A CLINICAL STUDY OF RESPIRATORY EXCHANGE DURING PROLONGED OPERATIONS WITH AN OPEN THORAX*

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THE ENDEAVOR OF EVERY SURGEON should be to carry his patient through an operation with the least possible disturbance of normal physiologic processes. Opening the thorax to obtain adequate exposure for upper abdominal operations has become commonplace. The physiologic disturbances produced by opening the thoracic cavity thus become of immediate concern to every surgeon utilizing a thoracic, or a thoraco-abdominal, incision. Beecher[1] has recently reported that severe respiratory acidosis may occur during the course of an operation with an open thorax. In an endeavor to obtain additional information concerning the altered conditions produced by opening the thorax, the following clinical study of respiratory gas exchange has been made.

METHOD

The present study is based on a series of 53 patients undergoing prolonged operations with an open thorax. The operations may be classified into three main groups: 13 pneumonectomies, nine lobectomies, and 31 miscellaneous operations. The 31 miscellaneous operations all involved an open thorax through a thoracic or a thoraco-abdominal incision, but in none of these patients was pulmonary tissue resected. A majority of these operations were esophagectomies, esophago-gastrectomies, or exploratory thoracotomies. Arterial blood samples were analyzed for oxygen content and capacity, carbon dioxide content, and pH. Arterial blood samples were drawn at the following times: (1) before the administration of the anesthetic; (2) after the patient was anesthetized and just before the pleura was opened while the respirations were still spontaneous and unassisted; (3) toward the end of the operation after several hours of controlled mechanical respiration with the pleural cavity open; and (4) one or two hours after operation with the patient in bed in an oxygen tent. Not all samples were obtained in every patient.

Anesthesia. The usual premedication was Sodium Pentobarbital, 0.1 Gm. the night preceding operation and an equal amount two hours before operation. Ten milligrams of morphine and 0.6 mg. atropine sulfate were given one hour preceding operation. A 7 or 9 mm. intratracheal tube with an inflatable cuff was introduced under topical Pontocaine anesthesia, or, in a few instances,

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under intravenous Sodium Pentothal anesthesia. If the tracheal tube was introduced under local anesthesia, general anesthesia was then induced with intravenous Sodium Pentothal. Anesthesia was continued with ether and oxygen in a closed rebreathing circuit. In the first few patients in the series, Sodium Pentothal was the sole anesthetic agent used. Ether was not used in these patients in order to avoid the technical difficulties of blood gas analysis when small amounts of ether were present in the blood sample. Later these difficulties were overcome.[5] The patient was then placed in a lateral position. The diseased lung was uppermost for the lobectomies and pneumonectomies. The esophagectomies and esophagogastrectomies were performed through a left thoracic or a left thoraco-abdominal incision. Nothing was placed beneath the thorax and the operating table was not broken, as is sometimes done in order to facilitate exposure by arching the upper hemithorax.

Respiration was unassisted until the parietal pleura was exposed. When the pleura was opened, the patient was given 3 to 6 mg. of d-Tubocurarine.[8] At this time, mechanical insufflation of the lungs was started by use of either the Mautz[6, 7] attachment to the standard Heidbrink machine, or the Crafoord[4] apparatus. The principle of these machines is the same. In each, the rebreathing bag is encased in an airtight container slightly larger than the fully inflated rebreathing bag. At the end of the inspiratory phase, a large outlet valve immediately releases the pressure upon the rebreathing bag, and the expiratory phase occurs into the flaccid rebreathing bag without resistance. The pressure used to compress the rebreathing bag can be read on a water manometer and is easily controlled by a valve. The pressure used was generally 10 cm. of water. This intermittent insufflation of the lungs was continued throughout the operation until the pleural cavity was closed. Additional d-Tubocurarine was given during the course of the operation in amounts sufficient to prevent the respiratory muscles from opposing the mechanical insufflation of the lungs. No curare was administered toward the end of the operation, and the patients immediately resumed spontaneous respiration when the pleural cavity was closed, at which time mechanical respiration was discontinued. Secretions were aspirated from the trachea when indicated during the course of the operation and in the postoperative period.

Blood Samples and Chemical Methods. A Cournand arterial needle was inserted into the brachial artery under local anesthesia before inducing general anesthesia. The arm was bandaged on a splint, and the needle was left in place until the postoperative blood sample had been drawn. If the needle became dislodged before the last blood sample was obtained, the remaining samples were drawn from the femoral artery. The blood was collected under oil in a syringe containing a small quantity of heparin to prevent coagulation and a few drops of mercury to aid in the mixing of the blood as the syringe was rotated. The analyses were started as soon as a sample was obtained, and the blood was kept packed in ice until duplicate analyses had been made.

