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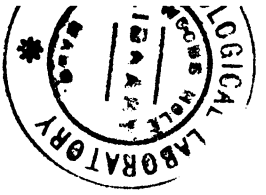

STUDIES OF THE ELASTIC PROPERTIES OF THE THORAX OF SUPINE ANAESTHETIZED PARALYSED HUMAN SUBJECTS

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When an elastic structure such as the thorax is inflated its elastic tissues are stretched. The recoil force produced by these tissues can be determined by measuring the inflating pressure within the airways. By recording the pressure at different inflation volumes a pressure-volume diagram may be constructed.

Rahn, Otis, Chadwick & Fenn (1946) made use of this principle when they studied the elastic properties of the thorax in conscious human subjects by determining the relaxation pressure curve. After the inspiration of different volumes of air, the airway is occluded; the respiratory muscles are then deliberately relaxed and the recoil pressure (relaxation pressure) which then develops is measured. In our experience there is a wide scatter of the individual pressure-volume points on repeated measurements. It would seem therefore that even trained and co-operative subjects are unable to relax their respiratory muscles completely or to the same extent at all times when pressure-volume measurements are being made.

Nims, Conner & Comroe (1955) have reported that the compliance of the thorax was lower in anaesthetized patients made apnoeic in various ways than in the same subjects when conscious; they suggested that some inspiratory activity was present during the pressure-volume determinations in the conscious state.

The present study of the thoracic compliance in anaesthetized paralysed subjects was undertaken in the belief that the results would represent the true total thoracic compliance, i.e. when uninfluenced by muscle activity, and that reliable data would be obtained for the compliance of the thoracic cage itself about which little has been reported. Our results confirm and extend those of Nims *et al.* (1955); we have, however, been led to believe that the mechanism of inflation when the thorax is ventilated by positive pressure is different from

that occurring during natural breathing, or during the relaxation pressure manoeuvre. We suggest that it is probably not justifiable to compare compliance measurements made by positive pressure breathing in paralysed subjects with those obtained when the thorax is expanded by the action of the respiratory muscles.

METHODS

Measurement of thoracic compliance

Method **1**; *prolonged positive pressure inflation.* Twelve subjects were studied in the supine position before a planned surgical operation. None had clinical or radiological evidence of pulmonary or cardiovascular disease. All received premedication with pethidine 100 mg + scopolamine 1/150th gr. (0·43 mg), or with Omnopon 1/3rd gr. (22 mg) (Roche Products, Ltd., ≒ morphine gr. 1/4) + scopolamine 1/150th gr. (0·43 mg). Half to one hour later anaesthesia was induced with sodium thiopentone intravenously. Succinyl choline chloride 50 mg (Scoline, Allen and Hanburys) was then administered and a cuffed endotracheal tube was inserted. The lungs

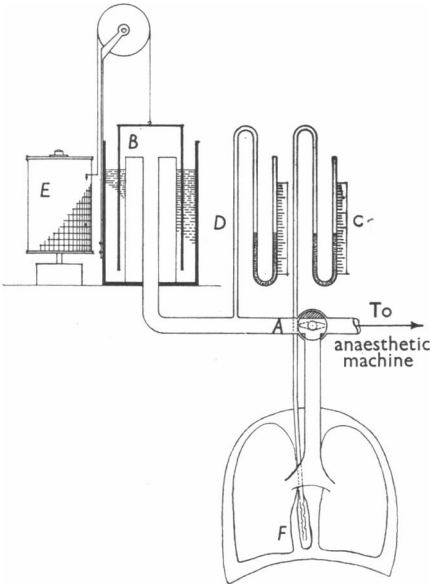

Fig. 1. The tap (*A*) enables the subject to be connected either with the anaesthetic machine or with the oxygen-filled spirometer (*B*), movements of which are recorded on the kymograph (*E*). Manual pressure on the spirometer bell results in inflation of the thorax, the recoil pressure of which can be recorded under static conditions from the water manometer (*D*). Changes in intra-oesophageal pressure are recorded on the water manometer (*C*) from a latex balloon.

were ventilated with pure oxygen for several minutes to reduce the alveolar nitrogen concentration. Complete muscular paralysis was then induced by intravenous injection of either succinyl choline 50–100 mg, tubocurarine 30 mg or gallamine triethiodide 80–100 mg intravenously. The subject was connected via the three-way tap *A* with the oxygen-filled spirometer *B* (Palmer) (Fig. 1). The pressure in the system was recorded with the water manometer *D*. The movements of the spirometer bell were recorded on the slowly moving kymograph *E*. There was a progressive

decrease in the volume of the spirometer as oxygen was absorbed by the subject by 'diffusion respiration' (Enghoff, Holmdahl & Risholm, 1951). The slope of the line recorded on the kymograph was an approximate measure of the rate of oxygen uptake. This sloping line and its projection were used as the base line for the measurement of all induced volume changes.

Inflation of the thorax to a new volume was produced by manual pressure on the spirometer bell which was then held steady. The reading was taken when the pressures in the system had become equilibrated as judged by the pressure in the water manometer *D* becoming approximately steady. A very slow fall in pressure did in fact continue to take place owing to the continuous oxygen uptake by the subject, but this further decline created no difficulty in determining the point of pressure equilibrium. Immediately after the reading had been noted the procedure was repeated by inflating the thorax to a new volume. In this way from five to seven pressure-volume measurements were made up to pressures of 20–22 cm H_2O.

