CIRCULATORY CHANGES DUE TO OPEN PNEUMOTHORAX IN SURGICAL PATIENTS

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Considerable information has appeared in the literature describing the effects upon the circulation of a closed pneumothorax in animals and unanesthetized man.[1-7] These studies revealed varying degrees of reduction of cardiac output after graded amounts of air were introduced into the thorax. Data concerning the acute circulatory changes caused by opening the pleura during thoracotomy in humans has not been published. A study[8] of the effects upon the circulation of thoracotomy in dogs was complicated by spontaneous respiration having been permitted when the chest wall was intact and then intermittent positive pressure breathing having been used after the pleura was opened. The precipitous fall in cardiac output recorded under these conditions could be attributed to the changes in pulmonary ventilation.

This study was undertaken to evaluate in anesthetized man the immediate effects upon the circulation of an open pneumothorax. Position, anesthetic agent, depth of anesthesia, alveolar ventilation, mean airway pressure, arterial oxygen saturation, arterial P_{CO_2} and surgical manipulation were controlled as rigidly as possible. The only variable was a wide incision through the parietal pleura. The cardiac output and related measurements were determined with the patient in the lateral position just before opening the pleura and immediately after the pleura was opened.

Method

Ten patients without cardiac disease were studied. Each patient served as his own control. Pentobarbital (50–100 mg.) and scopolamine (0.3–0.4 mg.) were administered forty-five minutes prior to the study; the dosages varied according to age, physical status and body weight. Cannulas were placed into an artery and vein. Endotracheal intubation was effected following induction of anesthesia with thiopental and succinylcholine. *d*-Tubocurare was administered intravenously in doses sufficient to maintain apnea. Anesthesia was maintained with cyclopropane at an EEG level 3 and a blood cyclopropane level of 8–15 mg. per cent. Pulmonary ventilation was controlled by means of a volume-limited, pressure-variable ventilator.[9] The appropriate tidal volume and respiration rate for each patient were chosen from the Radford nomogram.[10, 11] The arterial and intratracheal pressures were recorded continuously by Statham strain gauges on a Sanborn model 150 oscillograph along with the electroencephalogram and electrocardiogram. The mean arterial blood pressure was obtained by an electronic integrator and the mean airway pressure by planimetry of each respiratory cycle occurring during the cardiac output determination. The arterial blood *p*H, P_{CO_2} and cyclopropane concentra-

TABLE 1

Physical Characteristics of the Subjects

Experiment	Sex	Age (years)	B.S.A. ($m.^2$)	Diagnosis	Position (Side Up)
1	F	58	1.61	Bronchiectasis	left
2	F	66	1.70	Hiatus hernia	left
3	M	54	1.96	Ca of lung (rt.)	right
4	M	66	1.79	Ca of lung (lt.)	left
5	M	55	1.83	Ca of lung (rt.)	right
6	F	67	1.47	Bronchiectasis	right
7	M	42	1.86	Ca of lung (rt.)	right
8	F	43	1.45	Acholia of cardia	left
9	F	50	1.82	Ca of lung (lt.) (coin lesion)	left
10	M	46	2.21	Ca of lung (lt.)	left

B. S. A. = Body surface area.

Presented at the Annual Meeting of the American Society of Anesthesiologists, Inc., Miami Beach, Florida, October 8, 1959, and accepted for publication November 19, 1959. The authors are in the Departments of Anesthesiology and Surgery, Tufts University School of Medicine and New England Center Hospital, Boston, Massachusetts.