The pH of the blood was determined with a Cambridge glass electrode pH meter. The apparatus was calibrated with standard buffer solution before each determination. Duplicate determinations were made on each specimen, and checks to 0.02 unit were required. Oxygen content, oxygen capacity, and carbon dioxide content of whole blood were determined by the method of Van Slyke and Neill,[9] modified to compensate for the presence of small amounts ot ether, as described by Goldstein, *et al.*[5] The carbon dioxide content of the plasma was then read from the nomogram of Van Slyke and Sendroy[10] in volumes per 100 and converted to millimoles per liter by dividing by 2.226. The partial pressure of carbon dioxide (pCO_2) was then read from the nomogram of Van Slyke and Sendroy using the calculated value of the total plasma CO_2 in mM./L., [Total CO_2]p, and the observed pH. This nomogram is constructed from the Henderson-Hasselbalch equation:

$$pH = 6.10 + \log \frac{[\text{Total } CO_2]p - 0.0301 \quad pCO_2}{0.0301 \; pCO_2}$$

In those instances where the values were such that they could not be read on the nomogram, the pCO_2 was calculated directly from the Henderson-Hasselbalch equation. The bicarbonate value of the plasma, [HCO_3]p, is equal to the [Total CO_2]p minus the CO_2 dissolved in the plasma. The CO_2 dissolved in the plasma is equal to 0.0301 pCO_2. The bicarbonate value, [HCO_3]p, may thus be calculated from the equation:

$$[HCO_3]p = [\text{Total } CO_2]p - 0.0301 \; pCO_2$$

An increase or decrease in fixed acid in the blood can be determined by the difference in the bicarbonate value of the blood specimens at a standard pH of 7.4. In order to correct for the difference in the observed bicarbonate reading and the standard reading at 7.4, the buffer value of the blood must be known. Buffer value of a solution is defined as the amount of acid which must be added to cause a change of one pH unit. In plasma separated from red cells, the buffer value can be measured by the change in bicarbonate concentration when it is titrated with carbon dioxide and the buffer value can be calculated by dividing the change in bicarbonate by the change in pH. This value in normal plasma alone has been found to be about 8.6. However, in the presence of red cells an additional buffering effect is noted, and this has been found to be about 2.3 x (Hb expressed in mM./L.). Hb in millimoles per liter can be calculated from the oxygen capacity:

$$\frac{O_2 \text{ Capacity}}{1.34} = \text{Hb in Gm./100 cc.}$$

$$16.7 \text{ Gm. of Hb per liter} = 1 \text{ mM. hemoglobin}$$

Therefore:

$$\text{Hb mM./L.} = \frac{\text{Hb in Gm. per 100 cc. x 10}}{16.7}$$

The buffer value (β) of the blood can then be calculated from the equation:

$$\beta = 8.6 + 2.3 \text{ (Hb expressed in mM./L.)}$$

The value of the bicarbonate at pH 7.4 may be calculated by:

$$[HCO_3]p \text{ cor.} = [HCO_3]p + \beta \text{ (observed pH} - 7.4)$$

RESULTS

The results of this study of arterial blood in operations on 53 patients with an open thorax have been grouped in nine tables and are described under three headings: the oxygen saturation, the carbon dioxide tension and pH, and the change in concentration of fixed acids.

Oxygen Saturation. The ease with which the saturation of arterial blood with oxygen can be achieved during the course of an operation with an open thorax is illustrated in the first three tables. Table I presents this data in 13 pneumonectomies, Table II in nine lobectomies, and Table III in 16 miscellaneous operations with an open thorax in patients without significant pulmonary disease.

There was a mild degree of unsaturation of the arterial blood with oxygen before general anesthesia was induced in all three groups of patients. The average preoperative saturation was very similar in the three groups, 88.4 per cent in the pneumonectomies, 89.3 per cent in the lobectomies, and 88.9 per cent in the miscellaneous group. The cause for this moderate reduction in oxygen saturation is not obvious. It seems unlikely that it was due to pathologic changes in the lungs, because such pulmonary changes were not present in the third group (Table III) yet the degree of unsaturation was very similar. This preoperative reduction in arterial oxygen saturation may have been due to depression of respiratory activity by the preoperative medication.

Tables I, II, and III also list the arterial oxygen saturation in the three groups of patients some time after the induction of general anesthesia, and again toward the end of the operation. It can be seen that in these 38 patients, with three exceptions, there was a marked rise in the average oxygen saturation above the preoperative level after general anesthesia was induced. Patients L5, L6, and M9 showed a decrease in saturation. In the two latter, the decrease was certainly not large enough to be significant. In the great majority of patients a normal, or above normal, saturation was attained. In the 13 pneumonectomies, the average saturation was 97.5 per cent, in the nine lobectomies 96.3 per cent, and in the miscellaneous group 98.5 per cent. Some of the figures during operation referred to above were obtained before the pleura was opened, and some afterwards. The figures shown in Table VII, however, illustrate that the rise occurs with natural breathing in the lateral position before the start of controlled respiration with mechanical ventilation. Furthermore, in many of these patients, readings of the arterial saturation were also made with the Millikan oximeter. All such readings showed a rapid increase in the saturation of arterial blood with oxygen as soon as general anesthesia with oxygen and ether vapor had been established. The rise in saturation, therefore, was undoubtedly due to the increased alveolar oxygen tension.

