

The measurement of bronchial endomural or 'squeeze' pressure¹

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Although it has been customary for a long time to talk about 'bronchospasm', it is now realized that the bronchial obstruction which exists in patients with bronchospasm may be due to one or more of the following: 1 Swollen bronchial mucous membrane due either to oedema or to vascular engorgement or to both. This may be aggravated in expiration when the shortening and narrowing of the bronchus leads to folding of the mucous membrane; 2 viscid mucus; 3 contraction of the bronchial musculature; and 4 distortion of the respiratory passages associated with emphysema or scarring, which may lead to air trapping. This form of obstruction, in contradistinction to the others, is presumably not reversible.

In the work to be described we have tried to evolve a method for measuring the contraction of the bronchial musculature on expiration. But, because the elements in bronchial narrowing are complex, we have preferred to describe what we are measuring as 'bronchial squeeze' or, more elegantly, 'endomural bronchial pressure'.

THEORETICAL FACTORS AFFECTING WIDENING AND NARROWING OF THE BRONCHI In inspiration bronchi lengthen and widen; in expiration they shorten and narrow. The factors affecting the bronchial lumen are probably as follows (see Fig. 1).

NEGATIVE INTRAPLEURAL PRESSURE OR TENSION This becomes more negative on inspiration and less negative on expiration. Its tendency is to widen the lumen, the outward pull being greater on inspiration and less on expiration. This pull is conveyed to the wall of the bronchus by way of the elastic tissue of the lung.

INTRA-ALVEOLAR PRESSURE This is in the alveoli surrounding the bronchial wall and is linked to the pressure in the bronchial lumen.

PRESSURE IN THE BRONCHIAL LUMEN During inspiration this pressure will be above that in the alveoli, since air is flowing from the bronchi to the alveoli. Conversely, during expiration the alveolar pressure will be higher than that in the bronchial lumen, and the alveoli around the bronchus will produce some inward pressure on the bronchial wall. At the end of expiration and at the end of inspiration the pressure in the alveoli and in the bronchus will be approximately atmospheric, for the air has stopped moving. Owing to the complex pathways and variation in the dist

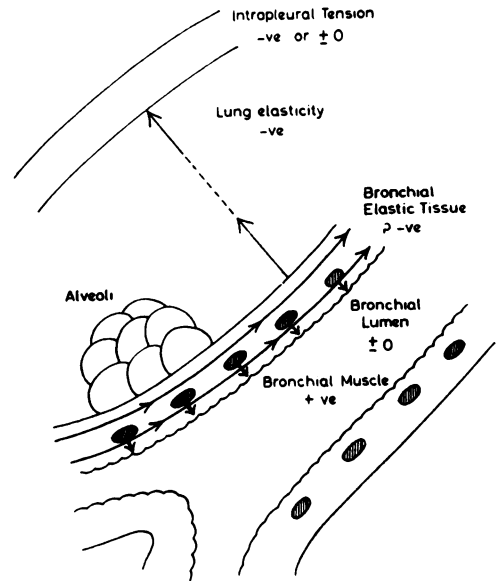


FIG. 1. Factors affecting transmural bronchial pressure at the end of expiration. For discussion see text.

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ance the air has to traverse, the alveolar pressure will reach atmospheric pressure later than the bronchial pressure. The delay will probably vary with the length of the air passages supplying particular groups of alveoli. This delay and variation is likely to be greater where there is distortion of the airways by disease. If there is marked air trapping, the alveolar pressure on the bronchial wall on expiration will be increased, though this increase will also be reflected in a rise in intrapleural pressure on expiration. It is probable that air trapping is most likely to affect the most distal thin-walled bronchial connexions (Campbell, Martin, and Riley, 1957). It may be that the function of bronchial muscle is to narrow the more proximal bronchi on expiration and so, by back pressure, hold open the distal thin-walled channels (Brocklehurst, personal communication).

ELASTIC TISSUE OF THE BRONCHIAL WALL This is mainly distributed longitudinally and is probably concerned with the shortening of the bronchi during expiration. On the analogy of a rubber tube pulled longitudinally, relaxation of this tension, as will occur when the rising diaphragm allows shortening of the bronchi, should tend to widen rather than narrow the bronchi on expiration. This force may therefore act in opposition to the others mentioned and help to give the bronchial wall stability.

