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Respirator-Induced Pneumothorax and Subcutaneous Emphysema: Experimental Overinflation of Cadaver Lungs

Resuscitation maneuvers (including positive-pressure ventilation and closed chest cardiac massage) may be complicated by the formation of pneumothorax (PT), mediastinal emphysema (ME), or subcutaneous emphysema (SCE) [*1–4*]. Proposed mechanisms of these untoward effects have included overinflation [*2–5*], violent inspiratory efforts during inflation [*4*], pulmonary parenchymal laceration due to rib fractures [*1*], and perforation of air passages during intracardiac injections [*1*].

Fresh, intact cadavers were intubated, and their lungs were overinflated to determine if overinflation alone would lead to the formation of PT, SCE, or both.

Materials and Methods

Twenty adult cadavers were selected using the following criteria.

1. The postmortem interval was less than three hours such that no rigor mortis was present to inhibit tracheal intubation.
2. There were no lesions of the chest, neck, or abdomen. Cadavers with tracheostomies, chest tubes, or recent chest or abdominal incisions were excluded.
3. The baseline postmortem chest X-ray revealed the following: (a) normally inflated lungs, (b) no evidence of PT, ME, or SCE, and (c) no parenchymal or pleural lesions.

Each cadaver was placed supine on an X-ray table (Fig. 1). A cuffed endotracheal tube was inserted using a laryngoscope. The cuff was inflated maximally to avoid leakage. Baseline AP chest and abdomen X-rays were taken. The height, weight, chest circumference, and abdomen circumference were recorded.

Dynamic Ventilation with Intermittent High Pressure

Fourteen intubated cadavers were attached to an Emerson volume respirator. The cadavers were ventilated at each of various volumes for 2 min. Peak pressures (as indicated on the gage attached to the machine) had a duration of 0.2 s. The volume was incremented by 100 to 400 cm^3 for successive periods of ventilation. At the end of each 2-min ventilation period the machine was shut off in the end-expiratory cycle (zero pressure) and a chest X-ray was taken.

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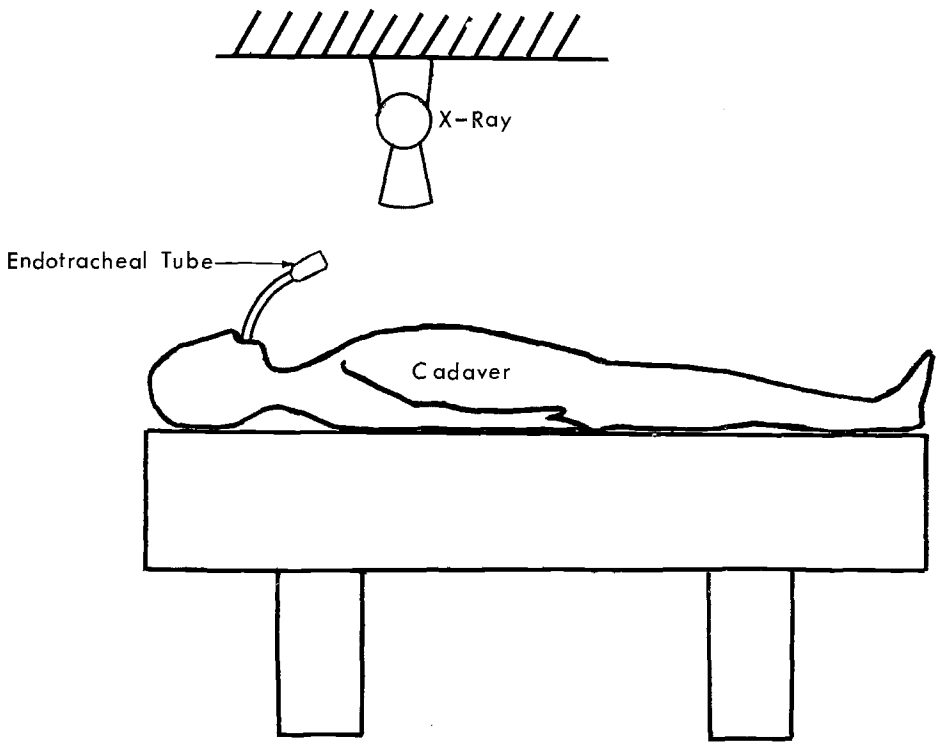