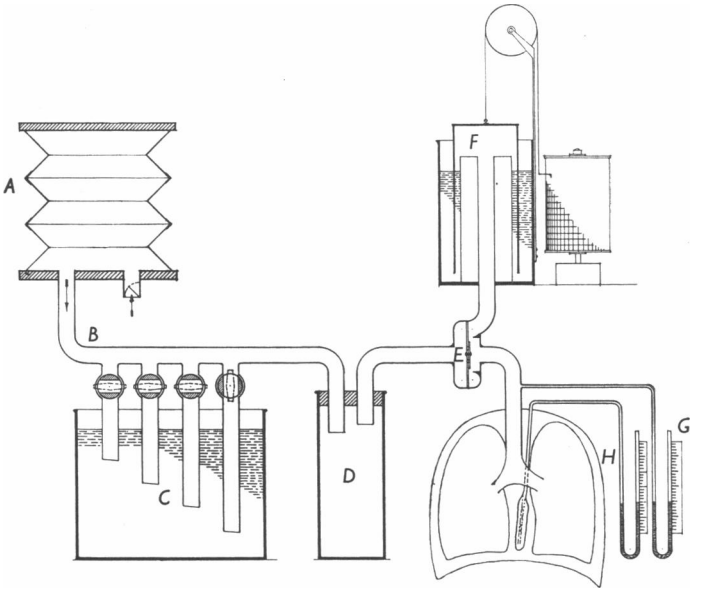

Fig. 2. The pump forces air through the water trap (*D*) and the B.O.C. valve (*E*) into the patient. Down-pipes fitted with taps lead from the tube (*B*) into water for varying depths (5–22 cm) (*C*). Opening one of these taps allows the blowing off of surplus air when the recoil pressure of the inflated thorax equals the depth of water in the down-pipe; the sustained intrapulmonary pressure during this period is recorded on the water manometer (*G*) and the change in intra-oesophageal pressure on the narrow-bore water manometer (*H*). During the inflow phase of the pump, the B.O.C. valve closes the connexion between the patient and the pump and opens a port enabling the subject to expire passively into the spirometer (*F*) recording on a kymograph.

The volume changes recorded on the kymograph were corrected for the water displacement which occurred in the spirometer owing to the pressure changes. This correction factor was obtained by closing tap *A* (to shut the spirometer off from the chest and the anaesthetic machine) and determining the pressure changes produced by different volume changes. No correction of the final volumes to body temperature and pressure, saturated with water vapour (BTPS), was made as the resulting change was so small as to be within the limits of experimental error of the method.

Method 2; *cyclical positive pressure inflation.* Another series of fifteen patients was studied in the supine posture before a planned surgical operation. The apparatus (Fig. 2), which was modified

from that described by Comroe, Nissell & Nims (1954), consisted of a pump *A* of fixed stroke volume (2·6 l.) set to cycle at 10/min (unless otherwise stated) with inflow and outflow phases of equal duration. The outlet from the pump was via a wide-bore brass tube (*B*) with 'down-pipes' extending into water for 5–20 cm (*C*). Each of the down-pipes was fitted with a tap; by selecting the tap to be opened the maximum pressure in the system could be varied. The brass tube led via a large water trap (*D*) to a B.O.C. valve (*E*) (British Oxygen Company's inspiratory/expiratory valve, Mark 2, ref. no. 304,378) which had an end-expiratory resistance of approximately 0·5 cm H_2O; thence a rubber tube led to the cuffed endotracheal tube inserted in the patient. During the compression (outflow) phase of the pump, the B.O.C. valve opened only to the patient, but during the return stroke (inflow) the valve closed the communication with the pump and opened a port, thus enabling the subject to expire passively into a well-balanced 6 l. Palmer spirometer (*F*). During the compression (outflow) phase of the pump, the recoil pressure of the thorax rose with inflation to a value equal to that of the height of the column of water in the down-pipe selected, after which the remainder of the air delivered from the pump was expelled through this down-pipe. The maximum pressure of inflation was thus maintained constant for a time (1–2·5 sec) which varied with the time taken for the recoil pressure of the thorax to reach the selected inflation pressure. A number of pressure-volume measurements were thus made. The pressure developed at the mouth during pump outflow was recorded by the manometer *G*, while the volume then introduced was recorded on the spirometer during the following passive expiration.

Measurement of intra-oesophageal pressure. Intra-oesophageal pressure measurements were made using a 70 cm polythene catheter with lateral perforations in the distal 10 cm which were surrounded by a thin latex rubber balloon whose diameters were 0·8 × 10 cm. The catheter was attached to a water manometer with a very small water displacement (0·03 ml./cm H_2O). The catheter was inserted in the oesophagus to a distance of 33–38 cm from the incisor teeth while enclosed in a large oesophageal tube which was subsequently withdrawn. The volume of air in the balloon was 1·0–1·5 ml. The accuracy of the balloon-catheter-manometer system was tested by comparing its ability to record pressure changes produced in a glass vessel containing the balloon and the distal part of the catheter, with those recorded directly from the glass vessel with a large bore water manometer. The readings were identical.

In four anaesthetized paralysed subjects in whom a thoracotomy was to be performed, a shallow pneumothorax was induced and the simultaneous changes in intra-oesophageal and intrapleural pressures were recorded at different volumes of inflation of the thorax.

Determination of the effect of anaesthesia and of muscular paralysis on the lung volume

In one subject in the supine posture we examined the effects on the lung volume of the induction of anaesthesia, and in three other subjects the effect of muscular paralysis on the lung volume. The studies were made before surgical operation and all the subjects received premedication as described previously.

The conscious subject breathed via a well-fitting anaesthetic mask from a closed circuit oxygen-filled system which included a 6 l. Palmer recording spirometer. The gas in the circuit was circulated at the rate of 40 l./min by means of a fan. Carbon dioxide was absorbed in a soda-lime container situated beyond the outlet from the fan. This system obviated the use of valves and thus had a negligible resistance to breathing. The spirogram of the subject was recorded in the conscious state and during the induction of anaesthesia with intravenous sodium thiopentone. The effect on the anaesthetized subject of muscular paralysis produced by intravenous succinyl choline chloride was recorded in the same way.

