Nutritional Evaluation during the Altered Physiological State after Injury

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THE RESPONSE to severe injury appears to be a response of every system, every organ, and presumably every cell in the body. It is a response, not of the moment, but of days or weeks. The magnitude and duration of this response appear to be directly proportional to the magnitude of the injury.

Since this response is primarily metabolic in nature, it is not surprising that almost every metabolic study reveals a deviation from the normal pre-injury state.

Most injuries include at least three components—the destruction of tissue, the loss of blood, and a break in the defense against bacteria. Each component of injury evokes additional facets of the metabolic response. For example, the loss of blood is associated not only with the inherent loss of its substances but with the changes brought on by renal vasoconstriction, by the autonomic response, by the adrenal cortical response, and by the increased production of red blood cells, albumin, fibrinogen, platelets, and other components. The destruction of tissue may be associated with the loss of water or plasma as local edema and with the increased metabolism of the cellular proliferation of wound healing. Infection increases the exudate, increases the destruction of tissue, and evokes leukocytic and antibody responses.

The above changes are used only as examples of the complex nature of injury and the responses which are evoked. Without an appreciation of this dynamic state, the physician may discover many pitfalls in his evaluation of the nutritional state of the postoperative or post-injury patient.

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POTASSIUM AND NITROGEN

Associated with tissue destruction there is an increased excretion of potassium and nitrogen in the urine. This appears to be due not only to the adrenal cortical response but also to the increased destruction of tissue (Fig. 1). A patient with an acute arterial injury and a resultant gangrenous limb may excrete far more potassium and nitrogen than does the patient with an amputation stump free of necrotic tissue.[1] This increased metabolic load coincides with a period of renal vasoconstriction and decreased glomerular filtration[2] so that in the absence of pre-existing renal disease, a relative retention of nitrogen or potassium may occur and be associated with rising plasma concentrations. A transient rise in the concentration of potassium in the plasma is not uncommon on the day of severe injury. Unless renal damage is severe, the plasma potassium concentration rapidly returns to a normal level.

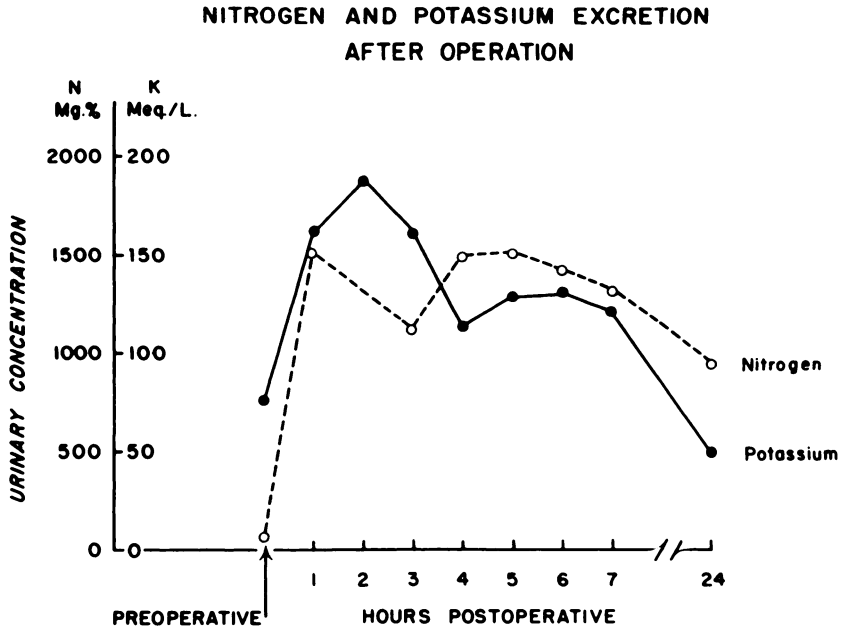
Even though the plasma potassium concentration returns to normal, the blood urea nitrogen concentration may continue to rise for several days, although the total nitrogen excreted in the urine remains elevated (Fig. 2). These findings, after very severe injury, do not suggest the presence of pre-existing renal disease, but if noted after minor trauma, strongly suggest the presence of a pre-existing impairment of the renal reserve.

SODIUM

Coincident with the potassium and nitrogen diuresis, there is a conservation of sodium and water. The net conservation of sodium over a period of two weeks may amount to 500–900 milliequivalents. In spite of sodium retention, the plasma sodium concentration often falls sharply within a period of a few hours, and may remain at subnormal levels

for several days.[1] Accompanying the fall in sodium concentration, there may be a transient decrease in the total calcium concentration in the plasma.[3]

taken for diabetes. Similarly, the diabetic patient undergoing operation may have a rise in his blood sugar concentration and may increase his insulin requirements during the



Nitrogen Balance

Preoperative (24 hours) +1.5 grams

Postoperative (24 hours) −19.7 grams

Potassium Balance

Preoperative (24 hours) +3.2 Meq.

Postoperative (24 hours) −174.9 Meq.

Figure 1

The tremendous mobilization of nitrogen and potassium by a patient who developed a gangrenous extremity as a result of an arterial embolus and unsuccessful embolectomy is indicated.