The average oxygen saturation towards the end of the operation was slightly lower, as shown in the next to the last columns in Tables I, II, and III.

TABLE I.—*Arterial Blood Saturation with Oxygen in Patients Before and During Pneumonectomy.*

Case Number	Before Operation	During Operation	Time After Start of Operation	During Operation	Time After Start of Operation
	Per Cent Saturation	Per Cent Saturation	Hours : Minutes	Per Cent Saturation	Hours : Minutes
P1	89.2	100.0	2:30	100.0	5:00
P2	84.0	....	....	92.4	3:00
P3	94.8	100.0	1:30	100.0	3:40
P4	88.9	99.3	2:12	98.9	4:15
P5	89.0	90.5	1:12	94.0	2:25
P6	89.2	99.0	2:00	97.1	3:15
P7	88.9	100.0	1:12	98.4	2:00
P8	90.3	....	....	98.7	2:00
P9	83.6	96.6	1:30	97.2	2:00
P10	83.8	96.1	1:15	82.3	4:15
P11	90.3	97.9	0:46	97.6	4:05
P12	86.5	96.8	1:10	92.6	3:40
P13	91.3	100.0	0:42	92.3	4:10
Average	88.4	97.5	1:27	95.5	3:22

This slight decrease in the average figures can probably be attributed to technical difficulties encountered in a few patients, *e.g.*, P10, L6, and M17.

There was a consistent decrease in the postoperative saturation of the arterial blood with oxygen in the 11 patients in whom this determination was made (Table VIII). The postoperative samples were obtained approximately two hours after operation with the patients in bed in an oxygen tent. The degree of oxygen saturation was very similar to that before operation. The decrease from the operative figures can be attributed to the fact that the gas

TABLE II.—*Arterial Blood Saturation with Oxygen in Patients Before and During Lobectomy.*

Case Number	Before Operation	During Operation	Time After Start of Operation	During Operation	Time After Start of Operation
	Per Cent Saturation	Per Cent Saturation	Hours : Minutes	Per Cent Saturation	Hours : Minutes
L1	84.8	87.0	2:00	87.2	2:00
L2	93.6	100.0	2:30	100.0	4:00
L3	89.4	92.0	2:00	92.3	4:00
L4	83.5	100.0	2:00	100.0	3:00
L5	91.3	87.0	2:00	88.5	5:00
L6	90.9	90.5	2:00	82.0	2:30
L7	84.8	100.0	1:12	100.0	3:45
L8	94.2	100.0	1:10	100.0	4:15
L	91.4	100.0	1:12	99.0	3:45
Average	89.3	96.3	1:27	94.3	3:35

mixture in the oxygen tent had a much lower percentage of oxygen than that in the closed anesthetic circuit.

Carbon Dioxide Tension and pH. The removal of carbon dioxide from the blood during operation could not be achieved with the same success as the introduction of oxygen. This fact is illustrated in Tables IV, V, and VI, which also show the changes in pH which occur in conjunction with the changes in carbon dioxide tension. The patients have been grouped under the same headings as in Tables I, II, and III. Table IV gives the data on the same 13 pneumonectomies, Table V gives the data on the same nine lobectomies, whereas Table VI includes a larger group of miscellaneous operations with an open thorax, some of whom had pathologic changes in the lungs, such as inoperable cancer of the lung, fibroid tuberculosis, or marked degrees of emphysema. The data in three patients, M27, M28, and M29, shown in Table

TABLE III.—*Arterial Blood Saturation with Oxygen in Patients Without Pulmonary Disease, Before and During Thoracic and Thoraco-abdominal Operations.*

Case Number	Before Operation	During Operation	Time After Start of Operation	During Operation	Time After Start of Operation
	Per Cent Saturation	Per Cent Saturation	Hours : Minutes	Per Cent Saturation	Hours : Minutes
M1	87.4	98.3	3:00	91.4	5:00
M3	84.9	98.7	4:00	100.0	6:00
M5	88.2	....	....	99.3	1:30
M6	86.7	100.0	4:00	100.0	6:00
M7	91.3	....	....	100.0	3:00
M9	91.2	90.0	2:00	93.6	4:00
M10	90.7	100 0	3:00	102.0	5:00
M11	86.7	97.0	2:00	9 .5	4:00
M14	89.7	....	....	100.0	2:00
M15	89.3	98.3	2:00	95.7	3:00
M17	87.9	100.0	2:00	86.9	4:30
M24	94.4	99.8	1:30	99.0	3:06
M25	89.7	97.3	1:10	100.0	5:42
M27	88.6	100.0	0:[illegible]5	93.4	3:25
M28	89.3	100.0	0:42	....	....
M29	92.2	96.9	1:20	100.0	6:30
Average	88.9	98.5	2:07	95.6	4:11

III, are omitted in Table VI because of failure to obtain determinations of carbon dioxide, or pH, or both.