Measurement of lung compliance in conscious supine subjects

Four subjects were studied within 3 hr before their surgical operation. The supine subject breathed from a closed circuit oxygen-filled recording spirometer system (as described), and at intervals was instructed to inspire varying measured volumes. The airway was then closed at the

mouth by a tap and the subject instructed to relax the respiratory muscles. The pressure at the mouth proximal to the obstruction was conveyed to one limb of a narrow-bore water manometer, and the intrathoracic pressure measured via an intra-oesophageal balloon and catheter was conveyed to the other limb of the same manometer. The pressure change recorded during relaxation was thus due to the recoil of the lung and was not dependent upon the degree of relaxation of the respiratory muscles. In this way a number of pressure-volume points were obtained.

RESULTS

Determination of the pressure-volume relationships of the thorax of anaesthetized and paralysed subjects using Method 1

In twelve anaesthetized paralysed subjects the chest was inflated with prolonged positive pressure as described under Method 1. A typical pressure-volume relationship over the range 0–20 cm H_2O is shown in Fig. 3. The points obtained in two separate determinations have been plotted. The pressure-volume curves of all the twelve subjects lay between the dotted lines shown in Fig. 4; the mean of all the curves is indicated by the continuous line. The pressure-volume curves of the two youngest subjects (13 and 17 years) lay at the lower part of the range. In four subjects the extent of the scatter of the curves was not reduced when the volume changes were expressed as a percentage of the vital capacity. In all cases the pressure-volume line was curved, though to varying degrees; the volume increment for a given increase of pressure rose as the inflation pressure was increased.

As the relationship of volume to pressure was not linear the compliance of the system cannot be expressed by a single value unless the pressure or volume employed is specifically stated. In order to compare results on different subjects or by different investigators, we have adopted the expression used by Nims *et al.* (1955), namely the ratio of the volume increase to pressure increase when the pressure was raised to 20 cm H_2O above the resting expiratory level. The mean value for our twelve subjects expressed in this way was 78 ml./cm H_2O at 20 cm H_2O. These results are similar to those of Nims *et al.* (1955) who, using a slightly different technique, obtained a mean value of 62 ml./cm H_2O at 20 cm H_2O in seven subjects.

Fenn, Otis, Rahn, Chadwick & Hegnauer (1947) have estimated that at a sustained intrapulmonary pressure of the 30 cm H_2O in the conscious subject, the volume of blood in the thorax is reduced by about 500 ml., probably owing to interference with the venous inflow. The quantitative relationship between rise of intrapulmonary pressure and the volume of blood 'displaced' has not so far been determined. It is probable, however, that the volume of blood 'displaced' from the thorax is dependent, within limits, on the duration of the positive intrapulmonary pressure. If the duration of thoracic distension is brief, probably blood 'displacement' would be minimized and a truer value for thoracic compliance might be obtained. For this reason, Method 2 was used in a further study of anaesthetized or anaesthetized and paralysed subjects.

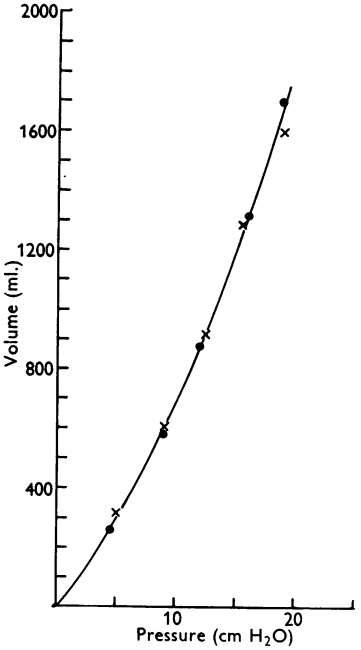

Fig. 3

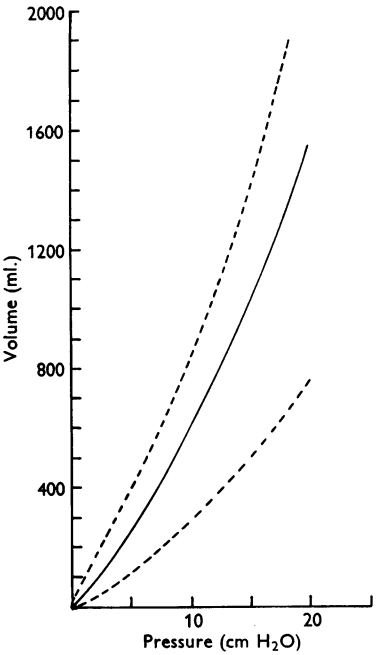

Fig. 4

Fig. 3. Pressure-volume relationship of the thorax of a supine anaesthetized paralysed human subject, obtained using Method 1 (prolonged positive pressure inflation). ● and × represent the individual pressure-volume points obtained in two successive determinations.

Fig. 4. The mean (——) and the range (----) of the pressure-volume curves of twelve supine anaesthetized paralysed human subjects obtained using Method 1, prolonged positive pressure inflation.

Determination of the pressure-volume characteristics of the thorax of anaesthetized and paralysed subjects using cyclical positive pressure inflation

Fifteen anaesthetized and paralysed subjects were studied in the supine position. Fig. 5 illustrates a typical result obtained during cyclical ventilation by measuring the volume of air expired following a 3 sec period of inflation at varying pressures. When this method was used the pressure in the lungs always returned, between the periods of inflation, to the resting level (+0·5 cm H_2O). In Method 1, by contrast, the inflation was sustained and was raised progressively during the course of the experiment. In this study (as when Method 1 was used) the individual pressure-volume points were reproducible within small limits (50 ml.). The reproducibility of our results using either Method 1 or 2 is in striking contrast with the wide scatter of the pressure-volume points obtained in repeated measurements in conscious subjects. This difference can be reasonably attributed to the inability of the conscious

