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Crush injuries of the chest

A follow-up study of patients treated in an artificial ventilation unit

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Fifty-four patients previously treated for a severe chest injury in an artificial ventilation unit have been followed up. An attempt has been made to determine their state of health and activity. Particular attention has been paid to their respiratory function. The significance of the findings has been discussed.

The effectiveness of the current management of crush injury of the chest is usually measured in terms of immediate survival (Avery, Mörch, and Benson, 1956; Griffiths, 1960; Reid and Baird, 1965; Lloyd, Crampton Smith, and O'Connor, 1965; Bargh, Griffiths, and Slawson, 1967), but there are no long-term reviews of patients with this injury treated by intermittent positive pressure ventilation (I.P.P.V.). A follow-up study of patients treated by this means in the Artificial Ventilation Unit (A.V.U.) in the Royal Infirmary of Edinburgh has therefore been made.

During the period April 1961 to December 1965, 86 patients suffering from a severe crush injury of the chest were treated. The injuries, management, and immediate progress of the first 64 of these patients have been described by Bargh *et al.* (1967). The remaining 22 were not dissimilar from this group. Table I summarizes the outcome of these 86 patients. Eleven patients died while still in the A.V.U. and one other patient died in hospital soon after leaving the Unit following a gastrectomy for haematemesis at the age of 85. Six patients are known to have died at a later date, one from the result of a further road accident, the other causes of death being myocardial infarction (two patients), status asthmaticus, bronchogenic carcinoma, and carcinoma of the colon.

The data presented here are based on the 54 surviving patients who had been treated one to six years previously and whom we were able to trace and interview. Forty-eight of these patients were male and their ages ranged from 6 to 75 years. Two patients had no evidence of rib fracture and 12 had bilateral rib fractures. Forty patients had a haemopneumothorax and 41 had paradoxical respiration. Table II details the major associated injuries.

TABLE I

OUTCOME OF 86 PATIENTS WITH CRUSH INJURY OF THE CHEST

	No.
Died in A.V.U.	11
Died in hospital after leaving A.V.U.	1
Died later	6
Traced	
Seen	54
Not seen	4
Untraced	10
Total	86

TABLE II

ASSOCIATED INJURIES IN 54 PATIENTS WITH CRUSH INJURY OF THE CHEST

	No.
Head injury	25
Abdominal injury	12
Limb fractures	33
Pelvic fracture	8
Spinal injury	8

All but one patient had a tracheostomy and patients were treated by I.P.P.V. for an average period of 12 days with a range of 3–35 days. Thirty-three patients had a laparotomy and one a thoracotomy. A neurosurgical procedure was carried out on three patients and an orthopaedic procedure on 26. The median duration of hospital stay was six weeks.

RESULTS

RESPIRATORY FUNCTION Respiratory assessment was made of the 54 patients by questioning, clinical

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examination, chest radiography, spirometry, and blood gas analysis.

Patients were assigned to one of three groups according to the presence and degree of the symptoms of cough, sputum, and dyspnoea. Group I consisted of patients with no cough, sputum or undue exertional dyspnoea. Group II consisted of those patients with slight cough and/or minimal sputum, and/or dyspnoea on moderate exercise. Group III consisted of patients with frequent cough and/or moderate to copious sputum and/or dyspnoea on slight exercise. Patients were placed in these purely arbitrary groups both before and after the accident (Table III). It can be seen that of 23 patients in group I before injury, 17 deteriorated afterwards, nine entering group II and eight entering group III. Of the 25 patients originally in group II, only seven are shown to have deteriorated into group III.

Fifteen patients complained of chest pain which was skeletal rather than anginal in character. In no case was this severe, but it occasionally led to some disability, *e.g.*, difficulty in lifting heavy weights. Eight patients were noted on clinical examination to have mild deformity of the chest wall. This usually took the form of asymmetry of the chest or flattening of the rib cage. Five patients, including three of the above, were noted to have a bony deformity on chest radiography. Thus a total of 10 patients had a deformity demonstrated either clinically or radiologically. In no patient was the chest deformity gross; certainly there was no evidence of marked traumatic thoracoplasty seen in any patient. Figure 1 shows the most severe deformity noted in the series. There was a tendency for chest pain to be related to bony deformity (Table IV), but the relationship was not statistically significant ($P=0{\cdot}1$).

In 10 patients the clinical signs of obstructive lung

TABLE III

GROUPING OF THE 54 PATIENTS, ON THE BASIS OF RESPIRATORY SYMPTOMATOLOGY, BEFORE AND SINCE ACCIDENT

		Before			
		Group 1	Group 2	Group 3	Total
After	Group 1	6	—	—	6
	Group 2	9	18	—	27
	Group 3	8	7	6	21
	Total	23	25	6	54

For groupings see text.