TABLE 2

HEMODYNAMIC DATA BEFORE AND AFTER OPEN PNEUMOTHORAX IN TEN SURGICAL PATIENTS

Exp.	C. I. (l. minute m.^{2})		S. V. I. (cc. m.^{2})		H. R. (beats minutes)		T. P. R. $\left(\frac{\text{dynes}}{\text{sec.-cm}^{-5}}\right)$		P_{mA} (mm. Hg)		M. C. T. (seconds)		I. B. V. I. (l. m.^{2})		L. V. W. $\left(\frac{\text{Kg-meters}}{\text{minutes}}\right)$	
	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
1	3.63	1.47	49	23	74	66	1,640	4,130	120	123	19.2	26.2	1.16	0.64	9.5	3.9
2	3.66	2.54	52	38	70	68	1,090	1,660	85	90	28.7	27.2	1.76	1.15	7.1	5.2
3	2.86	2.27	35	23	82	83	1,790	2,340	125	130	17.6	19.2	0.84	0.73	9.4	7.8
4	2.19	2.11	37	42	59	50	2,240	2,690	110	127	33.0	35.0	1.20	1.23	5.8	6.5
5	3.16	2.61	53	41	60	64	1,530	1,840	111	153	26.2	21.6	1.39	0.94	8.6	9.9
6	2.05	1.95	40	41	51	47	2,000	2,650	75	95	23.7	28.7	1.19	1.37	3.0	3.7
7	4.58	3.85	64	48	72	80	1,120	1,250	120	112	22.2	19.9	1.69	1.28	13.8	10.8
8	2.81	2.42	39	28	72	88	2,250	2,510	115	110	20.1	20.1	0.94	0.81	6.3	5.2
9	3.71	3.47	62	58	60	60	1,150	1,240	97	98	17.0	19.8	1.05	1.14	8.8	8.4
10	3.93	2.74	58	33	68	84	1,380	1,780	150	135	27.9	26.5	1.83	1.21	17.6	11.0
Mean	3.26	2.54	49.0	37.5	67	69	1,600	2,200	111	117	23.6	24.4	1.31	1.05	9.0	7.2
S.D.	±0.79	±0.70	±10.7	±11.1	±9	±14	+440	±860	±21	±20	±5.3	±5.2	±0.35	±0.25	±4.2	±2.8
S.E.	±0.25	±0.22	±3	±3	±3	±4	±140	±270	±7	±6	±1.6	±1.5	±0.11	±0.08	±1.3	±0.8
t		2.14		2.35		0.41		1.93		0.71		0.37		1.88		1.12
P		<0.05		<0.05		>0.5		=0.05		>0.4		>0.5		=0.05		=0.2

C. I. = cardiac index; S. V. I. = stroke volume index; H. R. = heart rate; T. P. R. = total peripheral resistance; P_{mA} = mean arterial blood pressure; M. C. T. = mean circulation time; I. B. V. I. = intrathoracic blood volume; L. V. W. = left ventricular work.

TABLE 3

ARTERIAL *p*H AND BLOOD GASES: BEFORE AND AFTER OPEN PNEUMOTHORAX IN TEN SURGICAL PATIENTS

Experiment	pH		Total CO_2 m.M. l.		P_{CO_2} (mm. Hg.)		O_2 Saturation (%)		Cyclopropane (mg. %)	
	Before	After	Before	After	Before	After	Before	After	Before	After
1	7.34	7.42	23.0	22.2	42	34	110	103	15	15
2	7.26	7.34	26.0	26.8	56	48	104	98	7	8
3	7.45	7.34	23.9	21.9	34	40	103	98	8	13
4	7.39	7.42	25.0	21.0	42	32	-	-	-	-
5	7.33	7.31	25.2	25.4	47	49	100	102	12	11
6	7.43	7.38	30.0	30.1	44	50	102	103	8	8
7	7.34	7.38	23.4	26.2	42	43	101	94	17	12
8	7.42	7.32	23.5	22.7	36	43	-	-	-	—
9	7.43	7.37	21.7	20.2	38	41	95	100	7	6
10	7.35	7.36	18.4	17.7	33	31	—	-	11	8
Mean	7.37	7.36	24.0	23.4	41	41	100^{+2}	100	11	10
S.E.	±0.02	±0.01	±0.9	±1.0	±2	±2	±2	±1	±1.4	±1.2

tion were maintained as constant as possible to eliminate respiratory acidosis and changing levels of anesthesia from influencing the results.

The patients were turned to either lateral position and prepared for operation. A routine postero-lateral thoracotomy incision was performed, in the fifth interspace and the pleura exposed. At this point surgical manipulation was stopped and after a ten-minute interval for stabilization the cardiac output was obtained by means of the dye dilution technique [12, 13, 14] using a Colson densitometer.[15] Samples of arterial blood were drawn for determination of *p*H, O_2 content, O_2 capacity, total CO_2 and cyclopropane by methods previously described.[16, 17] The pleura was then widely incised without further manipulation and after another ten-minute interval for stabilization, the cardiac output was again measured and blood samples obtained. The blood volume was maintained by replacing measured blood loss as it occurred by blood transfusion. The mean circulation time, intrathoracic blood volume, total peripheral resistance and left ventricular work were calculated according to the conventional formulas.[13, 14]