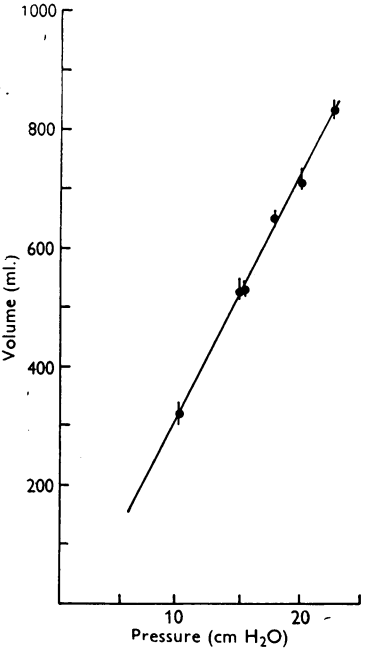

Fig. 5

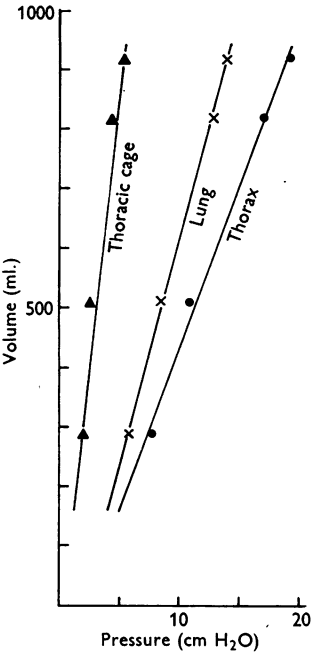

Fig. 6

Fig. 5. Pressure-volume relationship of the thorax of a supine anaesthetized paralysed human subject obtained using Method 2, cyclical positive pressure inflation. The points represent the mean of six volume measurements at each pressure, the range of the volumes being indicated by the vertical lines through each point.

Fig. 6. The pressure-volume relationship of the thorax, ●, lungs, ×, and thoracic cage, ▲, of a supine anaesthetized paralysed human subject obtained using Method 2 (cyclical positive pressure inflation).

TABLE 1. Compliance is expressed in ml./cm H_2O.

		Thorax		Lung		Thoracic cage	
Subject	Age and sex	V/P_{20}	V/P	V/P_{10}	V/P	V/P_5	V/P
A.T.	28, M.	70·5	77·5	92·5	104	260	297
H.C.	32, F.	48·5	54	94	118	100	100
J.F.	61, M.	58·5	67	88	104	144	183
M.B.	56, F.	50	55	65	78	180	192
S.M.	38, F.	45	49·5	59	72	158	162
A.N.	35, M.	35	41·5	45	55	85	88
L.G.	31, M.	67	74·5	85	95	266	350
D.W.	38, M.	61·5	71	130	155	105	123
M.G.	63, M.	77	81	93	98	314	369
A.G.	60, M.	51·5	57	82	93	116	145
D.S.	30, F.	57·5	65·5	66	77·5	260	289
F.G.	24, F.	57	60	97	106	94	118
D.K.	52, M.	59	69	83	92	140	175
W.B.	18, F.	43	48	52·5	62	215	240
G.H.	30, M.	80	89	130	144	180	225
Mean values		57	65	84	97	174	204

subject to relax or inhibit his respiratory muscles. The fifteen subjects studied differed in the slope of their individual pressure-volume lines ($\Delta V/\Delta P$); the range being from 41·5 to 89 ml./cm H_2O. The mean value for all the subjects was 65 ml./cm H_2O. Individual results are given in Table 1. There is a marked difference between this linear pressure-volume relationship and the curved pressure-volume line using Method 1, the difference becoming greater at higher inflation pressures. The results of Fenn *et al.* (1947) suggest that displacement of blood from the thorax by sustained positive pressure may be responsible for a large part of this difference. In addition, the possibility of viscous resistances being present under the conditions of Method 2 has been considered. However, after a minimum of 1 sec duration of sustained inflation pressure, it is thought unlikely that any appreciable viscous resistances would be present. We, therefore, attribute the major part of the difference between the pressure-volume relationship obtained by Methods 1 and 2 to blood displacement from the thorax.

It should be noted that the projection of the pressure-volume line downwards never passed through the zero point but cut the abscissa at approximately 2·0 cm H_2O. It follows that a region of low thoracic compliance must exist at the beginning of the pressure-volume plot, the exact shape of which we shall consider (p. 10). The question then arises how should the results be expressed. If the compliance is calculated from the values when the pressure was raised to 20 cm H_2O (V/P_{20}), a value is obtained which is modified by the region of low compliance to which we have drawn attention. If the compliance is expressed by the slope of the line, a higher value is obtained as it does not take into account the initial region of low compliance. The mean value V/P_{20} was 57 ml./cm H_2O. These results demonstrate that the thoracic compliance in anaesthetized subjects is markedly reduced as compared with the available data in conscious subjects, where the average value obtained was 117 ml./cm H_2O (see also p. 15).

Determination of the lung and thoracic cage components of the total thoracic compliance

In fifteen anaesthetized paralysed subjects cyclically inflated with positive pressure (Method 2) we measured the intra-oesophageal pressure in addition to the inflating pressure and the volume of air expired. We show on p. 13 that the intra-oesophageal pressure change accurately represents the change in intrathoracic pressure under these conditions. The pressure required to inflate the lung was taken as the difference between the inflating pressure and the intra-oesophageal pressure change. The pressure acting on the thoracic cage was the change in intra-oesophageal pressure produced during inflation.

Fig. 6 shows a typical result. The pressure-volume relationships of the

thorax, the lung and the thoracic cage are shown separately. The following points should be noted. (1) The pressure-volume relationship of the lung and the thoracic cage over the range studied (excluding the initial part of the plot) was linear. (2) The lung compliance was considerably lower than that of the thoracic cage. (3) The projection of the lung line downwards never passed through the zero point; the thoracic cage pressure-volume line passed closer to the zero point than the lung line, and sometimes through the zero point itself.

