There was no increase in the total carrier-rate during the stay of patients in the cubicle ward, and only a small rise in the percentage carrying staphylococci resistant to two or more antibiotics.

The rate of appearance of new strains of *Staph. aureus* on successive swabbing was investigated as a possible measure of the exposure of patients to infection. Isolation in cubicles did not have much effect on the rate of apparent acquisition of staphylococci sensitive to antibiotics or resistant only to penicillin, but it reduced considerably the rate for organisms resistant to two or more antibiotics.

It seemed likely that many of the "acquisitions" were artifacts, due to the intermittent isolation of organisms carried before admission. For this reason, the rate of apparent acquisition of multiple-resistant strains—which are less often found in the nose of patients on admission—is a more sensitive index of exposure to infection in hospital than the rate of apparent acquisition of all strains of *Staph. aureus*.

No likely source could be found for two-thirds of the apparent acquisitions. About half of the remainder could be attributed to infection from the staff and half to infection from patients in other cubicles. The rate of acquisition of strains

from known sources might be a valuable measure of the effect of ward design on the risk of acquiring a hospital staphylococcus.

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Crushed Chest Injury: Some Physiological Disturbances and Their Correction

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Although still a relatively uncommon injury, there is an increasing incidence of severe thoracic and pulmonary damage, due principally to high-speed motoring accidents. This type of injury, which was formerly associated with mining accidents, is commonly referred to as the crushed chest injury or stove-in-chest. There may be multiple posterior or anterior fractures of ribs and fractures of the sternum or a mixture of such fractures.

Griffiths (1960), reviewing the literature, has summarized the advantages of treatment by intermittent positive-pressure ventilation (I.P.P.V.) over mechanical fixation. Nevertheless, the results remain somewhat disappointing.

In a series of 33 cases treated in the Respiratory Emergency Unit of Glasgow Royal Infirmary during the past two years it has become evident that an explanation for many disappointing results can be traced to our previous inability to measure the severity of the *respiratory* and *metabolic* disturbances that result from this form of trauma. The recent development of simple biochemical techniques and their application at the bedside now make it possible to assess rapidly the severity of the initial disturbances, to aid the selection of the correct treatment, and to follow at frequent intervals the effectiveness of the treatment.

Respiratory and Metabolic Disturbances

The term "crushed chest injury" implies considerable violence to the thorax, and this must result in damage to the

lungs, as suggested by Harley (1961) (Fig. 1). The disruption of alveolar structure, interstitial and intra-alveolar haemorrhage, and atelectasis due to plugging of the bronchioles with secretions and debris are evident. Alteration in the alveolar ventilation/blood perfusion ratio (V/P ratio) is inevitable in the affected segments of the lungs. This leads to a reduction

FIG. 1.—Demonstrating damage to the lungs.

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in the oxygen content of the arterial blood, although carbon dioxide elimination may be virtually unimpaired (Comroe *et al.*, 1962). Continuing haemorrhage, lobular atelectasis due to secretions, haemothorax and/or pneumothorax, pain, and central depression of respiration due to injury or analgesic drugs will cause the oxygen content of the arterial blood to fall further. Finally, when lung failure is imminent the carbon dioxide content of the arterial blood will rise and acidosis will supervene.

The overall reflex response to this state of hypoxia and hypercarbia is an attempt to improve alveolar ventilation principally by increasing the rate of respiration. Thus the work of ventilation is increased, and therefore oxygen consumption will rise. The efficiency of this response is further impaired by the trauma to ribs and muscles of respiration, although Maloney *et al.* (1961) question the validity of intrapulmonary shunting of air due to a flail segment.

Severe loss of blood is a major problem in such patients, who have often sustained other multiple injuries. Vasoconstriction occurs, and the consequent reduction in arterial perfusion of the tissues results in a metabolic acidosis. If the haemorrhage is of sufficient magnitude the cardiac output falls, increasing the severity of the metabolic acidosis.

