Studies of Venous Pressure, Vital Capacity, Circulation Times and Electrocardiograms in the Course of Pulmonary Collapse Therapy*

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INTRODUCTION

This study was stimulated through the observation, by one of us (L.H.H.) of the appearance of the picture of right-sided heart failure in two patients who had been receiving artificial pneumothorax. Our purpose was to see if we could detect, early in the course of collapse therapy, any evidences of right heart strain. In addition to clinical observations we employed certain laboratory procedures which will be discussed below. Other similar studies have been carried out in this regard but as yet there has been nothing to demonstrate definite evidence of cardiac strain induced by collapse therapy.

Kroetz (quoted by Heise and Steidls) in 1922 was apparently the first to study the venous pressure of patients before and after pneumothorax. Greiletly-Bosirel (quoted by Heise and Steidls) found that as a rule the establishment of pneumothorax produces no modifications of venous pressure. Gerdier (quoted by Heise and Steidls) concluded that in pneumothorax venous pressure was a valuable indication of circulatory obstruction not always detected by clinical observation. He stated that venous pressure could predict that the collapse should be interrupted even when auscultation, x-ray studies and the respiratory rhythm failed to forecast disaster. However, others1-9 have failed to note any significant changes of venous pressure during collapse therapy. Studies of the circulation time1,6,7 have consistently shown a decrease with induction of collapse therapy but nothing to indicate right heart failure. Dock and Harrison11 found in rabbits an initially increased volume flow of blood through the lungs and a delayed decreased volume flow. Electrocardiographic studies3,6,10,14,16 have shown some changes which have been ascribed largely to rotation of the heart or the presence of air in the chest interfering with conduction of the heart current. In our present study we have

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employed all of these laboratory tests to determine the effect of pulmonary collapse on the circulation.

Method

The patients studied all had pulmonary tuberculosis of varying degree. A total of 32 patients were studied. They were classified in four groups based on the extent of parenchymal involvement as seen on the x-ray film. Groups I and II were based on the standards of the N.T.A.* classification. Group III did not follow this classification strictly because we felt that these patients with a cavity greater than 4 cm. in diameter would still remain in Group II providing there was not other parenchymal involvement to exceed the amount of lung tissue in one complete lung. Group III included those patients with involvement greater than the total tissue of one lung and Group IV included those patients with diffuse involvement of both lungs.

In the above groups there were thirty patients with artificial pneumothorax and two with unilateral phrenicotomy. All studies were carried out in a room at a uniform temperature of 26° C. The patient was allowed to rest for a full fifteen minutes before the tests were begun. The patient was first fluoroscoped and then vital capacity was determined using the McKesson apparatus and having the patient in an erect position. The venous pressure was then determined in the antecubital veins bilaterally, using an 18-gauge needle and reading the venous pressure against a column of 4 per cent sodium citrate solution. The level of the vein was adjusted approximately to the level of the right atrium using the angle of Louis as a measuring point. Two per cent novocaine was injected intradermally at the site of insertion of the measuring needle. All pressures were determined in the left arm first and then in the right. With the right needle in place a mixture of five cubic centimeters of 20 per cent decholin solution and five minims of ether was injected rapidly into the vein. The patient was instructed to indicate immediately when the sensations of ether and a bitter taste were perceived. The responses were timed by stop watch. Electrocardiograms were then taken with the patient in a recumbent position. None of these patients had any elevation of temperature above 99° F. at the time of the tests. They were all thoroughly instructed in what to expect, thus eliminating fear. All procedures were carried out on the same day. Control studies were carried out about a week after the patient's admission to the hospital. Follow-up studies were then done one month and five months following the institution of collapse therapy.

*National Tuberculosis Association.
Results

Of the 32 patients, 12 were in Group I, 9 were in Group II, 7 were in Group III, and 4 were in Group IV, according to the above-mentioned classification.

Vital Capacity of the Lung

The study of the vital capacity in these patients revealed a progressive linear decrease with the increased degree of collapse. These findings are well illustrated in Table I. As can also be noted there apparently was no significant reduction in the control vital capacity of the lung until the class three and four cases were studied.

