The following must be noted as incidental side benefits:

1. Doing all the surgery the night of admission with multiple operating teams drastically reduces operating time and essentially removes the problem of subsequent semiemergent operations that destroy operating room schedules for all surgeons.
2. Doing all the surgery the night of admission not only reduces the time in the hospital but also the time out of work.
3. Immediate fracture surgery not only reduces fracture complications and their increased time out of work but also improves extremity function considerably.[3]

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Discussion

DR. DONALD TRUNKEY (San Francisco, California): President Bricker, I rise to congratulate Dr. Border on a very nice study. I think this was a difficult study because of the heterogeneity of the patients and the multiple variables that he studied. Nevertheless, I believe he did a very careful analysis, and I think his conclusions are quite valid.

We have also advocated immediate internal fixation of long bone fractures as soon as possible after the injury. Priority is always given first to head injury, then torso injury, then peripheral vascular injuries, and then the long bone fractures.

We would delay the long bone fracture fixation only if the patient is hemodynamically unstable after treatment of his head injury or his torso injury. This delay should optimally not extend past 48 hours, and this time is used primarily to correct coagulation disorders and treat hypothermia.

As Dr. Border points in his paper, the mortality for an injury severity score of 35, which was his mean, is approximately 30% in the American College of Surgeons trauma outcome study, and he has reduced this very significantly by his approach.

I think this is extremely important. The reason is that 80% of late trauma deaths are due to sepsis and multiple organ failure. Our studies have shown that an injury severity score of 30 is associated with an immune failure. This is probably caused by the release of products of inflammation that increase with the severity of the injury. These products of inflammation include prostinoids, leukotrienes, kinins, monokines, and lymphokines, which brings me to my first question.

Dr. Border, did you measure any of these products of inflammation in your study and relate this to the groups that you studied?

My second question is: Did the antibiotic management of these patients vary among groups? Did that in any way alter your findings?

Thirdly, what was your nutrition management in these patients? Did that alter the outcome, and, finally, I can not help but think that part of your good outcome may be due to the reduced stay in the intensive care unit. It is my feeling that the intensive care unit is a hostile environment where we select out the opportunistic organisms. By reducing that stay, you reduce the chance of these patients becoming infected.

DR. J. C. MCDONALD (Shreveport, Louisiana): Dr. Border has asked me to comment on this paper, and I am happy to do so because I believe it to be a major contribution to the care of the multiply injured patient.

It is typical of Dr. Border and his group to report a modest series of only 59 patients, but to have over 29,000 data elements to consider in their presentation. The thesis of this paper is that early internal fixation of fractures of the femur reduces morbidity and mortality rates in patients with multiple system blunt trauma. Of special interest is the decrease in pulmonary failure, episodes of sepsis, and days of fever. This thesis seems to have been established very well by his presentation, but the larger question is why it is true. Dr. Border discusses several possibilities in his manuscript, but I would like to focus on the single effect on host defense mechanisms of a wound containing dead tissue and breakdown products of tissue and blood. In the interest of time, I will show only three examples.

(Slide) This example relates the magnitude of burn injury to capability of leukotaxis. All measurements were obtained within the first 24 to 48 hours after injury. The greater the burn, the greater the defect in leukotaxis.

(Slide) The next slide shows the changes in complement levels in the same situation, and the final slide (Slide) shows changes in serum fibronectin as related to severity of burn injury. Both demonstrate the same phenomenon—the greater the burn, the greater the defect.

At least in burn patients, these defects are usually not corrected until the wound is covered. It is likely that such changes occur as a consequence of other untreated wounds.

Thus, my question to Dr. Border is: Does he have any data relative to the changes of these standard measurements of nonspecific host resistance in his patients?

DR. LEE H. RILEY, JR. (Baltimore, Maryland): Dr. Border has asked that I comment on this paper, and I am very anxious to do so, because I think it is outstanding.

Dr. Border and his associates have shown that blunt multiple trauma produces a pulmonary failure state that lasts about 3 days, and that any prolongation of that pulmonary failure-septic state may be due to other factors. This study reports the influence of one such factor, the treatment of an associated fracture of the femur. The authors have found that immediate internal fixation of the fracture significantly decreases the duration of the pulmonary failure state, the cost of care, the length of hospital stay, and the number of complications associated with the fracture itself. Our limited experience agrees totally with this.

We look forward to other reports from this and other groups on the effect of other factors, such as the treatment of major spinal fractures and major pelvic fractures, on the duration of the pulmonary failure-septic state.

Equally important perhaps, this paper illustrates the benefits that accrue from trauma centers in which the disease of trauma is studied and managed in its totality and illustrates the continuing maturation of American surgery in which the study and treatment of trauma is a major and worthy goal for senior investigators and senior academic surgeons such as Dr. Border.

DR. JAMES CARRICO (Seattle, Washington): There is very little to add to what Dr. Trunkey and the previous discussants have said. I think this group is to be congratulated and recognized for their efforts in doing a very difficult study and for bringing to us data that others have suggested.

Our trauma team in Seattle (with Dr. Sigvard T. Hansen as chairman of orthopedics) has been practicing early fixation of fractures for about 10 years now. We agree totally with Dr. Border's approach to the problem.

At the American Association for the Surgery of Trauma meeting last August, the group from Dallas presented data that also agree and suggest that both the duration and incidence of the respiratory failure are reduced by such an approach.

My question for Dr. Border has to do with the way the study was designed and how the groups were selected. Clearly group 4 was not selected on any random basis. They were selected because they were more likely to have troubles. Should these not have been included in either the early fixation group or a prolonged traction group?

