Papers and Originals

Crush Injuries of the Chest

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Severe crush injuries of the chest have been associated with a high mortality rate (Griffiths, 1960). This was largely due to inadequate management of the respiratory problem present, and suggested that intermittent positive-pressure ventilation (I.P.P.V.) as used by Avery *et al.* (1956) might be the answer to this problem. Results of treatment by this method have been published (Windsor and Dwyer, 1961; Reid and Baird, 1965; Lloyd *et al.*, 1965). We here present a series of 64 patients with a severe chest injury, 61 of whom received I.P.P.V.

Material

The 64 patients in the series have been divided into groups according to the anatomical sites of injury (see Table II). This classification does not take into account fractures of bones of the limbs or of the pelvis. Forty-seven patients had injuries to the thorax alone and 17 had additional severe injuries.

An attempt has been made to assess the associated physiological disturbances. All 64 patients had evidence of respiratory dysfunction. Twenty-four had circulatory depression (shock), 11 had neurological disturbance, and 10 had shown signs of combined circulatory and neurological dysfunction. As would be expected, the incidence of multiple physiological upset was commoner in the group of patients with severe injuries outside the thorax.

Management

Emergency Treatment.—The emergency treatment followed the general lines described by Griffiths (1960). All 64 patients in the present series had tracheobronchial toilet. In 43 pleural drainage was performed; it was bilateral in 15. In 13 cases laparotomy was carried out; in eight of these damaged viscera were removed or repaired and in five the findings were negative or a retroperitoneal haematoma was noted. Three thoracotomies were performed—in one patient a tear of a lung was repaired and the other two had a lower lobectomy. In two patients neurosurgical procedures were carried out. Sixty-four patients continued to manifest signs of respiratory distress after such emergency measures had been carried out. They were therefore transferred to the artificial ventilation unit for I.P.P.V.

Management of the Respiratory Problem by I.P.P.V.—In 62 of the 64 patients a tracheostomy was performed—in 47 this was followed by immediate I.P.P.V., but in the remaining 15 I.P.P.V. was not instituted on the same day, though 13 of them required it later because of a deterioration in respiratory function. Apnoea was established in the first instance by manual hyperventilation through a Waters canister combined with the generous addition of intravenous opiates. When

apnoea was achieved the patient was connected to a mechanical ventilator which had been set to maintain him in a mild degree of respiratory alkalosis. Muscle relaxants were employed only when sedation and hyperventilation failed to achieve apnoea. These drugs were used on 19 patients; 11 of them required a muscle relaxant on at least one further occasion because of difficulty in synchronizing the patient with the ventilator. Thiopentone was used in four patients, usually to control extreme restlessness. In this series the average period of I.P.P.V. was 12 days, but three patients were ventilated for less than 24 hours and one patient was ventilated for 37 days.

General Care.—Routine investigations, such as chest radiography, serum electrolyte estimation, blood-gas analysis, and haematological examination, were carried out at least daily. The investigations were tailed off when the patient's condition was stabilized. Attention was paid to achieving satisfactory nutrition and fluid balance. The general care of the patients in this unit is discussed more fully by Bargh and Slawson (1965).

Complications Related to I.P.P.V.

The most serious complications which can develop are acute airway obstruction and tension pneumothorax. The commonest causes of acute airway obstruction are the accumulation of secretions as the result of inadequate suction, or the presence of tracheobronchial scabs where humidification is inadequate. Less frequently it may be due to aspiration of gastric contents past the cuff or to herniation of the cuff of the tracheostomy tube.

Tension pneumothorax may develop at any time in a patient with a chest injury when on a ventilator. Patients with a manifest pneumothorax on admission will have had pleural drainage performed, but this drain may block. Also, in a few patients with pre-existing pleural adhesions a functioning intrapleural drain may not prevent air accumulating in a separate pleural compartment. Where there is rupture of a lung or a bronchus even a functioning intrapleural drain may not be able to cope adequately with the air leak; in these cases thoracotomy is usually indicated.

The danger of the sudden development of a tension pneumothorax has led to two opposing practices. One view held is that all patients with a crush injury who receive artificial ventilation should have intrapleural drainage whether or not a pneumothorax is present (Campbell, 1966). The other view is that intrapleural drainage should be reserved for those patients who have a pneumothorax or a haemothorax. The former practice appears very sound, but may be associated with lung damage in the presence of pleural adhesions.