RESULTS

Pertinent data are summarized in tables 1, 2, 3 and 4. Alveolar ventilation was maintained within normal levels in most instances. Before the pleura was opened the mean arterial *p*H was 7.37 ± 0.02 (range: 7.26 to 7.45), the mean arterial P_{CO_2} was 41 ± 2 mm. Hg (range: 33 to 56 mm. Hg) and the mean arterial O_2 saturation was 100^{+2} ± 2 per cent (range: 95 to 100^{+10} per cent). After the pleura was opened the mean arterial *p*H was 7.36 ± 0.01 (range: 7.31 to 7.42), the mean arterial P_{CO_2} was 41 ± 2 mm. Hg (range: 31 to 49 mm. Hg) and the mean arterial O_2

TABLE 4

MEAN AIRWAY PRESSURE BEFORE AND AFTER OPEN PNEUMOTHORAX IN TEN SURGICAL PATIENTS

Experiment	Before Pneumothorax (cm. H_2O)	After Pneumothorax (cm. H_2O)	Δ (cm. H_2O)
1	1.2	1.3	+0.1
2	2.2	1.9	−0.3
3	5.6	4.3	−1.3
4	2.6	2.8	+0.2
5	3.1	2.3	−0.8
6	-	-	-
7	4.7	2.7	−2.0
8	3.2	2.4	−0.8
9	3.6	3.5	−0.1
10	4.1	3.8	−0.3
Mean ± S.E.	3.37 ± 0.44	2.78 ± 0.32	
t	= 1.083		
P	= 0.3		

saturation was 100 ± 1 per cent (range: 94 to 100[13] per cent).

In one experiment (2) the arterial P_{CO_2} was 56 mm. Hg before opening the pleura and in two other experiments (5 and 6) the P_{CO_2} was 49 and 50 mm. Hg, respectively, after opening the pleura. There was no statistically significant difference in the arterial *p*H, P_{CO_2} and O_2 saturation before and after the pleura was opened (table 3). The mean airway pressure before and after the pleura was opened was not significantly changed $P = 0.3$ (table 4). The mean per cent hemodynamic changes due to open pneumothorax are charted in figure 1, including twice the standard error of the mean per cent change referring to the prepneumothorax values as zero.

Cardiac Output. After the pleura was opened the cardiac output ranged from − 16 to − 59 per cent in five of the ten subjects and from − 4 to − 14 per cent in the remaining five patients. The average change was − 22 ± 5 per cent ($P < 0.05$), (table 2). A scattergram of the absolute values of the cardiac index before and after opening the pleura shows that the reduction is greater in those instances where the cardiac index ranged from 3.5 to 5.0 l. minute⁻¹ m.⁻² before the pleura was opened

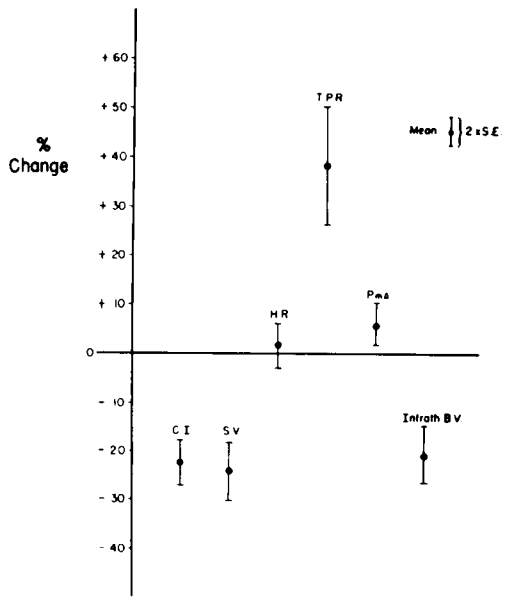

FIG. 1. Hemodynamic changes (per cent) in open pneumothorax (prepneumothorax values as zero).

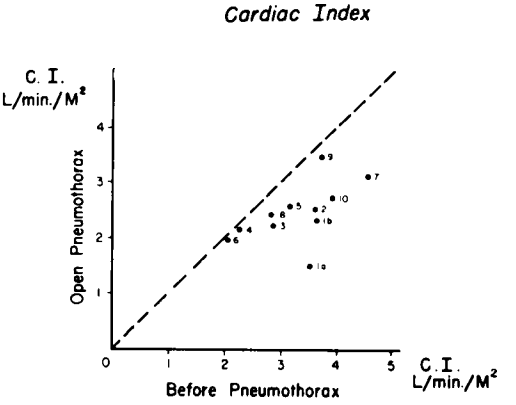

FIG. 2. Cardiac indices before and after open pneumothorax.