BRONCHIAL MUSCLE In the larger bronchi this is mainly disposed circularly with insertion into the inner surface of the bronchial cartilages. Contraction on expiration will tend to narrow the lumen, and relaxation on inspiration will allow the lumen to widen. As the bronchi become smaller peripherally, the bronchial muscle is inserted further and further forward in the cartilages so that the muscle tends to form more and more of a ring within them. In the smaller bronchi it is disposed in a helical manner (von Hayek, 1960). Distal to the orifice of the segmental bronchus the cartilage is mainly in isolated plates and the muscle circular or helical.

If this theoretical analysis is correct, during expiration the narrowing effect will be mediated by 1 decreasing intrapleural tension, with resulting relaxation of elastic tension of the lung and decreasing pull on the outer walls of the bronchus; 2 a rise of pressure in the alveoli above that in the bronchial lumen. This difference in pressure will disappear at the end of expiration when both pressures equalize at atmospheric pressure; and 3 contraction of bronchial muscle.

These forces will be acting against 1 the intrinsic rigidity of the bronchus which, below the largest bronchi with almost complete cartilage, is very slight (von Hayek, 1960); and 2 possibly the elastic tension of the bronchus which, as the tube shortens, will tend to widen it.

The reader may be interested to compare this theoretical analysis with that of Campbell *et al.* (1957), who have examined the problem from a somewhat different point of view. These authors were principally concerned with analysing the effect of maximal forced expiration on the bronchial lumen and attempted to express in algebraic terms the balance of forces affecting the bronchial wall. Their consideration was mainly of the effect of forced expiration when the 'intrathoracic pressure', as measured in the oesophagus, was demonstrably above the atmospheric. They assumed that this pressure acted directly on the outer wall of the bronchus, whereas we have suggested that the pressure on the outer wall of the bronchus might be similar to that within the alveoli. Both these assumptions may well be over-simplifications. In parenthesis, it is perhaps another dangerous over-simplification to talk about 'intrathoracic pressure' when there are obviously many intrathoracic pressures, in different parts of the pleura, in the alveoli, in the interstitial tissues of the lung, within the wall of the bronchus, in the lumen of the bronchus (and different in different parts of the lumen), and within the blood vessels.

Campbell *et al.* also made a number of assumptions about the intraluminal pressure in different forms of chest disease. Some of these assumptions might be justified but none has yet been proved. For the present we would prefer to leave the matter open. Nevertheless, Campbell *et al.* and ourselves have considered much the same potential factors. They attempted to reduce these to specific formulae, but for only a few of the terms employed were actual measurements possible. This was a most useful theoretical exercise and, probably knowingly and justifiably, an over-simplification. But numerical values for most of the terms were missing. The work reported here may be a first step towards providing values for some of the missing terms.

METHOD OF MEASUREMENT

In the method we have used, the pressure on the inner bronchial wall throughout the respiratory cycle is recorded by a balloon mounted on a bronchial probe and inserted into a basal segmental bronchus. This will be referred to as the 'balloon pressure'. At the same time we record

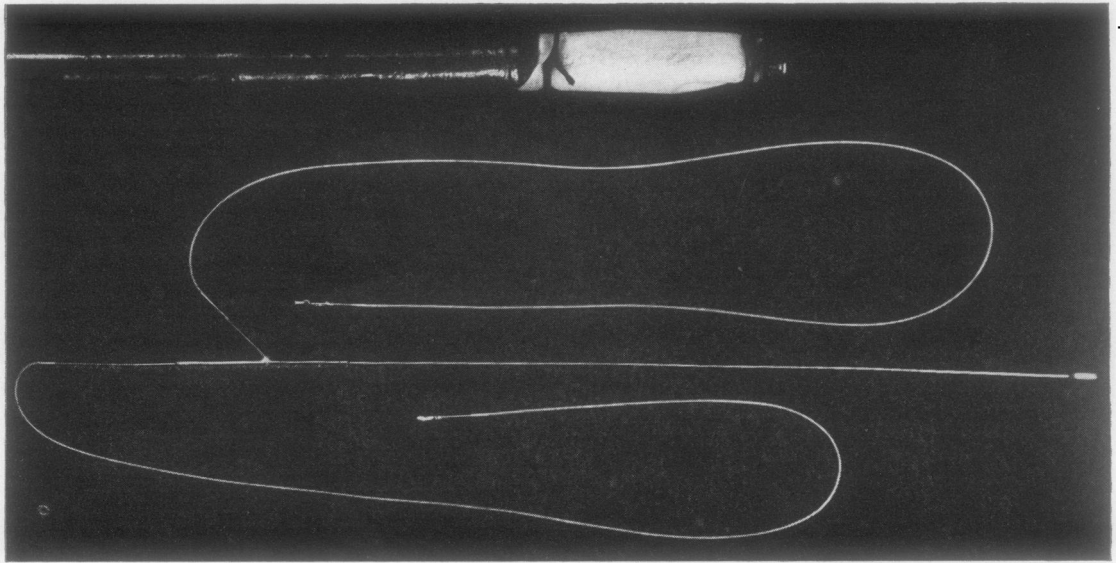


FIG. 2. Photograph of the bronchial probe. Above is an enlargement of the distal end of the probe.

