EFFECTS OF SURGICAL PNEUMOTHORAX ON PULMONARY VENTILATION

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THE effects of surgical pneumothorax on pulmonary ventilation and blood flow have been the subject of study for nearly three-quarters of a century. As early as 1896, Sackur[1] reported the open pneumothorax in animals resulted in arterial oxygen saturation as low as 50 per cent. During the past decade, many laboratory studies have been carried out to elucidate the disturbance in gas exchange produced by open pneumothorax.[2-12] However, the quantitative relationship between the degree of lung collapse and respiratory dysfunction remains unknown.

This study attempts to determine: (1) the effect of different degrees of collapse of the operated lung on arterial oxyygen saturation, *p*H and carbon dioxide tension; (2) the extent to which compensation can occur in the nonoperated lung; and (3) the effect of the duration of collapse upon compensatory mechanisms. A corollary to this study, the effects of apnea, suction and hyperventilation on arterial oxygen saturation, has been published.[13]

METHODS

The study was carried out in 27 patients who were good-risk operative candidates: 13 underwent pulmonary resection for tuberculosis, 7 for neoplastic disease, while 7 patients had nonpulmonary intrathoracic operations. Their ages ranged from 21 to 62 years. Preanesthetic medication consisted of optimal doses of pentobarbital, meperidine, and atropine. Induction of anesthesia was carried out as follows: after inhaling oxygen at high flows for three minutes to effect denitrogenation, topical anesthesia of the larynx, trachea and bronchi was effected by the trans-laryngeal technique; oxygen was continued for another five minutes; and then 100 to 200 mg. of thiopental followed by 40 to 60 mg. of succinylcholine were injected intravenously. When maximal relaxation developed, the patient's trachea was intubated with a Carlens endobronchial tube (14 cases) or an endotracheal tube and bronchial blocker (9 cases) or a Bonica endobronchial tube (4 cases).

Anesthesia was maintained with an 8 l./minute flow of a mixture of 75 per cent nitrous oxide–25 per cent oxygen administered with a semiclosed system with carbon dioxide absorption, supplemented by continuous intravenous infusion of thiopental, meperidine and succinylcholine. Ventilation was artificially controlled by means of a ventilator which was set to provide a slightly greater respiratory volume than the predicted values.[14] The respiratory rate was maintained at 14 per minute and tidal volume ranging between 450 and 600 ml. After the initial adjustments the same minute volume of ventilation was maintained throughout the study. Following collapse of the operated lung the positive pressure had to be increased to 20 to 25 cm. of water to maintain a tidal volume similar to the precollapsed value for both lungs.

The operation was carried out with the patient in the lateral position. After the thoracotomy was accomplished and the pleura opened, "complete" collapse of the upper (operated) lung was effected by gentle manipulation and aspiration of air. This collapse was then maintained by eliminating the ipsilateral lumen of the Carlens tube from the circuit, by inflating the bronchial blocker, or by advancing the Bonica tube from the trachea to the contralateral bronchus to effect endobronchial anesthesia. After the appropriate time had elapsed and blood samples taken as described below, the occlusion was eliminated

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TABLE 1

ARTERIAL BLOOD GAS STUDIES DURING INTRATHORACIC OPERATIONS

Values of Arterial Blood	Phases of the Study						
Percentage of Oxygen	*1* Pre-anesthetic	*2* Supine	*3* Lateral Position	*4* Pleura Opened Both Lungs Expanded	*5* Complete Collapse	*6* Partial Collapse	*7* Re-expanded
Saturation of Hemoglobin							
Mean	95.4	99.2*	98.9	99.2	92.1*	93.3*	99.2*
Range	6.0	3.6	3.5	4.2	10.0	7.0	2.4
S.D.	1.63	0.98	0.95	1.14	2.75	1.90	0.65
Carbon Dioxide Tension							
Mean	45.3	39.1†	38.1	36.7	40.9*	39.8*	37.3*
Range	15.7	10.2	10.0	19.5	14.8	12.5	12.8
S.D.	4.26	2.77	2.71	5.29	4.01	3.39	3.47
*p*H							
Mean	7.38	7.42*	7.42	7.42	7.39*	7.40*	7.42*
Range	0.19	0.13	0.09	0.10	0.10	0.10	0.08
S.D.	0.052	0.04	0.024	0.03	0.02	0.03	0.02

* $P < .01$—statistically significant difference.