In some patients difficulties are encountered in instituting or maintaining I.P.P.V. Common causes of difficulty are diminished lung compliance, increased airway resistance

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(whether due to secretions or bronchospasm), and increased muscle tone associated with effort and restlessness. If secretions are present they must be removed, if necessary by bronchoscopy. If the patient then remains difficult to settle on the ventilator muscle relaxants or, occasionally, barbiturates are used. In the presence of such difficulty blood-gas analysis may be of great value in determining the presence of hypoxia or hypercapnia. Once satisfactory ventilation is established unnecessary disturbance of the patient must be avoided. This is especially important on the first day on the ventilator and in patients with pre-existing lung disease. We consider frequent cuff deflation unnecessary and disturbing, particularly during the first 48 hours. Tracheal suction should be performed as necessary but not as a routine. If infusions are to be set up, injections given, blood samples taken, or nursing care carried out, all should be done at the same time to minimize the frequency with which the patient is disturbed. Occasionally patients placed on a ventilator rapidly become hypotensive. This may be due to hypovolaemia, to the too sudden drop of a raised P_{aCO_2} , or to the use of high ventilating pressures. If the fall is severe vasopressor drugs may be required.

Other Complications

These include anaemia, gastrointestinal stasis, renal failure, electrolyte disorders, and respiratory infection.

Anaemias.—Haemoglobin levels were measured regularly in these patients. The haemoglobin level dropped below 70% in 28 and below 60% in 10. In 28 patients blood transfusion was employed to treat the anaemia. During the 48 hours after trauma a drop in the haemoglobin level may occur owing to haemodilution, but we found that considerable falls occurred outside this period. There are several possible causes for this delayed fall in the haemoglobin level. (1) Continued bleeding: this is more likely to occur in patients with multiple injuries. (2) Infection: many of our patients who became anaemic had a chest infection or, less commonly, an infection elsewhere. (3) Renal failure: two patients who developed anaemia had severe renal failure requiring haemodialysis; in other patients less severe grades of renal failure were present. Haemoglobin estimations must be carried out regularly in all patients with multiple injuries, especially in those who develop renal failure or a severe infection.

Gastrointestinal Stasis.—This is a very serious complication, for not only does it result in fluid and electrolyte imbalance but the resultant abdominal distension embarrasses respiration and makes artificial ventilation more difficult. Furthermore, regurgitation of gastric contents may occur and these may be aspirated. Stasis occurred in 25 patients. The criterion of gastrointestinal stasis was a gastric aspirate greater than 500 ml. over a 24-hour period. Other signs, such as abdominal distension and muted bowel sounds, are less easy to quantitate. Five of the 25 had an aspirate greater than 1,000 ml. in 24 hours. In this group laparotomy was carried out in seven; in five there were positive findings, which included ruptured diaphragm with herniation of the stomach, traumatic perforation of the small bowel, bruising of the duodenum, ruptured spleen, and retroperitoneal haematoma.

Renal Failure.—A rise in blood urea above 40 mg./100 ml. was common. In 12 patients it rose above 100 mg. (Table I). In five of these renal failure was so severe that haemodialysis was performed. All 12 had a period of oliguria, usually lasting two to four days. The blood urea was usually first raised within 48 hours of the accident and the period of renal failure among the survivors varied from 6 to 25 days. The serum potassium level was raised above 5.5 mEq/l. in five patients, rising to 7 mEq/l. in one patient. The causes of renal failure in these 12 patients are considered under two headings in Table I. Factors which we believed to be primary causes of renal damage were oligaemic shock, circulatory insufficiency due to cardiac arrest, severe infection, and prolonged hypoxia and hypercapnia. Factors which contributed to the rise in blood urea without necessarily causing renal damage included the increased protein breakdown that follows injury, respiratory infection, inadequate fluid intake, and nephrectomy (Robson, 1966). Five of the 12 patients died, haemodialysis having been performed on two of them. The factors which determine renal failure occur soon after the injury, and a reduction in its incidence will largely depend on the speed and efficiency of the initial management.

Electrolyte Disorders.—Daily electrolyte estimations were performed in most of our patients and electrolyte disorders were seen to occur frequently. It was found, for example, that in 14 patients the serum potassium level exceeded 5.5 mEq/l. and in 23 the level fell below 3.5 mEq/l. Because these changes occur commonly, daily electrolyte studies are desirable, and these may have to be continued for as long as two to three weeks.

TABLE I.—Details of 12 Patients whose Blood Urea was Raised Above 100 mg./100 ml.

