Long-Term Pulmonary Function after Recovery from Pulmonary Contusion due to Blunt Chest Trauma

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ABSTRACT: **Background:** Blunt chest trauma can cause severe acute pulmonary dysfunction due to hemo/pneumothorax, rib fractures and lung contusion.

Objectives: To study the long-term effects on lung function tests after patients' recovery from severe chest trauma.

Methods: We investigated the outcome and lung function tests in 13 patients with severe blunt chest trauma and lung contusion.

Results: The study group comprised 9 men and 4 women with an average age of 44.6 ± 13 years (median 45 years). Ten had been injured in motor vehicle accidents and 3 had fallen from a height. In addition to lung contusion most of them had fractures of more than three ribs and hemo/pneumothorax. Ten patients were treated with chest drains. Mean intensive care unit stay was 11 days (range 0–90) and mechanical ventilation 19 (0–60) days. Ten patients had other concomitant injuries. Mean forced expiratory volume in the first second was 81.2 ± 15.3%, mean forced vital capacity was 85 ± 13%, residual volume was 143 ± 33.4%, total lung capacity was 101 ± 14% and carbon monoxide diffusion capacity 87 ± 24. Post-exercise oxygen saturation was normal in all patients (97 ± 1.5%), and mean oxygen consumption max/kg was 18 ± 4.3 ml/kg/min (60.2 ± 15%). FEV1 was significantly lower among smokers (71.1 ± 12.2 vs. 89.2 ± 13.6%, $P = 0.017$). There was a non-significant tendency towards lower FEV1 among patients who underwent mechanical ventilation.

Conclusions: Late after severe trauma involving lung contusion, substantial recovery was demonstrated with improved pulmonary function tests. These results encourage maximal intensive care in these patients. Further larger studies are required to investigate different factors affecting prognosis.

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Blunt chest trauma is a frequent injury in developed countries, with motor vehicle accidents being the most common cause. There is an estimated 7% risk of a serious thoracic injury with any motor vehicle accident [1,2]. Falls from a height, work accidents, recreation-related crush injuries, terror attacks and assaults are less common but substantial additional causes of blunt chest trauma [1-4].

Pulmonary contusion should be anticipated in any patient who sustains significant, high energy blunt chest impact [2]. The presence of rib fractures or a flail segment increases the odds for lung contusion, but lung contusion occurs also in about 35% in the absence of bony thoracic injury [2,3]. Despite the substantial data on blunt chest trauma, most concern the acute phase and there is limited and contradictory information about late sequelae and recovery. The aim of this study was to assess long-term lung function tests after severe chest trauma.

PATIENTS AND METHODS

Patients who survived severe blunt chest trauma and lung contusion during the period 2005 to 2006 were located with the help of the Rabin Medical Center computerized database. Thirteen patients who were eligible gave written informed consent to participate in the study. All patients underwent complete pulmonary function tests and cardiopulmonary exercise tests. The study was approved by the institutional review board.

PULMONARY FUNCTION TESTS

Pulmonary function tests were performed according to guidelines of the American Thoracic Society and the European Respiratory Society [5,6] with the Medical Graphics Pulmonary Function System (1070-series 2, St. Paul, MN, USA). Lung volumes were obtained by body plethysmography (model 1085, Medical Graphics). Maximal voluntary ventilation was assessed by asking the patient to breathe as rapidly and as deeply as possible for 12 seconds, and the result was multiplied by 5. Carbon monoxide diffusion capacity was measured with a gas mixture containing air, 10% helium, and

FEV1 = forced expiratory volume in the first second

0.3% carbon monoxide. Each measurement was adjusted to standard temperature and pressure. The predicted values of the parameters were obtained from the regression equations of the European Community for Coal and Steel.

CARDIOPULMONARY EXERCISE TEST

The cardiopulmonary exercise test was performed according to the ATS statement [7]. The test was done between 8:30 a.m. and 12:00 noon. Patients were encouraged to take their medications as usual. An incremental exercise test was administered first, according to the protocol of Wasserman et al. [8]. On arrival at the exercise laboratory, patients were connected to a 12-lead electrocardiogram (Cardiofax, Nihon Kohden, Tokyo, Japan) with a single-lead (v5) monitor (VC-22, Nihon Kohden). Oxygen saturation (SaO_2) was measured by pulse oximetry (Nellcor NPB-190, Pleasanton, CA., USA), and blood pressure with a sphygmomanometer. The patient was then positioned on an electrically braked cycle ergometer (Ergoline 800, Bitz, Germany). After a 2 minute rest (arms at sides), they were asked to perform unloaded pedaling for 2 minutes at a rate of 60 rpm. The load was then progressively increased by 15 watts/min (ramp protocol). The duration of the test was symptom limited; the endpoint was defined as the point at which the patient could not maintain a pedaling rate of more than 40 rpm.