Freeman (1963) has stressed the occurrence of metabolic acidosis as the consequence of stagnant hypoxia after severe haemorrhage. He has drawn attention to the great increase in the amount of dead-space ventilation that results. To maintain a normal oxygen content of arterial blood a considerable increase in total ventilation would therefore be required.

The acidity, the high potassium-ion content, and the citrate content of stored blood, which may be required in large volumes during the initial resuscitation, increase the problems (Bull, 1963).

Concomitant head injuries and unconsciousness are common. The effects of an obstructed airway in these cases are too well known to require further comment.

FIG. 2.—Summarizing the disturbances of the respiratory system.

FIG. 3.—Summarizing the disturbances of the cardiovascular system.

A degree of renal failure presumably related to the hypotension, acidosis, and hypoxia is often present, and, indeed, acute tubular necrosis may occur.

In reviewing the literature we have noted the emphasis on the extent of the bony injuries, particularly in the earlier reports. Little attention has been paid to the direct damage to the lungs and to the interrelationship of the disturbances of the respiratory and cardiovascular systems (Figs. 2 and 3).

Outline of Management

As rapidly as possible plasma and stored blood are used to correct the hypovolaemia. The consequent increase in metabolic acidosis justifies the empirical administration of sodium bicarbonate solution intravenously—for example, 100 mEq in an adult. A sample of arterial blood is obtained as soon as is feasible, and the following measurements are made: (1) pH, arterial carbon dioxide tension (Paco₂), standard bicarbonate, base excess, and buffer base (Astrup, 1959 ; Andersen *et al.*, 1960) ; (2) oxygen saturation (Sao₂)—by means of the Kipp haemoreflexor ; and (3) where possible the tension of oxygen in arterial blood (Pao₂). Collectively these measurements are referred to as blood-gas analysis.

As a result of biochemical and clinical assessment (including radiological examination) we have found it possible to place the cases in one of three arbitrary classes. This not only assists discussion but is also of some clinical value.

Class 1

The extent of the thoracic injury varies, and pneumothorax and/or haemothorax may be present. These patients have a reduced oxygen and carbon dioxide content in the arterial blood (Whitwam and Norman, 1964). Clinically this group is distinguished by the patient's ability to cough effectively so that the airway remains clear of secretions. Spontaneous respiration is always maintained.

The treatment of these patients is conservative, consisting of (a) broad-spectrum antibiotic, (b) frequent physiotherapy, and (c) continuous oxygen, which is administered as soon as possible after injury. We prefer the nasal catheter (or modifications) with low flow of oxygen because expectoration and eating are possible while breathing oxygen-enriched air.

We administer analgesics—for example, morphine (at first intravenously), and do not employ analgesic/antagonist mixtures, as their efficiency has been questioned (Telford and Keats, 1961).

Frequent blood-gas analyses are essential so that the patient's condition can be accurately and continually assessed. Early improvement is usually seen (Table I), as in the patient (Case 28) who had fractures of nine successive ribs and a haemopneumothorax which required drainage. However, it may be a few days before the arterial blood is fully oxygenated.

TABLE I				
Time from Admission	Pao ₂ (mm.Hg)	pH	Paco ₂ (mm.Hg)	Remarks
0	—	7.34	36	Breathing air
+ 2 hours	297	7.36	38.5	Breathing air/oxygen mixture
+ 4 hours	402	—	—	" " " "
+ 12 hours	—	7.39	44	" " " "

Class 2

These patients have usually sustained more serious injuries, including non-thoracic injuries. The level of consciousness may be impaired. The oxygen content of the arterial blood is reduced and the carbon dioxide content is also lowered,

although the latter is more difficult to assess in patients with pre-existing obstructive lung disease.

However, in contrast to those in Class 1, these patients are unable to cough effectively, and therefore require tracheostomy and aspiration of bronchial secretions to maintain a clear airway.

The general management is as outlined above, but because of the tracheostomy the oxygen or oxygen/air mixture must be humidified. The oxygen content of the arterial blood rises to normal, although this may occur during a few days rather than immediately. The carbon dioxide content of the arterial blood remains satisfactory because of the maintenance of a clear airway and reduction in dead space.