Venous Pressure and Circulation Times

In Table II we indicate the average circulation times and venous pressure ranges in the four classes of cases. It will be seen that there is a definite decrease in the circulation time in Group III and IV and definitely low venous pressure in Groups III and IV. The decrease in circulation time was accounted for chiefly by changes in the pulmonary circulation time. The changes incident to the establishment of pulmonary collapse are noted in Table III. The degrees of increase or decrease are noted as measured against the average control values for each class. There was no significant

| TABLE I |
|------------------|--------|--------|--------|--------|
| Class of Disease | 1 Per cent | 2 Per cent | 3 Per cent | 4 Per cent | All Groups Per cent |
| Average Control* Vital Capacity | 96 | 87.3 | 82.2 | 71 | 84 |
| Average Reduction in Vital Capacity | -20 | -24.3 | -18.2 | -18 | -20 |
| One Month Following Institution of Collapse Therapy | or | or | or | or | or |
| Average Reduction in Vital Capacity | 69 | 57 | 48 | 34 | 52 |
| Five Months Following Institution of Collapse Therapy | or | or | or | or | or |

*Throughout this study the average has been calculated by simple arithmetical division of the total for all cases divided by the number of cases. The differences between the individual cases was so little that we felt this would be sufficient in determining the significance of our findings. The few instances in which there was greater deviation are discussed in the text of the paper.
difference in the venous pressure between the right and left arms either before or after collapse. In Groups I and III there was definite decrease in circulation time which seemed to be chiefly accounted for by a decrease in the ether time. This was much less marked in Groups III and IV, the possible explanation for which will be noted later.

It will be seen that the changes which occurred in circulation time at the end of one month of collapse therapy remained about the same at the end of five months, although in many instances the degree of collapse was greater at the end of five months.

Electrocardiographic Changes

In general the changes were not marked and certainly were not consistent with any definite evidence of cardiac strain. There was no apparent correlation of the degree of pulmonary collapse, extent of the disease, the degree of mediastinal shift or the side of the collapse with any specific change in the tracings. Alterations in venous pressure or circulation time also had no consistent bearing on the appearance of the cardiogram.

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<th>TABLE II</th>
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<td>Circulation Time</td>
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<td>Class</td>
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<tr>
<td>Ether</td>
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<td>Decholin</td>
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<td>Venous Pressure</td>
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<table>
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<th>TABLE III</th>
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<tr>
<td>Average Change in Circulation Time</td>
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<td>Class</td>
</tr>
<tr>
<td>Ether</td>
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<tr>
<td>Ether</td>
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<td>Decholin</td>
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<table>
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<th>Average Change in Venous Pressure</th>
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<tr>
<td>1 mo.</td>
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<td>5 mo.</td>
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<tr>
<td>No</td>
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<td>+0.6 cm.</td>
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<td>-0.7 cm.</td>
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The average cardiac rate in these 32 cases was 88 per minute. Eleven of them were below 80 and some of these had far advanced disease. At the end of one month of collapse therapy there was uniform increase in the heart rate. At the end of five months, 13 patients still had increased heart rates, 7 were decreased, and 15 were unchanged. Again there seemed to be no correlation between the changes in heart rate and the stage of the disease. None of the patients developed any arrhythmia. Four cases showed development of some right axis deviation. This was most pronounced in a patient who had a minimal mitral lesion in addition to his tuberculosis. He has been receiving pneumothorax now for 16 months and has shown no clinical evidence of right heart strain. In three of these cases the pneumothorax was on the right side. In one case it was bilateral.

The most consistent changes observed at the end of one month of institution of collapse therapy were:
1. Decrease in the voltage of the R wave in 17 cases in Lead I and in 16 cases in Lead IV-F;
2. Decrease of the T wave in 15 cases in Lead I and in 18 cases in Lead IV-F;
3. In 9 cases, a decrease of T wave in Lead II;
4. In 10 cases, an increase of the R wave in Lead III which was accompanied in 5 cases by an increase in the S wave of Lead I;
5. In 6 cases, a decrease of the T wave in Lead III;
6. No change in 4 cases.

At the end of five months no further consistent changes were observed. An interesting observation was that the changes in Lead I seemed to be mirrored by the changes in Lead IV-F.

Discussion of Results

As stated at the outset of this paper, we attempted to demonstrate early evidence of right heart strain in the institution of collapse therapy. As can be gleaned from our results there is no evidence of right heart failure so far as these cases have been followed by the procedures herein utilized. It may be added that in no case was there clinical evidence of congestive failure, although some of the patients complained of dyspnea.