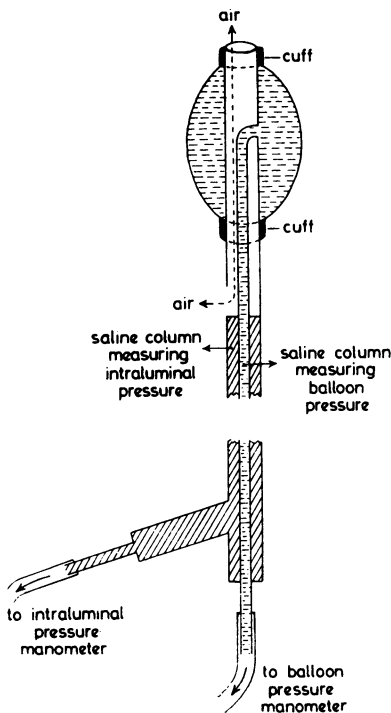


FIG. 3. Diagram of the bronchial probe.

both the pressure in the bronchial lumen immediately proximal to the balloon and, as a measure of intrapleural pressure, the pressure in the oesophagus.

PRINCIPLE OF THE BRONCHIAL PROBE (Figs. 2 and 3) The bronchial squeeze pressure and the intraluminal pressure in the bronchus are measured by means of the bronchial probe, which is usually inserted into one of the basal segmental bronchi. At the distal end of the probe is a balloon filled with saline, which is connected to the manometric system by a channel of saline running within the probe. The probe is hollow and air can move in and out of the segment blocked by the balloon through the lumen of the probe, escaping by three ports immediately proximally.

Proximal to these ports the lumen of the probe is filled with saline constituting the second channel. This measures the intraluminal pressure and is connected to the manometric system by way of a side-arm at the proximal end of the probe.

The probe is therefore two-channelled throughout its length. Distally, from the tip of the probe to the proximal port, one channel contains air and the other is the saline-filled balloon connexion. From the proximal port both channels contain saline; one is the balloon connexion and the other measures the intraluminal pressure in the bronchus.

DETAILS OF THE BRONCHIAL PROBE (Fig. 2) The hollow bronchial probe is made from chrome-plated brass

and is 74.5 cm. in length so as to allow for protrusion beyond the distal and proximal ends of a bronchoscope. The probe is 3 mm. in diameter with 2 mm. internal bore. Immediately proximal to the tip and 17 mm. proximal to this are two solid rubber collars, each 1.5 mm. in width and about 1 mm. in depth. A balloon is formed by passing over these two cuffs a length of thin Paul's tubing 5 mm. in diameter and binding this on to the probe by double loops of thread tied just proximal to the distal cuff and just distal to the proximal one. The proximal loop of thread is tied first and the rubber tube is filled with sterile saline by way of the channel running within the probe and opening distally by a port situated between the two cuffs. This channel is attached to the manometric system by a length of polythene tubing 1.5 mm. in bore. When all air bubbles have been expressed, the distal loop is tied and the redundant distal rubber tubing cut off with scissors. In this way a thin rubber balloon is formed, 17 mm. in length and, when loosely distended after tying, 4 mm. in diameter, bounded at each end by a solid rubber collar.

It was found by experiment that, with a moist jammed balloon and the rubber collars on the probe, the balloon pressure was not significantly affected by the pressure in the bronchial lumen.

The oesophageal pressure is measured by means of a saline-filled open-ended polythene tube, 1.5 mm. in bore, which is swallowed by the patient so that the tip lies a little above the diaphragm.

MANOMETERS (Fig. 4) Pressure changes are recorded by a three-channel photokymograph. This requires calibration on each occasion. The sensitivity is of the order of 1 cm. deflexion corresponding to 1 cm. of water.