FIG. 1—*Experimental setting.*

The maximum volume deliverable by the Emerson is 2200 cm^3. If upon reaching the 2200-cm^3 mark pressures were not high enough, the ventilation cycle was speeded up until complete expiration was not allowed. In this manner high pressures were obtainable in cadavers with large vital capacities, who would otherwise have easily tolerated the tidal volume of 2200 cm^3. If SCE or PT did not occur, vigorous external cardiac massage was performed for 2 min during maximal ventilation. Also, intracardiac injections were done during maximal ventilation on three of the cadavers. Ventilation was discontinued if SCE appeared.

Postmortem examination was performed in the routine fashion with additional procedures and emphasis as follows.

The cadaver was palpated for the crepitation of SCE. The body was inspected for asymmetry of the thorax, widening of the intercostal spaces, and protuberance of the abdomen. The chest and abdomen were percussed. A needle under water seal was inserted into each pleural cavity in search of air under pressure. Appearances of the subcutaneous tissues, mediastinum, and pleural cavities were noted. The pleural cavities were examined for fluid and adhesions, and the apparent extent of inflation of the lungs was observed. The visceral pleurae were checked for blebs, scars, and tears. The cut surfaces of the lungs were examined for the presence of emphysematous changes. The neck organs were inspected for evidence of "traumatic intubation" and for the presence of air in the tracheal submucosa.

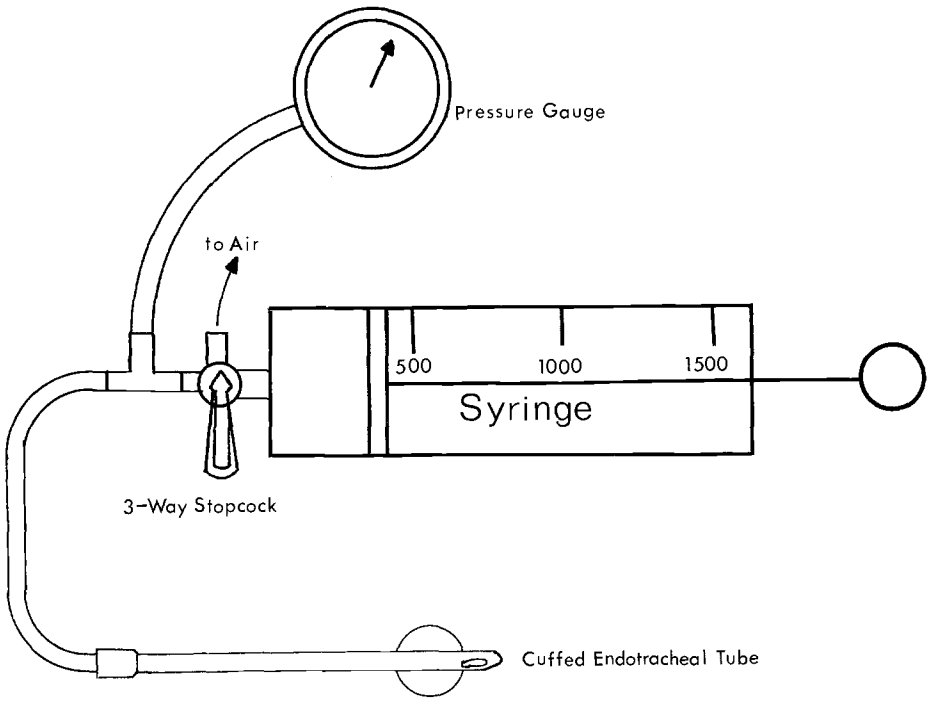

FIG. 2—*Static inflation apparatus.*

Static Stepwise Inflation with Sustained High Pressures

Six cadavers were connected to a 1500-cm^3 syringe equipped with stopcock and pressure gage (Fig. 2). Baseline X-rays and measurements were made, and inflation was done in increments of 100 to 200 cm^3. After each inflation increment the stopcock was closed and the pressure was recorded. Chest X-rays were taken after each 600 cm^3 (or after any unusual pressure change) with the stopcock closed (pressure sustained). Inflation was continued until SCE or PT occurred. If SCE occurred the stopcock was closed, a chest X-ray was taken, and the pressure was immediately released to avoid deforming the cadaver.

