BURN MORTALITY AND THE ANAESTHETIST

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THE MORTALITY FROM BURNS has diminished so little in the past twenty years that some feel that the point of diminishing returns has been reached. Anaesthetists can help in the attack on the hard core which remains.

CAUSES OF DEATHS FROM BURNS

Even 20 years ago shock accounted for only 20 per cent of all deaths from burns.[1] It is now even less of a major factor. However, shock and respiratory obstruction are said to be the commonest causes for death in the first week after a burn.[2] In the second week shock and sepsis are said to be equally important together with the very occasional bleeding gastrointestinal ulcer; between the third and ninth weeks sepsis kills most patients, while more deaths between the ninth and twelfth weeks are found to be due to viral hepatitis.[2]

Little advance seems possible in the management of the initial shock. Work has been done, however, which may be of value if the unfortunate necessity should arise to resuscitate and transport casualties with mass burns.[3] Those interested in hospital disaster plans will find interesting reading in the detailed account of the Coconut Grove Fire.[4]

Second-degree burns alone seldom constitute a problem although it has been nicely demonstrated that quite large amounts of the body's carbon dioxide can be lost in children from such lesions.[5] Patients with massive third-degree burns continue to die in spite of our best efforts. Some of the difficulties are illustrated in the following two case histories.

Case No. 1

This 10½-year-old boy (L.D.) was involved in a bad gasoline accident in mid-March, 1962. Extensive burns to the face and all four limbs, most of them third degree, covered about 55 per cent of his body surface area.

He was transferred from a country hospital to another Winnipeg hospital on April 1, 1962, where debridement was done on the 4th and skin grafting on the 7th of April under general anaesthesia.

Because of his deteriorating physical and mental state he was transferred to the Children's Hospital on April 8. The poor child was in an acute anxiety state, having seen his close relatives perish or get burned severely in the fire. Many visits were necessary to win his confidence. At first his favourite expression was a pathetic "Doctor, I don't want to die, I don't want to die."

He was given 10 anaesthetics at our hospital in a period extending just over 10 weeks, all, except one, quite uneventful. Skin taken from his sister was applied five days after admission, and the other procedures, except one which had to be abandoned, all involved extensive grafting. The technique of anaesthesia in all cases, except one, was that previously described by us.[6] A slow and gentle induction with cyclopropane

was followed by halothane, local analgesia to the pharynx and larynx with 4 per cent lidocaine spray and endotracheal intubation. It was impossible to measure the blood pressure clinically because of the burned arms. The heart and breath sounds were monitored by endo-oesophageal stethescope and Lead 1 or 2 electrocardiography. A mattress filled with warmed water at 97° F. helped to reduce heat loss. Blood replacement was generous, on the average two units of 400–500 ml. blood being given on each occasion. Veins were scarce, the jugulars being used four times. Intravenous meperidine 40–50 mg. was given slowly to ensure a pain-free and pleasant emergence.

Succinylcholine was studiously avoided except for one anaesthetic, given by a colleague, approximately six weeks after the burn, when cardiac arrest promptly ensued. The patient was resuscitated with difficulty by external cardiac compression and intracardiac epinephrine. He was disorientated for 24 hours afterwards. Nevertheless because of the overriding importance of grafting, anaesthesia was willingly undertaken two days later. At this point his confidence had been temporarily lost, veins were apparently all gone, and the chronic respiratory infection had worsened.

His condition steadily improved with seven major grafting procedures following the episode of cardiac arrest and he was sent home in high spirits on August 1 after four months in the Children's Hospital.

The successful management of this case was almost nullified by the use of succinylcholine on one occasion. It was a triumph of patience and hard work on the part of the whole team. The odds were high but the skin grafting was kept just ahead of the onslaught of the disease. The importance of early grafting is well expressed by Jackson who wrote: "The more ill the patient is, the more urgent it is not to delay grafting a day longer than necessary. Many a child will recover from pneumonia alone, who will not survive the lethal combination of pneumonia and an extensive granulating area."[7]

Unfortunately, in the second case reported here, the disease got ahead of the treatment.

Case No. 2

This boy, J.B., also 10½ years of age, suffered extensive gasoline burns of all four limbs and trunk on October 4, 1962. About 60 per cent of the body surface area was involved and most of the burns were third degree.

His resuscitation was satisfactorily conducted and his general condition seemed surprisingly good when he was anaesthetized on the 9th postburn day for change of dressings. His attitude was calm and co-operative, contrasting sharply with that of the first boy. Anaesthesia was again given on the 15th postburn day for extensive debridement. This was a major undertaking. A similar technique to that described above, avoiding succinylcholine, was used without difficulty.