STERILIZATION The bronchial probe and its connexions are sterilized in 2% Dettol in spirit. Dettol is drawn into the bronchial probe and tubing and the whole is placed in a long perspex holder filled with the sterilizing fluid. The channels are thoroughly washed through with saline before use. The manometers, Paul's tubing, and tying thread are sterilized in the autoclave. The saline reservoirs for filling the system, with their rubber tubing connexions, are sterilized by boiling. To assemble the apparatus and tie the balloon, gown, cap, and rubber gloves are worn with full sterile precautions.

PATIENTS INVESTIGATED

As this was a new method of measurement and its value not yet established, all the initial investigations were carried out on patients who were undergoing diagnostic bronchoscopy for a variety of reasons. The present paper is founded on investigations in eight patients examined with our final apparatus, together with very many laboratory investigations and 'mock-up' experiments involved in the evolution of a satisfactory technique.

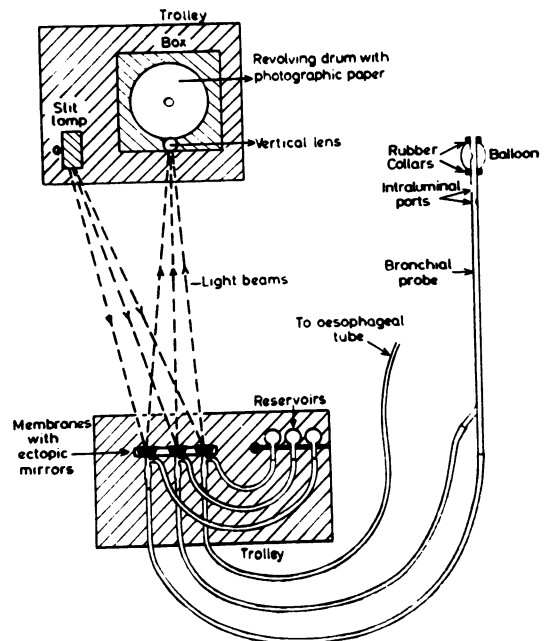


FIG. 4. Diagram of the manometer system as seen from above.

PROCEDURE

Compared with a routine bronchoscopy most of the additional time taken was spent in setting up the apparatus before the patient was brought to the theatre. The only added discomfort to the patient was the swallowing of the oesophageal tube, which was passed through the nose. This, of course, is no more uncomfortable than a gastric lavage. In fact we found that, if fluid is sucked through a straw as the tube is passed, it is swallowed with a minimum of trouble. It is, of course, important that the patient should do this before he sucks the anaesthetic lozenge. The tube was initially passed into the stomach and withdrawn into the oesophagus later (see below). The patient received the normal premedication with omnopon and scopolamine or omnopon and atropine, and a local anaesthetic spray containing adrenaline. We realize that these drugs may have had some effect on the results but this probably had little bearing on the conclusions to be drawn from the present paper.

Bronchoscopy was done in the usual way, the full diagnostic inspection being carried out before any measurements were made. Biopsy, if thought necessary, was deferred until after the measurements had been made. The presence of the oeso-

phageal tube caused no difficulty. The theatre was kept in darkness except for red lamps and the bronchoscopy light.

When the patient was on the bronchoscopy table the oesophageal tube was attached to the manometer and a little saline was run through to clear any mucus. The light spot was then watched and the tube was slowly withdrawn. As it passed from the stomach to the oesophagus the movement of the light spot with respiration suddenly reversed; the tube was then withdrawn another 2 cm. or so and secured at this level by adhesive strapping.

After the diagnostic bronchoscopy had been completed the probe was inserted into the anterior or lateral basal bronchus of the right lower lobe, whichever was the easier of access. It was passed gently into the bronchus until it jammed and, by adjusting the reservoir, the pressure within the balloon was set at +10 cm. of water. An experiment on a resected lung suggested that the balloon would become wedged so that its proximal border would lie about 2.5 cm. within the segmental bronchus.

Recordings were made with natural breathing, with deep breathing, and after a cough. Owing to their pre-medication one or two patients were breathing periodically (Cheyne-Stokes breathing). An example of the tracings obtained is shown in Figure 5.

