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The universal trauma model

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Dr. Charles L. Scudder, who is memorialized by this oration, was the founder member of the American College of Surgeons who was responsible for the establishment of the Committee on the Treatment of Fractures, which has evolved into the present Committee on Trauma. Dr. Scudder, a clinician who appreciated the importance of research, said in the first Oration on Fractures in 1929 that "The art of surgery is far in advance of all the sciences upon which its future depends. Until they stand abreast, the progress of surgery will be slow. Some day science will outdo the art and take its legitimate place as the basis of sound treatment."[1] During the intervening 55 years, the science of surgery has flourished and Dr. Scudder's prediction has been realized.

In his address, Dr. Scudder proposed that fractures could serve as a model of tissue repair. He stated, "The advancement of direct and indirect research into the processes of repair involving physical, chemical, physiological, and pathological studies . . . opens up fascinating and promising fields." As Dr. Scudder prophesied, not only have studies of fractures illuminated the process of tissue repair, but, ten years after Dr. Scudder's oration, Sir David Cuthbertson demonstrated that an animal model of long-bone fracture could be used to study the metabolic response to injury.[2]

Fracture injury, however, is limited as a general trauma model because of the highly specialized nature of the skeletal system, the difficulty in quantifying and standardizing the severity of injury within a group of patients to be studied, the brevity of the response to injury as a result of early fixation, the necessity for specialty-specific care, and the problem of isolating injury-specific effects in animal models of fracture injury to eliminate the confounding factors of pain and impaired mobility. Various combinations of those limitations also restrict the use of other subsets of trauma patients as a generic model. Therefore I propose the burn patient as the universal trauma model. The burn patient can also serve as an infection model.

Table I

Characteristics of a Useful Model of Injury

1. Accurately reproduces the effects of injury
   - A. Evokes both local and systemic responses
   - B. Magnitude of response proportional to severity of injury
   - C. Magnitude of response permits identification
   - D. Duration of response permits serial examination
2. Available to multiple investigators
3. Easily managed by investigators
4. Applicable to multiple species
   - A. Animal model free of confounding effects
   - B. Duration of survival permits longitudinal study

The trauma model

To be useful, a model must accurately reproduce the disease to be studied, should be available to many investigators, and must be readily managed by those investigators (Table I).[3] A model of injury should evoke both the local and systemic response to injury in a dose-related fashion, with the response to injury being of sufficient magnitude and duration to permit identification and serial examination. Lastly, for research purposes, the model should be applicable to many species, with the animal models free of confounding effects and surviving long enough to permit study of the natural history of the disease.

The burn patient in whom a local injury (the severity of which can be readily and reproducibly quantified) evokes a global systemic response (the magnitude and duration of which are proportional to the extent of injury) meets the criteria for a useful clinical model. The burn patient can also serve as a model of trauma epidemiology, demography, teaching, and research.

The opinions or assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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Validation of the burn patient as a model of both the local and systemic response to injury is provided by consideration of the alterations that occur at the site of injury and the pathophysiologic changes that occur in the cardiovascular system, the pulmonary system, the gastrointestinal system, the immune system, and the systems regulating metabolism. My predecessor and mentor, Dr. John Moncrief, in his Scudder Oration of 1977, detailed the local effects of thermal injury and pointed out the importance of avascularity in the burn wound and the similarity in terms of susceptibility to infection of burn-injured tissue and tissue damaged by mechanical trauma.[4] However, the wound per se in patients with burns of more than 25 percent of the body surface represents only the external manifestation of an injury that disturbs all other organ systems. In fact, the cutaneous damage from thermal injury, which is readily apparent, served to divert attention from the systemic effects of thermal injury for many years, resulting in failure to appreciate the hemodynamic consequences of burn injury.

