Gas Gangrene of the Abdominal Wall:

Management after Extensive Debridement

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TREATMENT of gas gangrene of the abdominal wall requires aggressive debridement. Even though oxygen at hyperbaric pressure and other adjunctive measures may help to decrease the extent of the excision that is needed to control clostridial infection,[1] a massive forfeiture of skin, muscle, and fascia may still be necessary.[8] When debridement includes a complete unilateral or bilateral extraperitoneal excision of the abdominal wall, an unusual problem is created for later reconstructon. After the procedure the abdominal viscera are contained only by a thin peritoneal sac, and so large an area of denuded peritoneum can be expected to keep its mechanical and bacteriologic integrity for only a few days.

Two recent patients illustrate several aspects of this problem. The first patient, whose overwhelming concurrent problems prevented closure by grafting shows the postoperative evolution of the wound created by abdominal wall debridement. The second demonstrates a successful method of closure in which a temporary mesh prosthesis was used to maintain mechanical strength of the abdominal wall until split thickness grafting was completed. Because total parenteral nutrition played an essential role in this patient's recovery, balance data are presented, showing that the catabolic process induced by sepsis and starvation was associated with nitrogen loss that if unreplaced could be expected to have caused death from starvation.

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Case Reports

Case 1. A 65-year-old man was admitted to the hospital because of painless hematuria; a Grade III transitional cell carcinoma infiltrating the bladder was found. His past history included asthma, with three courses of steroid therapy, and several episodes of nodal tachycardia. After a mechanical, but not an antibiotic preparation of the bowel, a total cystectomy, bilateral iliac lymph node dissection, and uretheral diversion to an ileal conduit were performed. No unusual technical problems were encountered during the operation, and the patient's first postoperative day was uneventful. Maximum temperature was 99° F. At 9:00 p.m. on the second postoperative day, the temperature rose to 103° F; there was tachycardia and leukocytosis. At this time the skin around the incision was observed to be slightly reddened and indurated, but its appearance did not seem sufficiently abnormal to explain his fever. Nevertheless, he was given large doses of penicillin and Chloromycetin and a search was made for other sites of infection—pulmonary, intraperitoneal, or renal. Just 4 hours later the abdominal wall showed unmistakable evidence of gas gangrene: bronze, brawny induration with crepitus. Gram-positive rods were found in a Gram-stained smear and cultures from the wound subsequently grew *Clostridium perfringens*. Abdominal wall debride-

ment was performed, at pressure, in the hyperbaric chamber.

A day later the debridement was extended, and at the end of the second procedure all layers of the abdominal wall except the peritoneum and a little posterior fascia adherent to it, had been excised from the costal margins to the symphysis. This operation was followed by a series of complications, and eventually, the patient's death. Acute renal failure, first oliguric, then polyuric, was the first; the metabolic consequences of uremia were controlled by hemodialysis. Respiratory failure, initially non-bacterial and characterized by decreased compliance and increased veno-arterial admixture (P_AO_2 58 mm. Hg after a 20-minute trial inhalation of 100% O_2) necessitated tracheostomy and mechanical ventilation. A progressive, and eventually complete motor and sensory loss below C5 was observed and was never satisfactorily explained. Daily transfusions were needed to replace blood lost from multiple gastric mucosal erosions. Eventually, recurrent episodes of Gram-negative bacterial bloodstream infection led to refractory hypotension and death.

During these events, it was possible to observe the consequences of peritoneal denudation. The bare peritoneal surface was treated at first with Sulfamylon applications, then with wet fine-mesh gauze. The original intent had been to apply split thickness grafts; because of the patient's progressive deterioration this was never done.

The day-by-day evolution of the peritoneal surface was as follows:

Day 1–3: Immediately after debridement, the peritoneum and adherent fascia were clean and glistening; their appearance was normal. There was, however, persistent bleeding from adjacent muscle which despite cautery and ligature required eight units of blood replacement.

Day 4: Crusting and eschar formation were observed under the Sulfamylon. Muscle bleeding continued; five units were replaced.

Day 7: Adherent fascia became black. Spotty areas of pertioneum were frankly necrotic, while other areas began to exude serous fluid. Cultures were sterile.

Day 9: Widespread full-thickness necrosis of the peritoneum was now evident.

Day 10: Dehiscence and evisceration occurred under the dressings resulting in bacterial peritonitis and death from septicemia.