Pleural puncture with a needle under water seal and autopsy examinations were done as in the machine-ventilated group.

Results

Dynamic Ventilation with Intermittent High Pressures

Pneumothorax was produced in only three of the 14 cadavers. SCE occurred in one of these at a pressure 20 cm H_2O greater than the pressure at which the PT occurred. These results are summarized in Table 1. The X-ray findings in the PT cases were identical to those described clinically [6], and included depression of the diaphragm, collapse of the lung, and contralateral mediastinal shift. An example of these changes can be seen in the chest X-rays of one cadaver (Fig. 3).

All pneumothoraces were left-sided. Adhesions were absent on the sides involved by PT and they were present on the opposite sides. The contralateral adhesions varied from

TABLE 1—*Results of dynamic ventilation with intermittent high pressures.*

Case Number	Age	Sex	Height, cm	Weight, lb	Vital Capacity, ml	Max Pressure Attained, cm H_2O	Result	Remarks on Adhesions
424Y70	80	F	148	77	2003	140	Left PT at 120, SCE at 140	Multiple apical fibrous adhesions, bilaterally
444Y70	79	F	169	145	2330	100	Negative	Moderate diaphragmatic fibrous adhesions, right
462Y70	52	M	175	187	3830	140	Negative	Extensive fibrous adhesions, diffuse, left
472A70	52	M	173	205	3780	160	Negative	No adhesions
485A70	83	M	166	105	3040	140	Negative	Multiple diffuse fibrous adhesions, left
526Y70	79	M	175	140	3330	140	Negative	Diffuse fibrous adhesions, right
567Y70	76	M	176	180	3400	145	Negative	No adhesions
664A70	64	M	165	163	3390	140	Left PT at 140	No adhesions
900Y71	59	F	167	144	2640	90	Negative	No adhesions
971Y71	85	M	185	192	3420	90	Left PT at 90	No adhesions; moderate panlobular emphysema
984Y71	59	M	164	160	3450	105	Negative	No adhesions
987Y71	69	F	166	180	2460	100	Negative	No adhesions
988Y71	24	M	176	109	4330	120	Negative	Extensive diffuse tumor adhesions
993Y71	71	F	171	147	2510	70	Negative	No adhesions; moderate panlobular emphysema

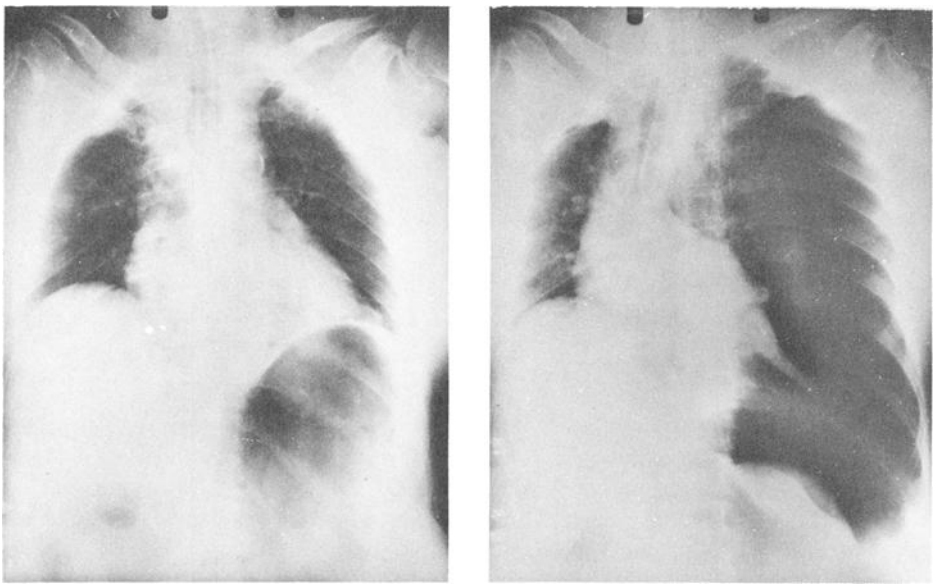

FIG. 3—*The preinflation X-ray is seen on the left. The postinflation X-ray on the right shows a left tension pneumothorax with depression of the left diaphragm and shift of mediastinal structures to the right.*