Some patients, however, deteriorate into Class 3.

Class 3

These patients have acute progressive (uncompensated) respiratory failure. The oxygen content of arterial blood is low, the pH is low, and the partial pressure of carbon dioxide is high. Metabolic acidosis is often present. Typically, multiple injuries are present, and these are both extensive and severe. Almost always there is a haemopneumothorax, occasionally bilateral. Hypovolaemic shock is present.

Intermittent positive-pressure ventilation is begun through a cuffed endotracheal tube employing 100% oxygen. The blood volume is restored as soon as possible. The patient's condition usually improves rapidly, so that tracheostomy, insertion of water-seal drain, laparotomy, etc., can be performed without undue haste.

Thereafter the patient is ventilated by a mechanical ventilator employing 100% oxygen, sedation, analgesia, and a muscle relaxant. It is rare for the oxygen content of the arterial blood to rise to normal for some days. The ventilator is adjusted to maintain the carbon dioxide level in arterial blood within normal limits as determined by frequent blood-gas analyses.

However, two types of patients require further discussion. (a) In the patient with severe pre-existing obstructive lung disease hyperventilation will not always correct the respiratory acidosis (Table II). In such cases the combination of ventilation and trihydroxymethylaminomethane is warranted (Nahas, 1963) (Table II). This patient (Case 29) subsequently made a good recovery, although splenectomy, and, later, laparotomy for division of adhesions, were required. (b) Occasionally the oxygen content of the arterial blood is very low, whereas the carbon dioxide content is only moderately raised (Case 5). This case has already been reported in detail (Kennedy *et al.*, 1963). A relatively satisfactory oxygen content of the arterial blood was not achieved until mechanical ventilation with pure oxygen was begun.

TABLE II

Time	SAO ₂ (%)	pH	Paco ₂ (mm. Hg)	*St. Bic. (mEq/ l.)	Ventilation
Admission	70	7.27	80	25	Spontaneous, breathing oxygen/air
+ 30 min. . .		7.26	82	25	Hyperventilation by anaesthetist with CO ₂ absorption and 100% O ₂
Trihydroxymethylaminomethane 18 g. infused					
+ 2 hr. 45 min.		7.37	67	35	Mechanical ventilation + 100% O ₂
+ 4 hr. 15 min.	94	7.50	50	37	

*St. Bic. = Standard bicarbonate.

Within a few days the dosage of sedative, analgesic, and muscle relaxant can be reduced and finally discontinued. After the oxygen content of the arterial blood has been normal for some days the concentration of oxygen in the inspired gas can be slowly reduced. Reduction in the oxygen content of the arterial blood again occurs if this is undertaken too rapidly. Finally the patient is ventilated with humidified air.

When "weaning" from the ventilator is started—that is, allowing the patient to breathe spontaneously for one to two minutes—a fall in oxygen content of the arterial blood occurs if the patient is breathing air. The patient must therefore breathe an oxygen-enriched atmosphere during "weaning," and this is allowed to continue only provided that the oxygen and carbon dioxide content of the arterial blood remains substantially normal.

Discussion of Results

During two years 33 cases of crushed chest injury were treated, and 21 (63%) survived (Table III, overleaf). All patients in Class 1—that is, those who required only conservative management—survived (Table IV). The survival rate of patients who could not cough effectively, and therefore required tracheostomy in addition to conservative management, was 62.5%. In this series the deaths in Class 2 occurred in elderly patients (69 to 80 years). The combined survival rate for 17 patients who did not require ventilatory therapy was 82.4% (Classes 1 and 2).