The average preoperative value of carbon dioxide tension was similar in all three groups. The median value was slightly higher in the group of 13 pneumonectomies than in the other two groups. The median preoperative value for pH was 7.38 in all three groups and the average value was essentially the same. Thus the preoperative median and average values for pH in these 48 patients were within normal limits, whereas the same values for carbon dioxide were very slightly above normal.

During the course of the operation, there was a tendency for the carbon dioxide tension to rise and the pH to fall. This tendency was most marked in the pneumonectomies, less marked in the lobectomies, and least in the miscellaneous group. In the pneumonectomies, the average rise in the pCO_2 was 29 mm. Hg., with an increase of 18 mm. Hg. in the median value. The average increase in pCO_2 in the lobectomies was 21 mm. Hg., with an increase in

TABLE IV.—*Pneumonectomy. pH and pCO_2 of Arterial Blood Before and Near Completion of Operation.*

Case Number	pH		pCO_2 mm. Hg		Time After Start of Operation
	Before Operation	Near End of Operation	Before Operation	Near End of Operation	Hours : Minutes
P1	7.40	7.37	47	54	5:00
P2	7.39	7.38	42	59	3:00
P3	7.44	7.28	35	55	3:40
P4	7.34	7.15	47	74	4:15
P5	7.35	6.99	48	115	2:25
P6	7.47	7.11	36	91	3:15
P7	7.40	7.02	44	124	2:00
P8	7.38	7.31	47	52	2:00
P9	7.36	7.29	47	57	2:00
P10	7.35	7.25	50	64	4:15
P11	7.38	7.37	44	43	4:05
P12	7.39	7.17	46	97	3:40
P13	7.37	7.18	46	75	4:10
Average	7.39	.22	45	74	3:22
Median	7.38	7.25	46	64	3:40

the median value of 17 mm. Hg. In the miscellaneous group, the average rise was 13 mm. Hg., while the median rise was only 10 mm. Hg. The average fall in pH in the group of pneumonectomies was 0.17 with a fall in the median value of 0.13. In the lobectomies, the average fall in pH was 0.12, with a

TABLE V.—*Lobectomy. pH and pCO_2 of Arterial Blood Before and Near Completion of Operation.*

Case Number	pH		pCO_2 mm. Hg		Time After Start of Operation
	Before Operation	Near End of Operation	Before Operation	Near End of Operation	Hours : Minutes
L1	7.38	7.29	42	60	2:00
L2	7.32	7.43	50	40	4:00
L3	7.38	7.12	45	90	4:00
L4	7.41	7.17	43	86	3:00
L5	7.39	7.10	43	115	5:00
L6	7.35	7.20	39	62	2:30
L7	7.39	.34	43	44	3:45
L8	7.37	7.29	42	47	4:15
L9	7.38	7.34	46	45	3:40
Average	7.37	7.25	44	65	3:34
Median	7.38	7.29	43	60	3:45

decrease in the median value of only 0.09. The miscellaneous group were quite similar to the lobectomies, with an average decrease of 0.12, and a decrease in the median value of 0.06. It is believed that the reference to average and median values is justified by the number of determinations which were made.

There were, of course, instances in which the ventilation was grossly inadequate, *e.g.*, P5, P7, L5, and M17. On the other hand, there were examples even in the pneumonectomy group, *e.g.*, P11, where the carbon dioxide tension was maintained at its preoperative value, or even lower. Similarly, there were several instances in which the pH fell to seriously low levels: P5, P7, and M17. In other instances, the pH was maintained at, or above, the preoperative level, *e.g.*, P2, P11, L2 and M10.