minimal diaphragmatic fibrous in one, to moderate diffuse fibrous in another. The pressures at which pneumothorax occurred were 90, 100, and 140 cm H_2O respectively. In the nonpneumothorax cases, pressures as high as 150 were tolerated despite vigorous external cardiac massage and intracardiac injection. Rib fractures were not produced despite attempts to do so. Tension pneumothorax was evident in all three PT cadavers when the water-sealed needle was inserted into the pleural cavity on the involved side.

None of the 14 cadavers had subcutaneous crepitation. The neck organs on all cadavers were normal. Four of the eleven nonpneumothorax cadavers had pleural adhesions varying from minimal posterior fibrous on one side, to extensive diffuse fibrous tumor adhesions bilaterally (in a case of metastatic synovial sarcoma). The remaining seven of the eleven nonpneumothorax cadavers had essentially normal lungs and pleurae.

Static Stepwise Inflation with Sustained High Pressures[2]

SCE occurred in all six cadavers; PT occurred in none. The sequence of events was generally the same in all six. As a volume approximately 1 to 1½ times the calculated vital capacity [7] was reached, a slight drop-off or, more often, a plateau of pressure occurred. Then there was a fairly sharp pressure increase for 300 cm^3 of inflation, followed by a precipitous pressure fall-off. SCE occurred during this fall-off. The SCE was manifested by obvious distension of the tissues around the base of the neck and the supraclavicular areas, with palpable crepitation extending inferiorly from the chin to the area just below the clavicles. X-ray examination confirmed the presence of SCE in all six cases. None had X-ray evidence of ME or PT. No air was found when the pleural cavities were needled or later opened. The anterior mediastinal tissues and subcutaneous fat had the bubbly, crepitant appearance of SCE; however, this apparent pneumomediastinum had not been

[2] Results are summarized in Table 2.

TABLE 2—*Results of static inflation with sustained high pressure.*

Case Number	Age	Sex	Height, cm	Weight, lb	Vital Capacity, ml	Pressure at Occurrence of SCE, cm H_2O	Volume at Occurrence of SCE, ml	Remarks on Adhesions
1068Y71	79	M	183	110	3500	104	3000	Adhesions unknown; no chest permission
1066Y71	66	M	168	115	3420	8	1500	No explanation for large compliance, early SCE
1071Y71	30	F	145	120	2720	130	4000	Bilateral radiation fibrosis; scant basilar adhesions, bilateral
1097A71	72	F	167	144	2420	102	6000	Diffuse fibrous adhesions, right; moderate medial fibrous adhesions, left
1135A71	54	M	160	148	3450	80	6600	No adhesions
1140Y71	64	F	167	110	2550	88	3200	No adhesions

evident on X-ray. The pleurae were intact and the lungs were fully inflated. One cadaver had changes of panlobular emphysema, moderate. All neck organs were normal. No lesion due to the endotracheal tube could be found. The laryngeal and tracheal mucosae were intact and free of submucosal air. One cadaver had moderate, diffuse, bilateral fibrous adhesions.

Discussion

These experiments were admittedly crude; however, the results may have significance in that they suggest different mechanisms for respirator-induced PT, versus respirator-induced SCE. Static overinflation of cadaver lungs with *sustained* high pressures always results in SCE. Dynamic overinflation of cadaver lungs with *intermittent* high pressures, on the other hand, occasionally results in pneumothorax. Fibrous pleural adhesions seem to protect against pneumothorax. In the following discussion I will compare the pathogenesis of pneumothorax with that of subcutaneous emphysema, and correlate these comparisons with clinical reports, as well as these cadaver findings.