Studies of the vital capacity revealed an almost linear reduction with the increase of pulmonary collapse. This is quite obvious since the vital capacity is dependent on the volume of respiratory surface. This finding has also been pointed out by Stewart and Baileye and Feinsilver. We feel that the determination of the vital capacity is of little value in determining the presence of involvement of pulmonary tissue unless this is rather extensive. Apparently the uninvolved portions of lung can compensate greatly until so much
lungs is involved as in the Group III and IV cases that there is a significant diminution in respiratory reserve with consequent reduction in vital capacity. These findings are corroborated by others.\(^3\)

On the whole, these patients in their control studies exhibited normal circulation times, using 13 seconds for the decholin time and 7 seconds for the ether time as normals. There were occasional patients in whom the circulation time seemed to be a little prolonged but there were no particular factors with which to correlate these findings. The circulation times in the Groups III and IV patients seemed to be more rapid. The fact that this was chiefly accounted for by a decrease in the ether time would suggest that extent of involvement of the pulmonary tissue may have sufficiently diminished the size of the vascular bed to account for the above reduction. The circulation times were also uniformly decreased with collapse therapy. This seemed to be most marked during the first month and less at the end of five months. The fact that this was less marked in the Group III and IV cases was probably due to the greater difficulties in obtaining good collapse in these cases. The changes again were due principally to reduction in the ether time and again we explain the changes through a reduction in the pulmonary vascular bed. The fact that these changes are less marked at the end of five months in view of increased pulmonary collapse can be explained only on the basis of change in heart rate since at the end of one month the heart rates were uniformly increased over the control levels, whereas at the end of five months in only less than half of the cases was there still an increase in heart rate. Feinsilver\(^1\) has noted essentially similar findings. Others\(^6,7\) have noted very little change in the circulation times.

Venous pressure readings in the controls were essentially normal. They tended to be generally lower in the Group III and IV cases. It has been pointed out\(^13\) that there is some evidence of emaciation being a factor in lowering of venous pressure. This is the only factor we could evolve as an explanation for the above since such things as vis-a-tergo or intrathoracic pressure could not be demonstrated to be essentially different in the Group III and IV and in the Group I and II cases. With collapse therapy, there was in general a slight rise in venous pressure at first but this promptly subsided. These changes were not large enough to be considered of any significance. In one of the Group III cases, there was a significant rise of venous pressure bilaterally (3 cm.), which was maintained at the end of five months. However, we could demonstrate no evidence of right heart failure to explain this. There was one other patient in Group I who had a rise in venous pressure of 3 cm. bilaterally at the end of one month but this promptly
returned to normal levels at the end of five months. There was no mediastinal shift or any particular adhesions to explain the temporary but definite rise in this case. It has been found by others\textsuperscript{1,6,9} that there were no significant changes in venous pressure incident to collapse therapy. We noted that the degree of mediastinal shift has no particular effect on the venous pressure. A few cases with marked pleural effusion and pronounced mediastinal shift showed little or no alteration in the venous pressure.

Hurst and Brand\textsuperscript{4} have pointed out that high tension pneumothorax may cause a rise in venous pressure on the same side, with or without mediastinal shift. They feel that venous pressure changes occur on the side of the more extensive tuberculous lesion or collapse measure and explain it on the basis of local obstruction of the subclavian or innominate vein. This phenomenon did not occur in our studies so far as we have carried them. It is not surprising that we did not observe more changes indicative of increased tension in the pulmonary circuit since it has been pointed out\textsuperscript{13} that the vascular bed of the lungs must be reduced by about 60 per cent before the pressure is increased in the lesser circulation.

The electrocardiographic studies revealed even less evidence of any cardiac strain than did the above discussed procedures. As previously mentioned the most consistent findings were decrease of the R and T waves in Lead I and IV-F. As Master\textsuperscript{14} has suggested the low voltage of the Q-R-S complex and the T waves is probably to be accounted for either by the rotation of the heart or by the fact that air in the chest is a poor conductor for the heart current. We are inclined to lean to the latter explanation since in these cases we were not able to demonstrate actual cardiac rotation fluoroscopically. In four patients there was development of right axis deviation, the pneumothorax being on the right side in three cases and bilateral in one case. In Master's\textsuperscript{14} cases all of the patients with right pneumothorax showed a right axis deviation of the Q-R-S complex. He feels that in right pneumothorax particularly that a rotation of the heart occurs and probably accounts for the above. He followed two cases who showed disappearance of the above changes with disappearance of the pneumothorax. His study, however, was concerned with only seven cases, five of which were spontaneous pneumothorax. He found the most marked changes with displacement of the mediastinum. We could not conclude that in one case with marked mediastinal shift that any greater change was noted in the electrocardiogram. Anderson\textsuperscript{3} found that neither the degree of pulmonary collapse maintained nor the duration of the collapse therapy had any definite relationship to the form of the electrocardiogram. Again his study was different from ours in that he did not compare the
electrocardiograms of the same patient before and after pneumothorax. Hansen and King\textsuperscript{16} felt that the change observed, most frequently in the R and T waves, were due to change in position and not myocardial damage. Similar conclusions have been reached by others.\textsuperscript{6} Boas and Mann\textsuperscript{15} feel however that displacement of the heart in their experience had no effect on the result of the electrocardiograph. Our studies seemed to corroborate the latter statement. Simon and Baum\textsuperscript{10} consider the presence of adhesions as important in producing predominance of either ventricle. This could not be demonstrated in our series. It appears that in those cases in which axis deviation appeared that it was due to rotation of the heart. The change in the R and T waves was most likely on the basis of presence of air in the chest.