TABLE IV.—Summarizing Patients in Their Respective Classes

Class	No.	Surviving		No. With Obstructive Lung Disease Prior to Injury	No. With Serious Non-Thoracic Injury	Remarks
		No.	%			
1	9	9	100	4	3	Only one patient with obstructive lung disease had other serious injury
2	8	5	62.5	4	3	The 3 patients who died were aged over 69 and had obstructive lung disease
3	16	7	43.75	8	12	Two patients who had hepatorenal failure died suddenly. Only one patient was aged over 70, and he died.
Total	33	21	63	16	18	

The survival rate in 16 patients who required mechanical ventilation was 44%; 12 cases (75%) had serious non-thoracic injuries. Of the 24 patients who required tracheostomy or tracheostomy plus ventilation 50% survived (Classes 2 and 3).

The incidence of pre-existing obstructive lung disease was almost 50% in each class. Its influence on prognosis in this series is difficult to assess. The three patients in Class 2 who died had pre-existing obstructive lung disease but were elderly; on the other hand, three of the seven survivors in Class 3 had clinical evidence of this disease.

Acute tubular necrosis requiring dialysis occurred in three patients. It was always possible to dialyse these patients, although they required simultaneous mechanical ventilation. Two patients had combined hepato-renal failure, and they died suddenly. One of these patients had been dialysed on one occasion.

With one exception all patients with a tracheostomy developed a serious pulmonary infection. The commonest organism was penicillin-resistant *Staphylococcus aureus*. Despite aseptic precautions during aspiration of bronchial secretions the incidence of pulmonary infection has remained high. Barrier nursing of infected patients in individual cubicles cannot be undertaken at present in this unit.

We have routinely administered sodium bicarbonate solution to correct metabolic acidosis rather than the amine buffer trihydroxymethylaminomethane, although this practice is open to several theoretical objections. The principal objection is that sodium bicarbonate is a weak titrating agent producing excess carbon dioxide, whereas trihydroxymethylaminomethane is an intracellular as well as an extracellular buffer, although respiratory depression can follow its administration (Nahas, 1963).

As has been implied before, frequent blood-gas analysis is necessary to detect any deterioration in the patient's respiratory state. We have employed oxygen saturation as a measurement of change of oxygen content of arterial blood, although direct measurement of the oxygen tension in arterial blood is more sensitive because of the shape of the oxygen-dissociation curve of blood.

Indeed to focus attention on the prime importance of the trauma to the lung and to concentrate treatment on the respira-

tory disability we suggest that a better term than crushed chest injury is acute traumatic lung failure.

Summary

The pulmonary pathology following crushed chest injuries is discussed with emphasis on the consequent derangements of the respiratory and cardiovascular systems. A classification is