Physiologic responses to injury

Physiologic studies conducted by numerous investigators in a number of clinics and laboratories have shown that the hemodynamic response to burn injury[5,6] faithfully mimics that resulting from other trauma, as Bredenberg et al have described in a model of hemorrhagic shock (Table II).[7] The pathophysiologic changes in injured patients that compensate for a diminished circulating blood volume are proportional to the severity of the injury. In mechanical-trauma patients, especially those with rapid hemorrhage, compensatory capacity is more apt to be exceeded and hypotension occurs more frequently than in burn patients.

The alterations in pulmonary function that are present in burn patients following resuscitation also reproduce those that occur from other forms of injury.[8,9] In both groups of patients, these changes in pulmonary function represent the combined effects of the injury per se and of resuscitation. Pulmonary function, as manifested by the blood-gas status in all burn and trauma patients, may be further influenced by associated injury (such as inhalation injury in the burn patient[10] and pulmonary contusion in the mechanical-trauma patient[11]), post-injury hypermetabolism, pharmacologic agents that exaggerate hyperventilation,[12] and such complications as infection.

The gastrointestinal tract appears to have a well-defined and limited spectrum of response to any injury of sufficient magnitude to exert systemic effects, i.e., loss of motility, focal mucosal ischemia, and loss of cellular integrity. This response is manifested by gastritic and duodenitic mucosal change characterized by erosions of the upper gastrointestinal tract in burn and trauma patients at an incidence approaching 100 percent in patients with burns of over 40 percent of the total body surface.[13,14] These early changes may progress to frank ulceration with associated bleeding or perforation, especially in those patients who develop complications.

The immunologic consequences of burn injury affect all limbs of the immune system and, again, resemble those of other forms of injury.[15,16] Although

Table II

Hemodynamic Changes

	Burn Injury	Other Trauma
Blood volume	↓	↓
Left ventricular end diastolic volume	↓	↓
Pulse rate	↑	↑
Blood pressure	↓	↓↓
Systemic vascular resistance	↑	↑
Pulmonary vascular resistance	↑	↑
Cardiac output	↓	↓

Table III

Metabolic Changes: "Flow" Phase

	Burn Injury	Other Trauma
Temperature	↑↑	↑
Glucose flow	↑	↑
Proteolysis	↑	↑
Metabolic rate	↑↑	↑
Weight change	↓	↓

the qualitative responses of the humoral component and cellular components of the immune system are similar in burn patients and in patients with other injuries, certain quantitative differences exist that may be injury-severity-dependent or, alternatively, a manifestation of the extensive open wound and the invariable bacterial population of such wounds in the burn patient. Depression of all the immunoglobulin fractions occurs in burn patients but involves predominantly the immunoglobulin M (IgM) fraction in patients with other forms of trauma.[17,18] Depression of complement activity is universal following trauma, regardless of etiology, as are changes in the distribution of lymphocyte subpopulations[19] and lymphocyte activity as manifested by the emergence of suppressor cells[20] and depression of transformation.[21]

Neutrophil changes in terms of cell density[22] and chemotaxis also occur independent of the agent of injury.[23] The depression of neutrophil oxygenation activity that occurs after burn injury has yet to be verified in other types of trauma patients.[24] Changes in the humoral expression of cellular activity are also influenced by injury. A biphasic fibronectin response is apparently common to all forms of injury.[25] The impairment of Interleukin-2 production, recently identified in burn patients by Mannick et al,[26] awaits confirmation as a universal response to injury.

Although all wounds disrupt the cutaneous mechanical barrier, the magnitude of such disruption is clearly greater in the burn patient with a large surface wound; this predisposes such patients to develop local infections.

The pioneering metabolic studies of Cuthbertson have been extended to a variety of trauma patients by a number of investigators (e.g., Moore, Kinney, Egdahl, Soroff, Mason, and Wilmore). Those investigators have defined the components and consequences of post-injury hypermetabolism characterized by increased glucose flow and accelerated proteolysis and manifested by an elevated body temperature and weight loss (Table III). As in the case of other organ system effects, injury of any kind evokes a stereotyped biphasic pattern of hypofunction followed by hyperfunction.