† $P < .001$—highly statistically significant difference.

and the operated lung was re-expanded to about 65 to 75 per cent of its total volume.

Blood samples were obtained from the brachial artery at the following stages: (1) during preanesthetic period following a 10-minute period during which the patient was undisturbed and 20 minutes after each of the following stages; (2) onset of anesthesia in supine patient; (3) after lateral positioning; (4) after chest was opened; (5) after complete collapse of operated lung; (6) after partial collapse; and (7) after complete expansion. In 9 patients measurements were also made after 45 to 60 minutes of complete collapse. In four instances measurements were also made after the patient's lungs had been ventilated with 100 per cent oxygen administered for 20 minutes during complete collapse of the lung, but after measurements of phase (5) had been made.

The arterial blood samples were analyzed for oxygen saturation, carbon dioxide tension and *p*H by standard techniques [15–18] and the data analyzed by the Student *t*-test for differences of the means.

RESULTS

Data on oxygen saturation, carbon dioxide tension and *p*H of arterial blood for all of the seven phases of the study were obtained on 19 patients. A summary of these data is contained in table 1 and depicted in figure 1. The patients manifested a mild degree of hypoventilation during the preanesthetic period; this was eliminated by hyperventilation effected during induction and maintenance during the second, third and fourth phases. There was no statistically significant difference between these values with the patient supine, with the patient in the lateral recumbent position, or when the pleura was

TABLE 2

COMPARISON OF EFFECTS OF SHORT AND PROLONGED COLLAPSE

Values of Arterial Blood	Phases of the Study			Statistical Analysis
	(*a*) Pre-collapse	(*b*) Brief Collapse	(*c*) Prolonged Collapse	
S_{aO_2}				
Mean	98.9	92.2	92.7	(*a*) vs. (*b*) $P < .01$
Range	3.7	5.0	7.5	(*b*) vs. (*c*) N.S.
S.D.	1.8	2.4	3.5	
P_{aCO_2}				
Mean	34.4	41.4	43.2	(*a*) vs. (*b*) $P < .01$
Range	13.5	8.2	7.4	(*b*) vs. (*c*) N.S.
S.D.	6.5	4.0	3.6	
*p*H				
Mean	7.42	7.38	7.37	(*a*) vs. (*b*) $P < .01$
Range	0.05	0.07	0.05	(*b*) vs. (*c*) N.S.
S.D.	0.02	0.03	0.02	

opened with both lungs fully inflated. In all of these circumstances hyperventilation increased the arterial oxygen saturation above normal and produced a mild respiratory alkalosis.

During complete collapse of the lung (phase 5) the arterial oxygen saturation was below the precollapse values and even below the preanesthetic values, arterial carbon dioxide tension increased slightly and the *p*H decreased slightly. There was a small (but significant) difference between complete collapse (phase 5) and partial collapse (phase 6).

Table 2 and figure 2 contain values of the effects of brief and prolonged total collapse of the operated lung. No significant difference between the two stages was noted.