The patient continued to do better than expected until burn baths were begun on the 17th day after the accident. The nurses noted that, following the first bath, he had "runny brown stools," "the occasional burp," and by 11:45 p.m. the same evening "chills." Burn bathing was again carried out the following day in order to get him ready for grafting as soon as possible. The nurses noted the same evening that he was "drowsy, incoherent, and having chills." Next day after bathing he was difficult to rouse, refusing all solids, voiding dark amber urine, and had a temperature of 105° F. Deterioration was now rapid. He began to vomit dark brown material, his respiratory rate was counted at 68/minute, and he was perspiring profusely and was irrational. Death occurred on the 20th postburn day. This was only the third day after his condition had suddenly begun to worsen.

The rapid decline of this patient in spite of adequate resuscitation in the early shock period and when he was apparently doing quite well clinically illustrates the most difficult problem in these cases.

Late or Septic Shock

The efficiency of resuscitation has been assessed by Unger and Haynes[8] by daily measurement of blood pressure, blood volume, peripheral resistance, and cardiac output in severely burned patients. In the cases they describe, treatment in the shock period immediately after burns raised the cardiac index well above normal. Blood pressure at an early stage was well maintained in spite of decreased total blood volume, peripheral resistance being elevated. In the first few days, with continuing treatment, the cardiac output continued to go up, the peripheral resistance kept falling, and the blood pressure remained fairly steady, while blood volume rose at an approximate rate of three per cent per day for about one week. Correlation with the details of treatment was not presented. In spite of these satisfactory haemodynamic findings it was clinically observed that peripheral circulation seemed rather poor. At the beginning of the terminal phase, patients showed normal blood pressure, a high cardiac index, an expanded blood volume, and a good urine output. However there was a spiking fever, tachycardia, and abdominal distension. To use their own words: "The debacle began with hypotension." Attempts to treat this hypotension with levophed brought the patient no benefit, and were rather illogical anyway. Sympathetic blocking agents might possibly have been of benefit at an earlier stage to ensure more even blood perfusion. At this late stage drugs acting on the cardiovascular system seem unlikely to halt the deadly menace of septicaemia.

The picture of spiking fever, tachycardia, abdominal distension, disorientation, and chills as seen in our second patient and described by Unger and Haynes[8] is an ominous one. Septicaemia in the 1950's was said to be mostly due to *Staphylococcus aureus* but in the 1960's, *Pseudomonas* seems to be the predominant organism.[9] Modern antibiotics may have helped to bring this resistant invader to the fore. Recovery from Gram-negative septicaemia appears to be unknown at the moment. The only hope seems to rest with a specific anti-serum, which has been shown to be much more effective than gamma-globulin in experimental animals.[10]

Autopsy Findings

Sometimes at autopsy evidence of damage is widespread.[6] In others, as in the second case reported here, disappointingly little comes to light. The thesis of Delarue *et al.*[11] analyses with great care the findings throughout the body. Thrombo-embolic phenomena occur widely in bad burns and these workers were impressed by the lethal effect of septicaemia, although they did not isolate the causative organism. Autopsies should be planned to give maximum information, especially in the respiratory tract.[2] Recent workers stress over and over again the importance of respiratory tract infection and a careful scrutiny of this system should be done with microscopic examination of sections at every level. Even when clinical evidence is absent, extensive pathology may be present, especially in burns involving the so-called "respiratory area" of the face around the nose and mouth.[1,6] Genito-urinary tract infection is also apparently more commonly found at autopsy than might be expected and may be a neglected source of septicaemia.[2]

Adrenal Function in the Burned Patient

(*a*) *Adrenal Medulla*

After severe burns the urinary catecholamines have been found to increase markedly.[12,13] Most patients who died, however, showed a terminal decrease in output of urinary epinephrine and also a subnormal epinephrine content in the adrenal medulla at autopsy. Epinephrine is normally resynthesized very rapidly and this picture is therefore thought to represent adrenal medullary failure.

(*b*) *Adrenal Cortex*

Unlike the medulla, the adrenal cortex is essential to life. The work of Feller[14] suggests that patients with third-degree burns of more than 40 per cent of the body surface area may be unable to cope with the stress without hydrocortisone.

The first few days after severe burns the urinary levels of both 17-ketosteroids and 17-hydroxysteroids were found to be high. When these values fell towards the normal range in the face of clinical signs of continuing stress, then a fatal outcome was imminent. At this point an ACTH test did not produce the expected rise in 17-hydroxysteroids, suggesting adrenal cortex insufficiency. Feller considered this to be a differential failure since ketosteroids did increase more than hydroxysteroids. Some of the patients who showed this deficient response to the ACTH test were given intravenous hydrocortisone; survival time over those who did not receive this drug was very significantly increased but the final outcome was nevertheless the same.