That the lateral position in itself was not responsible for the increase in carbon dioxide tension and the decrease in pH appears evident from the data

TABLE VI.—*Miscellaneous Operations with Open Thorax pH and pCO_2 of Arterial Blood Before and Near Completion of Operation.*

Case Number	pH Before Operation	pH Near End of Operation	pCO_2 mm. Hg. Before Operation	pCO_2 mm. Hg. Near End of Operation	Time After Start of Operation Hours : Minutes
M1	7.44	7.34	34	46	5:00
M2	7.41	7.34	41	46	2:00
M3	7.40	7.34	42	46	6:00
M4	7.40	7.19	42	68	1:30
M5	7.42	7.36	43	49	1:30
M6	7.33	7.19	50	77	6:00
M7	7.38	7.33	43	49	3:00
M8	7.37	7.22	43	65	3:00
M9	7.41	7.14	42	90	4:00
M10	7.35	7.43	50	34	5:00
M11	7.48	7.20	45	95	4:00
M12	7.33	7.15	56	89	2:00
M13	7.51	7.32	31	51	2:30
M14	7.37	7.32	37	42	2:00
M15	7.40	7.38	40	42	3:00
M16	7.44	7.34	41	52	1:00
M17	7.37	6.94	40	134	4:30
M18	7.33	7.20	54	74	3:00
M19	7.29	7.19	60	68	3:15
M20	7.38	7.33	50	52	2:30
M21	7.29	7.13	54	75	3:09
M22	7.38	7.23	45	65	2:25
M23	7.39	7.37	39	42	4:00
M24	7.42	7.34	36	41	3:06
M25	7.31	7.30	52	44	5:42
M26	7.39	7.22	35	56	3:35
Average	7.38	7.26	44	57	3:20
Median	7.38	7.32	42	52	3:00

presented in Table VII. In this table, the preoperative values of pCO_2 and pH are compared with those made on samples obtained just before opening the pleura, approximately one hour after the start of the operation. Figures were obtained in four pneumonectomies, one lobectomy, and 12 miscellaneous operations. There was no change in the average value of pCO_2, and an increase in the median value of only 1 mm. Hg. In one instance, M22, there was an increase in pCO_2 of 20 mm. Hg. Thus, although inadequate ventilation may occur in the lateral position, it is not a consistent finding. The pH showed an average decrease of 0.06, with a decrease in the median value of 0.09. Compared with the figures in Tables IV, V, and VI, these changes cannot be regarded as particularly significant. It seems obvious that the changes in carbon dioxide tension and pH during the course of the operation were largely a result of the open thorax, and not the lateral position upon the operating table.

In 11 patients, arterial blood samples were obtained approximately two hours after the end of the operation, when the patient was in bed in an oxygen tent. Two of these patients had pneumonectomies, two lobectomies, and seven had miscellaneous operations. Table VIII shows a comparison between the

TABLE VII.—*Effect of Lateral Position Before Opening Pleura—Respiration Not Assisted.*

	Arterial Blood						
	pH		pCO_2 mm. Hg.		Oxygen Saturation Per Cent		Time After Start of Operation
Case Number	Before Operation	Before Opening Pleura	Before Operation	Before Opening Pleura	Before Operation	Before Opening Pleura	Hours : Minutes
P10	7.35	7.36	50	41	84	96	1:15
P11	7.38	7.43	44	32	90	98	0:46
P12	7.39	7.32	46	47	87	97	1:10
P13	7.37	7.32	46	50	91	100	0:42
L8	7.37	7.28	42	46	94	100	1:10
M19	7.29	7.17	60	77	82	86	0:55
M20	7.38	7.39	50	47	83	96	1:00
M21	7.29	7.22	54	47	77	97	0:40
M22	7.38	7.21	45	65	84	94	0:45
M23	7.39	7.26	39	57	86	89	1:10
M25	7.31	7.24	52	60	90	97	1:10
M26	7.39	7.23	35	48	87	91	1:10
M27	7.34	7.27	49	51	89	100	0:55
M28	7.40	7.39	43	39	89	100	0:42
M29	7.31	7.37	52	42	92	97	1:20
M30	7.44	7.37	34	32	89	97	1:00
M31	7.36	7.25	46	51	89	100	1:00
Average	7.36	7.30	49	49	87	96	0:59
Median	7.37	7.28	46	47	89	97	1:00

values for carbon dioxide tension and pH before, during, and after operation on these patients. The average and median values for pCO_2 were lower, and for pH slightly higher, than those obtained near the end of the operation. The average pCO_2 was still 5 mm. Hg. above its preoperative value, and the average pH was 0.06 below the preoperative value. In general, where the respiratory acidosis was most marked near the end of the operation (P12 and M21) return towards normal, or preoperative values, was most marked. As stated above, the oxygen saturation decreased in the postoperative period below that present near the end of the operation.

Fixed Acids. The change in the concentration of fixed acids in the arterial blood was calculated in the group which showed the most severe respiratory acidosis, *i.e.*, the 13 pneumonectomies. Table IX presents the data on the case with the least respiratory acidosis, P11, the case with the greatest respiratory acidosis, P7, and the average values for the group as a whole. As can be seen in the table, the average increase in fixed acids for the group as a whole near the end of the operation was only 2.7 millimoles per liter.