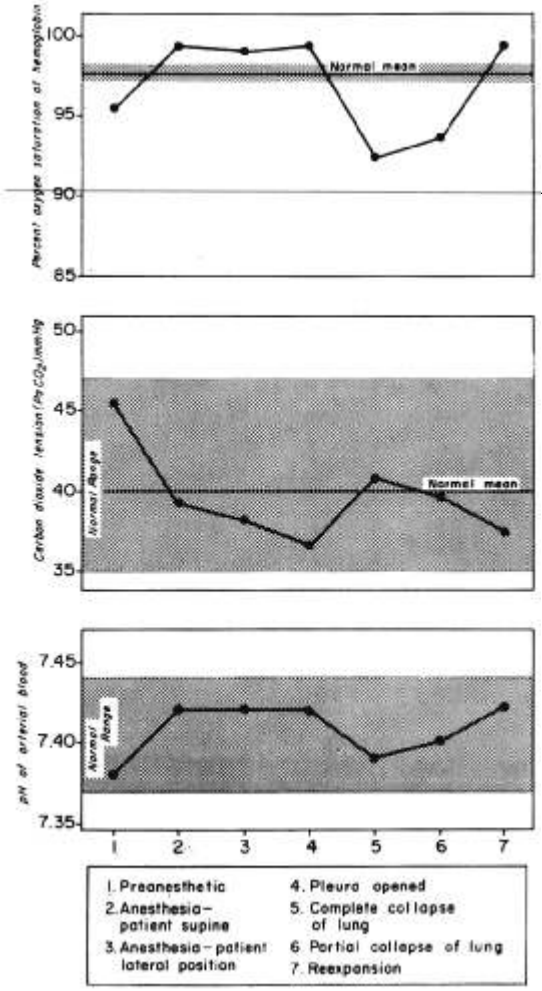

FIG. 1. Arterial blood oxygen saturation, carbon dioxide tension and *p*H of 19 patients during various stages of intrathoracic surgery.

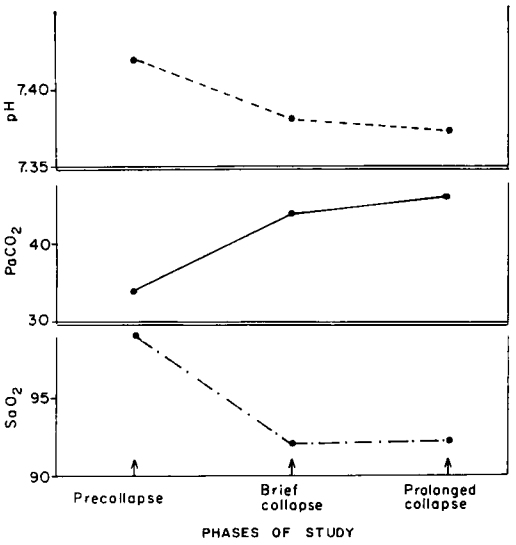

FIG. 2. Arterial blood oxygen saturation, carbon dioxide tension and *p*H of 9 patients before the operated lung was completely collapsed, after 20 minutes of collapse (brief collapse) and after 45–60 minutes of collapse (prolonged collapse).

Table 3 contains values of oxygen saturation with the patient's lungs ventilated with a mixture containing 75 per cent nitrous oxide and 25 per cent oxygen, and with the same patient ventilated with 100 per cent oxygen. The difference between the effects of the two mixtures is statistically significant.

Table 4 contains values of 4 other patients who had extensive pulmonary fibrosis and bronchiectasis of the operated lung and minimal or no disease of the contralateral lung. It is to be noted that the desaturation of oxygen during total collapse, as occurred in the patients reported in table 1, did not take place in these patients.

DISCUSSION

There is general agreement that surgical pneumothorax creates a shunt-like effect so that some of the venous blood passes through the pulmonary circulation to the left side of the heart without absorbing oxygen or eliminating carbon dioxide. It would be expected that as a result, arterial CO_2 tension would rise and oxygen saturation would fall. Some authorities believe that the rise and fall is

TABLE 3

EFFECTS OF HIGH OXYGEN CONCENTRATIONS ON OXYGEN SATURATION DURING TOTAL COLLAPSE OF LUNG

Case	Inspired Gas Mixture	
	75% N_2O–25% O_2	100% O_2
8	85.0	94.3
10	88.2	96.7
11	91.5	97.0
16	93.8	98.4
17	90.5	97.8
Mean	89.8	96.8*

* $P = <0.01$.

proportionate to the amount of lung collapsed,[19-21] a concept that has caused many surgeons to compromise operating conditions in their desire to minimize the amount of ventilatory dysfunction. However, if this were the case, non-ventilation of a whole lung should result in marked oxygen desaturation and severe acidosis. This does not occur as our data as well as those of other attest.[22-25] This decrease in the magnitude of the arteriovenous shunt is effected primarily by diversion of blood from the collapsed lung tissue to the ventilated lung as a result of: (1) effects of gravity; (2) mechanical factors; and (3) biochemical mechanisms.