(figure 2). When the cardiac index during the control state was below 3.5 l./minute/m.,² the reduction was less.

Stroke Volume. The average stroke volume of the group was reduced − 23 ± 4 per cent ($P < 0.05$) (table 2). The decrease of the stroke volume ranged from − 6 to − 53 per cent in eight of the ten subjects. In one patient there was no change and in one other there was an increase of 13 per cent.

Heart Rate. The heart rate was not significantly changed after the pleura was opened. The average change was + 7 ± 4 per cent ($P > 0.5$).

Total Peripheral Resistance. The calculated total peripheral resistance was increased in all instances after producing an open pneumothorax. Seven of the ten subjects had an increase ranging from + 20 to + 150 per cent. In the other three subjects the increase ranged from + 8 to 11 per cent. The average total peripheral resistance of the ten patients was increased 38 ± 12 per cent ($P = 0.05$).

Mean Circulation Time. The mean circulation time was not significantly changed when the pleura was opened (average + 7 ± 5 per cent) ($P > 0.5$).

Intrathoracic Blood Volume. The average intrathoracic blood volume of the ten subjects was reduced 20 ± 6 per cent following an open pneumothorax ($P = 0.05$). In seven of the ten subjects the reduction of the intrathoracic blood volume ranged from − 14 to − 45 per cent and in three patients there was an increase of 2, 8 and 15 per cent respectively.

Left Ventricular Work. In seven subjects the left ventricular work ranged from − 4 to − 59 per cent. This change was primarily due to the reduction in cardiac output. In three subjects the left ventricular work was increased 10, 15 and 20 per cent and was related to the increase of the mean arterial blood pressure. The average reduction was 20 ± 7 per cent and was not statistically significant ($P > 0.2$).

Correlation of the Cardiac Output, Stroke Volume and Intrathoracic Blood Volume. During the open pneumothorax, the correlation coefficient of the decrease in cardiac output and stroke volume was + 0.76, cardiac output and intrathoracic blood volume was + 0.73 and stroke volume and intrathoracic blood volume was + 0.76. These values are significant at the 5 per cent level.

Discussion

The change in cardiac output and total peripheral resistance following an open pneumothorax in anesthetized man was similar in many respects to the reported changes following closed pneumothorax in unanesthetized man.[4, 5, 7] The 22 per cent decrease in cardiac output was due to the lowered stroke volume. The maintenance of the blood pressure was due to the increase in total peripheral resistance.

The probable causes for these changes were a decrease in lung size, an increase in pulmonary vascular resistance and the influence of atmospheric pressure on the mediastinal contents. Earlier investigators had related an increase in pulmonary vascular resistance to a decrease in lung size of the isolated lung.[18-21] Recently, Simmons and Hemingway [1] studied the acute effects of a closed pneumothorax upon the dogs pulmonary circulation by injecting into a pleural cavity a volume of air equal to the functional residual capacity. They observed an increase in pulmonary vascular resistance and believed it was due to a mechanical effect of a decreased lung size. We have observed that the lung retracted and decreased in size without evidence of atelectasis after the pleura was opened during unilateral thoracotomy. This may be due to elimination of negative intrapleural pressure, although a negative intrapleural pressure only occurs during the expiratory phase with intermittent positive pressure breathing. The use of intermittent positive pressure breathing prevented atelectasis, but did not prevent the lung from decreasing in size.

Hilton [6] reported a decrease in cardiac output in goats following induction of a closed pneumothorax. He related this change to the increased intrathoracic pressure, causing a decrease in the effective filling pressure and stroke volume. Ferguson, Shadle and Gregg [22] observed marked differences in the dog between the performance of the heart in open and closed chest preparations. The average values for stroke work index and stroke volume index in the closed chest preparations were more than four times that when the chest was open. However, in their study, the important factor in reducing the stroke volume was the increase in heart rate, three times greater in the animals with open chests than in the intact dogs. Evidence has been presented that the heart decreases in size after the pleura is opened in anesthetized animals [23, 24] and most likely a similar change takes place during an open pneumothorax in man.

The effect of a lowered intrathoracic temperature after thoracotomy on pulmonary vascular resistance and total peripheral resistance is unknown. Reflex pulmonary vasoconstriction initiated by the effect of cold air on receptors in the chest wall has not been described. During these studies the operating room temperature was 70 F.