TABLE VIII.—*pH, pCO_2 and Oxygen Saturation of Arterial Blood Before Operation, Near End of Operation and Postoperatively.*

	pH			pCO_2 in mm. Hg.			Oxygen Saturation Per Cent			Postoperative Blood Sample
Case Number	Before Operation	Near End of Operation	Post-Operative	Before Operation	Near End of Operation	Post-Operative	Before Operation	Near End of Operation	Post-Operative	Time After End of Operation Hours:Minutes
P11	7.38	7.37	7.37	44	43	45	90	98	92	1:45
P12	7.39	7.17	7.31	46	97	45	87	93	85	1:35
L8	7.37	7.29	7.32	42	47	49	94	100	97	2:10
L9	7.38	7.34	7.34	46	45	48	91	99	92	2:10
M19	7.29	7.19	7.23	60	68	62	82	88	86	1:45
M20	7.38	7.33	7.28	50	52	60	83	93	78	1:50
M21	7.29	7.13	7.29	54	75	52	77	85	82	1:55
M22	7.38	7.23	7.20	45	65	63	84	60	68	1:50
M23	7.39	7.37	7.39	39	42	39	86	89	82	2:00
M24	7.42	7.34	7.35	36	41	37	94	99	85	1:30
M26	7 39	7.22	7.31	35	56	48	87	93	89	2:25
Average	7.37	7.27	7.31	45	57	50	87	90	85	1:54
Median	7.38	7.29	7.31	45	52	48	86	93	85	1:50

TABLE IX.—*Change in Fixed Acid of Arterial Blood During Pneumonectomy-mechanical Respiration—Lateral Position.*

	Case P11 with Minimum Respiratory Acidosis		Case P7 with Maximum Respiratory Acidosis		Average 13 Patients	
Blood Plasma	Before Operation	Near End of Operation	Before Operation	Near End of Operation	Before Operation	Near End of Operation
pH B	7.38	7.37	7.40	7.02	7.39	7.22
pCO_2 mm. Hg B	44.0	43.0	44.0	124.0	45.0	74.0
Total CO_2 mM/L P	26.3	23.8	27.0	34.0	26.0	28.8
HCO_3 mM/L P	25.0	22.5	25.7	30.3	24.6	26.6
Hb gm. per cent B	14.4	13.3	11.2	12.8	13.9	14.0
Hb mM/L B	8.6	8.0	6.7	7.7	8.3	8.4
Buffer value β	28.4	27.0	24.0	26.3	27.7	27.9
Correction for pH mM/L	−0.6	−0.8	0.0	−10.0	−0.3	−5.0
HCO_3 Corrected mM/L p	24.4	21.7	25.7	20.3	24.3	21.6
Change in fixed acid mM/L	....	+2.7	....	+5.4	....	+2.7

DISCUSSION

With the method of anesthesia employed, there was no difficulty in maintaining adequate oxygenation during the course of long operations with an open thorax. Indeed the saturation of arterial blood with oxygen was higher during the operative procedure than it was in either the preoperative or the postoperative state. The increase in saturation occurred with the patients in the lateral position with unassisted respiration before opening the pleura. Oximeter readings indicated that the rise in saturation occurred promptly with induction of general anesthesia with ether vapor and oxygen. It appears reasonable, therefore, to attribute this satisfactory degree of oxygenation during the operation to the increased oxygen tension in the alveolar air which follows the breathing of an anesthetic mixture rich in oxygen.

The average preoperative saturation of arterial blood with oxygen was somewhat below normal. As the condition was present even when there was no significant pulmonary disease (Table III), it may have been due to the preoperative medication. The average postoperative oxygen saturation was below normal and very similar to the preoperative saturation. As all the postoperative arterial blood samples were taken with the patient in an oxygen tent with an unobstructed airway, it would appear advisable to provide a higher content of oxygen than that afforded by the ordinary inefficient oxygen tent.

In all the patients reported here there was a tendency toward the development of respiratory acidosis during the course of the operation with an open thorax. The tendency was most marked in the group of pneumonectomies, less marked in the group of lobectomies, and least obvious in the group of miscellaneous operations. The respiratory acidosis was indicated by an increase in the carbon dioxide tension of the arterial blood, and was associated with a decrease in the pH. That there was no significant associated metabolic acidosis is evident from calculation of the change in concentration of the fixed acids in the group of pneumonectomies, the group showing the most marked respiratory acidosis. In our group of 13 pneumonectomies the average increase in fixed acids was 2.7 millimoles per liter, a figure which lies within the normal daily variation. Beecher,[1] in a similar calculation, noted an average increase in the fixed acids in six pneumonectomies of 9.3 millimoles per liter. The increase was less marked in the lobectomies and least marked in the miscellaneous operations in his series. Beecher suggested that possibly the high tension of carbon dioxide encountered in his series might interfere with cellular oxidation to the extent that the fixed acids accumulate. This explanation may well be valid, as in our series of pneumonectomies with mechanical ventilation of the lungs, the increase in the carbon dioxide tension was less than half that reported by Beecher in pneumonectomies with unassisted respiration.