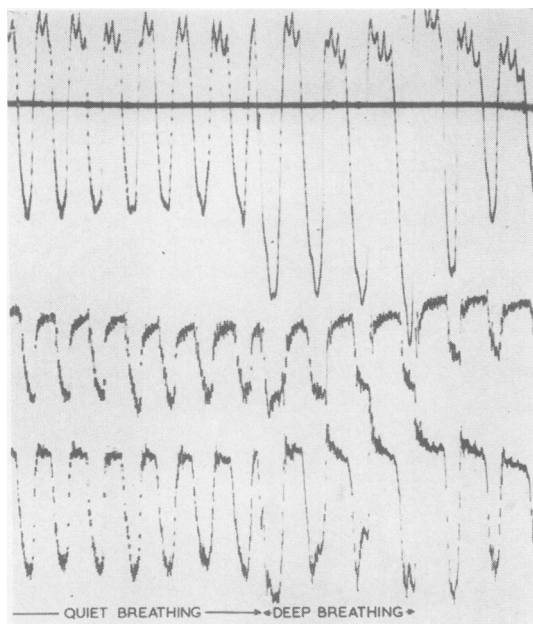


FIG. 5. Example of pressure tracing. The upper trace represents intra-oesophageal, the middle trace bronchial intraluminal, and the lower trace endomural bronchial squeeze (balloon) pressure. Each trace has to be separately calibrated; one cannot make direct comparisons between the pressures as recorded on the tracing. The smaller waves are due to cardiac and vascular pulsations.

When the measurements were complete the probe and the oesophageal tube were withdrawn. The apparatus was then calibrated. Within a range of -15 to +15 cm. of water the relationship between readings and true pressures has been found to be linear. A graph was drawn for each manometer and from this the height of each wave on the tracing could be translated into centimetres of water. Alternatively, a conversion factor could be used.

RESULTS

CONTRACTION OF BRONCHIAL MUSCLE The present paper mainly examines whether there is likely to be an element of muscular contraction in the 'bronchial squeeze', as measured by the variations in balloon pressure on respiration. In the following analysis we are concerned not with pressures, in terms of height below or above atmospheric pressure, but with the range of pressure recorded with each breath. This, of course, is measured by recording the heights of the waves on the tracing and translating these, by means of the calibration graphs, into terms of centimetres of water.

INTERRELATIONSHIP OF THE PRESSURES MEASURED If the bronchial squeeze pressure variation depended mainly on the intrapleural pressure, one would then expect to find a constant ratio between the intrapleural pressure, as measured in the oesophagus, and the balloon pressure in the bronchus. For this purpose it is not of vital importance if the pressure in the lower part of the oesophagus does not represent exactly the pressure in all parts of the intrapleural space as long as, in the circumstances of the investigation, it has a constant relationship to it. This we have no reason to doubt.

Similarly, if the bronchial squeeze mainly depended on the intraluminal pressure in the bronchus, we would expect to find a constant ratio between this and the balloon pressure. It must, of course, be realized that the intraluminal pressure, as measured proximal to the balloon, is lower than the intraluminal pressure distal to the balloon. By 'mock-up' experiment, breathing through an artificial 'bronchus', it was found that, with shallow and deep breathing, the ranges of the two pressures had a constant relationship at similar depths of respiration (coefficient of correlation: 0.93 to 0.99 in different experiments). As the lumen by which air is transferred from the distal to the proximal bronchus is constant, being formed by the distal lumen of the bronchial probe, this close relationship is to be expected, provided there is no great variation in the rate of air flow. We think therefore, in the following analysis, that the intra-

luminal pressure measured can be assumed to vary directly with the intraluminal pressure affecting the bronchial wall in the region of the balloon.

As would be expected, all the three pressures measured tended to vary together. The deeper the breath, the greater the range of intrapleural, intraluminal, and balloon pressures. This is well seen in Fig. 6, in which are graphed the variations in pressure range in successive breaths with normal

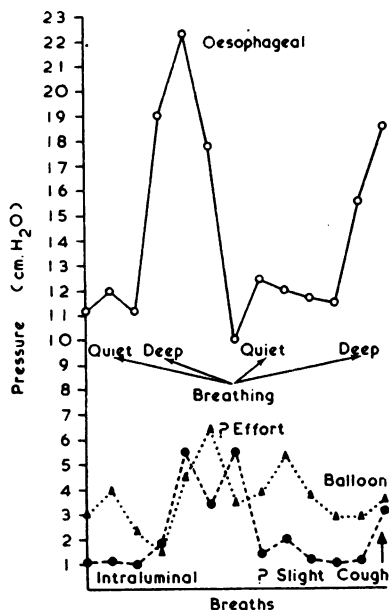


FIG. 6. Ranges, in successive breaths, of oesophageal, intraluminal, and balloon pressures, showing changes with voluntary deep breathing.

