Venous Pressure as a Guide to Pneumoperitoneum Therapy in Pulmonary Emphysema*

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The striking improvement of symptoms in most patients with pulmonary emphysema treated with pneumoperitoneum has been correlated with changes in pulmonary function by Carter, Gaensler et al.1,2 Although the circulatory changes in pulmonary emphysema have been the subject of intensive investigation,3-7 there has been no report, so far, dealing with the hemodynamic effects of pneumoperitoneum in this disorder. The measurement of the venous pressure in the upper extremity in the course of pneumoperitoneum administration seemed to be one of the simplest methods to determine relative intrathoracic pressure changes. The results of these determinations are the subject of this preliminary report.

Methods

An unselected group of 10 patients, with marked pulmonary insufficiency secondary to far advanced pulmonary emphysema, was treated with pneumoperitoneum. In the course of their treatment, venous pressure determinations were performed during the administration of pneumoperitoneum. A calibrated Statham pressure transducer was used for continuous pressure registration which was recorded through a Sanborn Strain Gauge Amplifier on a Sanborn Visocardiette. The zero point of the transducer was chosen at a point 5 cm. below the costo-chondral junction of the second rib of the sternum. We were aware of the fallacy of this method for the determination of the level of the right auricle in patients with an increased anterior-posterior diameter of the chest due to pulmonary emphysema. Since we are, however, mostly interested in changes of venous pressure rather than absolute values, this method seemed adequate for our purpose. Peak pressures in inspiration and expiration and planimetrically calculated

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mean pressures were averaged from the record during a control period of 15 minutes, following 30 minutes of complete bed rest, and after stabilization of the record following introduction of 300, 600, 900, 1200 and 1500 cc. of air in the abdomen. The symptom changes occurring during the administration of pneumoperitoneum were noted for correlation with the venous pressure changes. In two patients the experimental procedure was repeated and a total of 13 experiments will be reported.

Results

Of 13 experiments there were six in which an average fall in mean venous pressure of 19 mm. of water (range -12 to -37 mm. water) occurred in the course of pneumoperitoneum. In three instances the change did not exceed ± 10 mm. of water (range -5 to +9 mm. water) (Figures 1, 2, 3, and 4).

In four experiments there was an average rise of mean venous pressure of 29 mm. of water (range plus 18 to plus 56 mm. water). In one patient with a significant drop in venous pressure at one time repetition of the experiment showed no change on another occasion. A second patient demonstrated no significant mean venous pressure change on one occasion and a significant rise during a subsequent determination.

FIGURE 1

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM IN TWO PATIENTS WITH EMPHYSEMA

PATIENT WITH POOR RESPONSE TO PNEUMOPERITONEUM

PATIENT IMPROVED

VENOUS PRESSURE

150 -
140 -
130 -
120 -
110 -
100 -
90 -
80 -
70 -
60 -
50 -
40 -
30 -
20 -
10 -

mm H2O

0 300 600 900 1200 1500

CC AIR INTRODUCED
In eight experiments a rise occurred when 600 cc. of air or more was introduced into the abdomen. In only two cases did the rise exceed the control value of venous pressure (Figure 5).

All seven patients in whom nine experiments showed a drop or no change in venous pressure initially during pneumoperitoneum administration responded very well to this form of treatment clinically. There was markedly decreased dyspnea on rest and effort. In one of this group pneumoperitoneum had to be abandoned because of air in the scrotum. The one patient whose venous pressure did not change when pneumoperitoneum was first initiated had to be discontinued three months later, subsequent to the determination, during which he manifested a rise in venous pressure, because of increasing dyspnea following the introduction of air in the abdomen.

In the remaining three patients who manifested rises in venous pressure following pneumoperitoneum, this procedure had to be abandoned early because of severe respiratory embarrassment following the procedure. Two of these patients had slightly elevated venous pressures during the control period and in all three the

**FIGURE 2**

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM
IN TWO PATIENTS WITH EMPHYSEMA

<table>
<thead>
<tr>
<th>CC AIR INTRODUCED</th>
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</thead>
<tbody>
<tr>
<td>0</td>
</tr>
<tr>
<td>V.P. IN EXP.</td>
</tr>
<tr>
<td>V.P. IN INSPI.</td>
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<tr>
<td>MEAN V.P.</td>
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<table>
<thead>
<tr>
<th>VENOUS PRESSURE (MM H2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>180</td>
</tr>
</tbody>
</table>