The acidosis is then clearly respiratory and is due to the increased carbon dioxide tension in the alveolar air resulting from inadequate pulmonary ventilation. Beecher[2] has shown that the carbon dioxide tension does not rise in operations on patients in the supine position with a closed thorax, under ether oxygen anesthesia with an intratracheal tube. Ether is therefore apparently not a factor in the acidosis and pulmonary ventilation is adequate under these conditions. We also have found that under the same conditions, with the exception that the patient was in the lateral and not the supine position, the pulmonary ventilation was adequate and that there was no significant increase in the carbon dioxide tension of the arterial blood (Table VII). On the other hand, Beecher[1] observed respiratory acidosis with patients in the lateral position and the diseased lung uppermost. In these patients, however, the lateral position was modified by placing a blanket roll or pillow, approximately five inches in diameter, beneath the thorax, placing the operating table in about five degrees Trendelenberg position, and breaking the table ten to 15 degrees at the head, and five degrees at the foot. Under these circumstances, ten of the 11 patients studied showed an average increase of 14.6 mm. Hg. in the carbon dioxide tension of the arterial blood. In the 17 patients whom we studied in the lateral position before opening the pleura, there was no increase in the average carbon dioxide tension. In some of these patients, *e.g.,* M25, M27, M28, and M29, there was no significant disease of the uppermost lung. Yet, even in the four pneumonectomies and one lobectomy in Table VII, there was no significant increase in carbon dioxide tension. The modified lateral position used by Beecher may be responsible for the difference in results. There can be no doubt that the lateral position interferes with the outward and cephalic movement of the ribs on the lower side of the thorax. It seems equally obvious that the placing of a roll or pad beneath the thorax, placing the operating table in a slightly head-down position and angulating the table beneath the thorax, would tend to produce further interference with the ventilation of the lower lung. We have not found such curvature of the thorax essential for good operative exposure and consequently have not employed it.

With one pleural cavity widely opened and no pleural adhesions, the respiratory muscles immediately become ineffective in the movement of gases in and out of the lungs. Under these circumstances, the lung fails to expand when the diaphragm moves downward and the ribs move outward. This is not only true on the side of the opened pleura, but also to a greater or lesser extent on the side of the unopened pleura, because the mediastinum moves to this side with inspiration, resulting in only partial expansion of the lower lung. It is thus obvious that pulmonary ventilation, solely dependent upon the action of the respiratory muscles, in the presence of an open thorax will be markedly impaired. The degree of impairment of pulmonary ventilation will be dependent upon a number of factors, such as the presence or absence of adhesions to the chest wall or diaphragm, the degree of mobility of the mediastinum, the presence or absence of an opening in the opposite pleural cavity and whether or not the position on the operating table contributes to the interference with inflation and deflation of the lung.

The presence of a severe acidosis at the conclusion of a long operation with an open thorax is certainly undesirable, and may contribute to some of the hitherto unexplained fatalities which occur. Unfortunately, we do not have data on a control series of patients with unassisted respiration. Beecher,[1] however, has reported such a series. The degree of respiratory acidosis which he reported in five pneumonectomies was approximately twice as great as in the 13 pneumonectomies reported here in whom mechanical ventilation was used. Again, the respiratory acidosis in the eight lobectomies reported by Beecher was approximately 50 per cent greater than in the nine lobectomies reported here. The five miscellaneous operations reported by Beecher showed approximately the same degree of respiratory acidosis as the 26 miscellaneous operations reported here. More recently, Beecher[3] has reported similar studies on pneumonectomies and lobectomies with assisted respiration in the lateral position and with unassisted respiration in the Overholt prone position. The three pneumonectomies with assisted respiration showed approximately the same degree of acidosis as in the 13 pneumonectomies with mechanical respiration reported here. On the other hand, the ten lobectomies with assisted respiration showed no significant respiratory acidosis, in contrast to the nine lobectomies reported here with mechanical ventilation of the lungs, who showed an average increase in carbon dioxide tension of 21 mm. Hg. and an average decrease in pH of 0.12. Beecher also reported five pneumonectomies and four lobectomies in the prone position with unassisted respiration who exhibited no significant respiratory acidosis. The disparity between the number of cases referred to above as reported by Beecher and those published by him[1, 3] arises from the fact that we have only referred to those cases where determinations of pH and carbon dioxide tension were made preoperatively and near the end of operation.