Cardiopulmonary data were collected and analyzed with an exercise metabolic unit (CPX, Medical Graphics). Heart rate, minute ventilation, tidal volume, oxygen consumption, carbon dioxide production, respiratory rate, total ventilation, oxygen pulse, and oxygen saturation were recorded and calculated over 30 second intervals using standard formulas. Blood pressure was measured with a sphygmomanometer at rest and every 2 minutes until peak exercise. The dyspnea index (VE/MVV), expressed in percent, was calculated manually.

ATS = American Thoracic Society

STATISTICAL ANALYSIS

Variables are shown as means ± standard deviations. One tailed *t*-test equal variances not assumed was used to analyze differences between patients who smoked and non-smokers, and mechanically ventilated patients versus non-ventilated patients. A P value < 0.05 was considered statistically significant. Analyses were carried out using the use SPSS version 12.0.1 for Windows.

RESULTS

The study group consisted of 9 men and 4 women, with an average age of 44.6 ± 13 years. Six patients smoked and seven were non-smokers. Ten had been injured in motor vehicle accidents and 3 had fallen from a height [Table 1].

In addition to lung contusion, most of them had fractures of more than three ribs and hemo/pneumothorax. Ten patients were treated with chest drains. The mean stay in the intensive care unit was 11 days (range 0–90), and mechanical

VE = minute ventilation
MVV = maximal voluntary ventilation

Table 1. Clinical characteristics of the study population (n=13)

Patient no.	Gender/ Age	Cause	Smoking	Time elapsed (mos)	Lung contusion	> 3 ribs	Sternum fracture	Hemo/ pneumothorax	Mechanical ventilation	ICU days	Hospital stay
1	M/23	Road accident	No	18	Yes	Yes		Yes	Yes	3	15
2	F/32	Road accident	No	24	Yes	Yes		Yes	No	1	9
3	F/40	Road accident	No	12	Yes	Yes		Yes	No	0	5
4	M/42	Road accident	Yes	12	Yes	Yes	Yes		No	0	12
5	M/35	Fall from height	No	12	Yes	No	Yes	Yes	Yes	16	30
6	M/25	Road accident	Yes	48	Yes	Yes		Yes	Yes	5	16
7	F/45	Road accident	Yes	12	Yes	Yes		Yes	Yes	20	36
8	M/49	Road accident	Yes	12	Yes	No	Yes		No	0	1
9	F/60	Fall from height	No	12	Yes	Yes		Yes	No	1	7
10	M/50	Road accident	Yes	10	Yes	Yes		Yes	Yes	90	97
11	M/66	Road accident	Yes	12	Yes	Yes		Yes	No	0	26
12	M/52	Fall from height	No	15	Yes	Yes			No	0	20
13	M/61	Road accident	No	20	Yes	Yes		yes	Yes	7	25

ICU = Intensive care unit

ventilation 19 days (range 0–60). Ten patients had concomitant injuries including cardiac contusion (n=2), fracture of the clavicle (n=2) and fracture of the scapula (n=1).

After 12–48 months pulmonary function tests and cardiopulmonary exercise test were performed. Mean FEV1 was 81.2 ± 15.3% of normal, mean forced vital capacity 85 ± 13%, total lung capacity 101 ± 14%, residual volume 143 ± 33.4%, and carbon monoxide diffusion capacity 87 ± 24%. Post-exercise oxygen saturation was normal in all patients (97 ± 1.5%), mean peak oxygen consumption was 18 ± 4.3 ml/kg/min (60.2 ± 15%) [Table 2]. FEV1 was significantly lower among smokers (71.1 ± 12.2% vs. 89.2 ± 13.6%, $P = 0.017$), but there was only a non-significant tendency towards lower FEV1 among patients who underwent mechanical ventilation (86.7 ± 15.6% vs.74.6 ± 13.4%, $P = 0.08$).

DISCUSSION

Our results show that most survivors of severe chest trauma have a good chance of recovery with near normal pulmonary function tests and fair exercise capacity. These results are consistent with a previous study by Livingston and colleagues [9], showing improvement in pulmonary function tests after 6 to 18 months. In a study of blast injury survivors, there was resolution of lung contusion within 5 months, near normal pulmonary function tests and good exercise capacity after one year [10]. Landercasper et al. [11] demonstrated long-term disability after flail chest injury with abnormal spirometry tests in 57% of long-term survivors. In that study the magnitude of the abnormalities and the number of smokers were not mentioned, and lung diffusion capacity was normal in most patients. According to the discussion that followed, more than half the patients were smokers and they had a worse performance. Kishikawa and team [12] found no difference in FEV1 and vital capacity among patients with chest trauma with or without pulmonary contusion (both in the normal range), but they did find lower functional residual capacity and lower oxygen saturation in the supine position among patients with pulmonary contusion. Most of their patients were smokers, although this finding was similar among patients with and without pulmonary contusion. In our study, smokers had worse spirometry results, which could be related to previous disease or incomplete recovery after contusion. Svennevig et al. [13] found that severe injury to the chest frequently causes respiratory symptoms. However, objective tests were only moderately reduced when compared with normal values.