The overall response to injury, i.e., hypermetabolism, sets the injured patient apart from many other surgical patients in whom the adaptation to partial starvation is hypometabolism. Among all trauma patients, the burn patient should perhaps be regarded as a metabolic caricature, since the metabolic rate in patients with burns of more than 50 percent of the body surface exceeds that encountered in any other group of patients.[27]

The similarity of these changes in each organ system has made it possible to apply the results of studies in the burn model to other injured patients and, conversely, apply the results of studies of other injury models to advance our understanding of the burn patient. The usefulness of such models is well illustrated by the advances in patient care that have resulted from the transfer of investigative findings between burn patients and other types of injured patients.

Fluid resuscitation

The need for fluid resuscitation in burn patients was clinically evident as early as the mid-19th century when the fluid deficit of the burn patient was recognized as similar to that of cholera patients.[28] It remained for Underhill to measure fluid loss in patients with extensive burns in 1930,[29] the same year that Alfred Blalock confirmed decreased blood volume as the cause of hypotension following mechan-

ical trauma and identified a disproportionate plasma loss.[30]

In the 1940s and early 1950s, Cope and Moore,[31] Harkins,[32] and Evans[33] quantified the volume deficits and physiologic changes consequent to burn injury. The observed blood volume changes were used to design formulae by which fluid requirements in burn patients could be estimated. Those formulae were subsequently modified by Artz et al[34] and Moyer et al[35] on the basis of clinical experience.

More recent physiologic studies in burn patients and patients with traumatic blood loss have identified the primacy of electrolyte-containing fluid in restoring cardiac output and correcting early postinjury fluid deficits,[36,37] as indicated by the random distribution of high and low colloid-ratio data points in the regression describing transcapillary fluid shifts during the first 24 hours of burn resuscitation (Figure 1). Such studies have provided the basis for resuscitation regimens relying on the administration of noncolloid-balanced electrolyte solutions in the first 24 hours after injury.[38,6] Adequate and timely resuscitation has significantly decreased acute renal failure as a complication of all forms of trauma.

As shock and renal failure have receded with the use of fluid resuscitation, the pulmonary system has emerged as the weak link in the physiologic chain. Several investigators at our institute and other centers have studied the effects of resuscitation on subsequent complications, such as pulmonary edema, which may lead to the development of respiratory insufficiency in the burn patient or other types of injured patients. The temporal association of the use of resuscitation fluids emphasizing electrolyte containing fluids and minimizing colloid containing fluids and the increase in pulmonary complications (presently the most frequent cause of morbidity and mortality in the burn patient[39] and, I suspect, in other trauma patients as well) have led to the incrimination of intravascular hypoproteinemia as a significant causative factor.

Several groups have found that the fluids administered during resuscitation exert little influence on lung water.[40,41] Lam and Goodwin have reported a low correlation between lung water expressed as

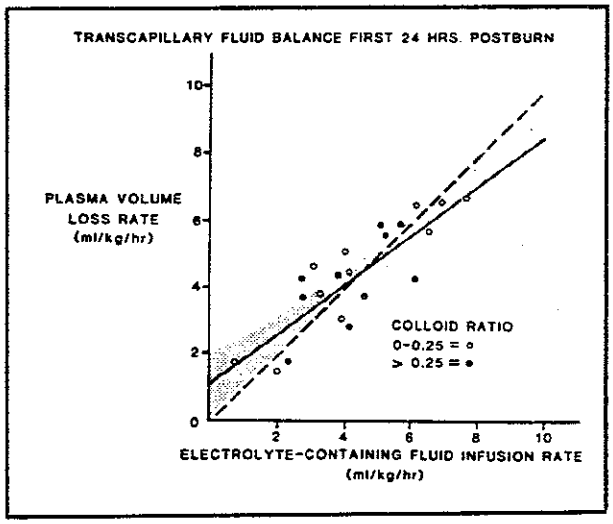

Figure 1. *Transcapillary fluid balance in the first 24 hours postburn is related to the infusion rate of salt-containing fluids (balanced salt solutions and colloid containing fluids). Individual data points for periods during which either less than or more than 25% of the infused fluids were colloid-containing fluids (a colloid ratio of < 0.25 or > 0.25, respectively) are randomly distributed within the regression of plasma volume loss rate on salt-containing fluid infusion rate. That finding indicates that during the first postburn day colloid-containing fluid is no more effective in restoring plasma volume than balanced salt solution. (Regression indicated by solid line and line of identity by dashed line. Stippling indicates 95% confidence limits of regression.)*