Case No.	Age	Day on which B.U. was First Over 40 mg./100 ml.	Duration of Renal Failure (Days)	Duration of Oliguric Phase (Days)	Primary Cause of Renal Failure	Other Factors which Contributed to Rise in B.U.	Dialysis	Potassium (mEq/l.)	Outcome
3	54	Day 2	15	3	Oligaemic shock		1 X	5.8	Recovered
21	53	Day 3*	24	2	" "	Chest infection— <i>Staph. aureus</i>	2 X	5.1	"
27	73	Day 2	34	7	Oligaemic shock. <i>Ps. pyocyanea</i> septicaemia	<i>Ps. pyocyanea</i> septicaemia	2 X	6.5	Died
31	29	Day 3*	4	4	Oligaemic shock	Nephrectomy	1 X	6.5	"
66	34	Day 2	8	2	"	"	—	5.8	"
98	57	Day 2	12	1	Circulatory insufficiency due to cardiac arrest	Pneumonia— <i>Staph. aureus</i> . Inadequate fluid intake for several days	—	4.4	Recovered
103	58	Day 2	11	1	"	Chest infection— <i>Staph. aureus</i>	—	—	"
112	41	Day 2	20	2	Oligaemic shock	Chest infection— <i>Staph. aureus</i> pneumococci and	1 X	5.0	
122	74	Day 4	15	3	Oligaemic shock. <i>Staph. aureus</i> chest infection	Chest infection— <i>Staph. aureus</i> . Excess protein intake	—	7.0	Died
142	73	Day 2	6	2	Uncertain. ? Oligaemic shock. ? Respiratory acidosis	Pneumococcal chest infection	—	5.1	Recovered
172	58	Day 2	25	4	Uncertain. ? Oligaemic shock. ? Chronic renal insufficiency secondary to reduced cardiac output due to aortic incompetence	Chest infection— <i>Ps. pyocyanea</i>	—	4.7	
193	15	Day 3	3	5	Circulatory insufficiency due to 2 cardiac arrests. Persistent anoxia. Persistent respiratory acidosis	—	—	4.9	Died

* First recorded result.

Respiratory Infection.—In a retrospective study it is often rather difficult to assess whether or not there has been a respiratory infection. Thirty-four patients received antibiotic treatment and were presumed to have a respiratory infection. This constitutes 53% of the total. By far the commonest infecting organism was *Staphylococcus aureus*, other important organisms being pneumococci and *Pseudomonas pyocyanea*.

Results

Table II summarizes the results in the present series. It can be seen that in the group of patients with injuries to the thorax alone the mortality rate was 13%, whereas in the group with severe associated injuries the rate was 24%. The overall mortality rate was 16%.

TABLE II.—Anatomical Distribution of Injuries and Mortality Rate in 64 Patients with Severe Crushed Chest Injury

Sites of Injury	No. of Cases	Died
Thorax only	47	6 (13%)
Thorax + abdominal injury	6	3
Thorax + head injury	10	1 (24%)
Thorax + abdominal + head injury ..	1	0
Total	64	10 (16%)

Causes of Death

The causes of death were assessed on the basis of the site and extent of injury, the clinical course of the condition, and the necropsy findings. In some patients the cause of death was obvious. In others multiple factors were involved and it was not always possible to be certain of the exact mechanism of death. Table III lists the causes of death in the group of patients with injuries to the chest only. Two patients had severe injury to the thoracic aorta. One of them (Case 71) died as the result of haemorrhage; the other (Case 27) developed a dissecting aneurysm which compressed the left main bronchus and left pulmonary veins, with resultant bronchopneumonia and pulmonary oedema.

TABLE III.—Causes of Death in Six Patients with Chest Injury Only

Case No.	Age	Cause of Death	Time of Death in Days After Accident
27	73	Traumatic aneurysm of thoracic aorta	33
71	46	Rupture of aorta	1
122	74	Multiple	18
125	48	Respiratory failure	6
193	15	" "	7
199	44	" "	4

Case 122.—A 74-year-old woman died as the result of multiple factors, including bronchopneumonia and a severe infection at the site of a compound fracture of the tibia. She appears to have been overtransfused during the initial resuscitation, and this may well have adversely affected her progress.

Case 125.—A previously healthy 48-year-old man was admitted to hospital with many fractured ribs and a fractured pubic ramus. He died on the sixth day. Post-mortem examination showed a traumatic haematoma of the left upper lobe and consolidation of both lungs associated with oedema, haemorrhage, and a severe generalized bronchitis. He was probably overtransfused during the initial resuscitation.