With the patient in the lateral position, the resistance to blood flow in the vessels of the lower lung is decreased and perfusion pressure is increased, resulting in a diversion of blood from the upper to the lower lung.[26-28] Moreover, collapse of the operated lung enhances this redistribution by markedly increasing the vascular resistance in the operated lung several fold. This is brought about following removal of air from the alveoli by the recoil of the elastic fibers which not only compress the alveoli but contract around the blood vessels and cause them to become more tortuous.[29, 30, 31] Hypoxic alveolar gas mixtures also produce constriction of pulmonary precapillary vessels, probably through reflex mechanisms.[2-11, 32-34]

Our data suggest that during complete collapse of the operated lung the three aforementioned factors combined to cause most of the blood to be diverted to the unaffected lung. When this relative over-perfusion of the unaffected lung was accompanied by hyperventilation, deviation of the ventilation-perfusion ratio was lessened and the arterial oxygen saturation was only slightly decreased while the carbon dioxide tension and *p*H were maintained within normal ranges. Since these parameters were similar during partial and complete collapse, it may be assumed that the benefit of some ventilation in the partial-collapse state was offset by smaller redistribution of blood, probably because the second of the three factors mentioned above was not operating to a maximum degree. The lack of difference between brief and prolonged collapse noted in 9 of our patients (table 2, fig. 2) and other data[4, 35] suggest that these mechanisms are effective within 10 to 15 minutes and persist for some time.

Our results also suggest that there are other factors which influence the magnitude of biochemical deviation during surgical pneumothorax. These include: (1) the condition of the operated lung, (2) the adequacy of ventilation, and (3) the concentration of oxygen in the anesthetic mixture. The data in table 3 suggests that patients with extensive chronic diseases of the operated lung but minimal or no disease of the contralateral lung have already compensated so that exclusion of the diseased lung from ventilation produces minimal or no disturbance. Similar findings have been reported by others.[25, 36, 37] It has long been known that tuberculosis causes narrowing and even obliteration of pulmonary vessels.[38]

In the early phase of the study it was noted that unless the ventilation of the unoperated lung during collapse was equal to or

TABLE 4

EFFECT OF EXTENSIVE PULMONARY DISEASE OF OPERATED LUNG
(Minimal or No Disease of Contralateral Lung)

Case	S_{aO_2}		P_{aCO_2}		*p*H	
	Preat.	Atel.	Preat.	Atel.	Preat.	Atel.
1	98.8	99.0	39.2	38.4	7.40	7.41
2	96.4	96.4	39.0	38.6	7.41	7.39
3	99.3	99.0	35.5	36.2	7.46	7.40
4	96.6	97.0	38.8	39.0	7.44	7.44
Mean	97.8	97.85*	38.1	38.0*	7.43	7.41*

* No significant difference.

exceeded the normal for both lungs, the patients promptly developed hypoxia and hypercarbia. When using the 25 per cent oxygen mixture this "compensatory" hyperventilation of the contralateral lung was apparently more effective in preventing a rise in Pa_{CO_2} than a fall in the oxygen saturation. An explanation of this is found in the differences in the dissociation curves of oxygen and carbon dioxide.[39-41] With hyperventilation of the one lung a more than normal amount of carbon dioxide in the blood passing through functioning alveoli can be washed out, thus compensating for the unaltered amount of CO_2 in the shunted venous blood in the collapsed lung.[41] On the other hand, hyperventilation with air cannot materially increase the oxygen saturation of arterial blood of the ventilated lung because it is nearly saturated normally.[39] Under such circumstances, the arterial oxygen tension will depend wholly upon the redistribution of pulmonary blood flow. In the normal individual this is sufficient to maintain oxygen saturation at levels of 92–93 per cent during complete collapse of one lung.[42]