**PATIENT WITH POOR RESPONSE TO PNEUMOPERITONEUM**

**PATIENT IMPROVED**
FIGURE 3
MEAN VENOUS PRESSURES DURING PNEUMOPERITONEUM
IN PATIENTS WITH PULMONARY EMPHYSEMA

--- POOR RESULTS, TREATMENT DISCONTINUED
--- IMPROVED CASES

FIGURE 4
VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM

<table>
<thead>
<tr>
<th></th>
<th>BEFORE PNEUMOPERITONEUM</th>
<th>PNEUMOPERITONEUM 900cc FILL</th>
<th>PNEUMOPERITONEUM 1200cc FILL</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTRA-ABDOMINAL PRESSURE</td>
<td>+5 +7</td>
<td>48.4 mm</td>
<td>53.6 mm</td>
</tr>
<tr>
<td>Inspiratory V.P.</td>
<td>63.8 mm</td>
<td>76.1 mm</td>
<td>81.3 mm</td>
</tr>
<tr>
<td>Expiratory V.P.</td>
<td>50.9 mm</td>
<td>48.4 mm</td>
<td>53.6 mm</td>
</tr>
<tr>
<td>Venous Pulse Pressure</td>
<td>38.7 mm</td>
<td>27.7 mm</td>
<td>27.9 mm</td>
</tr>
<tr>
<td>Mean V.P.</td>
<td>63.9 mm</td>
<td>62.1 mm</td>
<td>673 mm</td>
</tr>
</tbody>
</table>
venous pressure rose to 120 mm. of water or more following the introduction of air into the abdomen.

Increased dyspnea also occurred in the group which responded well to treatment when in seven of the nine experiments a venous pressure rise occurred with increased air in the abdomen, after the venous pressure had reached its optimum low level. The symptoms could be alleviated by removal of some of the excess air from the abdomen at the end of the procedure.

Discussion

By introducing 100 cc. of air into the peritoneum of normal animals, Overholt has shown that the intra-abdominal pressure can be raised from subatmospheric levels to above atmosphere. Pressure tracings revealed inspiratory and expiratory intra-abdominal pressure changes which paralleled the respective intrapleural pressures. The presence of intrathoracic hypertension in emphysema was demonstrated experimentally by Kauntz and his group when they obtained an increase in intrapleural pressure.

FIGURE 5

VENOUS PRESSURE CHANGES DURING PNEUMOPERITONEUM IN A PATIENT WITH PULMONARY EMPHYSEMA
and venous pressure in dogs with partial obstruction of expiration over a period of weeks, in which partial enlargement of the lungs occurred. Duomarco and his collaborators\textsuperscript{10} demonstrated the existence of abdominal hypertension in subjects with intrathoracic hypertension, thus demonstrating a close relationship between intrapleural and intra-abdominal pressure in patients with uncomplicated emphysema.

A discrepancy between changes in intra-abdominal and intrathoracic pressure in patients with pulmonary emphysema was observed by Alexander.\textsuperscript{11} A belt was applied to the lower part of the abdomen, thus increasing the intra-abdominal pressure in emphysematous patients. The elevation of the diaphragm caused increased excursion of the leaves and restoration of the previously elevated intrapleural pressure to normal subatmospheric levels. This change was accompanied by a drop in venous pressure. Symptomatic relief occurred in these patients.

In some cases of emphysema, pneumoperitoneum or increased abdominal pressure exerted by an abdominal belt produces a lowering of the venous pressure as a result of decreasing intrapleural pressure. A more advantageous mechanism for ventilating the lungs is produced in that the ventilating cycle is accomplished by contraction of the uplifted diaphragm rather than by contraction of abdominal muscles and lower intercostals to create increased intra-abdominal pressure. Ventilation then occurs with an intrapleural pressure swing below the atmosphere rather than partly above and partly below. In these cases contraction of the diaphragm is followed by expansion of the lung.

In those cases in which the venous pressure is elevated, the uplifted diaphragm does not contract and elevate, either because of atrophic changes in the diaphragm or inadequate elasticity in the lungs. The pressure is, therefore, transmitted to the lungs and initiates effects similar to pressure breathing. In a paper by Barach, Martin and Eckman\textsuperscript{12} in which venous pressure changes were observed as a result of pressure breathing, an increase in venous pressure of 40 per cent of the applied pressure was found in normal subjects, but in patients with congestive heart failure, in whom there was decreased pulmonary elasticity, the rise in venous pressure was in the neighborhood of 70 per cent of the applied pressures. In addition, marked prolongation of circulation time took place in the subjects with heart disease. It would appear likely that patients in whom a rise in venous pressure, instead of a fall, took place after pneumoperitoneum or after an excess of air was introduced, would endure the same physiologic effects as pressure breathing, namely, a blockade of blood into the right heart with a slowed circulation time and decreased cardiac output.
Under these circumstances, a stagnant type of anoxia would contribute to dyspnea. The factor of the function of the right heart in delivering blood to the lungs at an increased intrapulmonary pressure must also be considered. In those instances in which right ventricular insufficiency is present, either overt or latent, the increased venous pressure may develop in part as a result of inability to empty the right ventricle under conditions of increased intrapulmonary pressure.

