenous glucose production, a modest protein-sparing effect. The urinary urea nitrogen is about 40% less when dextrose is given in this way [10]. Because of the dissolution of cells, which flood the extracellular water with potassium, it is advisable to refrain from giving potassium for the first 24 to 48 hours postoperatively.

Energy and protein supplied either by the enteral or parenteral route can prevent completely the loss of protein after uncomplicated major surgery. Unfortunately, there is no obvious clinical benefit either early or late from preventing protein loss [22], although some suggest that specially formulated enteral diets may have a place in reducing postoperative complications [23]. It has been shown, however, that the wound healing response is enhanced by postoperative nutrition, and with large granulating wounds it may be of some clinical benefit [14, 24]. Hormonal manipulation has also been tried. Insulin added to total parenteral nutrition (TPN) regimens and intramuscular GH administration result in reduced postoperative protein loss, preservation of body composition, and increased hand grip strength [25, 26]. Much more work is required, however, before this protocol can be used routinely for major surgery [27].

Role of Nurse, Physiotherapist, and Dietitian. The best metabolic care, as the patient passes into the "turning phase" and beyond is encouragement of positive attitudes by the nursing staff, insistence upon increasing mobilization by the physiotherapist, and provision of attractive and nourishing food by the dietitian. Indeed the key of success is the resumption of eating as the anabolic phase commences. Early on, the patient prefers carbohydrates to protein, but by the middle of the second week a balanced diet, equivalent in energy and protein content to that being consumed at home just prior to admission, should be consumed (Fig. 8). Some patients with artificial dentures experience gum shrinkage after surgery, and the discomfort of chewing in these circumstances limits protein intake. Provision of special dietary supplements in patients who are slow to reach energy and protein balance appears to be beneficial [28].

Can Anything Be Done to Limit Postoperative Fatigue? Although careful preoperative counseling can limit the toll of surgery by shortening the hospital stay, it has not been shown that it has any effect on the magnitude or duration of postoperative fatigue. Postoperative nutrition sufficient to abolish net protein loss similarly has not been shown to limit postoperative fatigue. On the other hand, it appears that subjects who are fit prior to surgery of modest magnitude experience little if any fatigue postoperatively.

Minimally Invasive Surgery and Metabolic Response

Minimally invasive surgery, in particular laparoscopic cholecystectomy, in which the gallbladder is removed through tiny abdominal incisions, is of particular practical and scientific interest. Even though a general anesthetic is required and the procedure may be prolonged, it seems to be associated with shortened convalescence and minimal postoperative fatigue [29]. Although preliminary investigations suggest that the neuroendocrine responses are the same as those that occur after open operations [30], full scientific evaluation of the metabolic effects of these procedures and the postoperative physiologic and psychological effects is awaited.

Résumé

Après avoir passé en revue les connaissances actuelles de la réponse métabolique à la chirurgie abdominale, nous présentons deux nouvelles études dan lesquelles les changements dans

la composition corporelle, les fonctions physiologique et psychologique ont été suivies pendant un an après une chirurgie abdominale majeure. Dans la première, comportant 46 patients, la perte de poids corporel moyenne a été de 3000 g (valeur maximale à deux semaines), composé de 1400 g de lipides, 600 g de protéines, et de 1000 g d'eau. La composition corporelle est revenue à la normale six mois plus tard chez la plupart des patients. La deuxième étude a intéressé 74 patients, où le déficit de la fonction musculaire squelletique et la fatigue ont été retrouvés au maximum au 7è jour postopératoire. Le retour aux taux préopératoires a été observé pour la plupart des patients 30 jours après et pour pratiquement tous les opérés, 90 jours après. Nous basant sur les résultats de cette étude, nous avons établi une politique de soutien métabolique pour tout patient ayant une intervention de chirurgie abdominale majeure.

Resumen

Después de revisar el conocimiento actual sobre la respuesta metabólica a la cirugía abdominal, presentamos dos nuevos estudios en los cuales se determinaron los cambios en composición corporal, función fisiológica y estado psicológico por un periodo de hasta un año luego de cirugía mayor. En el primer estudio se midieron tales cambios en 46 pacientes en la fase perioperatorio y a diferentes intervalos hasta un año después de la operación. Se encontró una pérdida promedio de peso corporal de 3000 g (máxima a las dos semanas) y la composición tisular de tal pérdida representada por 1400 g de grasa, 600 g de proteína y 100 g de agua. La composición corporal retornó a su estado normal en el curso de seis meses en la mayoría de los pacientes.

En el segundo estudio, sobre 84 pacientes, se encontraron máximos déficits de la función muscular esquelética y fatiga en el 7° día postoperatorío. La mayoría de los pacientes retornaron a sus niveles preoperatorios de función muscular y de fatiga a los 30 días después de la cirugía y casi todos presentaban valores normales a los 90 días.

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