Alveolar rupture with extravasation of air occurs in two principal ways. First, peripheral alveoli can rupture into the pleura and then into the pleural cavity leading to PT [*8*]. Secondly, alveoli adjacent to vessels can rupture into the perivascular spaces, allowing escape of air along parenchymal vasculature [*4*]. It has been shown in cats [*7*] that overinflation via a cannulated bronchus causes air to dissect along the perivascular spaces to the hilus and then into the mediastinum. These two types of alveolar rupture, peripheral into the pleural cavity, and juxtavascular into the perivascular spaces, result in two patterns of air extravasation. Peripheral rupture results in immediate PT, whereas juxtavascular rupture leads to ME and SCE, which occasionally progresses to PT if the pressure is great enough. These two patterns are seen clinically as follows.

Clinically, rupture of peripheral alveoli with resulting PT is seen in many diseases of the pleura and pulmonary parenchyma, as well as occurring spontaneously [*9*]. Reports of respirator complications favor the occurrence of pneumothorax over that of SCE. Some of these reports deal with patients having parenchymal or pleural disease; however, several authors report pneumothorax complicating ventilation of apparently normal respiratory systems. Rupture of alveoli results when the alveoli are overdistended. Overdistension of a peripheral alveolus would require an elevated transalveolar pressure [*4*]. The transalveolar pressure is the difference between the intra-alveolar pressure and the intrapleural pressure. If a patient makes a violent inspiratory effort during pressure ventilation, the negative intrapleural pressure generated results in a markedly elevated transalveolar pressure. Conversely, a patient who attempts to exhale during pressure inflation ("fighting the machine") has a positive intrapleural pressure and a very low transalveolar pressure. The chest wall and diaphragm of a cadaver act as supporting structures only. They could never exert a negative intrapleural pressure. It is thus understandable why PT did not occur in cadavers during static inflations with sustained high pressures. It is not clear why, in the dynamic ventilation with intermittent high pressures, pneumothoraces were occasionally produced.

Returning to clinical reports of respirator-associated pneumothorax and SCE, not all reports favor the occurrence of PT over SCE. A recent paper describes patients who had been given continuous positive-pressure ventilation for intractable respiratory failure [*10*]. The mean end-expiratory pressure was 13 cm H_2O. Four of these patients developed complications thought to be due directly to the pressure ventilation. Mild to extensive SCE occurred in three of the four (with ME in one of these), and tension PT occurred in

the fourth. Thus, SCE was favored over PT in this series of respirator complications in which sustained end-expiratory pressure was utilized. Their clinical use of elevated end-expiratory pressure mimicked the effect I achieved by static inflation with sustained high pressure. The clinical appearance of SCE rather than PT correlated with my findings. The question now arises as to the reliability of postmortem clinical correlations.

Hartung has measured respiratory function parameters on cadavers [*11*]. He states that compliance curves obtained in cadavers are essentially the same as those for unconscious or drug-relaxed living patients. Compliances obtained on healthy volunteers are somewhat higher than those of cadavers, and this is probably due to very slight voluntary inspiratory effort during inflation.

Summary and Conclusions

Postmortem ventilation studies were performed on fresh, intact cadavers to evaluate the effects of overinflation. Sustained pressures beyond the "physiologic" range resulted in subcutaneous emphysema (SCE) in all six cadavers. Intermittent high pressures, on the other hand, caused pneumothorax (PT) in three of thirteen cadavers, and SCE in none. PT and SCE seem to have different pathogeneses. PT probably results from intermittent excessive ventilation pressure, or violent inspiration attempts in the living patient, with resulting rupture of peripheral alveoli. SCE, on the other hand, occurs when sustained high inflation pressure causes air to enter perivascular spaces and dissect towards the hilus and then into the mediastinum and subcutaneous tissues. These findings correlate with clinical reports in that intermittent positive pressure therapy is sometimes complicated by pneumothorax, whereas continuous positive pressure therapy has resulted in the complication of SCE.

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