the lung tissue volume ratio and colloid oncotic pressure in the first 24 hours postburn.[42] Those investigators have found that resuscitation of burn patients with fluids containing colloid in an amount comparable to the Evans formula did not protect the lung and the incidence of both pulmonary edema and mortality was higher in those patients than in patients who received only fluids containing electrolytes in the first 24 hours.[43] Adequate resuscitation was effected with either fluid regimen, although less resuscitation fluid was required to achieve the desired urinary output during the first 24 hours in patients who received colloid. No consistently superior effect

of colloid resuscitation on cardiac output was identified.

In the patients who received only electrolyte-containing fluids in the first 24 hours, lung water remained constant during the first seven days postburn, while it progressively increased in patients who had received colloid-containing fluids (Table IV). These and similar findings in studies of patients with trauma-related hemorrhage have further defined the limits of asanguinous noncolloid resuscitation and provided a sound physiologic foundation for the regimens presently employed to replace the blood and plasma losses that occur in all injured patients.

Enteric response

As early as 1823, Swan[44] reported frank ulceration of the gastric mucosa in a burn patient 19 years before Curling reported his 10 cases of ulcer disease of the upper gastrointestinal tract following burn injury.[45] This enteric response to injury and sepsis has been noted in a variety of trauma patients in whom the incidence has been reported to range from two percent of general trauma patients to 21 percent of burn patients and 54 percent of patients with intracranial lesions.[46]

Table IV

Changes in Lung Water in Burn Patients as Related to First 24 Hour Resuscitation

	Lung Water (ml/ml)	
Time Postburn	Patients Receiving Colloid-Containing Fluids (n = 25)	Patients Receiving Only Crystalloid (n = 25)
12 hours	0.13	0.13
24 hours	0.125	0.123
36 hours	0.120	0.124
48 hours	0.123	0.138
60 hours	0.141	0.138
72 hours	0.145	0.140
120 hours	0.167	0.149
168 hours	0.173	0.137

Adapted from
Goodwin CW, Dorethy J, Lam V, et al
Ann Surg 197:525, 1984

The natural history of this form of ulcer disease in burn patients has been defined by serial endoscopic examinations, which have documented progression of gastritic and duodenitic changes to frank ulceration in those patients with severe injury and those in whom complications developed.[14] A similar pathologic progression has been observed in other injured patients.[47] Additional studies at our institute confirmed the importance of gastric acidity in stress ulcer formation[48] and the beneficial effect of prophylactic antacid therapy in preventing progression of the mucosal disease.[49] In the burn patient, the histamine H_2 receptor antagonist cimetidine has been found to provide equal protection.[50]

Such regimens of prophylaxis, commonly employed in all severely injured patients and many nonsurgical critically ill patients as well, have significantly reduced the occurrence of bleeding and perforation as complications of stress ulcers. In burn patients who have received prophylaxis, the incidence of those complications has varied between one percent and two percent on an annual basis[51] and operative intervention for treatment of stress-ulcer complications has been necessary in only two of 2,043 patients treated during the past 8½ years.