Case 193.—A 15-year-old boy was crushed between a lorry and a wall. He had a ruptured right lower lobe bronchus and required a right lower lobectomy. Cardiac arrest occurred twice before the thoracotomy. Next day he was conscious, was rational, and had normal blood gases. He became increasingly difficult to ventilate, and high pressures had to be used. On the sixth day renal failure supervened and he died one day later. Post-mortem examination showed gross consolidation of both lungs. There was obvious contusion of the left lower and right middle lobes and widespread evidence of intra-alveolar haemorrhage such as might be caused by a blast injury. In addition, there was bilateral bronchopneumonia and pulmonary oedema. It is of interest to note that this boy had

no fractured ribs. His case is a good example of the compression type of chest injury in which the lungs are severely traumatized by compression of the thoracic cage in the absence of fractures of the ribs or sternum.

Case 199.—A previously healthy 44-year-old man sustained a severe chest injury and required a left lower lobectomy because of extensive laceration of this lobe. There was severe bruising of the remaining lung tissue, and, despite hyperventilation with pure oxygen, he deteriorated and died on the fourth day of respiratory failure.

Of these six deaths two (Cases 27 and 71) were almost certainly inevitable and three (Cases 122, 193, and 199) were probably unavoidable. We feel that in Case 125 overtransfusion played a significant part in the respiratory failure and that the patient might have survived.

Table IV lists the causes of death in the four patients who had severe associated injuries.

TABLE IV.—Causes of Death in Four Patients With a Chest Injury and Severe Associated Injuries

Case No.	Age	Cause of Death	Time of Death in Days After Accident
31	29	Renal failure and pulmonary embolus	6
57	65	Multiple	11
66	34	" "	11
128	23	Severe head injury	5

Case 31.—A 29-year-old man was transferred in a moribund state from a distant peripheral hospital for haemodialysis. He died soon after admission.

Case 57.—A 65-year-old man with a history of myocardial infarction had an emergency splenectomy, and later developed a gastric fistula with peritonitis.

Case 66.—A 34-year-old man had a fractured right femur and 10 fractured ribs on the right side. He was severely shocked and in respiratory distress. Next day he developed signs of intra-abdominal bleeding. A laparotomy was carried out and a lacerated right kidney removed. This proved to be a very difficult operation with considerable loss of blood. Later he developed uraemia, the blood urea rising to 160 mg./100 ml. on the eighth day. On the eleventh day he suddenly collapsed and died. Post-mortem examination showed moderate collapse and oedema of both lungs, together with pneumonic consolidation of the left lower lobe. There was no evidence of obstruction of any of the major bronchi. A laceration of the upper surface of the right lobe of the liver was present, with a large blood clot in the vicinity. Death probably resulted from a combination of uraemia, respiratory difficulty, and unsuspected blood loss.

Case 128.—A racing driver, this man died from the effects of a severe head injury.

The deaths of three patients (Cases 31, 57, and 128) were probably inevitable. The other patient (Case 66) was a previously healthy young man, and, taking into consideration the nature of his injuries, we feel that he should have survived.

Discussion

A major problem in the management of patients with severe injuries of the chest is deciding which of them require I.P.P.V. In order to overcome this difficulty different authors have attempted to classify patients with crush injuries of the chest into classes 1, 2, and 3 (Reid and Baird, 1965) and into mild, moderate, and severe groups (Lloyd *et al.*, 1965) based mainly on the results of blood-gas analysis. They regard a raised PaCO_2 as being the main indication for deciding on I.P.P.V. In this series the decision to institute I.P.P.V. has been based on the clinical rather than the biochemical findings. Thus the persistence of dyspnoea and paradox, after correction of airway obstruction and collapse of lung, has been regarded as the indication for I.P.P.V. even if blood-gas analysis shows a low PaCO_2 . We believe that I.P.P.V. does more than correct a respiratory acidosis. It abolishes respiratory effort, reduces pulmonary plethora and oedema, and effects internal splinting of the displaced segment of the chest wall with resultant control

of paradox and reduction in residual deformity. It reduces pain and allows humane sedation without fear of respiratory depression. If a trained medical and nursing staff and proper facilities—that is, a respiratory unit—are available, and they should be, the hazards of I.P.P.V. are negligible.