These regions of low compliance at low inflation pressures make it difficult to express the results quantitatively. While the main part of the pressure-volume line probably gives a true measure of the compliance of the lung or thoracic cage under these conditions, it does not include the initial region of low compliance. We were unable to study the thoracic cage compliance at inflation pressures much above 5 cm H_2O because with our technique we were unable to raise the intra-oesophageal (=intra-thoracic) pressure by more than this amount. The fact that the projection downwards of the thoracic cage line frequently passed to the right of the zero point indicated that the thoracic cage often had a region of low compliance at low inflation pressures though to a smaller extent than the lungs.

We have therefore treated our data for thoracic cage compliance in the same way as for total compliance and have calculated the ratio of volume to pressure at 5 cm H_2O (V/P_5) as well as the slope of the main part of the pressure-volume line (V/P). Similarly, we have calculated two values for the lung compliance, namely the ratio volume/pressure at 10 cm H_2O (V/P_{10}), and the slope of the main part of the line ($\Delta V/\Delta P$). Individual data are given in Table 2. The mean value for the slopes of the pressure-volume lines of the fifteen subjects was 97 ml./cm H_2O for the lungs, and 204 ml./cm H_2O for the thoracic cage. When the regions of low compliance were included, as described above, the mean value of V/P_{10} for the lung was 84 ml./cm H_2O; V/P_5 for the thoracic cage was 174 ml./cm H_2O. It will be seen that both the lung, and, to a lesser extent, the thoracic cage compliances were lower than the corresponding published values in conscious subjects. Thus, Mead & Whittenberger (Ferris, Mead, Whittenberger & Saxton, 1952) reported that the thoracic cage compliance in eight conscious subjects had a mean value of 238 ml./cm H_2O and that the lung compliance had a similar value.

Nims *et al.* (1955) attributed the lower total compliance of the thorax in anaesthetized subjects as compared with conscious subjects, to the presence of inspiratory muscle tone in the latter group. It might seem to follow from their interpretation of their results that under anaesthesia there is a decreased thoracic cage compliance. In view of these observations and speculations we were surprised to find that the major factor contributing to the decreased compliance under anaesthesia appeared to be a decrease in *lung* compliance.

As these conclusions, however, were based on a comparison of the mean values of two different groups of subjects, it was desirable to obtain compliance data in the same subjects conscious and subsequently anaesthetized and paralysed. Since it was not possible to train them in the relaxation pressure manoeuvre in advance, lung compliance alone was measured in the conscious subject by a technique which required no previous training.

Comparison of lung compliance in conscious and in anaesthetized paralysed subjects

Lung relaxation pressure curves were obtained as described in four supine subjects within 3 hr before surgical operation. Lung compliance was also determined in these subjects after anaesthesia and muscular paralysis using cyclical positive pressure inflation (Method 2). Fig. 7 shows a typical result. The mean value for the lung compliance in the four conscious subjects was 154 ml./cm H_2O (range 120–200) and after anaesthesia and paralysis, 109 ml./cm H_2O) range 95–144). Thus a decrease in the lung compliance of the order of 30% occurred when the subjects were anaesthetized, paralysed and inflated with cyclical positive pressure. These results confirm that lung compliance is lower in anaesthetized paralysed subjects though the magnitude of the decrease is considerably smaller than had been inferred from published data.

The pressure-volume relationship of the thorax of anaesthetized and paralysed subjects at low inflation volumes

This more detailed examination of the pressure-volume relationship at low inflation pressures was undertaken in two subjects in view of the unexpected finding reported above of a region of low compliance at the origin of the pressure-volume plot. The procedure described in Method 1 was used except that the intrapulmonary pressure was allowed to fall to zero between readings. Simultaneous recordings of changes in intra-oesophageal pressure were made. Above 3 cm H_2O the pressure-volume plot obtained by this modification of Method 1 was identical with the plot when Method 2 was used. Fig. 8 shows this result. At 3 cm H_2O the total compliance of the thorax was approximately 27 ml./cm H_2O. We separated the total thoracic compliance into its lung and thoracic cage components as usual by reference to the changes in intra-oesophageal pressure. We found that the initial low total compliance was due mainly to an initial low lung compliance (over the first 3 cm H_2O). In the other subject studied in this way, the inflexion in the pressure-volume plot occurred at approximately 5 cm H_2O; the more detailed breakdown of the total compliance was not carried out in this case. At these low inflation pressures, however, a small error in the recording of the intra-oesophageal pressure, or in the transmission of intrathoracic pressure change to the oesophagus, would

greatly alter the position of the pressure-volume lines of the lung and thoracic cage. However, the usual finding that the lung pressure-volume line beyond the region of low compliance, when projected downward, cut the abscissa further from the zero point than did the thoracic cage line, suggests that the lung is mainly responsible for the initial low compliance.

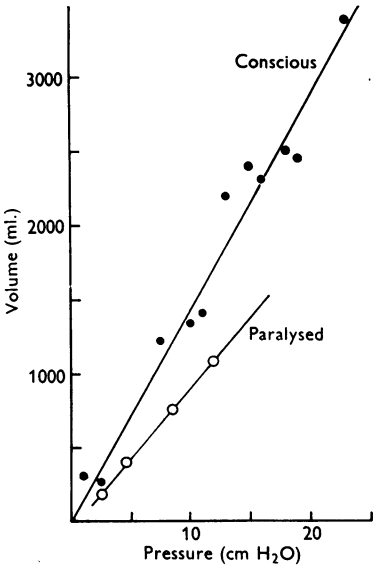

Fig. 7

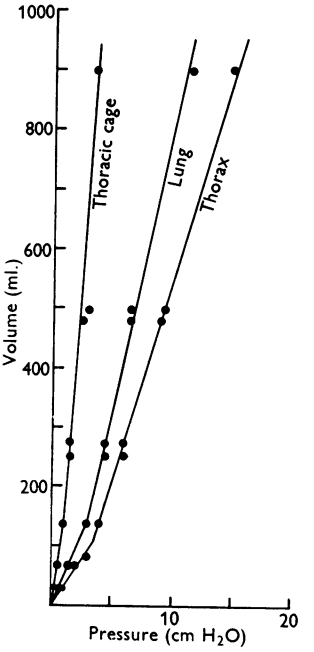

Fig. 8

Fig. 7. Pressure-volume relationships of the lung of a supine subject when conscious, and subsequently when anaesthetized and paralysed.