This is in agreement with the observations of Gillanders, who found that no rise in venous pressure was obtained when pressure was applied to the abdomen of normal or uncomplicated emphysematous subjects; whereas, in the presence of right side heart failure in patients with emphysema, this maneuver led to a marked rise in venous pressure.

Training in diaphragmatic breathing has recently been emphasized as a factor of considerable value in the treatment of patients with pulmonary emphysema. Conceivably, the decreased tone of the diaphragm in those cases in which diaphragmatic breathing has not taken place, may account for the failure of the development of a negative pressure or an increased negative pressure within the lungs after pneumoperitoneum. If increased diaphragmatic training were to take place, patients of this kind may, on subsequent trials of pneumoperitoneum be found to benefit by the procedure as a result of more efficient contraction of the elevated diaphragm.

SUMMARY

1) Venous pressures were determined during the administration of pneumoperitoneum in 10 patients with pulmonary emphysema.

2) Six patients showed a consistent drop or no change in venous pressure. All derived excellent benefits from pneumoperitoneum.

3) In three patients in whom a rise in venous pressure was recorded, pneumoperitoneum had to be discontinued because of respiratory embarrassment following it.

4) One patient who on one occasion had no change in venous pressure tolerated pneumoperitoneum well for three months. Subsequently, dyspnea became so incapacitating following the procedure that it had to be abandoned. A rise in venous pressure was noted during the last pneumoperitoneum.

5) An increase in diaphragmatic excursion is felt to be responsible for the lowering of venous pressure in the successful cases of pneumoperitoneum therapy.

6) Direct transmission of the intra-abdominal pressure increase to the intrapleural space through a fixed atrophic diaphragm, in the presence of markedly reduced pulmonary elasticity, blocks the...
flow of blood into the right heart with resultant increased venous pressure, decreased right cardiac output and stagnant anoxia. This accounts for the respiratory embarrassment in these patients following pneumoperitoneum.

7) Changes in venous pressures during pneumoperitoneum seem to be a good prognostic index for emphysematous patients in whom this form of treatment is contemplated.

RESUMEN

1) Las presiones venosas fueron determinadas durante la administración de neumoperitoneo, en 10 pacientes con enfisema pulmonar.

2) Seis enfermos mostraron una baja continua o ningún cambio en la presión venosa. Todos obtuvieron excelentes beneficios del neumoperitoneo.

3) En tres de ellos en los cuales se notó un aumento en la presión venosa, el neumoperitoneo tuvo que ser suspendido debido al impedimento respiratorio que siguió.

4) Un paciente que en una ocasión no sufrió cambio en la presión venosa, toleró el neumoperitoneo bien por tres meses. Más tarde, la disnea vino a ser tan incapacitante al seguir el procedimiento, que éste tuvo que ser abandonado. Un aumento en la presión venosa fue notado durante el último neumoperitoneo.

5) Se cree que un aumento en la excursión del diafragma es el responsable de la baja de la presión en los casos con éxito de terapéutica del neumoperitoneo.

6) La transmisión directa de la presión intra-abdominal aumenta hacia el espacio intrapleural a través de un diafragma atrófico fijo, en la presencia de una reducción marcada de la elasticidad pulmonar, perjudica el paso de la sangre al corazón derecho con el aumento resultante de presión venosa, decrecimiento del rendimiento cardíaco y la anoxia por étasis. Esto produce el impedimento respiratorio que siguió al neumo-peritoneo en esos pacientes.

7) Parece ser que los cambios en la presión venosa durante el neumoperitoneo son un buen índice de pronóstico para los pacientes enfisematosos en los cuales esta forma de tratamiento es usada.

RESUME

1) On a mesuré la pression veineuse au cours de l’exécution d’un pneumopéritoine chez 10 malades atteints d’emphysème pulmonaire.

2) Chez six malades, on constata soit une chute nette, soit une absence de transformation de la pression veineuse. Tous tirèrent un très grand bénéfice de l’action du pneumopéritoine.
3) Chez trois d'entre eux, on constata une augmentation de la pression veineuse. Il fallut alors cesser le pneumopéricône, car il était suivi de troubles respiratoires.