Ventilation with mixtures containing a high concentration of oxygen was apparently effective in minimizing the oxygen desaturation. It is well known that high oxygen concentrations increase the oxygen saturation of hemoglobin to 100 per cent and increases the amount of oxygen dissolved in the plasma several fold. This extra oxygen in the plasma is readily available not only to the tissues but also to increase the oxygen saturation of hemoglobin of venous blood. That such is the case is attested by the prompt and sustained increase of the oxygen saturation of arterial blood of about 7 per cent in our 5 patients in whom the oxygen concentration was increased from 25 to 100 per cent during total collapse (table 3). Similar findings were reported by Stephen and associates.[43] Hyperoxia also helps the redistribution of blood from the operated to the contralateral lung.[4] This greater redistribution of blood, together with the increased oxygen saturation and the increase in the amount of physically dissolved oxygen from 0.3 ml. to approximately 2 ml. per 100 ml. of blood would make it possible for complete compensation to take place, so that the arterial blood would be 98 per cent saturated.

It appears that it is posible to compensate for complete collapse of one lung during intrathoracic operations. On the other hand, there are patients who cannot tolerate even partial collapse. The obvious conclusion is that if it is needed, complete collapse of the lung can be effected with no greater hazard to the patient than imposed by partial collapse provided: (1) the condition of the contralateral lung is good; (2) hyperventilation of the contralateral lung is effected; and (3) high oxygen concentrations are used in the anesthetic mixtures. Although during the past decade nitrous oxide-oxygen mixtures for intrathoracic surgery have been popular, such mixtures alone do not permit adequate compensation for the oxygen desaturation effected by collapse. Since halothane is nonexplosive and permits a high concentration of oxygen, it might prove a better anesthetic in circumstances where complete collapse of the lung is required.

Summary

The oxygen saturation, carbon dioxide tension and *p*H of arterial blood were measured in patients undergoing intrathoracic operations. These data indicate that partial and complete collapse of the operated lung caused a ventilation-perfusion abnormality so that blood flowing through the deflated lung could not adequately transfer gases.

Under the conditions of this study there was a small, but statistically significant difference between partial and complete collapse of the lung on arterial oxygen, carbon dioxide and *p*H. These biochemical parameters were not significantly influenced by the duration of the collapse of the lung.

Hyperventilation effectively minimized accumulation of carbon dioxide. With the technique used in this study the carbon dioxide content and the *p*H of the blood remained within normal limits throughout the period of investigation. Hyperventilation with mixtures containing 25 per cent oxygen was not effective in eliminating oxygen desaturation of hemoglobin. However, the magnitude of desaturation was significantly decreased by hyperventilating with 100 per cent oxygen.

In 4 patients with extensive disease of the operated lung and minimal or no disease of the contralateral lung arterial oxygen desaturation was not observed.

The possible mechanisms by which compensation for the collapse of the lung takes place have been discussed.

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CARBONIC ANHYDRASE When the red cells are in contact with the respiratory tissue of the lungs the carbonic anhydrase activity of the blood is increased five-fold. The attempt to explain this phenomenon on the basis of summation of the effects of two enzymes—the carbonic anhydrase of the red cells and an enzyme probably contained in the respiratory tissue of the lung and with the power of causing dehydration of carbonic acid—has not been successful, for it has been shown that the lungs do not contain carbonic anhydrase. The active reaction of the lung tissue of warm-blooded animals has shifted towards the alkaline side by comparison with that of the blood in the lung capillaries; this phenomenon, which is specific for homoiothermic animals, is evidently associated with the high intensity of the gas exchange. The catalytic process of dehydration of carbonic acid takes place in the lungs of warm-blooded animals at *p*H 8.0, *i.e.*, in optimal conditions for the action of carbonic anhydrase. The experimentally established five-fold increase in the carbonic anhydrase activity of the blood in minced lung tissue, consequently, is explained by the fact that the surface layer of the red cell, which contains the carbonic anhydrase, is in direct contact with the lung tissue which possesses an alkaline reaction. The catalytic process of dehydration of carbonic acid *in vivo* under the action of carbonic anhydrase now takes place at the border of the phases between the red cell and the respiratory tissue of the lung in the optimal zone of *p*H 8.0, whereas the formation of undissociated carbonic acid from its ions takes place within the red cell at *p*H 7.3. (*Trincher, K. S.: Cause of Increased Carbonic Anhydrase Activity in Lungs, Fiziol. Zh. im. Sech. 46: 726, 1906.*)