THE ELECTROCARDIOGRAM IN PNEUMOPERITONEUM, PNEUMOTHORAX, AND PHRENIC CRUSH

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This study was undertaken in order to determine the electrocardiographic changes that take place in mechanical procedures that cause displacement of the heart such as pneumoperitoneum, artificial pneumothorax, and phrenic crush, and to decide if these procedures produced any evidence of cor pulmonale. A number of patients suffering from pulmonary tuberculosis who required artificial pneumoperitoneum or artificial pneumothorax or phrenic crush were studied. In each case cardiograms were taken in the supine position with one pillow under the head. Then the therapeutic procedure indicated was carried out, and when it was considered that the optimum effect had been secured further cardiograms were taken. In 10 instances, however, the control cardiograms were taken after the therapeutic procedure was discontinued and had apparently ceased to have any effect. Fifty-six patients were under the care of the author and full clinical data of these cases are available. In a further series of ten patients who were under the care of Dr. R. J. Grove-White and others at Tan Tock Seng Hospital, Singapore, clinical data were not obtained. Where several therapeutic measures were employed they were considered as separate cases.

The heights or depths of P, Q, R, S–T, and T were measured from the reference level of the T–P segment, i.e. just before the commencement of the P wave, and of the S–T segment at a point 0·04 sec. after the end of the QRS complex. All measurements of voltage were carried out with a Beck's illuminating hand microphone with a scale reading 1/10 mm. The shape of the S–T segments and T waves was studied with a hand lens. The P–R, Q–T, and R–R intervals were read in 1/100 sec. with a hand lens in standard limb lead II, and the Q–T ratio calculated from a nomogram (Goldberger, 1949). The cases were divided into the following groups:

<table>
<thead>
<tr>
<th>Cases</th>
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<tr>
<td>i  Pneumoperitoneum</td>
<td>38</td>
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<tr>
<td>ii Pneumoperitoneum and right phrenic crush</td>
<td>16</td>
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<tr>
<td>iii Right phrenic crush</td>
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<td>iv Right-sided artificial pneumothorax</td>
<td>10</td>
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<tr>
<td>v  Left-sided artificial pneumothorax</td>
<td>10</td>
</tr>
<tr>
<td>vi Pneumoperitoneum and left phrenic crush</td>
<td>6</td>
</tr>
<tr>
<td>vii Left phrenic crush</td>
<td>5</td>
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Total 100

In assembling the data for assessment of their significance the following criteria were used: only differences of 1 mm. or more were considered significant in the case of P, Q, R, S, T, and S–T. In the case of the P–R interval a difference of 0·04 sec. or more and in the case of the Q–T ratio a difference of 0·02 or more were considered significant.
RESULTS

There were no significant changes in the P wave, P–R interval, or the Q–T ratio. The S–T segment showed no significant elevation or depression and in no case did the S–T and T simulate the appearance seen in coronary disease. There was no evidence of cor pulmonale electrographically or clinically. The main changes were in the QRS complex and are summarized as follows.

(i) Pneumoperitoneum produced an increase in the height of R in I, aVL, V2, V3, and V4 and a decrease in the height of R in II and aVF (Fig. 1–4).

(ii) Pneumoperitoneum and right phrenic crush produced an increase in the height of R in I, V2, and V4 and a decrease in the height of R in II, aVF, and III (Fig. 5 and 6).

(iii) Right phrenic crush produced a decrease in the height of R in V3 and V4 and an increase in the depth of S in V2.

(iv) Right-sided artificial pneumothorax produced an increase in the height of R in V4 and V5.

(v) Left-sided artificial pneumothorax produced a decrease in the height of R in I, V4, and V6 (Fig. 7–10).

(vi) Pneumoperitoneum and left phrenic crush produced an increase in the height of R in I, aVL, V2, V3, and V4 and a decrease in the depth of S in V1.

(vii) Left phrenic crush produced an increase in the height of R in I, and a decrease in the height of R in V4 and V6.

DISCUSSION

The mechanical principles governing the movement of the heart in the three planes should be considered. The heart is more or less tetrahedral in shape with the angles and margins rounded off, and its mobility is much greater at the apex than at the base, being restricted at the base by the attachment of the vascular pedicle. It is enclosed in a semi-rigid compartment—the thorax, and on either side are the spongy lungs which offer considerable resistance to shifting of the heart.
Fig. 3.—X-ray picture of same case as Fig. 1 after pneumoperitoneum.

Fig. 4.—Electrocardiogram of same patient as Fig. 2 after pneumoperitoneum. Note deep Q3 with flat T. aVL shows taller R than before PP due to shift to the left. V2 shows R=S due to counter-clockwise rotation.

Fig. 5.—X-ray picture of same patient as in Fig. 1 after pneumoperitoneum + Right phrenic crush.