TABLE III.—*Clinical Details of Patients*

Case No.	Age	Pulmonary Trauma on X-ray	Obstructive Lung Disease	Head Injury	Bony Thoracic Injury	Other Injuries	Renal or Hepatic Failure	Tracheostomy	I.P.P.V.	Class	Result
1	46	Yes	Yes	Concussion	None	Facio-maxillary and forearm fractures. Haematuria	Oliguria only	*E.T.T.	10 hrs.	3	Survived
2	20	Yes	None	Concussion	None	Rupture of spleen. Laceration of liver. Retroperitoneal haematoma. Fractured femur	Renal and hepatic failure	Yes	6 days	3	Died
3	57	None	Yes	None	7 ribs	Retroperitoneal haemorrhage. Fractured pelvis and humerus	One renal dialysis required. Hepatic failure	Yes	13 days	3	Died
4	55	Yes	Yes	None	Multiple fractures of ribs and sternum	Fractured patella	Transient jaundice	Yes	19 days	3	Survived
5	20	Yes	None	Unconscious	None	Peritoneal haemorrhage. Laceration of liver	Required renal dialysis on 10 occasions	Yes	33 days	3	Survived
6	80	Yes	Yes	None	6 ribs (L.), 2 ribs (R.) fractured	Ruptured diaphragm. Compound fracture of lower leg	Transient oliguria	Yes	Nil	2	Died
7	59	None	None	None	3 fractured ribs. Pneumothorax	Fractured lower leg	Transient oliguria	Yes	Nil	2	Survived
8	69	Yes	Yes	None	6 fractured ribs. Pneumothorax	None	None	Yes	Nil	2	Died
9	46	Yes	Yes	None	Fracture of 3 ribs and scapula	None	None	No	Nil	1	Survived
10	62	Yes	Yes	Fracture of skull and brain damage	Fractured ribs. Pneumothorax	Pott's fracture	Oliguria only	Yes	7 days	3	Died
11	25	Yes	None	None	Paralysis of hemidiaphragm and extensive contusion of lung	Fractured left femur	None	Yes	1 day	3	Died
12	48	Yes	None	Intracranial injuries	Multiple fracture of ribs	Fractures of pelvis and forearm. Haematuria	Oliguria only	Yes	2 days	3	Died
13	62	Yes	Yes	Intracranial damage suspected	Multiple fractures	None	Required renal dialysis twice	Yes	15 days	3	Died
14	42	Yes	Yes	Fracture of mandible. Intracranial injury	Multiple fractures of ribs	None	Oliguria only	Yes	6 days	3	Survived residual cerebral damage
15	45	Yes	None	None	Multiple fractures of ribs	Bilateral fracture of patellae	None	Yes		2	Survived
16	47	Yes	None	None	Multiple fractures of ribs. Tension pneumothorax	None	None	Yes	5 days	3	Survived
17	53	Yes	Yes	None	Multiple fractures of ribs. Pneumothorax	None	None	No	Nil	1	Survived
18	31	Yes	None	None	Multiple fractures of ribs	Fractures of lumbar transverse process, sacrum, pelvis, and bones of R. foot	Fall in urinary urea concentration	No	Nil	1	Survived
19	83	None	Yes	None	Multiple fractures of ribs	None	None	Yes	3 days	3	Died
20	31	Yes	None	None	Multiple fractures of ribs	Fractures of pelvis and lower leg. Facio-maxillary injuries	None	Yes	1 day	3	Died
21	52	Yes	None	Facio-maxillary fractures	Multiple fractures of ribs	None	None	No	Nil	1	Survived
22	34	Yes	None	None	Multiple fractures of ribs. Fracture of scapula. Laceration of R. main bronchus. Pneumectomy.	None	None	Yes	11 days	3	Survived
23	40	Yes	None	None	Multiple fractures of ribs	None	None	No	Nil	1	Survived
24	74	Yes	Yes	Lacerations	Multiple fractures of ribs	Fractures of pelvis and legs	None	Yes	Nil	2	Died
25	35	None	None	None	Multiple fractures of ribs	None	None	Yes	1 day	3	Died
26	59	Yes	Yes	None	Multiple fractures of ribs	Limb fractures. Brachial artery thrombosis. Infarction of arm	None	No	Nil	1	Survived
27	5½	Yes	None	None	None, but R. pleural effusion and pulmonary contusion	Laceration of liver	None	No	Nil	1	Survived
28	47	Yes	Yes	None	Multiple fractures of ribs. Haemo-pneumothorax	None	None	No	Nil	1	Survived
29	32	Yes	Yes	None	Multiple fractures of ribs. Haemo-pneumothorax	Ruptured spleen. Later required division of abdomen adhesions	None	Yes	14 days	3	Survived
30	18	None	None	Severe facio-maxillary	None	Fractured left femur	None	Yes		2	Survived
31	35	Yes	None	None	Fractured rib and scapula. Patchy atelectasis	None	None	No	Nil	1	Survived
32	70	Yes	None	None	Multiple fractures of ribs	None	None	Yes	Nil	2	Survived
33	70	Yes	Yes	None	Multiple fractures of ribs	None	None	Yes	Nil	2	Survived

*E.T.T. = Endotracheal tube.

suggested based principally on the degree of abnormality of blood-gas levels, and not on the extent of thoracic bony injuries. A series of 33 cases treated in the Respiratory Emergency Unit of Glasgow Royal Infirmary is presented and briefly discussed. A plea is made for frequent blood-gas analysis in the assessment of treatment of these cases.