TABLE IV

PATIENTS WITH CRUSH INJURY OF THE CHEST
SKELETAL CHEST PAIN : BONY DEFORMITY OF THE CHEST

	Skeletal Chest Pain	No Skeletal Chest Pain	Total
Bony chest deformity ..	5	5	10
No bony chest deformity ..	10	34	44
Total	15	39	54

Fourfold table test (*Documenta Geigy Scientific Tables*, 1962) $P=0{\cdot}$

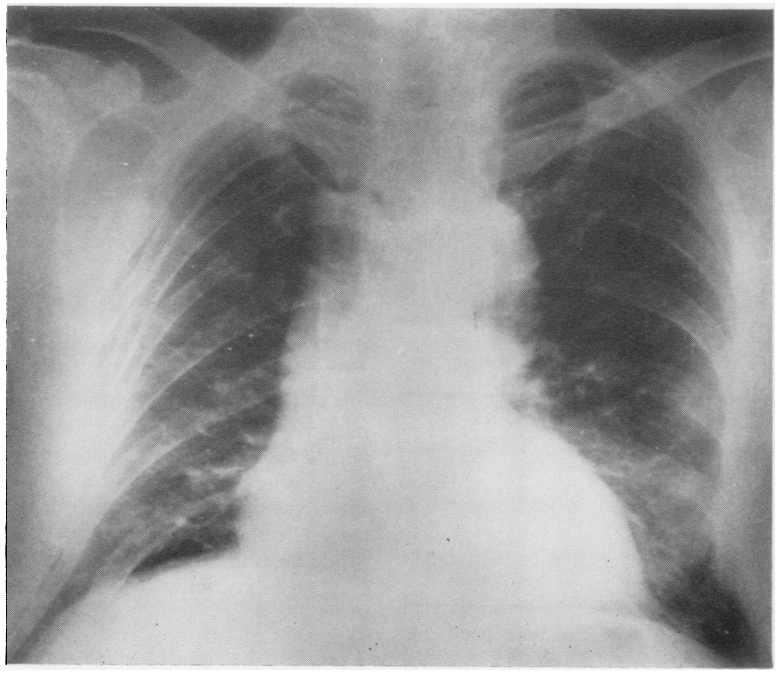

FIG. 1. *Chest radiograph.*

disease were present ; in five of these marked bronchospasm was noted.

Chest radiography was carried out in 51 patients. Five had evidence of chest wall deformity as described above. Pleural thickening was a common feature, being seen in 28 cases. Three patients had the radiological signs of emphysema and four had small areas of fibrosis or collapse. The chest radiographs of one patient revealed what appeared to be an aneurysm of the thoracic aorta ; this appearance had also been present in films performed after the chest injury and there had been no increase in the size of this lesion.

Lung function tests were carried out in all but two patients. The tests used were forced vital capacity (F.V.C.) and forced expiratory volume in one second ($F.E.V._{\cdot 1}$). Figure 2 is a histogram of the ratio of the actual F.V.C. to that predicted by sex, age, and height from the data of Needham, Rogan, and McDonald (1954). The mean ratio is 88·6%. Figure 3 is a histogram of the ratio of $F.E.V._{\cdot 1}$ to the actual F.V.C. The mean ratio is 70·2%. These histograms show that in the series as a whole a slight degree of both restrictive and obstructive lung disease is present. As one would expect, those patients with clinical evidence of emphysema are those with the greatest reduction in F.E.V.