Fig. 6.—Electrocardiogram of same patient as in Fig. 2 after pneumoperitoneum + right phrenic crush. Note Q3; V2 again shows S>R due to clockwise rotation.
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Fig. 7.—X-ray picture, showing artificial pneumothorax (left).

Fig. 8.—Electrocardiogram taken while the patient was having artificial pneumothorax (left). AVL and I show low R; V4 shows R = S.

Fig. 9.—X-ray picture of same patient as in Fig. 7, showing the left lung re-expanded.

Fig. 10.—Electrocardiogram of same patient as in Fig. 8 after re-expansion of lung. AVL and I show taller R due to a shift to the left; V4 shows R much greater than S due to counter-clockwise rotation. Discontinuation of artificial pneumothorax and re-expansion of lung caused a shift to the left and counter-clockwise rotation.
even when full of air. The direct effect of a right-sided pneumothorax would be to displace the apex to the left, but the displacement of the heart to the left is much less than the compression of the right lung as seen in the skigrams when the right lung is well collapsed by artificial pneumothorax. This is partly due to the fact that the lung, the right lung in this instance, is more compressible than the heart is moveable, and partly to the fact that the left lung and the semi-rigid thoracic wall on the left side offer a resistance to the movement of the heart towards the left. The heart is usually pointing forwards and so the force exerted by a right-sided pneumothorax which pushes the heart to the left, when it meets with resistance, will make the heart rotate forward, i.e. in a counter-clockwise rotation, so that a shift of the heart to the left will be automatically followed by counter-clockwise rotation (Meek and Wilson, 1925). In a left-sided pneumothorax the reverse changes will follow, but as shown above the displacement of the heart is much less, because when the heart is pushed to the right the resistance of the right lung, the vertebral column and the sternum make movement difficult, resulting in marked clockwise rotation. Pneumoperitoneum, because of the rise of the diaphragm, causes a shift of the apex to the left, counter-clockwise rotation and forward rotation of the apex, if the apex was originally pointing forwards, and backward rotation if the apex was originally pointing backwards. Right phrenic crush superimposed on a pneumoperitoneum causes a rise of the right dome of the diaphragm. This causes an increase in the transverse diameter of the right auricle, displacement of the apex to the right, which in turn, is followed by clockwise rotation, and backward rotation of the apex. Left phrenic crush will cause the same result as pneumoperitoneum, and if pneumoperitoneum has already been performed, the results will be accentuated. Further, the interposition of air between the heart and the electrode may cause a lowered voltage in the lead concerned so that lowered voltage may be expected in some of the air-introducing procedures, particularly in left-sided artificial pneumothorax as the air then envelopes the heart on three sides.

The main electrocardiographic changes produced can be accounted for on mechanical grounds.
(1) Pneumoperitoneum. A shift to the left would account for an increased R in aVL and I, counter-clockwise rotation for an increased R in V2, V3, and V4, and interposition of air between the heart and the electrode for decreased R in aVF and II.
(2) Pneumoperitoneum and right phrenic crush. An increased R in I, V2, and V4, and a decreased R in II, III, and aVF can be explained as in (1) above.
(3) Right phrenic crush. A decreased R in V3 and V4 and an increased S in V2 could result from clockwise rotation.
(4) Right-sided artificial pneumothorax. An increased R in V4 and V5 could result from counter-clockwise rotation.
(5) Left-sided artificial pneumothorax. A decreased R in I, V4, and V6 could result from a shift to the right and clockwise rotation or low voltage due to the interposition of air between the heart and the electrode.
(6) Pneumoperitoneum and left phrenic crush. A shift to the left would account for an increased R in aVL and I and counter-clockwise rotation for an increased R in V2, V3, and V4 and a decreased S in V1.
(7) Left phrenic crush. A shift to the left would account for an increased RI and the interposition of air between the heart and the electrode for a decreased R in V4 and V6.