Because of the small number of cases, the comparisons drawn between average figures in the preceding paragraph may not be valid. But the conclusion seems inescapable that in the lateral position, ventilation of the lungs is improved when normal breathing is augmented by manual compression of the rebreathing bag in the anesthetic circuit, or when ventilation of the lungs is completely taken over by mechanical means. At first glance, it would appear that the prone position with unassisted respiration is more successful in avoiding respiratory acidosis than the lateral position with either assisted respiration or mechanical ventilation. However, further studies must be done before such a conclusion is warranted. Furthermore, in the cases reported by Beecher there is a striking disparity between the preoperative pH values in the cases with unassisted respiration and the preoperative pH values of the cases in the prone position. The difference in these values in the pneumonectomies was 0.12 and in the lobectomies 0.16. The cases in the prone position in each instance showed the lower average preoperative value.

We have been unable to find any studies on respiratory acidosis during pnemonectomies, or other open thoracotomies, with patients in the supine position. The present authors are firmly convinced, however, that neither the supine nor prone positions afford the same advantageous exposure for the performance of a difficult pneumonectomy that the lateral position does. We feel, therefore, that a more strenuous effort should be made to improve the ventilation during the course of operations with an open thorax with the patient in the lateral position, rather than abandon a position which affords the best surgical exposure in favor of a position which improves ventilation.

We hold no particular brief for mechanical ventilation of the lungs over manual compression of the rebreathing bag. It is obvious that both can accomplish the same desired objective of improving pulmonary ventilation. On the other hand, we can see no objection to mechanical ventilation, which has been used satisfactorily for many years in laboratory experiments on animals. It frees the anesthetist from the tiresome repetitive task of squeezing a rubber bag 14 to 16 times a minute. The rate and depth of ventilation are more uniform and can be more accurately controlled with a mechanical apparatus. We have used mechanical ventilation at the Jefferson Hospital for over two and a half years in all operations involving an open thorax with complete satisfaction. Such apparatuses do not involve any additional hazard of explosion, as no electric motor is necessary in the operating theatre. Any source of compressed air is suitable, either piped into the operating room, taken from tanks, or drawn from a rubber hose leading from a small electric air-compressor in an adjoining room.

Finally, whatever method of pulmonary ventilation, or whatever position is used in operations with an open thorax, there is always the possibility that an occasional patient may develop severe respiratory acidosis. We know of no clinical signs or symptoms which can be depended upon to recognize such acidosis. Unfortunately, an increase in carbon dioxide tension, or a decrease in pH, produces no color change in the skin and mucous membranes such as results from a decrease in the saturation of hemoglobin with oxygen. The intermittent sampling of arterial blood is troublesome and time-consuming. It is to be hoped therefore that some method of continuous, or intermittent and rapid, determination of the carbon dioxide content of expired air may be developed. Such a method will enable the anesthetist and the surgeon to recognize the development of respiratory acidosis and to take steps to correct this serious and often unrecognized condition.

SUMMARY

1. A clinical study of the respiratory gas exchange has been made during the course of 53 operations involving an open thoracotomy. These operations comprised 13 pneumonectomies, nine lobectomies and 31 miscellaneous operations. All patients were operated upon in the lateral position. While the pleura was open, curare was administered and pulmonary ventilation was maintained by mechanical insufflation. Samples of arterial blood were drawn preoperatively, before opening the pleura, toward the end of the operation, and approximately two hours postoperatively. The blood samples were analyzed for oxygen content and capacity, carbon dioxide content, and pH. From these determinations, the carbon dioxide tension and, in the pneumonectomies, the change in concentration of the fixed acids were calculated.

2. Satisfactory oxygenation was maintained throughout the operative period, whereas the preoperative and postoperative levels of oxygenation were both somewhat below normal.

3. There was a tendency towards the development of respiratory acidosis during the course of these operations. This tendency was most marked in the pneumonectomies, less marked in the lobectomies, and least obvious in the group of miscellaneous operations.

4. In the group of pneumonectomies, in which respiratory acidosis was most marked, there was no significant associated metabolic acidosis.

CONCLUSIONS

Respiratory acidosis may occur in any operation involving an open thorax. The acidosis is a result of inadequate pulmonary ventilation. With the patient in the lateral position ventilation should be manually assisted, or taken over by mechanical means. The development of a practical method for the early detection of respiratory acidosis constitutes a challenge to those interested in the future development of this field of surgery.

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DISCUSSION.—DR. JOHN H. GIBBON, JR.: We do not mean to imply by this study that mechanical insufflation is the only method of insuring adequate pulmonary ventilation. However, in our hands it has been of great help.

We do wish to emphasize that you cannot regard the patient's condition as good merely because he is not cyanotic. In all of the patients reported here there was adequate saturation of the blood with oxygen. The lowering of the pH and the increase in the carbon dioxide tension of the blood does not produce any obvious signs or symptoms in a patient under anesthesia such as you would expect to find in an unanesthetized patient.

When the thorax is widely opened, the problem consists in being sure that the patient has adequate ventilation and exchange. Only in that way can you avoid respiratory acidosis, and I think for the present that a good bit of attention will have to be paid to the pH of the blood during prolonged operations with the thorax opened.