4) Chez l'un des malades, qui n'avait pas eu de modification de la pression veineuse, le pneumopéricône fut parfaitement toléré pendant trois mois. Ulterieurement la dyspnée consécutve au pneumopéricône était telle qu'on dut l'abandonner. Au cours de la dernière insufflation du pneumopéricône, on constata une augmentation de la pression veineuse.

5) Les auteurs pensent que l'augmentation de l'élévation diaphragmatique est la cause de l'abaissement de la pression veineuse dans les cas où le pneumopéricône a donné de bons résultats.

6) La transmission de l'augmentation de la pression abdominale se propage à l'espace intra-péritonal à travers le diaphragme s'il est fixé et atrophiqne, et la réduction notable de l'élasticité pulmonaire plaque le flux sanguin dans le coeur droit. Il en résulte une augmentation de la pression veineuse, une diminution de débit du coeur droit et une anoxie de stase. Ceci rend compte des troubles respiratoires qui sont, chez ces malades, consécutifs au pneumopéricône.

7) Les modifications de la pression veineuse au cours du pneumopéricône semblent être un élément favorable de pronostic chez les malades emphysémateux qui sont soumis à ce traitement.

REFERENCES


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Discussion
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I have been using pneumoperitoneum for this purpose in my office and in the emphysema clinic at Mount Sinai Hospital in Milwaukee for the past two years. Venous pressure measurements have been made with a water manometer before and periodically during the pneumoperitoneum regimen. Our experience parallels that he presented.

With reference to the volume of the pneumoperitoneum inflations we have arrived at the same conclusions empirically as the authors. That is to say, patients derive greater benefit from small inflations than large.

Of 17 patients with chronic so-called hypertrophic pulmonary emphysema 11 received pneumoperitoneum. One suffered acute appendicitis after six months of pneumoperitoneum. Inflations were necessarily discontinued during surgery and convalescence. They were reestablished four weeks after appendectomy. In the interim, there was recurrence of symptoms of emphysema.

All 11 have enjoyed a favorable response to pneumoperitoneum. One has reported slight relief and 10 enjoy satisfactory relief. To offer an idea of satisfactory relief, may I cite the following cases: One became totally disabled with emphysema and was obliged to sell his welding shop and his home. He moved to a trailer and was unable to walk its length without dyspnea and cyanosis. Today he is building a house. A few weeks ago he changed the motor in his car. Another with emphysema spent an entire year before pneumoperitoneum confined to his home with oxygen continuously available. Since pneumoperitoneum he has dispensed with the oxygen tank. At first he required transportation to the clinic in an automobile, but now he comes in a street car.

One may readily understand the rationale of therapeutic pneumoperitoneum when considering the anatomic and functional changes characteristic of pulmonary emphysema: (1) There is destruction of the perialveolar and peribronchial elastic fibers; (2) there is rupture of large numbers of alveoli; (3) the intra-
pleural negative pressure becomes less negative or even atmospheric; (4) there is a loss of the contralateral contractility of the lung; (5) the diaphragm occupies a constant low inspiratory position. It is well known that the diaphragm is responsible for from 37 to 47 per cent of the ventilatory function of the lungs; (6) the return of the venous blood from the periphery to the right side of the heart is decreased; and (7) the blood flow from the right side of the heart to the lung is diminished.

Dyspnea and cyanosis result from the loss of functionally competent alveoli, dislocation of the diaphragm downward, and interference with the normal circulation of the blood from the periphery to the heart and from the heart to the lung.

Empirical observations prove that artificial pneumoperitoneum corrects the functional derangements responsible for the clinical manifestations of pulmonary emphysema. This may be partial or complete. Injection of moderate amounts of air into the abdominal cavity causes a rise of the diaphragm which is brought about by the following factors: (1) Neutralization of the sub-diaphragmatic negative pressure. (2) Support of the pulling effect of the intrapleural negative pressure. (3) The diaphragm is displaced upward by the positive intra-abdominal pressure.

When the diaphragm approaches or is restored to its normal position the intrapleural pressure becomes more negative than prior to treatment. The diaphragm regains its piston-like motion during the two respiratory phases. The venous blood flow from the periphery to the heart is facilitated together with an accelerated flow of blood from the right side of the heart to the lung.

These changes are easily demonstrated clinically and herein lies the rationale for the use of therapeutic pneumoperitoneum in hypertrophic pulmonary emphysema.