Glucose

The blood glucose concentration rises with injury as a result of the responses of the sympathetic nervous system and the adrenal cortex. The fasting blood sugar may remain high throughout the week after injury, slowly subsiding toward the normal level. During this time the glucose tolerance curve is "diabetic" in type.[4] The stress response, being antagonistic to the effect of insulin, is associated with a decreased sensitivity to insulin. Because of the high blood sugar concentration, the normal response to trauma may be mis-

stress response. As a possibly related phenomenon, the serum amylase concentration often falls after nonspecific trauma.[5]

The rise in the blood sugar concentration during acute pancreatitis has often been attributed to an insulin insufficiency. A transient diabetic state due to the destruction of the islet cells is probably quite infrequent. Instead, the elevated fasting blood sugar level, the glucosuria, and the transient diabetic-type glucose tolerance curve are probably nonspecific indications of the severe trauma of acute pancreatitis.

Protein

Although the changes in carbohydrate metabolism may be most marked immediately after injury, the changes in protein metabolism become increasingly evident during the ensuing days. Not only is the nitrogen excretion increased, but the concentration of the serum proteins changes in a predictable manner. Following abdominal trauma, there is a rapid, progressive decrease in the albumin-globulin ratio, the ratio often falling to less than one by the third day post-injury. Following injuries of the extremities, the changes are less marked but are qualitatively similar.[6] Electrophoretic studies indicate a decrease in the relative proportion of albumin and an increase in the alpha$_1$ and alpha$_2$ globulin fractions.[6] These progressive changes reflect not only the continuing response of the patient but probably also reflect the continuing nature of the injury—that is, the continuing loss of albumin. Thus the low albumin-globulin ratio after injury does not necessarily reflect a pre-existing state of malnutrition.

A second aspect of the changes in protein metabolism is reflected in the excretion of creatinine and creatine after injury. Severe trauma to any part of the body may result in a sharp rise in the total urinary excretion of creatinine, an excretion which may reach a level of 4.0 to 6.0 grams per day. Similarly, the excretion of creatine may reach a level of 3.0 to 4.0 grams per day, but appears to follow injuries which involve the destruction of large masses of muscle. The latter observation, therefore, has been made in patients with injuries to the extremities in contradistinction to injuries of the viscera.[7]

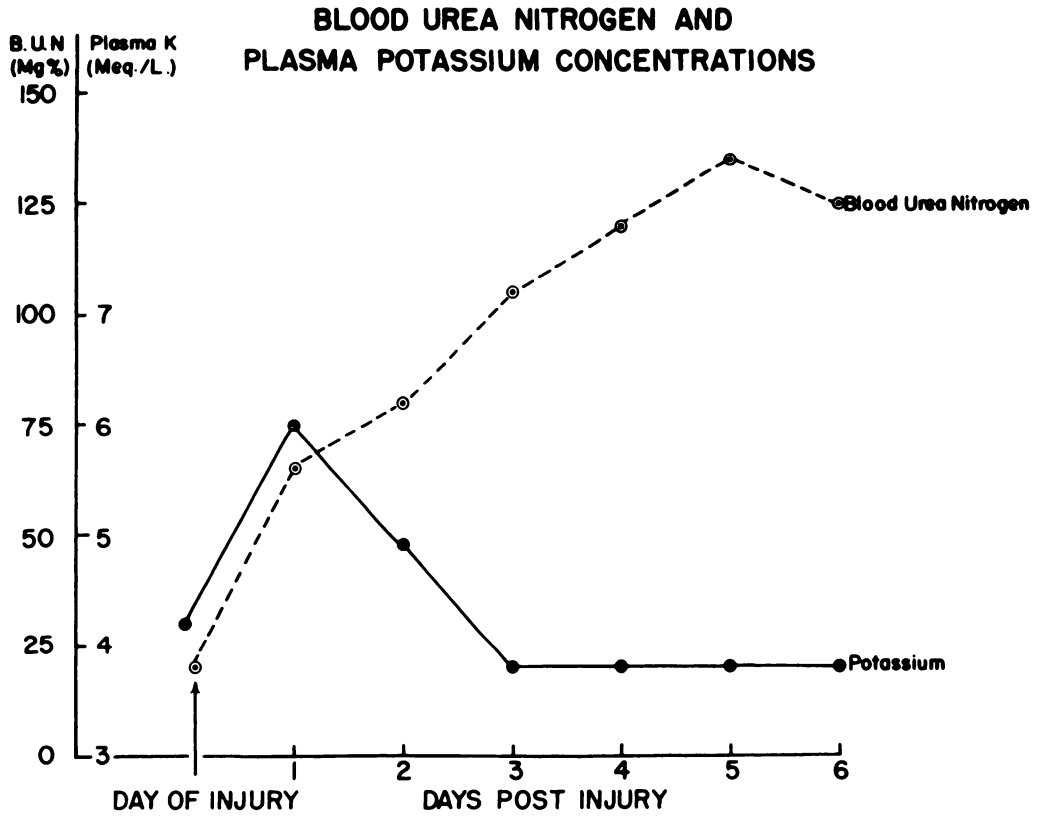


Figure 2

This figure demonstrates the transient rise in the plasma potassium concentration and the prolonged rise in the blood urea nitrogen concentration in a non-oliguric battle casualty.