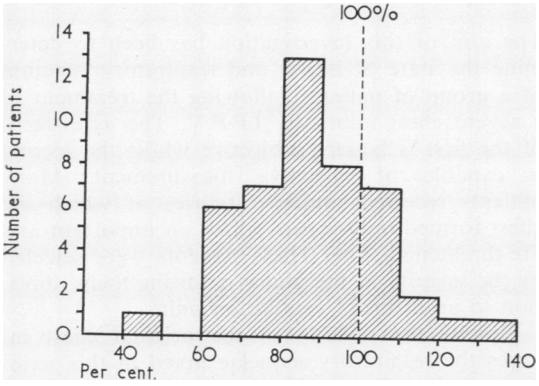

FIG. 2. *Ratio of measured vital capacity to predicted vital capacity.*

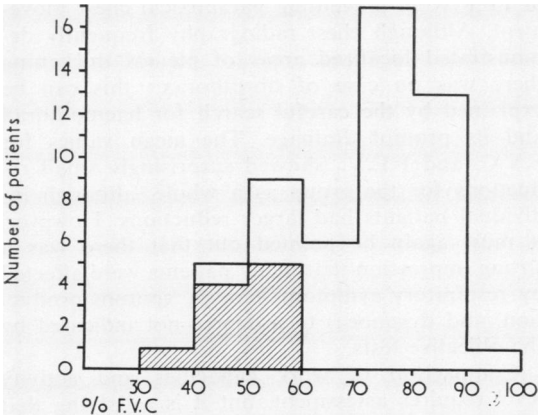

FIG. 3. *Histogram showing $F.E.V._{\cdot 1 \cdot 0}/F.V.C.$ %. Hatched area represents patients with clinical evidence of obstructive lung disease.*

An attempt was made to determine whether the degree of chest trauma influenced lung function as measured at review. It was found that there was no relation between the number of ribs fractured and F.V.C. or $F.E.V._{\cdot 1}$. Bilateral rib fractures did not affect lung function more than unilateral fractures.

The mean F.V.C. in the 10 patients who had a bony deformity of the chest at review was 82·8% of predicted, as compared with 90·0% in the remainder. This difference is not statistically significant (t=0·91).

Arterial blood gas analysis was performed in 35 patients and the results are summarized in Table V.

TABLE V

PATIENTS WITH CRUSH INJURY OF THE CHEST: BLOOD GASES

pHa	30	4	0	1
(ranges in units)	(7·36–7·44)	(7·30–7·35)	(<7·30)	(7·44–7·48)
$PaCO_2$	21	8	0	6
(ranges in Torr)	(36–44)	(45–50)	(>50)	(30–35)
PaO_2[1]	11	13	6	3
(ranges in Torr)	(90–110)	(80–90)	(70–80)	(60–70)

[1] PaO_2 was estimated in only 33 patients.

GENERAL PHYSICAL STATUS An attempt has been made to compare the present level of activity and work capacity with that before the accident. Nineteen patients considered their general status to be the same, 17 thought it less, and 18 much less. The common causes of decrease in activity were respiratory (19), orthopaedic (15), and neurological (6), either singly or together. Of the 40 patients who have worked since their accident, the delay in return to work varied considerably and nine of these did not return to work for more than nine months. The most important causes of delay were orthopaedic injury (14 patients) and respiratory deficiency (9 patients). Respiratory causes were divided between dyspnoea (7 patients) and chest pain (2 patients). Neurological causes (5 patients) tended to be associated with the longest delays.

Table VI shows the work status of the 54 patients before and since the accident. Thirty patients, including three unemployed, had a work status which was unaltered by the accident. Two patients now have

TABLE VI

WORK STATUS OF 54 PATIENTS WITH CHEST INJURY BEFORE AND AFTER ACCIDENT

Present Employment	Previous Employment				
	Heavy	Moderate	Light	No Job	Total after Accidents
Heavy	8	1	0	0	9
Moderate	2	5	0	0	7
Light	7	2	14	1	24
No job	5	1	5	3	14
Total before accident ..	22	9	19	4	54

more arduous employment while 11 have less. Causes of this deterioration in work status were given as respiratory (4), orthopaedic (3), neurological (1), and from causes unrelated to the injury (2). In one patient the cause was uncertain. The remaining 11 patients, all of whom worked prior to the accident, are now unemployed. Five of these were unemployed on account of a neurological deficit and two because of an orthopaedic disability. In two patients the unemployment was unconnected with their injury and in the remaining two the cause of unemployment was uncertain.

A specific inquiry was made from the patients and from their own doctors with regard to respiratory or cardiovascular disease. Since the accident three patients have suffered from acute bronchitis, one patient has had a pleurisy, and another has had pneumonia. Apart from these five episodes there was no obvious increase in acute chest infections in this series. Three patients gave a past history of pulmonary tuberculosis and none of these has had a relapse. One patient has had a myocardial infarct since the accident.

Since many patients sustained orthopaedic or neurological damage at the time of, or following, injury, it is worth summarizing the findings in these systems at review. Sixteen had residual orthopaedic lesions, including shortening of a leg following a fractured femur, stiff elbows and shoulders, and deformities of phalanges. It is noteworthy that eight of them considered that the lesion restricted their physical activity and/or work capacity.

As the result of spinal fractures, two patients are now paraplegic and a further two have minimal neurological changes in the lower limbs. Three patients had a severe neurological deficit as a result of a head injury, in one case a hemiplegia and obvious mental slowness, in another expressive dysphasia, and in the third hemianopia, nystagmus, and difficulty in balancing. A further three patients showed more minor deficits as a result of head injury. Four patients had peripheral nerve palsies. Table VII gives a composite picture of the respiratory, orthopaedic, and neurological deficits encountered in the present series. It can be seen that rather more than half the patients had either an orthopaedic or neurological deficit at review.