An attempt will now be made to analyse previous work in the light of the above hypothesis. Master (1928) found S–T deviation, right axis deviation, low voltage and TIII inversion in pneumothorax. Case 1 (right pneumothorax) showed S–T abnormality and large amplitude of QRS in lead III. There was right ventricular hypertrophy and failure as shown by the forceful apex beat and the enlarged liver in his cases, which would account for right axis deviation and the S–T deviation; the large QRS in III is explained by the fact that the air in right-sided pneumothorax is interposed between the two electrodes of I and II but not III which is connected to the left leg and left arm. Case 2 after right-sided artificial pneumothorax showed low voltage in all three standard
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limb leads and flat TIII. The low voltage in I and II was due to the interposition of air between the electrodes and in lead III both the low QRS and flat T were due to the fact that the apex had shifted to the left causing R and T to become taller in aVL, which would in turn cause QRS and T to be lower in III. The above explanation would probably hold for the other cases which are not reported in detail. Benatt and Berg (1945) found that artificial pneumoperitoneum produced a QTIII pattern and a flattening of S–TII. The QTIII is due to the fact that pneumoperitoneum causes a shift of the apex to the left, giving rise to a taller R and a taller T in aVL. The S–T flattening is only an impression created by the lowering of the T wave. Littmann (1946) found absence of R waves and presence of inverted T waves in CF2 and CF3 in the supine position only, in spontaneous pneumothorax complicated by mediastinal emphysema. On studying the electrodiagrams of the first case reported in this paper, it is found that CF2 and CF3 have inverted P waves, QS and inverted T, i.e. the pattern that is normally seen in V1; lead CF1 is not recorded nor are the V leads. The explanation may be as follows: due to the left pneumothorax the heart is shifted to the right; such a shift is always accompanied by clockwise rotation, which would result in V2 and V3 recording similar patterns to V1. Now CF2 and CF3 really equal V2–VF and V3–VF respectively. In the supine position the air in the mediastinum would accumulate anterior to the heart and cause backward rotation of the apex so that the leg would face the epicardial surface of the right ventricle and give an rS complex of low voltage due to the fact that air is a poor conductor —this would not affect the form of the resultant complex which would therefore be similar to V1.

Armen and Frank (1949) investigated 45 cases of pneumothorax and found, in right-sided artificial pneumothorax, depression of QRSI (8 out of 20 cases), depression of P waves (11 out of 20 cases), and depression or flattening of TI (10 out of 20 cases). The depression of QRSI can be due to the fact that the negativity of VR is lessened due to low voltage, the depression of P waves to low voltage, and the depressed TI follows the depression of QRSI. In left-sided artificial pneumothorax they found low voltage of QRSI, depression of P1, depression of T1 and inversion of T in CF2, CF4, and CF5. It is quite obvious that all these changes can result from lowered voltage. There was no S–T elevation or depression in any of the cases. The findings of Rauchwerger and Erskine (1949) and of Silverberg and Feldman (1950) in chest surgery are not easy to explain. In any case chest surgery cannot be expected to produce uncomplicated positional change of the heart as many other factors are involved, e.g. the presence or absence of segmental or lobar collapse, bronchiectasis, fibrosis of the lung and effusion.

Evans and Black (1950) investigating 10 cases of artificial pneumoperitoneum found abnormally large Q waves in the aësophageal leads and have explained this change as resulting from forward displacement of the apex. Pollak (1951) investigated 19 cases of pneumoperitoneum and found that no significant changes resulted in the recumbent position; in the sitting position, however, QIII was found in 4, TIII in 13, and left axis deviation in 9 cases. When a normal person sits up the diaphragm descends due to the “falling away” of the abdominal contents, and quite independently, the heart assumes a more vertical position. In the presence of pneumoperitoneum, however, the diaphragm will not descend as much as usual because of the presence of air underneath it but the force of gravity will make the heart try to assume a more vertical position, therefore the apex will shift to the left and counter-clockwise rotation occur. VL will show taller R and T; this accounts for QTIII and left axis deviation. Weinshel et al. (1951) found that, after right pneumothorax the electrical position of the heart became more vertical and the transition zone shifted to the left in 5 out of 9 cases; it is not possible to explain this without more data. In left pneumothorax the amplitude of the QRS deflection in the precordial leads was diminished; this was due to low voltage resulting from interposition of air between the heart and the electrode. Pneumoperitoneum caused a shift to the left and counter-clockwise rotation as expected. Weiss (1951) in a study of 10 cases of pneumoperitoneum and pneumonectomy thought that the therapeutic measures themselves had little effect on the electrocardiogram, but that the changes found were the result of subclinical cor pulmonale; these findings are not borne out by the present study. Rubin and Most (1952) studying 12 patients with pneumoperitoneum found that 7 cases developed deep Q waves...
in the oesophageal leads; this is due to forward rotation of the apex and upward displacement of the heart so that the oesophageal electrode would be placed below the level of the heart.

It will be seen that in most cases an explanation is possible on the hypothesis that has been put forward. A full explanation cannot be expected in the absence of complete data in each case including a twelve-lead electrocardiographic study before and after the carrying out of the therapeutic measure and this is not available in many of the previous publications.

**Summary**

A number of cases were studied electrocardiographically before and after undergoing pneumoperitoneum, artificial pneumothorax, or phrenic crush.

It is submitted that the main changes were the result of the mechanical effects produced by the therapeutic procedure employed.

I have to thank the patients for their co-operation, Dr. W. J. Vickers, the Director Medical Services, Singapore, for allowing me to take electrocardiograms of patients at Tan Tock Seng Hospital, Dr. R. J. Grove-White for his valuable assistance in the collection of data at Tan Tock Seng Hospital, the X-Ray Departments of the General Hospital, Tan Tock Seng Hospital and of the Singapore Anti-Tuberculosis Association for the skiagrams, and Miss Lim Kim Chwee for having taken the electrocardiograms.

**References**