Other Changes

Payne and Krauel,[8] studying one component of lipid metabolism demonstrated further alterations in the metabolism of this component after injury. Studying the lymph before and after thermal injury, they found that the marked increase in lymph flow from the injured extremity was associated with a striking increase in the local mobilization of cholesterol, neutral fats, and phospholipids.

The metabolic response is demonstrated not only by these broad patterns but also in the response of the individual organs and systems.

As a response to combat injury, including hemorrhage, Scott and Crosby[9] demonstrated a decrease in the clotting time and a rise in the platelet count and fibrinogen concentration. Immediately after injury and resuscitation, the prothrombin activity fell to approximately 50 per cent of normal. This defect corrected itself within one to three days but was followed shortly by a second fall of similar magnitude. Recovery to normal then gradually progressed. This defect could not be prevented by the administration of vitamin K. Rather than being a pure deficiency of prothrombin, it appeared to be a deficiency in the activity of the accelerator globulins. This decreased prothrombin activity after injury is not indicative of pre-existing hepatic disease.

It is well known that hemorrhage is followed within a few hours by hemodilution. Patients who have had a mass of muscle destroyed can have their hematocrit and blood volume maintained by transfusion, but shortly after the transfusion is completed the hematocrit will begin to fall, often resulting in a rather marked anemia as the red blood cells are lost.[10] Conversely, patients with severe abdominal injuries, like patients with severe thermal injuries, lose more plasma than red cells in the postoperative period, often demonstrating an increase in the hematocrit after transfusion has been discontinued.

Injury is followed by a transient decrease in the mean corpuscular volume.[11] The significance of this observation is unknown but the finding suggests that these cells lose water to the extracellular compartment, a shift which, if it occurred from all the cells in the body, might explain the concurrent fall in plasma sodium concentration. Reticulocytosis reflects the bone marrow's response to hemorrhage; the reticulocyte count was 3 to 5 per cent in one study of the severely injured.[11]

Following severe trauma, the leukocytic response is often quite striking; a count of 20,000 to 50,000 cells per cubic milliliter was often found in the combat casualty a few hours after injury.[11] The count often demonstrated a moderate drop while the patient was under anesthesia and undergoing operation. Thereafter, it again rose. Occasionally there would be a precipitous drop in the white cell count to 500–1000 cells per cubic milliliter, a reaction associated with a fall in the platelet count, a fall in the blood pressure, and sometimes a demonstrable bacteremia. This response, the hemoclastic reaction, denotes a rather grave prognosis. The eosinopenia after trauma is so well known as to need no comment.

Liver Function

As a result of hepatic ischemia and of the hemolysis occurring after transfusion and in hematomas, a rise in the serum bilirubin follows severe injury and rapid transfusion.[12,13] On the day of injury and resuscitation, the serum bilirubin may rise steadily to a level of 2.0–5.0 mg per 100 ml, reaching its maximum about six hours after operation and then characteristically subsiding rapidly. This increment is predominantly in the indirect, protein-bound fraction.

Other hepatic function studies may lead to false interpretation unless the response to injury is appreciated. The cephalin flocculation reaction is increased following injury.[12] The magnitude and duration of this change, as with so many of the above changes, appears proportional to the magnitude of the original injury. The thymol turbidity test does not demonstrate an abnormal reaction after injury. These two tests, usually employed to demonstrate disease of the hepatic cells, appear to be based fundamentally on an undefined relationship of the albumin-globulin fractions. The above response in the cephalin flocculation test may not reflect hepatic injury but instead may reflect the acute extrahepatic shifts in the albumin and globulin fractions.

Bromsulfalein retention can almost invariably be demonstrated after severe injury.[12,13] A retention of 15 to 40 per cent 45 minutes after the intravenous injection of 5 milligrams of bromsulfalein per kilogram of body weight has been described on the day following severe trauma, an observation more frequently found after abdominal trauma than after injury to the extremities. During the week after injury, the excretion of bromsulfalein gradually returns toward normal.

The pitfalls in interpreting prothrombin activity as a test of hepatic function after trauma have been discussed previously.

Studies of the gastrointestinal system have demonstrated a transient achlorhydria after trauma,[14] the frequent failure of roentgenographic visualization of the gallbladder,[15] and a decreased gastrointestinal absorption of water.[16] Renal function studies demonstrate the frequency of albuminuria,[13] the decreased renal clearance,[2] and the above-mentioned rise in the concentration of nonprotein nitrogen products in the blood. The adrenal cortical response, the basis for some of the previously mentioned observations, is well recognized.

These are all part of the body's response to severe trauma. They do not represent preexisting disease or detectable post-injury complication. To fail to recognize the body's total response to trauma will subject the clinician to many pitfalls in the evaluation of the patient in the post-injury state.

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