SUMMARY

Simultaneous studies of the venous pressure, circulation time, vital capacity of the lung, extent of pulmonary involvement, and electrocardiograms have been made in a series of 32 cases with pulmonary tuberculosis.

These studies were carried out as controls and again at the end of one month and five months after institution of collapse therapy. In 30 cases collapse was induced by artificial pneumothorax and in two cases by phrenicotomy. Factors of emotion, environmental temperature, and body temperature were noted and controlled. The control studies were carried out after the patient had been in the hospital for a week.

The purpose of the study was to try to demonstrate any evidence of right heart failure early in the course of collapse therapy. These patients were observed clinically as well as through the laboratory.

CONCLUSIONS

1) In no case was there clinical or laboratory evidence of right heart strain.

2) Decrease of vital capacity of the lung is directly proportional to the degree of collapse. Vital capacity does not seem to be materially affected in the controls until there is extensive pulmonary involvement.

3) The venous pressure is not significantly affected by pneumothorax or phrenicotomy.

4) The circulation times are decreased initially by pulmonary collapse chiefly due to a decrease in the ether time. It is suggested that this results from a decrease in the pulmonary vascular bed and an increase in heart rate.

5) The electrocardiogram has shown no evidence indicative of
right heart strain. The changes noted apparently are due to rotation of the heart and presence of air in the chest.

6) Only through more prolonged studies of these patients, may it be possible to detect any changes in the procedures herein employed which may indicate evidence of right heart failure before frank clinical features appear.

RESUMEN

En una serie de 32 casos de tuberculosis pulmonar se han llevado a cabo estudios simultáneos de la presión venosa, velocidad de circulación, capacidad vital del pulmón, extensión de la invasión pulmonar y electrocardiografías.

Se hicieron estos estudios, primero para que sirvieran de pauta, y se repitieron después de un mes y después de cinco meses de haberse iniciado la colapsoterapia. En 30 casos la forma de colapso empleada fue el neumotórax artificial, y en dos casos la frenicotomía. Se tomó en cuenta y se corrigió el factor emocional y, también, la temperatura del ambiente y del cuerpo. Se llevaron a cabo los estudios reguladores después de que el paciente había estado una semana en el hospital.

El propósito del estudio fue el de tratar de demostrar tempranamente en el curso de la colapsoterapia signos de insuficiencia del corazón derecho. Se observó a estos pacientes tanto clínicamente como con exámenes de laboratorio.

CONCLUSIONES

1) En ningún caso hubo signos clínicos o de laboratorio perjudiciales al corazón derecho.

2) La disminución de la capacidad vital del pulmón es directamente proporcional al grado de colapso. No pareció ser afectada notablemente la capacidad vital de los reguladores sino cuando existía invasión pulmonar extensa.

3) Ni el neumotórax ni la frenicotomía afectan importantemente la presión venosa.

4) El colapso pulmonar causa inicialmente una disminución en el tiempo de circulación debida, principalmente, a disminución del tiempo del éter. Se supone que esto es debido a disminución en la red vascular pulmonar y a aumento en el número de latidos del corazón.

5) La electrocardiografía no ha revelado ningún signo indicativo de daño al corazón derecho. Las alteraciones observadas se deben aparentemente a la rotación del corazón y a la presencia de aire en el tórax.

6) Sólo mediante estudios más prolongados de estos pacientes
podría ser posible descubrir en los procedimientos aquí empleados alteraciones que pudieran indicar insuficiencia del corazón derecho antes de que aparezcan francos signos clínicos.

REFERENCES


14 Master, A. M.: "Electrocardiographic Changes in Pneumothorax in which Heart has been Rotated; Similarity of Some of these Changes to those Indicating Myocardial Involvement," Am. Heart J., 3:472 (April), 1938.