One limitation of the present work is that some of the patients who agreed to participate in this study were deconditioned according to the results of the cardiopulmonary exercise test (relatively low anaerobic threshold and high reserves). This may have contributed to lower peak oxygen consumption, although most values are still acceptable and enable good performance.

In conclusion, in a cohort of 13 survivors of severe thoracic trauma with lung contusion, substantial physiological recovery is demonstrated with good pulmonary function tests. These results encourage maximal intensive care in these patients. Further larger studies are required to investigate different factors affecting prognosis.

Table 2. Pulmonary function tests of the study population (n=13)

Patient no.	FEV1 (%)	FVC (%)	FEV_1/ FVC %	RV (%)	ERV (%)	TLC (%)	DLCO (%)
1	79	81	83	175	100	96	86
2	88	89	85	107	122	95	63
3	92	94	85	129	85	104	111
4	69	80	71	201	38	115	119
5	89	95	78	133	110	106	97
6	60	73	71	195	37	99	79
7	61	78	67	123	112	89	65
8	92	100	75	134	76	106	101
9	115	109	87	146	112	136	108
10	69	61	91	152	135	86	31
11	80	87	71	106	39	89	93
12	71	67	85	166	42	102	92
13	90	92	78	97	130	88	80
Mean ± SD	81±15	85 ±13	79± 8	143±33	87±37	101±14	87±24

DLCO = diffusion capacity of carbonic anhydrase, ERV = expiratory reserve volume, FEV1 = forced expiratory volume in one second, FVC = mean forced vital capacity, TLC = total lung capacity, RV = residual volume.

Table 3. Cardiopulmonary exercise parameters of the study population (n=13)

Patient no.	VO_2max (%)	VO_2max (ml/min/kg)	AT (%)	SaO_2 (%)	Post-test SaO_2 (%)	O_2 pulse	Breathing reserve (L)
1	60	23.6	32	99	99	12	67.6
2	51.8	15.1	27	99	99	7	60
3	68	19.2	31	98	98	8	38
4	45.8	16	28	98	99	9	55
5	32	12.3	22	98	98	8	84
6	67.4	26.9	37	98	98	12	16.4
7	51.4	13.2	27	99	98	6	30.7
8	83.7	19.9	*	97	97	13	73
9	71.6	16.8	50	98	97	5	40.8
10	48.3	15.21	22	98	97	7	22.8
11	84.4	17.8	70	96	95	18	65
12	50.2	12.9	*	98	97	12	28
13	72.4	20.3	43	98	98	11	38
Mean±SD	60±15	18±4	58±10	98±0.5	97±2	10±4	48±21

VO_2max = maximal oxygen consumption, AT = anaerobic threshold, SaO^2 = oxygen saturation

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References

1. Newman RJ, Jones IS. A prospective study of 413 consecutive car occupants with chest injuries. *J Trauma* 1984; 24(2): 129-35.
2. Wanek S, Mayberry JC. Blunt thoracic trauma: flail chest, pulmonary contusion, and blast injury. *Crit Care Clin* 2004; 20(1): 71-81.
3. Shorr RM, Crittenden M, Indeck M, Hartunian SL, Rodriguez A. Blunt thoracic trauma. Analysis of 515 patients. *Ann Surg* 1987; 206(2): 200-5.
4. LoCicero J 3rd, Mattox KL. Epidemiology of chest trauma. *Surg Clin North Am* 1989; 69(1): 15-19.
5. Miller MR, Crapo R, Hankinson J, Brusasco V, Burgos F, et al. General considerations for lung function testing. *Eur Respir J* 2005; 26: 153-61.
6. Miller MR, Hankinson J, Brusasco V, et al. Standardisation of spirometry. *Eur Respir J* 2005; 26(2): 319-38.
7. ATS/ACCP statement on cardiopulmonary exercise testing. *Am J Respir Crit Care Med* 2003; 167: 211-77.
8. Wasserman K. Anaerobic threshold and cardiovascular function. *Mondaldi Arch Chest Dis* 2002; 58: 1-5.
9. Livingston DH, Richardson JD. Pulmonary disability after severe blunt chest trauma. *J Trauma* 1990; 30(5): 562-6; discussion 566-7.
10. Hirshberg B, Oppenheim-Eden A, Pizov R, et al. Recovery from blast lung injury: one-year follow-up. *Chest* 1999; 116(6): 1683-8.
11. Landercasper J, Cogbill TH, Lindesmith LA. Long-term disability after flail chest injury. *J Trauma* 1984; 24(5): 410-13; discussion 413-14.
12. Kishikawa M, Yoshioka T, Shimazu T, et al. Pulmonary contusion causes long-term respiratory dysfunction with decreased functional residual capacity. *J Trauma* 1991; 31(9): 1203-8; discussion 1208-10.
13. Svennevig JL, Vaage J, Westheim A, Hafsahl G, Refsum HE. Late sequelae of lung contusion. *Injury* 1989; 20(5): 253-6.