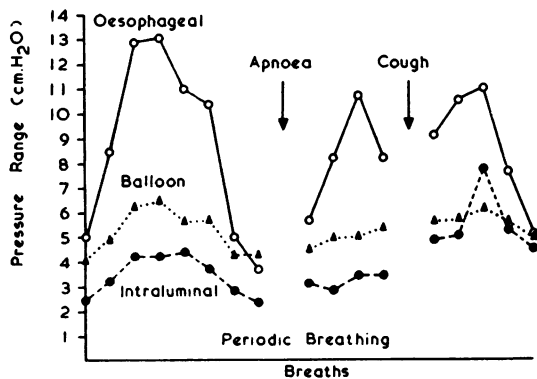


FIG. 7. Ranges, in successive breaths, of oesophageal, intraluminal, and balloon pressures, showing changes associated with periodic breathing. Pressures during coughing were off the trace.

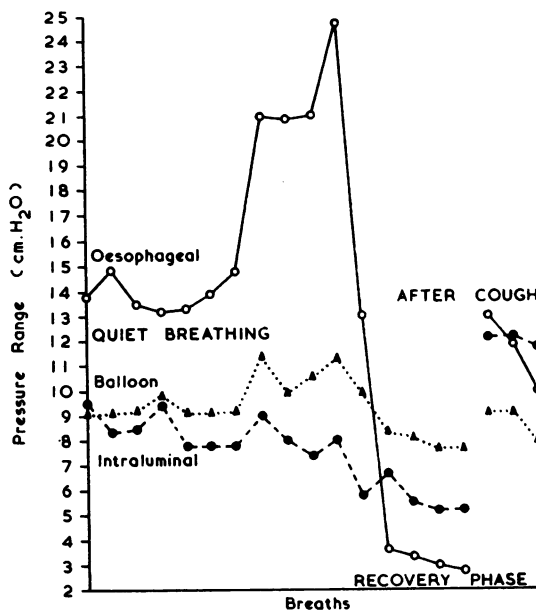


FIG. 8. The effect of deep breathing is shown in the central part of the graph, associated with a rise in oesophageal pressure range. Note the marked fall in oesophageal pressure range in the recovery phase and the rise after coughing.

breathing and deep breathing. It is also seen in Fig. 7, which represents similar variations in a patient with periodic breathing induced by pre-medication.

Nevertheless the three pressures do not move exactly together in all circumstances. Figure 8 shows that, in this patient after voluntary deep breathing, the range of oesophageal pressure with each breath dropped very markedly during the recovery phase of shallow breathing, whereas there was only a slight drop in the intraluminal and balloon pressures. After the patient had been requested to cough, both the intraluminal and the intra-oesophageal pressures rose more than the balloon pressure. Pressures during the cough are usually off the film and have not yet been measured.

BALLOON/OESOPHAGEAL PRESSURE RATIOS As stated above, if the balloon pressure were determined by the intrapleural pressure, as measured by the intra-oesophageal pressure, the ratio between the balloon and the intra-oesophageal pressures should be constant. Figure 9 shows for the same patient as in Fig. 8 the ratios of balloon to oesophageal pressure in successive breaths. During quiet breathing this ratio varied from 0.63 to 0.76.

During deep breathing it varied from 0.45 to 0.52, but during the recovery phase after deep breathing the balloon pressure was no less than two and a half times the oesophageal pressure in four successive breaths. During recovery after cough the ratio was back to 0.71 to 0.76.

Figure 9 also shows the ratio between balloon and intraluminal pressures. In quiet breathing it varied from 1.0 to 1.1, in deep breathing was constantly at 1.2, but in the recovery phase after deep breathing it varied from 1.2 to 1.7. During recovery after coughing it ranged from 0.6 to 0.75.

It is clear from this analysis that in this patient the balloon pressure did not depend directly on either the oesophageal or the intraluminal pressure. Nevertheless the balloon pressure may have been dependent on some interrelationship between the oesophageal and intraluminal pressures. This possibility is examined in Figure 10. In general, of course, the higher the intraluminal and oesophageal pressure the higher was the balloon pressure reading, but it will be seen, for instance, that six balloon pressures of 9.1 cm. of water were recorded, corresponding to intraluminal pressures ranging from 7.7 to 12 cm. of water and intra-oesophageal pressures varying from 11.7 to 14.8