Several points relative to massive abdominal wall defects were made obvious by this man's course. First, peritoneum widely exposed is not viable, if there was any thought that it might be. Second, continued bleeding from divided muscle, commonly a problem after extensive debridement for gas gangrene, and uncertainty that the infection had been controlled led to a decision against immediate grafting. After 3 days the denuded peritoneum developed an eschar which made it unsuitable for grafting and the chance was lost. The eschar separated in about 10 days, but to reveal abdominal viscera, not granulating peritoneum, and the resulting generalized peritonitis killed the patient.

Case 2. A 69-year-old man was admitted 3 days after right colectomy for a small ascending colon carcinoma. No metastatic disease had been found at operation and all nodes in the surgical specimen were negative. On the third postoperative day, he became restless and disoriented, febrile (101° F), oliguric, and was observed to be jaundiced. Examination of the abdominal wound suggested Clostridial infection. He was given penicillin, polyvalent Clostridial antitoxin, and transferred, as a candidate for hyperbaric oxygen therapy. A smear from the wound taken at the time of admission showed abundant Gram-positive rods with bizarre morphology due to penicillin therapy, but which grew in culture characteristic *Clostridium perfringens*.

All abdominal wall muscles on the right side were necrotic and were debrided. On the left,

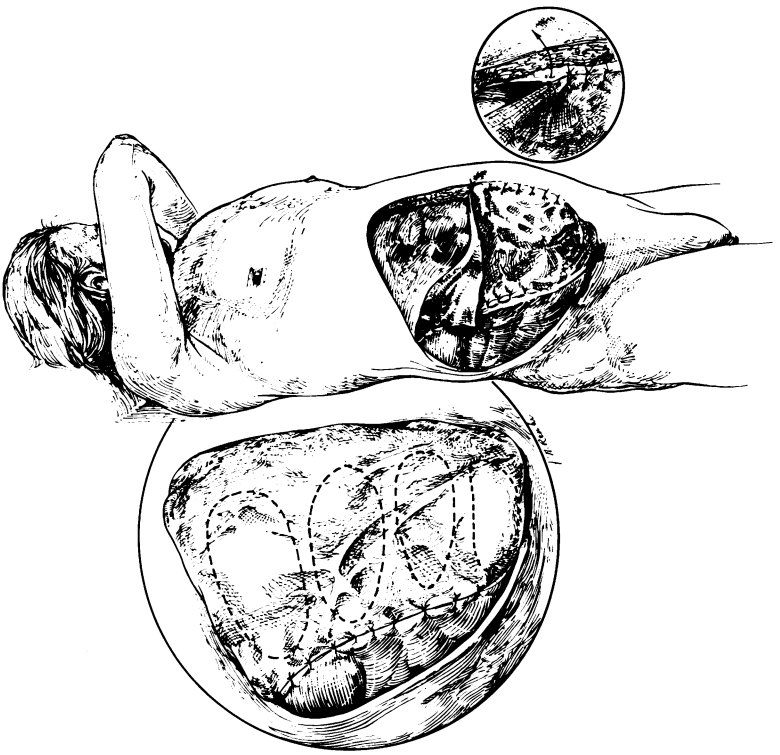

FIG. 1. Temporary mesh closure of abdominal wall defect. Above: mesh applied to cover and reinforce peritoneum. Below: windows cut to allow later application of split-thickness grafts to peritoneum.

only the rectus was excised: the left oblique and transversus muscles had normal color and reacted to stimulation. The peritoneal layer was left intact and was reinforced with a sheet of Mersilene mesh external to it, sutured to the posterior rectus fascia on the left, the investing fascia of quadratus lumborum on the right, and the inguinal ligament inferiorly. The superior border was free. This patient was not treated with hyperbaric oxygen.

No insurmountable obstacles were encountered postoperatively. An episode of pulmonary edema was treated with ethacrynic acid. Postoperative blood loss from the wound was replaced with two transfusions. A total of 1,000 cc. of bright blood appeared in naso-gastric suction, was replaced, and this bleeding subsided. Acute arrythmias were controlled with intravenous lidocaine. A probable pulmonary embolus was treated with heparin, but without venous interruption, and did not recur.

Management of the peritoneal defect was as follows:

The Mersilene prosthesis was placed, as described, to prevent early evisceration (Fig. 1). The bared peritoneum included the previously made paramedian incision and although its continuous suture was reinforced, it had little mechanical strength.