Fig. 8. Pressure-volume relationship of the thorax of a supine anaesthetized paralysed human subject to illustrate the region of low compliance at low inflation pressures.

We have demonstrated that the low thoracic compliance at the origin of the pressure-volume plot was related to the absolute resting expiratory level. Using Method 2 we prevented the deflation of the thorax to the resting level by placing a weight on the spirometer bell so as to maintain a positive intrapulmonary pressure of 6 cm H_2O at the end of expiration. Under these conditions the downward projection of the pressure-volume plot passed through the zero point. The significance of these findings is discussed on p. 17.

The effect of anaesthesia and muscular paralysis upon the lung volume

To make the comparison of the pressure-volume plots in conscious and unconscious supine subjects valid, we have to show that the functional

residual capacity was the same in the two circumstances as the thoracic compliance is not constant over the whole range of total lung volume. The experiments described below demonstrate that such was in fact the case. Fig. 9 shows a spirogram obtained in a conscious subject using the oxygen-filled closed circuit spirometer system with carbon dioxide absorber (see Methods). The record shows the variable end-points usually considered to be characteristic of the apprehensive subject. Sodium thiopentone (0·4 g) was administered during the period marked by the arrows; with the onset of anaesthesia a regular tracing with a smooth base line was obtained. At this stage there was a slight fall in the resting expiratory level (R.E.L.) of the order

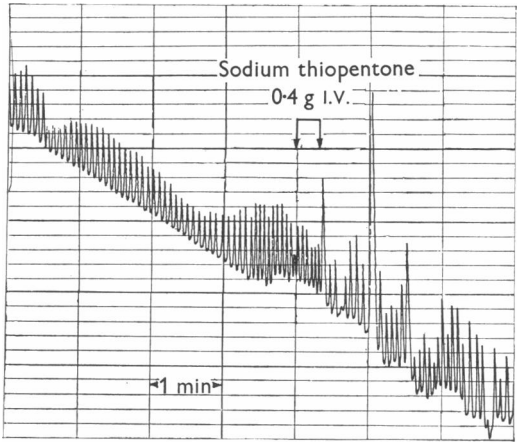

Fig. 9. The effect of anaesthesia upon the resting expiratory level of a supine human subject. The irregular tracing starting in the lower right corner was obtained with the subject conscious. Between the arrows 0·4 g sodium thiopentone was injected intravenously. The onset of anaesthesia was associated with a small drop (approximately 200 ml.) in the resting expiratory level and was followed by a regular spirogram.

of 200 ml.; the previous elevation of the R.E.L. was perhaps due to the subject's anxiety about the whole procedure. Whatever its cause, the recorded drop in the R.E.L. was too small to account for the low thoracic compliance after anaesthesia. Fig. 10 shows the effect of complete respiratory paralysis with succinyl choline on the spirogram of an anaesthetized subject; no significant change in the R.E.L. occurred. Similar results were obtained in three other subjects.

Comparison of intrapleural and intra-oesophageal pressures during cyclical positive pressure ventilation

Four subjects were studied before thoracotomy. In three of the subjects (aged 48–56 years) the operation was being performed for a small apparently localized peripheral carcinoma of the lung. No evidence of pleural disease was

found at operation. The subjects were in the supine position, anaesthetized and paralysed. Intermittent positive pressure ventilation was carried out by pressing on the anaesthetic bag. We were struck by the fact that in three of the subjects when the pleural cavity was first entered, a sucking-in of air did not take place, by contrast with what is always seen in the conscious subject in the lateral recumbent position. This finding was accounted for when the resting intra-pleural pressure (in the pneumothorax) was recorded. It was atmospheric pressure in two subjects and less than -1 cm H_2O in a third.

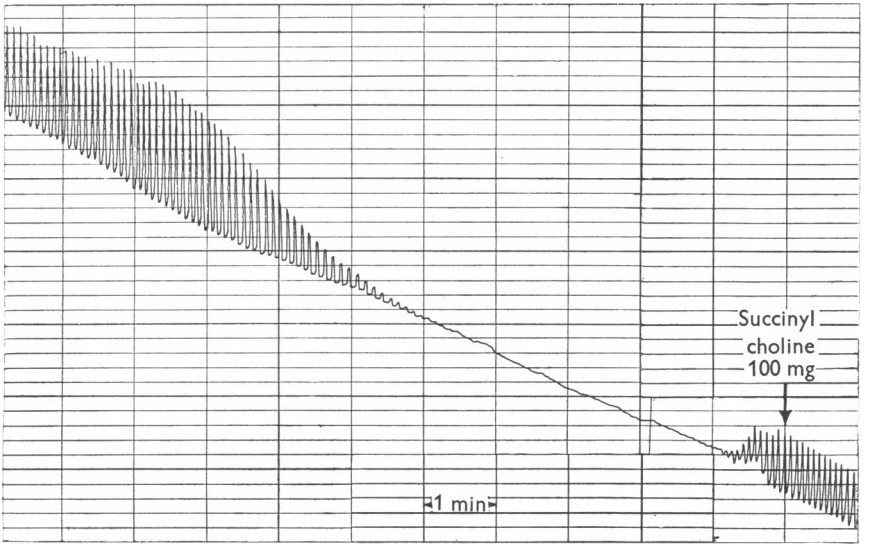

Fig. 10. The effect of complete muscular paralysis upon the resting expiratory level. The spirogram (starting in the lower right corner) was obtained in a supine anaesthetized human subject; at the arrow, 100 mg succinyl choline was injected intravenously which resulted in complete cessation of recordable respiratory movements after $\frac{3}{4}$ min; no significant change in the resting expiratory level occurred; oxygenation of the subject was maintained by 'diffusion respiration'.