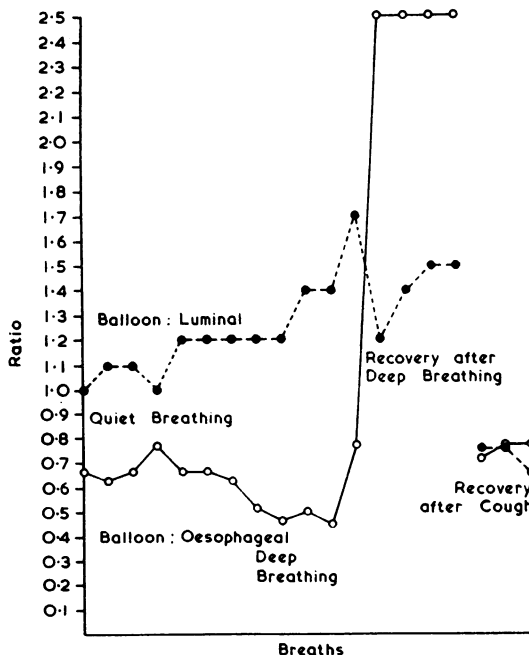


FIG. 9. Ratios of balloon to luminal and balloon to oesophageal pressure ranges in successive breaths. Note the remarkable change in balloon/oesophageal pressure ratio during the recovery phase.

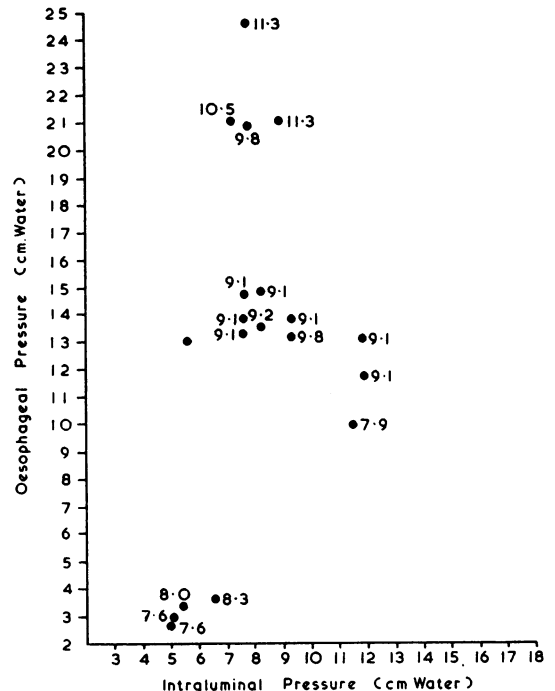


FIG. 10. Relationship between balloon pressure ranges and those recorded in the oesophagus and the bronchial lumen. Each dot represents a respiration. The figure beside the dot is the balloon pressure range recorded for that breath, the position of the dot being determined by the corresponding oesophageal and intraluminal pressure ranges. The figure suggests the relative independence of the balloon pressure range from the oesophageal or intraluminal pressure range.

cm. of water. A balloon pressure of 9.8 cm. of water was recorded, which corresponded to an intraluminal pressure of 9.4 and an oesophageal pressure of 13.1 cm. of water. Another balloon pressure of 9.8 cm. of water corresponded to an intraluminal pressure of 7.8 and an oesophageal pressure of 20.8 cm. of water. It seems clear therefore that there is a factor, other than the oesophageal and intraluminal pressures, which is determining the balloon pressure, and we think that this factor is the contraction of the bronchial muscle.

Although there is no space to reproduce all the graphs, similar ratios have been worked out for other patients and have shown similar, although less dramatic, variations. Table I shows how, with similar balloon pressures, there can be a wide variation of the corresponding intra-oesophageal and intraluminal pressures.

The suggestion that the balloon pressure is determined to an important extent by contraction

TABLE I

RANGES OF OESOPHAGEAL AND INTRALUMINAL PRESSURES CORRESPONDING TO A GIVEN BALLOON PRESSURE (CM. WATER)

Patient	Sample Balloon Pressures	No. of Readings	Range of Corresponding Oesophageal Pressures	Range of Corresponding Intraluminal Pressures
W. McL.	3.1-3.0	3	11.2-15.6	1.0-1.1
	3.6-3.7	2	10.1-18.6	3.3-5.6
T.R.	3.5-3.6	6	5.8- 7.5	2.5-3.2
	3.9-4.0	6	7.7- 9.0	2.6-3.5
T.W.	4.1	6	4.8- 6.3	Not available
	5.5-5.6	4 ¹	2.5- 8.6	0.9-1.4
W. Mc.	4.2-4.3	2	16.9-18.7	Not available
	7.5-7.6	2	20.4-20.8	1.3-1.8
D.C.	5.0-5.1	3	5.1-10.7	2.9-4.5
	5.6	3	7.6-10.9	4.4-5.3
R.B.	7.9-8.0	2	3.3- 9.9	5.5-11.6
	9.1	6	11.7-14.8	7.7-12.0
	9.8	3	13.0-20.8	5.7-9.4
J.D.	14.3-14.4	3	² 3.0- 6.2	2.6-2.8
	17.2-17.3	4	² 3.4- 6.2	2.8-3.3
	30.5	2	² 10.0	4.7-5.0
	32.1	2	² 10.4-13.0	7.1-10.4