The dangers of hydrocortisone might make one hesitate. Feller[14] reported that neither delayed healing nor an increased susceptibility to infection was manifest. No mention was made of gastrointestinal ulceration: possible side-effects may well have to be disregarded in the treatment of these desperate situations.

Succinylcholine in the Burned Patient

Cardiac arrest following succinylcholine administration, as in the first case described here, has aroused considerable interest recently.[6,15] Can this occurrence be explained now by what is known of the widespread body changes? The similarity in all reported cases is striking.[6] The presence of low plasma pseudocholinesterase and of a low true cholinesterase due to liver damage has been described.[15] Although this might increase the duration, would it increase the immediate intensity of action of succinylcholine on the heart of the burned patient? Myocardial damage due to burns, though perhaps contributing, is unconvincing as a cause in view of the fact that recovery from the cardiac arrests which have occurred has been prompt and complete following adequate resuscitation. All these patients have a marked tachycardia and the resemblance to the deaths that have occurred during induction with chloroform has been noted.[16] Until we know more about the cardiovascular actions of succinylcholine in the normal patient we are not likely to find out why it produces such dramatic effects in these particular cases.

Second Thoughts on the Use of Halothane in Burned Children

Evidence that halothane may have considerable liver toxicity in certain circumstances is coming in.[17,18,19] In an excellent discussion[19] of other aetiological factors and the possibility of coincidence it has been pointed out that halothane has been given probably to more than six million patients. Apparently only 19 fatal cases have been reported so far and it is impossible to be certain that halothane was responsible for any of them. However, the question is once more sub judice; liver damage is common in bad burns and the circumstantial evidence makes it necessary to abandon the drug in these cases, perhaps temporarily, and with genuine regret. Case histories of postoperative complications will have to be examined again very carefully. For example, in the second case described here, deterioration set in three days after general anaesthesia employing halothane. No liver pathology was described, but how carefully was it sought? Unfortunately microscopy was not done on this occasion. The usual liver function tests have been shown once again to be unreliable in assessing minor degrees of damage. For this reason one cannot adopt methoxyflurane as being any safer than halothane at present. It appears, then, that no inhalation anaesthetic agent at the moment surpasses cyclopropane for these extensively burned patients.

Summary

The anaesthetist must be prepared to play his part in attempting to reduce the hard core of mortality from burns which remains. Early skin grafting is important even in the presence of marked pulmonary infection. Colonization of large wounds left ungrafted is often followed by fatal septicaemia. The deterioration from this condition is often sudden and rapid. Septicaemia due to *Pseudomonas aeruginosa* is increasing in incidence and only the use of a specific anti-serum seems to offer any hope against it at the moment.

Pathology is widespread and complex. We should encourage careful planning of autopsies and focus special attention on the respiratory tract.

Failure of both adrenal medulla and cortex has been described. The use of hydrocortisone may be necessary with third-degree burns covering more than 40 per cent of the body surface area.

Succinylcholine should not be used in the burned patient, certainly not in the extensive and long-standing case. The mechanism of cardiac arrest due to this drug has not been satisfactorily explained.

Recent reports on the possible hepatotoxicity of halothane preclude its use in bad burns; it seems no wiser to use methoxyflurane either at the moment.

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RÉSUMÉ

L'anesthésiste doit se préparer à jouer un rôle pour essayer de réduire le taux très élevé de mortalité qui subsiste chez les brûlés. Il est de première importance de faire des greffes cutanées aussitôt que possible, même s'il existe une infection respiratoire. Une contamination de plaies étendues non greffées entraîne souvent une septicémie fatale. Dans ces cas, le malade dépérit soudainement et rapidement. La fréquence des septicémies aux pseudomonas aeruginosa augmente et, actuellement, le seul espoir que nous ayons pour juguler cette infection est l'usage d'un antisérum.

La pathologie se généralise de façon complexe. Il faudrait tenter d'obtenir des autopsies soignées et orienter la recherche particulièrement sur le système respiratoire.

Certains ont décrit une dégénérescence simultanée de la corticale et de la médullaire surrénaliennes. Chez les brûlés du troisième degré dont la surface des lésions atteint 40 pour cent de la surface corporelle, il peut être nécessaire de recourir à l'usage de l'hydrocortisone.

Il faut s'abstenir d'utiliser la succinylcholine chez les brûlés particulièrement s'il s'agit d'un grand brûlé et si l'anesthésie doit se prolonger. La façon dont la succinylcholine entraîne un arrêt cardiaque ne s'explique pas de façon satisfaisante.

Récemment, on a cité des cas de séquelles hépatiques à la suite d'anesthésies au fluothane; en conséquence, chez des brûlés, il serait déconseillé d'utiliser cet agent; il serait également plus sage de ne pas employer le méthoxyflurane.

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