The simultaneously recorded intra-oesophageal pressure at the resting expiratory position was positive, the value being 3–5 cm H_2O. Cyclical positive pressure ventilation was carried out and intrapleural and intra-oesophageal pressures were recorded simultaneously by two independent observers. In two of the subjects the pressure changes were identical at both sites; in the third the intra-oesophageal pressure changes were consistently $\frac{1}{2}$–1 cm H_2O lower than the intrapleural.

In a fourth subject aged 21 years, in whom thoracotomy was being performed for a neurofibroma, the resting intrapleural pressure was $-3{\cdot}75$ cm H_2O. For technical reasons, it was not possible to make a comparison of

intrapleural and intra-oesophageal pressure changes in this subject. These results validate the use of the intra-oesophageal pressure changes as a measure of the intrathoracic pressure changes.

Comparison of thoracic compliance in anaesthetized and paralysed subjects

We thought it desirable to extend the work reported above by studying the thoracic compliance (using Method 2) in subjects, first when they were anaesthetized only, and then when they were anaesthetized and paralysed. In this way we hoped to obtain information about the occurrence of respiratory reflexes set up by inflation of the thorax in man.

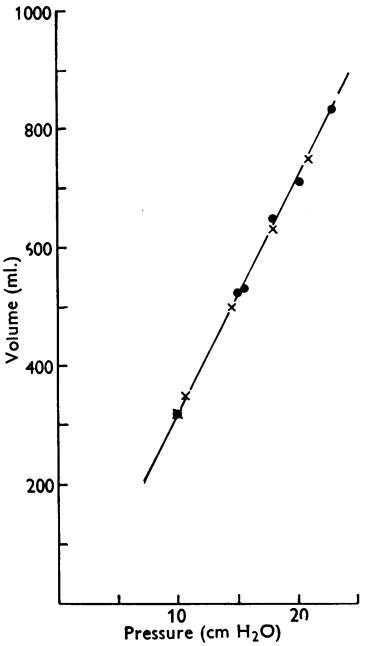

Fig. 11. The pressure-volume relationship of the thorax of a supine, lightly anaesthetized human subject ventilated with cyclical positive pressure (Method 2) is represented by the crosses (×), complete muscular paralysis was then induced with succinyl choline and the pressure-volume relationship again determined (represented by the dots ●); the pressure-volume relationship was identical.

Van Liew (1954) found that the thoracic compliance in anaesthetized dogs was increased after cutting the vagi. He therefore concluded that during inflation of the chest afferent vagal impulses were set up which caused active expiration. Nims *et al.* (1955) refer to animal experiments of their own in which similar results were obtained. In the anaesthetized paralysed subjects such expiratory reflexes could obviously play no part.

Our procedure was to determine the pressure-volume relationship in the anaesthetized subject and then immediately afterwards after complete paralysis of all his respiratory muscles with succinyl choline. Fig. 11, which represents a typical result, shows that the pressure-volume relationship is indistinguishable in the anaesthetized and anaesthetized paralysed subject. We have thus been unable to detect any reflex expiratory contraction in response to thoracic inflation.

DISCUSSION

We have shown that the total thoracic compliance in anaesthetized paralysed subjects cyclically ventilated in the supine position is lower than the reported values in conscious supine subjects (Rahn *et al.* 1946). Our results using Method 1 confirm those of Nims *et al.* (1955), though the method we used (prolonged positive pressure inflation) differed slightly from theirs. When using this method, the shape of the pressure-volume relationship is probably influenced by 'displacement' of blood from the chest. To minimize this complication we employed our Method 2, cyclical positive pressure ventilation. A different pressure-volume relationship was obtained by this technique indicating that the thoracic compliance values obtained by artificial respiration techniques are not absolute but depend in part on the conditions of measurement, which should therefore always be stated.

There are a number of determinations in the literature of thoracic compliance in the supine posture in conscious subjects (Rahn *et al.* 1946; Otis, Rahn & Fenn, 1946; Nims *et al.* 1955; Otis, Fenn & Rahn, 1950; Ferris *et al.* 1952). When the mean values quoted by all these investigators are averaged they give an average mean value of 124 ml./cm H_2O at 20 cm H_2O. In five subjects who were studied using cyclical ventilation in a tank respirator, the mean value was 117 ml./cm H_2O. It should be borne in mind that the tank respirator produces, as far as the thorax is concerned, cyclical *positive* pressure ventilation; this group of results in conscious subjects can therefore be directly compared with ours in anaesthetized paralysed subjects, as our Method 2 likewise produced cyclical positive pressure.

We must now consider how the decreased total, lung and thoracic cage compliances in anaesthetized paralysed subjects is brought about. Nims *et al.* (1955) consider the following possible factors: (1) bronchial obstruction; (2) pulmonary congestion; (3) changes in skeletal muscle tone; (4) changes in vagal reflexes. We have reviewed the possible part played by these factors in our experiments.

(1) *Bronchial obstruction.* There was never any suggestion clinically of bronchial obstruction which is a very infrequent complication of sodium thiopentone administration. Bronchospasm occasionally occurs following curare administration and has been observed with succinyl choline when

administered to cats (personal observation). But as we obtained identical results in anaesthetized and in anaesthetized paralysed subjects, the neuromuscular blocking agents could not have acted in any specific way. Conclusive evidence has been obtained that viscous resistances were absent during measurements made using Method 2 (Campbell, Howell & Peckett, unpublished).

(2) *Pulmonary congestion.* There was no clinical evidence of pulmonary congestion at any time during the period of study. Johnson (1951) has reported a 12% decrease in pulmonary blood volume following barbiturate anaesthesia and muscular paralysis. In addition, it is likely that positive pressure inflation would antagonize any tendency for pulmonary congestion to occur.