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Diagnosis and Treatment of Erythema Induratum (Bazin)

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Bazin (1861) gave the name of erythema induratum to a nodular eruption which occurred on the lower legs of young women who had tuberculosis. He commented that the condition was not rare. In the next 30 years the disease attracted little notice until, with the discovery of the tubercle bacillus, interest revived and attempts were made, with varying success, to isolate the bacillus from the lesions.

Later, other clinical conditions were described resembling erythema induratum but which were not tuberculous. This, and the declining incidence of tuberculosis generally, may be the reasons why erythema induratum does not now receive sufficient consideration. We wish to draw attention to erythema induratum particularly because of the influx of tuberculosis-prone individuals from overseas. We believe that Bazin's disease is not uncommon in Britain to-day and that it is often misdiagnosed. Even when the diagnosis has been made patients may not receive adequate antituberculous therapy because the physician may not be convinced that he is dealing with a manifestation of active tuberculosis.

Cases.—Table I is a summary of the case histories and investigations in 12 patients with erythema induratum (Bazin). Table II gives their treatment and the clinical response.

Differential Diagnosis

Disorders of the lower legs characterized by indurated plaques or nodules with or without ulceration present a somewhat complex diagnostic problem. The concept that all were not of the Bazin type was first discussed by Audry (1898), and later by Galloway (1899). However, Whitfield's (1901) name is particularly associated with a form of erythema induratum that is not tuberculous. Montgomery *et al.* (1944) stated that the following nodular vascular disorders of the legs have many features in common: nodular vasculitis, erythema induratum, erythrocyanosis and perniosis, erythema nodosum, panniculitis, recurrent idiopathic thrombophlebitis, and indurated cellulitis secondary to chronic venous stasis. Nodular vasculitis was the term they proposed for the type of case described by Whitfield.

Wilkinson (1954) expanded the concept of nodular vasculitis to include a group of disorders with a common vascular basis. In most of these he stressed the aetiological importance of cold

and peripheral venous stasis. He remarked on the additional effect of tuberculosis in some cases, and stated that "The question resolves itself into what part, if any, the tuberculous process plays in a given lesion, even if the disease is active in the patient. One cannot afford to be too sceptical."

As a result of the present study we believe that the role of tuberculosis should be emphasized, and that a case of erythema induratum should be considered to be of tuberculous origin unless reasons for excluding this are particularly strong. Telford's (1937) view that erythema induratum is the same condition as erythrocyanosis and is never tuberculous cannot be accepted. Nor do we agree with Sandberg and Adams (1962), who regard erythema induratum merely as a descriptive term that does not denote a specific tuberculous aetiology.

In our cases wrong diagnoses were prominent and as a result several patients had prolonged disability. Emphasis that Bazin's disease occurs in young women whereas nodular vasculitis is seen in the middle-aged may account for the diagnosis of nodular vasculitis made in some of our patients without sufficient investigation. Two-thirds of our patients were over the age of 35, and it is important to realize that erythema induratum is not confined to younger age groups but may occur later, even in the sixties. One patient of Montgomery *et al.* (1944), a woman aged 54—diagnosed as having nodular vasculitis because clinical, pathological, and bacteriological investigation had failed to disclose any evidence of tuberculosis—developed a tuberculous cervical adenitis within six months, and then showed a strongly positive tuberculin reaction. In addition to nodular vasculitis other diagnoses had included thrombophlebitis migrans, perniosis, erythema nodosum, and collagen disease, and the patients had failed to respond to treatment. Those previously recognized as having Bazin's disease had received insufficient or inadequate antituberculous therapy and had relapsed.

Clinical Features

Most patients showed typical clinical features of erythema induratum. Frequently they presented with crops of small, tender, and painful erythematous or dusky nodules of 1 cm. diameter or less occurring on the lower legs. Some of these nodules ulcerated, often with aggravation of pain. The influence of cold and peripheral vascular stasis could be observed in half of those patients who were worse in winter and in whom nodules tended to occur particularly in the calves and back of the ankles. Females outnumbered men by 11 to 1.

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