So my real question is: were your patients selected on some random basis or is there a bias in the selection? While we are all enthusiasts about this data (which fits our biases) we need to be sure that we are not misleading ourselves.

DR. LEONARD F. PELTIER (Tucson, Arizona): I believe that orthopedists generally are in full agreement with Dr. Border, and in this respect I think we are somewhat ahead of the general surgeons.

I particularly appreciate his emphasis on getting patients out of bed and providing us the scientific basis for this, since most of us have always felt that patients die in bed. Therefore, the initial treatment should be directed toward the immediate rigid internal fixation to allow early mobilization.

As Dr. Border has pointed out, the literature takes these problems up in a segmental form, and the patient's treatment is often rendered segmentally by a pick-up group of specialists anxious to get their piece of the action and get on home.

We do not train surgeons in this country as they do in Europe, and this is probably rightly so since the increasing technical sophistication and specialization make it impossible for any one surgeon to master the entire spectrum of care. What we should do is field teams of able, dedicated specialists who are accustomed to working together and who understand the problems and priorities of their colleagues.

Dr. Border has formed such a team and others must follow his example. While the development of trauma centers does not insure the formation of such a team, it is often a first step in that direction. The benefits of the approach described by Dr. Border are obvious to everyone.

DR. JOHN R. BORDER (Closing discussion): I had forgotten to mention the pulmonary thrombemboli problem in the formal paper. Group I had no pulmonary emboli, Group II had two documented pulmonary emboli for 20 patients. Group III with 9 patients had two documented pulmonary emboli. Group IV had no pulmonary emboli. Thus, the pulmonary emboli problem also increases with duration of femoral traction and may be prevented by immediate internal fixation of the fracture. Stated differently, we have had for several years no pulmonary emboli with immediate internal fixation of fractures in a much larger case series than presented here and have observed pulmonary emboli only when fractures were treated nonoperatively.

The series presented here has a number of exclusion criteria. In fact, granted our case material, those exclusion criteria eliminated one patient. That patient died of a massive head injury at 24 to 36 hours. Thus, the series presented, apart from the one case, is a complete sequential series.

Dr. Trunkey and Dr. McDonald, I strongly agree with your comments. Fracture hematoma is retained necrotic tissue. It produces all the bad effects of burn eschar, dead legs, and dead bowel. These include complement consumption with release of split complement products. This allows activated aggregated leukocyte emboli to damage pulmonary function and produce the acute respiratory distress syndrome, as discussed by a host of authors, one of whom is Peter Ward. It also produces, as the burn wound does, severe generalized immune system suppression over a period of 3 to 5 days, as discussed by Mannick and associates. This occurs concomitantly with establishment of the inflammatory response in the fracture area. One, therefore, has with delayed fracture surgery a suppressed antibacterial system, increased intraoperative and postoperative bleeding, together with colonization of wounds by virulent hospital bacteria. These conditions, plus the change in the condition of the fracture, that also occur with the passage of time lead to increased fracture complications in terms of obtaining union and in terms of infection.

Our work shows that immediate fracture surgery can be conducted with minimal intraoperative and postoperative bleeding prior to hospital bacteria contamination on the background of much better systemic immune function with significantly fewer fracture complications. This would be expected from basic principles.

Dr. Trunkey has mentioned the problem of the bacteremias, which also increase with days of femur traction. It is my feeling that antibiotics have little to do with this and that ingestion of food has everything to

do with it. It is to be noted not only that our femur traction groups were on the ventilator longer but that this tracheal intubation with the associated medications for the endotracheal tube also prevented ingestion of food. The enterocyte is exquisitely sensitive to the presence or absence of a mixed meal in terms of cell division and maintenance of a barrier that excludes the gut contents of endotoxin and bacteria. The accidental injury and subsequent therapy creates a colonic stasis, as described by Condon and associates, that prevents colonic emptying and maintains a high penetration pressure for endotoxin and bacteria. This is further aided by the increased morphine and demerol requirements of the patient in traction. It is yet further exaggerated by the secondary protein catabolism and colonic stasis that accompanies the second traumatic insult of delayed fracture surgery. It is my feeling that the bacteremias observed here reflect the effects of gut mucosa protein malnutrition with a high colonic level of bacteria and endotoxin that produces increased entry of gut bacteria into the blood stream. The essential point is that immediate fixation of the fractures reduces the duration of these effects so that amino acids from muscle may maintain the gut mucosa barrier to prevent penetration. This is the same primacy of high protein enteral support to prevent secondary septic problems that was discussed by Alexander for burns at this meeting in 1980. Some additional analogous effects were discussed by Alexander and his associates at the 1984 meeting. Further discussion of the same topic occurred in the subsequent 1985 meeting of the Surgical Infection Society.

Group IV, Dr. Carrico, was a group of more severely injured patients with special injuries and complications. Four out of seven of these patients had immediate internal fixation, with the other three being delayed only 2 or 3 days. It is striking that, even with these special problems, the patients in Group IV required less intensive care than the much less severely injured patients in Group III and about the same amount of care as those in Group II. Thus, even Group IV illustrates the basic thesis developed around Group I.

I began this study in 1974 when I was amazed at how different the course was in patients who had immediate internal fixation from those who had traction. I have lectured on this topic yearly for many years now in Association for the Study of Internal Fixation Courses on the operative fixation of fractures. Granted this background, even I am amazed at the size of the differences between groups and the magnitude of the statistical significance. I have now spent several months writing a chapter on this topic. The differences observed here are exactly those to be expected from the basic biological literature related to the topic. It therefore seems to me that we are dealing with a basic biological response to retained necrotic tissue that will be true of burns, pancreatitis, hematomas of all kinds, necrotic legs, and bowel.