(3) *Changes in lung volume.* No significant change in lung volume occurred during anaesthesia and paralysis.

(4) *Changes in muscle tone and reflex effects.* As we obtained identical results in the anaesthetized and anaesthetized paralysed subject, it follows that changes in muscle tone produced by any means, including reflex effects via the vagi, played no part in producing the decreased thoracic compliance after anaesthesia.

It seems at first sight that the difference in the thoracic compliance between conscious and anaesthetized subjects is due to the presence of inspiratory muscle activity in the conscious subject during the measurement of compliance. If this were so, then under anaesthesia only the thoracic cage compliance should decrease. Direct measurement of the two components of the total compliance showed, however, that the lung compliance was also signficantly decreased, compared both with the values reported for normal conscious subjects (Mead & Whittenberger, cf. Ferris *et al.* 1952) and with the same subjects when conscious. The change in the thoracic cage compliance can be attributed to the loss of inspiratory muscle activity following anaesthesia when artificial ventilation is performed. But the explanation of the decreased lung compliance which apparently occurs when the conscious subject is anaesthetized and then artificially ventilated is more obscure.

The crucial question to be considered is whether one is justified in comparing the results obtained from studies in the conscious state with those obtained during positive pressure inflation of the thorax in anaesthetized or anaesthetized paralysed subjects. We would suggest that when the respiratory muscles are not in action and the thorax is ventilated with intermittent positive pressure, the entire mechanical situation may be so altered that it is no longer comparable with that present in the conscious (and probably also in the unconscious) naturally respiring subject.

When a person breathes normally, the shape of the thorax is altered by muscle action and the external configuration of the lungs has to follow that of the thoracic cage; the change in the shape of the thorax is determined by the

resultant of the particular pattern of muscular action employed and the visco-elastic resistances of the lungs and of the thoracic cage. When the paralysed subject is ventilated by positive pressure, the pattern of inflation of the lungs is no longer dependent on the changing shape of the thoracic cage. The two components of the thorax now present a single visco-elastic resistance to the inflating pressure and the pattern of inflation of the thorax will depend on the magnitude and distribution of their combined resistances. As the thorax almost certainly has a different shape in the two circumstances, the distribution of the inspired volume would also be different. The distribution of naturally inspired gas is relatively even (Briscoe, Becklake & Rose, 1951); but any major deviation from this pattern of inflation would presumably result in overventilation (i.e. overdistension) of some regions of the lungs and under-ventilation of others. There would thus be a lower effective volume of lung being inflated and the compliance measured under such circumstances would be lower than that of the same lung when evenly, i.e., naturally, inflated. The decrease in thoracic cage compliance under anaesthesia might also be due to an alteration in the pattern of expansion, but whether this or the absence of inspiratory muscle activity when the unconscious subject is artificially inflated is the main contributing factor is not known.

A further unexpected finding was the region of low total compliance at volumes immediately above the resting expiratory level. Direct measurement suggested that this was largely due to a low lung compliance. In the course of the experiments to validate the use of the changes in intra-oesophageal pressure as a measure of the changes in intrathoracic pressure, we observed in three subjects that the intrapleural pressure was at or very close to atmospheric pressure. As the elastic tissues of the lung were not stretched at this level, an important factor normally concerned with the maintenance of the patency of the smaller airways was absent, so that collapse of the less rigid airways might occur. We suggest therefore that the low compliance at the origin of the pressure-volume plot is due to the pressure required to open these collapsed units.

The constant finding of an initial region of low compliance, even though the intrathoracic pressure was sometimes below atmospheric at the resting expiratory level, suggests that progressive collapse of some of the airways occurred while the intraluminar pressure was positive relative to the surroundings. This closure of the airways against positive pressure resembles the critical closing pressures described for certain blood vessels of the rabbit and frog by Nichol, Girling, Jerrard, Claxton & Burton (1951), and the pressures at which this closure occurred appears to have the same order of magnitude (3–5 cm H_2O). The absence of a low initial compliance region when a positive end-of-expiration intrapulmonary pressure of 6 cm H_2O was maintained is consistent with this interpretation.

SUMMARY

1. Thoracic compliance was determined by two methods in twenty-three anaesthetized paralysed supine human subjects.

2. Using prolonged positive pressure inflation the pressure-volume relationship was curved. Over 0–20 cm H_2O inflation pressure the mean total thoracic compliance was 78 ml./cm H_2O.

3. Using cyclical positive pressure inflation, the pressure-volume relationship was linear between 5 and 20 cm H_2O inflation pressure. Over the range 0–20 cm H_2O the mean compliance was 57 ml./cm H_2O. There was a region of low thoracic compliance at the origin of the pressure-volume plot; an alternative expression which excluded this region was derived from the slope of the pressure-volume line.

4. The separate lung and thoracic cage compliances were determined. The mean lung compliance was 84 ml./cm H_2O and the mean thoracic cage compliance was 174 ml./cm H_2O. The total and component thoracic compliances are lower than published values in conscious subjects.

5. No reflex expiratory activity occurred in anaesthetized subjects in response to thoracic inflation.

6. No significant change in the functional residual capacity occurred during the induction of anaesthesia and subsequently during complete muscular paralysis.

7. It is suggested that the low compliance of the lung in the artificially ventilated subjects may be due to an alteration in the distribution of inflation.

8. The initial low compliance at the origin of the pressure-volume plot is attributed to collapse of some airways over a transmural pressure range of about +3–5 cm H_2O.

The late Professor Samson Wright gave us immeasurable help and guidance during this work and we would like to record our gratitude. We would like to thank Professor Alan Kekwick, Dr Bernard Johnson and Dr O. P. Dinnick for their encouragement and for their help in the selection of the cases. In the earlier part of this work Dr Moran Campbell gave valuable help and advice. Part of this work was done while one of us (B. W. P.) was Research Assistant to the Poliomyelitis Unit at the Western Fever Hospital.

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