Posttraumatic immunosuppression

Global suppression of the immune system appears to be important in regard to the frequency with which sepsis occurs as a cause of morbidity and mortality in patients with extensive burns. Reduction of one form of infection has typically served only to accentuate other forms of sepsis. In the case of burn patients, burn-wound infection, which has been strikingly reduced by effective topical chemotherapy, has been replaced by pneumonia as the most frequent cause of death due to infection. Although the cause of death has changed, the survival of patients with massive burns has been little affected.[15]

The successive predominance of different infec-

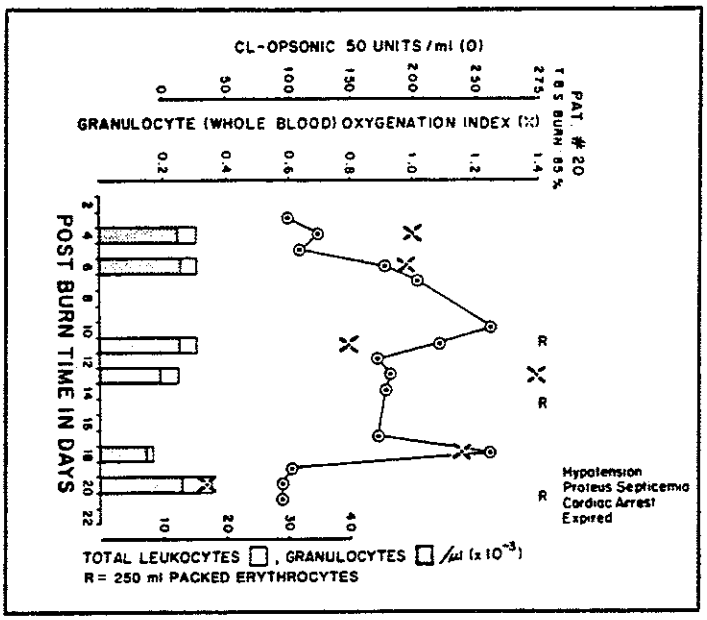

Figure 2. *Neutrophil function per se (oxygenation index) and serum opsonic activity (opsonic capacity) can both be assessed by measurement of chemiluminescence. These indices of neutrophil function change in relation to clinical events such as sepsis. Note rise in opsonic capacity following initial depression and the profound depression of both opsonic capacity and oxygenation index on the 20th postburn day at a time when Proteus septicemia was documented two days before the death of this patient with burns of 85% of the total body surface.*

tions and different causative organisms is a reflection of burn-size-related immunosuppression in such patients. Both the humoral and cellular components of this state of increased host susceptibility have been examined, and several laboratories have recently placed emphasis on defining changes in the function of granulocytes that represent the cellular limb of the "first line" information/effector system of host defense.

Investigators at our institute have described not only cyclic changes in neutrophil number but alterations in chemotaxis, both of which occur in proportion to injury severity.[22,52] Allen and others at our laboratory, using chemiluminescence as an index of granulocyte function, have described changes in oxygenation activity in association with septic complications (Fig 2).[24]

Although such an association does not differentiate cause from effect, recent studies using granulocyte enzyme-specific chemilumigenic probes have identified depression of oxidase activity disproportionate to granulocyte number as predictive of a fatal outcome. The depression of oxidase activity commonly precedes what appears to be preterminal depression of granulocyte peroxidase activity as a reflection of marrow exhaustion.

The documentation of these and other alterations in function of the immune system in all patients with severe injury has focused attention on means to augment function of the immune system. Investigators in many laboratories are presently evaluating the effectiveness of immunomodulators, vaccines, and serologic therapy, such as administration of IgG in improving the resistance of the severely injured host to opportunistic pathogens.[53]

Metabolic response

In terms of the metabolic response to injury, the burn patient has served as a reliable model in which the biphasic response pattern originally described by Cuthbertson has been studied in detail. The effect of injury on the hormones regulating metabolic processes, their influence on the distribution and utilization of nutrients, their changes across time, and their interactions as exemplified by the reciprocal relationship of catecholamines and thyroid hormone have been defined in burn patients.[54] The information derived from such studies has been used

to develop physiologically sound regimens of metabolic support, including provision of nutrient substrates to meet the specific needs of injured patients.