Patients were questioned regarding changes in their memory, mental ability, and power of concentration.

TABLE VII

COMPOSITE PICTURE OF INCIDENCE OF RESPIRATORY SYMPTOMATOLOGY AND OF ORTHOPAEDIC AND NEUROLOGICAL DEFICITS SEEN AT REVIEW IN THE 54 PATIENTS

Respiratory Grading at Review	No Orthopaedic nor Neurological Deficit	Orthopaedic and/or Minor Neurological Deficit	Serious Neurological Deficit	Total
Group 1 ..	3	3	0	6
Group 2 ..	11	13	3	27
Group 3 ..	11	8	2	21
Total ..	25	24	5	54

Many claimed some deterioration in these functions and, while no attempt was made to assess quantitatively these functions, it was felt that a number of these patients possibly sustained minor intellectual changes, probably from a head injury or hypoxia.

Seventeen patients in the present series had an E.C.G. performed while receiving treatment for their chest injury. Nine had a normal E.C.G. (if we include two with a sinus tachycardia), while four showed evidence of ST changes and three had a cardiac arrhythmia. One patient, with aortic stenosis and incompetence, had evidence of left ventricular hypertrophy. The eight patients who had an abnormal E.C.G. originally had this procedure repeated at review. With the exception of the patient who had changes associated with aortic valve disease, all the E.C.G.s were within normal limits. This implies that the changes seen in the original E.C.G.s were temporary and were probably due to hypoxia, pericardial irritation, or myocardial bruising.

DISCUSSION

The aim of this investigation has been to determine the state of health and respiratory function of a group of patients following the treatment of a severe chest injury by I.P.P.V. The assessment of the first is largely subjective whilst the second is capable of objective measurement. Many patients received multiple injuries of which the chest formed only a part, albeit an important and life-threatening one. These patients were selected by the severity of the injury requiring their admission to an artificial ventilation unit.

Despite the severity of the original chest injuries the respiratory sequelae noted in this series have not been gross. Only one in five patients had evidence of chest wall deformity and in none was this severe, a tribute presumably to the efficiency of I.P.P.V. in preventing paradoxical chest movement. Although chest radiography frequently demonstrated localized areas of pleural thickening there was no case of fibrothorax; this can be explained by the careful search for haemothorax and its prompt drainage. The mean values for F.V.C. and $F.E.V._1$ showed surprisingly small reductions for the group as a whole, although individual patients had larger reductions. However, it must again be pointed out that there was a strong impression that many patients were affected by respiratory symptoms (cough, sputum production, and dyspnoea) to a degree not indicated by the objective tests.

Comparison of work capacities and activity level requires assessment, but it is satisfying that more than half the patients were still in the same job and that one-third considered their level of activity to be unchanged. A few patients were

severely incapacitated, in keeping with the multiplicity of injuries. The most important cause of marked disability was a neurological lesion such as paraplegia or severe brain damage. Respiratory disability, although a cause of deterioration, did not often lead to total incapacity.

If morbidity is to be kept as low as possible it is essential to treat, promptly and efficiently, not only the chest injury but also any associated injuries or physiological derangement.

We wish to acknowledge gratefully the help given by Dr. J. G. Duncan, who studied and reported all the chest radiographs performed in this series.

REFERENCES

Avery, E. E., Mörch, E. T., and Benson, D. W. (1956). Critically crushed chests: a new method of treatment with continuous mechanical hyperventilation to produce alkalotic apnea and internal pneumatic stabilization. *J. thorac. Surg.*, **32**, 291.

Bargh, W., Griffiths, H. W. C., and Slawson, K. B. (1967). Crush injuries of the chest. *Brit. med. J.*, **2**, 131.

Griffiths, H. W. C. (1960). Crush injuries of the chest. *J. roy. Coll. Surg. Edinb.*, **6**, 13.

Lloyd, J. W., Crampton Smith, A., and O'Connor, B. T. (1965). Classification of chest injuries as an aid to treatment. *Brit. med. J.*, **1**, 1518.

Needham, C. D., Rogan, M. C., and McDonald, I. (1954). Normal standards for lung volumes, intrapulmonary gas-mixing, and maximum breathing capacity. *Thorax*, **9**, 313.

Reid, J. M., and Baird, W. L. M. (1965). Crushed chest injury: some physiological disturbances and their correction. *Brit. med. J.*, **1**, 1105.