Though blood-gas analysis is not used as the main indication for I.P.P.V., it has been found to be of great value in the management of a patient on a ventilator. This is particularly so in the "difficult" patient—that is, one with chronic lung disease or a low compliance from any other cause. Here frequent adjustments of inspiratory flow rates and inflation pressures may be necessary to achieve adequate ventilation, and the results of blood analysis are then invaluable. It is also very useful in the interpretation of certain clinical signs. For example, if varying levels of consciousness are occurring, are these due to a concurrent head injury or to hypoxia and acidosis? Or, if the patient's blood pressure is falling, is this due to excessive ventilation or hypovolaemia? In the patient with renal failure is the restlessness respiratory or renal in origin? In these situations blood-gas studies may be the only means of deciding.

As the management of the respiratory problems improves the mortality rate will depend more and more on the severity of the associated injuries. For this reason patients in this series have been classified according to the sites of injury—that is, thoracic only or thoracic together with other major injuries. Furthermore, as patients die from dysfunctions rather than injuries, we have attempted to quantitate these dysfunctions.

Ten patients died. In four of these (Cases 27, 31, 71, and 128) death resulted from dysfunctions which could not be influenced by I.P.P.V. Three deaths (Cases 125, 193, and 199) were due to failure of respiration with which I.P.P.V. with pure oxygen failed to cope. The remaining three deaths (Cases 57, 66, and 122) were due to multiple factors, of which respiratory dysfunction contributed its share.

It is impossible to compare the results in this series with the results of current authors (Reid and Baird, 1965; Lloyd *et al.*, 1965; Campbell, 1966). The indications for I.P.P.V. are different, and, furthermore, the mortality rate will, in part, depend on the presence of other serious injuries—for example, head or abdominal visceral injuries. However, if the results of this series are compared with those recorded by Griffiths in 1960 a marked improvement in the mortality rate is obvious. This is largely due to the efficient management of the respiratory problem.

Summary

Sixty-four patients with severe crush injuries of the chest are reviewed. They have been classified according to the anatomical site of injury. The great majority of patients received I.P.P.V., the indications for this being on clinical rather than biochemical features—particularly the persistence of dyspnoea and paradoxical respiration after the correction of airway obstruction and collapse of the lung. Serious complications encountered included anaemia, gastrointestinal stasis, renal failure, electrolyte disorders, and respiratory infection. The overall mortality rate was 16%. The value of I.P.P.V. in abolishing respiratory effort, effecting internal splinting of the chest wall, and reducing pain is emphasized.

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Insulin Resistance

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Insulin resistance is usually defined as a state in which the daily insulin requirement exceeds 200 units for more than two days (Root, 1929; Joslin *et al.*, 1959). We have used a somewhat narrower definition in the cases described below, and have restricted the term to diabetics whose daily insulin requirement exceeded 200 units, was not attributable to severe ketosis or infection, and persisted for longer than one week. The incidence of this state is difficult to ascertain, but it is undoubtedly low. Smelo (1948) collected a series of 54 cases of insulin resistance, and Shipp *et al.* (1965) reviewed 55 cases published since 1948 and described 34 cases seen in the Joslin Clinic between 1940 and 1960; the latter constituted 0.1% of the total number of diabetics seen during this period. An earlier paper (Oakley *et al.*, 1959) presented the results of immunological and other studies in 13 cases of insulin resistance, in six of which treatment with prednisone was given; these have been followed up and are included in the present study together with 28 additional cases.

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Insulin resistance may be due to a number of different causes, some of which, such as ketosis, hormone antagonism, and infection, have long been recognized. The condition may also exist for no demonstrable reason. Field and Stetten (1956) demonstrated the presence of an antagonist in the alpha fraction of the serum globulin in severe ketosis. An antagonist to insulin has also been found by Vallance-Owen *et al.* (1958) in the albumin fraction of plasma from uncontrolled insulin-requiring diabetics and also from normal subjects. In the experimental field Young (1937) produced a degree of insulin resistance in the metahypophyseal phase of the diabetes induced in dogs by administration of anterior pituitary extract; the same state has been shown to follow the administration of growth hormone. In man human growth hormone may increase insulin requirement, but not to resistant levels (personal observation). An insulin antagonist, placental lactogen, has been shown by Kalkhoff *et al.* (1964) to be produced in increasing amounts during both normal and diabetic pregnancy; this may account not only for the progressive rise in insulin requirement generally observed in diabetic pregnancy, which may reach resistant levels, but also for its sudden fall after delivery. Insulin resistance may rarely be associated with severe liver disease, including haemochromatosis (Colwell and Weiger, 1956), generalized