A number of studies have demonstrated that injury alters a variety of central nervous system regulatory mechanisms (Table V). In burn patients with chemical hypothyroidism, inappropriately low thyroid-stimulating hormone levels have been observed.[55] Shirani and Vaughan have recently reported that the suppression of triiodothyronine (T_3) and tetraiodothyronine (T_4) concentrations in critically ill burn patients, and a murine burn model as well, occurs in association with an increase in dialyzable fractions and little if any change in T_3 uptake.[56] They have proposed that a circulating inhibitor of thyroid-hormone binding is responsible for these changes as described in other patients with a variety of non-thyroidal illnesses. Further evidence of altered hypothalamic function has been found in an animal model in which burn injury depressed pineal melatonin levels and in burned humans in whom the nocturnal plasma melatonin surge was blunted.[57]

Table V

Postburn Alterations of Central Nervous System Function

I. Thermoregulatory dysfunction
   A. Elevated temperature of comfort
   B. Elevated upper critical temperature
II. Decrease in metabolic rate in response to administration of narcotics
III. Decreased stimulated growth hormone response
IV. Dissociation of ACTH (corticotropin) and cortisol levels
V. Suppressed thyroid-stimulating hormone response to chemical hypothyroidism
VI. Inappropriate antidiuretic hormone secretion
V. Altered pineal function
   A. Decreased daytime pineal melatonin content in hamster
   B. Blunted nocturnal plasma melatonin surge in man

In the area of metabolic research, the applicability of the burn model to multiple species has been particularly well illustrated in studies examining the effect of injury on thermoregulation and the energy costs of the injury per se. Clinical studies indicating that burn patients are internally warm and not externally cold[58] have been transposed to and extended in the laboratory using a unique large-animal metabolic chamber.[59] Burn injury in either goats or pigs is followed by a temperature-independent increase in resting energy expenditure just as it is in burned man.

Current studies at our laboratory by Aulick and others using groups of 39 rats (a metabolic mass equal to one pig, which we now term the standard pig unit or SPU) have again confirmed an injury proportional temperature-sensitive but not temperature-dependent increase in metabolic rate and alteration of thermoregulatory set points. In this model, the application of topical antimicrobial agents reduces the magnitude of post-injury increase in metabolic rate to a level that appears to represent the direct metabolic expense of the injury. This recent finding also suggests that the intrinsic metabolic response to injury can be modified by host-microbial interactions.

Surgical education

The burn patient is also a useful model for teaching the total management of trauma, one of the seven primary components of general surgery as defined by the American Board of Surgery.[60] The sigmoid relationship between magnitude of pathophysiologic change and extent of injury in the burn patient facilitates the development of programs of graduated complexity of care and progressive responsibility applicable to undergraduate, graduate, and postgraduate training (Fig 3).

At all levels of surgical education, the burn patient is unequalled for the study of wound biology, post-traumatic organ-specific dysfunction, and the organ-system interactions that determine the whole-body response to injury. The use of the burn patient

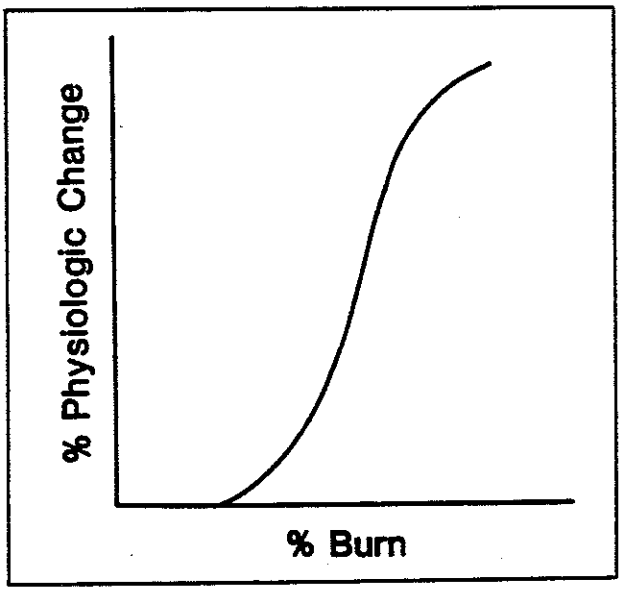

Figure 3. *This idealized sigmoid dose response curve not only describes the effect of extent of burn on physiologic change but the effect of burn size on occurrence and duration of organ dysfunction and the effect of burn size on mortality.*

as a teaching model is supported by the commonality of physiologic changes and clinical care needs of all trauma patients; this makes the knowledge gained and the skills acquired in caring for burn patients applicable to all other critically ill patients.