On the third postoperative day windows were made in the Mersilene and split-thickness skin grafts applied to the peritoneum. Almost complete take was obtained over the muscle laterally. Take on the peritoneum appeared at first to be satisfactory but the grafts sloughed after 5 to 10 days, as the peritoneum underlying them became necrotic. The eventual take of the first set of grafts placed directly on peritoneum was not more than 20%. For the following 30 days the original peritoneal grafts were debrided as necessary and immediately replaced by grafts on the underlying viscera: liver, transverse colon, and small bowel. The patient's ileocolic anastomosis presented itself in the wound, and fistula developed from it which was troublesome only when the patient was fed orally. As the grafts gained strength, the mesh was gradually cut away; eventually, permanent coverage was obtained in all areas and the mesh was completely removed.

Total parenteral nutrition was employed for 30 days. Accelerated catabolism of the patient's body cell mass, evidenced by his high urine nitrogen excretion (Fig. 2), made adequate nitrogen intake urgently necessary, yet attempts to feed the patient by mouth increased output from the fistula to an unmanageable degree. Furthermore,

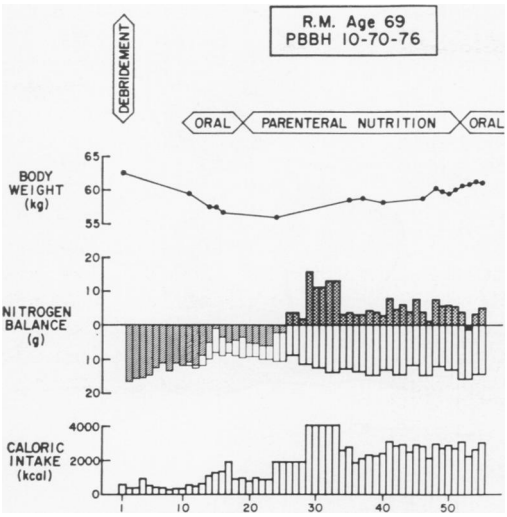

FIG. 2. Body weight, nitrogen intake and output, and total caloric intake for Case 2. Some urine nitrogen determinations were made on 2-day pools—these are plotted as Gm./24 hrs.

the increased peristalsis that followed food intake dislodged grafts and caused prolapse of small bowel loops from the wound. Management was much simplified by keeping the bowel at rest. Nasogastric suction was begun, and a parenteral feeding mixture administered through a subclavian vein catheter (usually a 7:3 V:V mixture of protein hydrolysate (Amigen) and 50% glucose solution).

After skin grafting was complete, the fistula was closed, and oral feeding reinstituted. The patient was fitted with a corset, padded to fit his abdominal wall defect, and although he has an impressively large ventral hernia it is contained by the prosthesis, and the grafts show no tendency to ulceration or pressure erosion in spite of activities that include driving and gardening.

Methods and Results

Clinical nitrogen balance was followed without food analysis. All oral intake was weighed or measured and its nitrogen content calculated from standard tables. The nitrogen content of the parenteral feeding mixture and of 24-hour urine collections was determined by Kjeldahl method. The nitrogen excretion represented by the patient's few and scanty stools, and by colonic fistula drainage, has not been included, but may be assumed negligible. Day-by-day nitrogen balance also reflects changes in urea concentration or the body water pool in which nitrogenous metabolites are distributed. The patient's blood urea nitrogen did fluctuate during the balance period but was normal at the beginning and end (18 and 15 mg./100 ml.).

The body composition data of Table 1 were determined by the simultaneous multiple-isotope dilution method of Moore *et al.*[5]; details of the technic are as described by Morgan, Boyden, and Moore.[6] These compositional measurements were made after the second patient had completely recovered, when his body weight was 6 Kg. greater than when his acute illness began; they define the upper limit of our estimate of triglyceride and protein substrate available to him when he began to fast. Body fat was 25.7 Kg., equivalent to more than 230,000 calories, far more than necessary for the patient's energy needs during a 30-day fast, even when maximum reasonable allowance is made for the included 'essential' lipid and increased energy demands due to catechol or fever-induced hypermetabolism. The patient's cell mass, derived from measurement of exchangeable potassium and the usual ratio of potassium to nitrogen and nitrogen to wet weight in lean tissue, was 23.3 Kg., and this component of the body, rather than caloric stores, would have limited the patient's survival in starvation.

Discussion

Abdominal Wall Reconstruction

The problem posed by these two patients is not a new one; several approaches to it are possible. Mladick *et al.*[4] reported a patient in whom the entire abdominal wall—including peritoneum—was excised. Closure was accomplished by allowing the exposed visceral peritoneum to granulate for 3 weeks, then placing split thickness grafts

on the granulations. This procedure was entirely successful, and is the first report of a patient surviving an abdominal wall excision of this magnitude. Nevertheless, the approach taken in Case 2 offers the potential advantages of retaining intact peritoneum as a bacteriologic barrier for the duration of its viability, shown by Case 1 to be about a week, and to permit grafts, when placed, to remain exposed under the prosthetic mesh.