Other factors considered as having a negligible effect on the changes in cardiac output were stroke volume and total peripheral resistance after the pleura was opened. The influence of the anesthetic agent or variations of depth of anesthesia was excluded because anesthesia was maintained at a constant light level and the control values were obtained during the anesthetized state. The patient's position on the operating room table was not a factor because the control values were obtained in the lateral position. The elimination of a fluctuating negative intrapleural pressure was disregarded also because intermittent positive pressure breathing was used before and after the pleura was opened. The diaphragm and chest cage did not participate in the changes because of complete curarization throughout the entire procedure. The mean

airway pressure constantly below 5 cm. H_2O before and after the pleura was opened, therefore, does not account for these hemodynamic changes. Alveolar ventilation was not influenced by the opening of the pleura. The tidal volume and respiratory frequency of the ventilator were unchanged before and after the pleura was opened and in most instances the arterial *p*H, P_{CO_2} and O_2 saturation remained within normal levels during the open pneumothorax. Theye and Fowler[25] recently have shown that the ventilatory requirements are not influenced specifically by effects of either lateral position or open pneumothorax.

During light cyclopropane anesthesia, the circulatory adaptation mechanisms remained intact.[14] This was evidenced by the increase in the total peripheral resistance and the maintenance of the systemic blood pressure after the pleura was opened.

The opening of the pleura in relatively healthy patients results in a statistically significant fall in cardiac output and stroke volume but compensating mechanisms are present to maintain the blood pressure by increasing the total peripheral resistance. It is conceivable that, with other factors involved (*i.e.*, blood loss, surgical retraction, increased mean airway pressure, inadequate pulmonary ventilation and deep anesthesia) the cardiac output could be further reduced and the regulating mechanisms depressed, causing an acute hypotension.

Summary

The immediate effect on the circulation of opening the pleura was studied in ten patients during cyclopropane anesthesia. The cardiac output was reduced and the total peripheral resistance was increased in each instance. However, the arterial blood pressure and heart rate were unchanged. The reduction of the cardiac output was positively correlated with the decrease in stroke volume and the intrathoracic blood volume. The position of the patient, anesthetic agent, depth of anesthesia, airway pressure and ventilation were controlled, and therefore were excluded as the causes of these circulatory changes. The probable mechanisms were discussed.

Aided by research grants from the National Institutes of Health, Public Health Service, H-1711, and the Massachusetts Heart Association, Inc.

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GERIATRIC ANESTHESIA Any anesthetic agent and any method of administration that is satisfactory in the young may be employed in the old. The principal difference is that the older patient requires smaller doses of sedative, lighter planes of anesthesia, and smaller amounts of muscle relaxants. The numerous psychologic differences in the geriatric patient and the effects of these psychologic changes on bodily functions must be considered. The aging heart possesses less reserve and does not adjust as well to stress. Coronary sclerosis is frequent. Cardiac output is less than in the young. The blood volume is frequently low and may lead to disturbances in both fluid and electrolyte balance and hemorrhage. Anemia is more common in the elderly. A rigid thoracic cage and loss of elasticity of the lung are almost invariably seen in the elderly patient. This is accompanied by a decrease in permeability of the alveolar membranes, a reduction of vital capacity and an increase in the ratio of residual air to total lung capacity. Controlled or assisted respiration is imperative to insure satisfactory ventilation. Due to changes in the kidneys, the elimination of drugs is slower and their effects last longer than in younger patients. Psychologic preparation during the preoperative visit is of the utmost importance in the geriatric patient. Preoperative medication should be given earlier than to young patients since absorption is slower. (*Greisheimer, E. M.: Psychologic and Physiologic Aspects of Geriatric Anesthesia, Geriatrics 14: 634 (Oct.) 1959.*)

STATUS EPILEPTICUS A nine year old boy with status epilepticus was treated with muscle relaxants and artificial respiration. The etiology of the convulsions was presumably brain edema following drainage of two cerebral abscesses. Total curarization was maintained for eight hours. Suxamethonium was used with hyperventilation. The relaxant was then removed and the child kept anesthetized with thiopental. Large doses of anticonvulsants were ineffective, but since the convulsions were not too severe no further attempt was made to stop them. Artificial respiration was withdrawn at about 15 hours. Twenty-four hours after the removal of the endotracheal tube, tracheal edema developed to the point where tracheostomy was needed. The child finally made a complete recovery except for slight dysphasia and a right facial paresis and was discharged in six weeks. (*Evanson, J. M.: Treatment of Status Epilepticus by Muscle Relaxants and Artificial Respiration, Lancet 2: 72 (Aug. 1) 1959.*)