For the undergraduate, burn injury illustrates the relationship between injury and response, and burn care is a good example of the clinical applications of the basic sciences in the fields of resuscitation, infection control, wound care, and general support. What differences there are between burn and other trauma patients are quantitative rather than qualitative and represent advantages in terms of teaching during the entry years of graduate training and of the relative ease with which longitudinal clinical investigations can be carried out (Table VI).

The magnitude and duration of change represent challenges at any level of surgical education and permit observation of the organ-system adaptations and interactions that may occur with greater velocity but be of lesser duration in other trauma patients.

At the graduate level of surgical training, early exposure to burn patients provides experience in planning and conducting resuscitation, physiologic monitoring of organ function, wound care and closure, diagnosis of the myriad complications of injury, and provision of the metabolic support required by the severely injured patient.

In later graduate years, care of burn patients provides experience in endoscopy of the respiratory tract and both the upper and lower gastrointestinal tract, the treatment of associated injuries and life-threatening complications, and burn-specific surgical procedures. At this level of training, surgical judgment is refined in terms of balancing operative risks and benefits when assessing the indications and timing for burn-wound excisions and other operative procedures in critically ill patients.

At the postgraduate or fellowship level, training can be focused on refining diagnostic and therapeutic skills. Maturation of surgical judgment occurs in the course of designing treatment modifications to meet specific needs of individual patients. Most important for the postgraduate trainee is research experience and the development of investigative skills of broad usefulness. Studies of burn patients facilitate the development of these skills by virtue of the relative ease with which the physiologic changes can be studied and the applica-

Table VI

The Burn Patient as a Trauma Teaching Model

Similarities:

- Patient response related to injury dose
- Requirement for early resuscitation
- Multiple organ dysfunction
- Requirement for local wound care

Differences:

- Velocity of early changes
- Magnitude and duration of organ dysfunction
- Extent of local wound
- Susceptibility to infection

bility of burn study methods and techniques to other trauma patients.

In the course of clinical investigations, the postgraduate trainee rapidly acquires an understanding of the dose-relatedness of injury and response and of the importance of patient stratification and homogeneity as related to the various factors influencing anticipated mortality, a principle frequently ignored but essential for meaningful studies of any category of trauma patients.

The ready availability of established animal models of burn injury facilitates the laboratory study of clinically relevant problems; this has been important in bringing about improvements in burn and trauma care. The use of specific models to examine various aspects of burn injury provides the postgraduate trainee with an appreciation of the importance of species-specificity and proper species selection for modeling in surgical research.

Epidemiology

The burn patient is also a model of trauma epidemiology and demography. Trauma is the most frequent cause of death in the United States in people from age one to 44.[61] In the population at large, death due to injury (more than 160,000 individuals die annually) ranks number four among all causes of death.[62] The incidence and predominant cause of trauma are age- and sex-dependent, and trauma death rates are statistically influenced by age, sex, race, economic status, and geography.[63]

The societal costs, including use of medical and other resources as well as loss of productivity due to motor-vehicle injuries alone have been estimated to be between $15- and $25-billion annually.[64] Since deaths due to motor-vehicle injuries represent 33 percent of all injury-related deaths, one might estimate yearly societal costs of all trauma to be between $45- and $75-billion, and Munoz has recently reported that in 1982 those costs were approximately $61-billion.[65]