Schmitt [9] described another variation of the use of mesh, here for closure of large abdominal wounds that are similar to the defects produced by debridements for gas gangrene. Mesh is sutured across the defect, granulations are allowed to grow through interstices of the mesh, and split grafts are placed on them. Complications of infected sinus formation and excessively long delay in grafting because of "gathering" or "corrugation" of the prosthetic material, we feel, make this method less desirable than temporary use of a prosthetic mesh. Such reconstructive closure with removal of the mesh before grafting has also been performed successfully by McNally.[3]

A parallel is seen between this type of acquired abdominal wall defect and its congenital counterpart, the omphalocele. The behavior of the avascular omphalocele sac in neonatal life closely parallels that of the denuded adult peritoneal surface after debridement. The omphalocele quickly loses viability after birth and sloughs to reveal bare viscera, usually during the first week of life. Modern methods of treatment, especially that of Schuster,[10] employ mesh prostheses as temporary adjuncts in the repair of a large defect. Nevertheless, though the patho-physiologic features of the congenital and acquired defects are similar, an important difference exists in the fact that omphalocele closure, ideally, requires full-thickness anatomic closure while for the acquired defect in the massive and rigid adult frame one must settle for less.

TABLE 1. *Body Composition*: (R. M., PBBH #10-70-76, Male, Age 71, December 5, 1969)

	Observed	Predicted
Body weight	68.3 Kg.	
Body water	31.2 l.	36.0 l.
Body fat	25.7 Kg.	19.1 Kg.
Fat free body	42.6 Kg.	49.2 Kg.
Extracellular water	15.0 l.	17.5 l.
Exchangeable sodium	2,717 mEq.	2,967 mEq.
Exchangeable chloride	1,969 mEq.	2,154 mEq.
Intracellular water	16.2 l.	18.4 l.
Exchangeable potassium	2,550 mEq.	2,836 mEq.
Body cell mass	23.3 Kg.	25.9 Kg.

Parenteral Maintenance

Although it has been known for a long time that grafts will take when placed on omentum, liver, intestine, and other viscera,[2] active peristalsis tends to dislodge even small grafts. The second patient's 30-day period without oral intake was of major assistance in accomplishing wound closure. A normal man can tolerate fasting for this period, but the sick surgical patient cannot and parenteral alimentation is essential. Body compositional measurements for the patient of Case 2 show that his cumulative nitrogen loss, if unreplaced, would have been in the range associated with death from starvation.

Normal man invokes very effective mechanisms to prevent loss of body cell mass, i.e., nitrogen stores, when challenged by starvation. The total urine nitrogen of fasting volunteers decreases to levels in the range of 3 Gm. in 24 hr.[7] representing the protein catabolism necessary for essential gluconeogenesis. Energy requirements are met principally by oxidation of fat; residual obligate glucose requirements being further reduced by adaptation of the central nervous system to utilization of substrate other than glucose.

Nitrogen is conserved less effectively when starvation is combined with sepsis or trauma. Figure 2 shows daily urinary nitrogen loss to average 10 Gm./24 hr. for

this patient, even when glucose was supplied in an amount adequate for those tissues having an absolute requirement for it. Ten Gm. of urinary nitrogen represents approximately 300 Gm. of wet lean tissue oxidized; if this loss had continued unreplaced for the subsequent 30 days, a total of 9 Kg. of body cell mass would have been expended. But, the organism cannot survive loss of more than a ¼ to ⅓ of its total nitrogen even though adequate calories are supplied.

The patient's initial body composition can be estimated; the estimate was confirmed by determination of the body composition in a state of health one year postoperatively (Table 1). At this time his measured exchangeable potassium was 2,550 mEq., equivalent to body cell mass of 23.3 Kg., and it can be seen that this protein reserve would not have carried him through 30 days without nitrogen intake.

Summary

A satisfactory method for closure of a large full-thickness abdominal wall defect after debridement for clostridial myositis is described. Prosthetic mesh is sutured in the defect to prevent evisceration, and split thickness skin grafts are placed through windows cut in the mesh. Grafts are replaced, as necessary, to maintain a bacteriologic barrier until the underlying viscera are firmly matted, granulating, and complete coverage is possible. Gastrointestinal decompression and total parenteral nutrition were essential adjuncts to treatment of the wound in the patient described.

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