Physician contacts comprise one component of societal costs and, in the year 1980, 99-million physician contacts were related to trauma, exceeding by 27-million, or 38 percent, those related to heart disease, the second most common cause of physician contacts.[66] A study in two Northeastern Ohio metropolitan statistical areas that examined physician contacts at the emergency room level found an overall annual rate of injury-related emergency room visits of 194 per 1,000 population, which represents approximately 44-million injury-related emergency room visits each year out of the total U. S. population of approximately 227-million.[67] Recently published data from that study indicate that only five percent of all patients who visited emergency rooms for treatment required hospital admission; the admission rate ranged from two percent in patients aged five to nine to 21 percent in patients aged 65 to 74 and 34 percent in patients over 75 years old.[68]

Table VII

Burn Injury in the United States Annually

Total Burns (estimated)	2,000,000
Burns Admitted to Hospital	106,000
Burn and Fire Deaths	6,000

For comparison, more than two-million individuals are burned in this country per year, 6,000 of whom die (Table VII).[69] Burn injury and fire-related deaths represent the fourth most common cause of death due to unintentional injury in this country and are exceeded only by motor-vehicle accidents, falls, and drownings.

The vast majority of burn patients have relatively minor injuries and do not require hospital care but, as is true for other types of trauma patients, five percent of burn patients (or approximately 106,000)[70] have injuries of such magnitude or distribution or complicating diseases and injuries that hospital admission is necessary. These patients receive care for an average of 12 days,[71] or an aggregate of more than one-million hospital days each year.

The burn patient population thus represents a model of the total trauma population with regard to distribution of injury severity within the injured population, the hospital admission rate, and the de-

pendency of incidence, etiology, and etiology-specific death rates on age, sex, race, economic status, occupation, and geography.[71]

Burn facilities

The epidemiologic characteristics of burn injury, the graduated physiologic response of burn patients, and the laboratory and people-intensive nature of burn-patient care have led over the past 40 years to a hierarchical organization of burn-treatment facilities. The American Burn Association directory of burn-treatment facilities lists 183 such facilities in the United States, of which 138 include designated burn beds (for Canada there are 24 burn-care facilities of which 17 include designated burn beds).[72]

Within this group of hospitals there are 38 university-related burn centers in which there is ongoing graduate surgical training and identifiable research activity. These centers, which serve as regional tertiary referral centers, have been the training ground of the directors and staff members of other burn-treatment facilities, as exemplified by our alumni who direct 17 of these 38 centers. It is also from these centers that research-generated information has flowed to improve care throughout the entire system.

The development of the general trauma-care system, restrained by a variety of professional and non-professional considerations, is now proceeding according to the same hierarchical pattern.[73] Although the distribution of injury severity within the general trauma population resembles that within the burn population, the greater volume of mechanical trauma and the frequent need for prompt surgical intervention will justify and necessitate establishment of a system with a greater density of trauma centers. An informal survey by the College's Committee on Trauma appears to bear this out by the classification of approximately 50 trauma-care facilities as Level I centers analogous to regional burn centers.[74]

The application of data from clinical and laboratory studies of burns and other injuries to patient care provided within the burn-care system has resulted in a significant decrease in burn mortality, as confirmed by the increase in the extent of burn associated with a 50 percent mortality today as compared to four decades ago (Table VIII).

Table VIII

Change in Burn Patient Survival 1945 - 1983

	LA_{50}	
Age group	1945-1947	1979-1983
Pediatric	51.0	45.21
Young adults	43.0	60.81
Older adults	23.0	39.19

The fidelity of the burn model, in terms of epidemiology and demography, clinical care, research, teaching, and the organization of care has permitted the ready transfer of these benefits to trauma patients and corresponding improvement in their survival.

This fruitful exchange will be further enhanced by the continued development of a hierarchical trauma-care system that emphasizes the training and research center. Clarification of the pathophysiologic mechanisms determining the outcome of injury brought about by multidisciplinary care and research at such centers will, as Dr. Scudder predicted, form the basis of improved treatment to enhance survival of all trauma patients and reduce the societal costs of accidental injury.

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