¹ Three readings of intraluminal pressure ; ² oesophageal calibration estimated

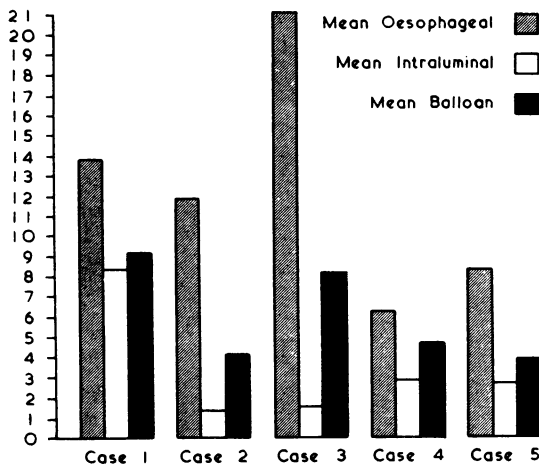


FIG. 11. Mean oesophageal, intraluminal, and balloon pressure ranges in five patients breathing quietly, showing the lack of direct relationship.

of the bronchial muscle is borne out by comparisons between patients. Figure 11 shows the mean oesophageal, intraluminal, and balloon pressures during quiet breathing in five patients. There is obviously quite a wide variation in the relationship between either the oesophageal or the intraluminal pressure and the balloon pressure.

DISCUSSION

The present paper is mainly intended to report a method for measuring bronchial squeeze pressure

directly as a potential tool for the elucidation of some of the problems of disordered lung function.

The evidence we have adduced suggests that an important element in 'bronchial squeeze' is contraction of bronchial muscle. Indeed the fact that this pressure seems to vary a good deal less than the oesophageal pressure suggests that it may be relatively independent of intrapleural pressure and perhaps also relatively independent of the pressure in the bronchial lumen. It must, of course, be clear that at best we have been measuring variations in bronchial muscular tone with respiration and not the tone itself. To put it in an extreme form, if the bronchial muscle remained maximally contracted throughout respiration, there would be no change in the balloon pressure from this cause. Nevertheless it seems probable, though it is certainly not established, that the greater the degree of 'bronchospasm' the greater the variation in pressure with respiration. Measurements on a few patients suggest an increase in balloon pressure range and in the ratio of balloon to oesophageal pressures, with increasing severity of bronchitis.

It is not at present possible to say how far our measurements of 'bronchial squeeze' are representative of the bronchial tree as a whole. The pressure exerted by bronchial muscular contraction might be less in the peripheral bronchi. There is also the possibility that the presence of the probe leads to local muscle spasm. There is often a temporary increase of oesophageal pressure, and probably of the squeeze and intraluminal pressures, after insertion of the probe. This is probably merely the usual stimulus to respiration of almost any change in a patient's environment, and it settles down quite rapidly. We feel we are entitled to assume, at least provisionally, that we are genuinely showing qualitative differences between patients and in the same patient under different conditions.

As has already been pointed out, the intraluminal pressure measured proximal to the balloon will be different from the pressure distal to it, though varying proportionally. Even the distal pressure may be different from that in other similar segments because of the presence of the probe in the bronchus. Nevertheless, if the pressure in that segment becomes less than that in the neighbouring segments, the segment should become smaller until the pressure equates, and *vice versa*. Whether this theoretical adjustment is relatively immediate, it is difficult to say. In any case, so far as the present paper is concerned, we are dealing only with the possible effect of the local intraluminal pressure on the local bronchial squeeze pressure as measured.

At present we do not have enough data to determine the normal range of bronchial squeeze pressure. None of our patients was strictly normal. The most normal, a patient with mild sarcoidosis, showed a pressure range of 2 to 4 cm. of water when breathing naturally at bronchoscopy. The patient with clinically most severe bronchitis and 'bronchospasm' and with a very irritable bronchial mucous membrane showed a pressure varying from 14 to 32 cm. of water under the same conditions. The ranges of the ratio of balloon pressure to oesophageal pressure in these two patients were 0.1 to 0.4 and 2.1 to 5.3 respectively.

An absolute rise of balloon pressure above the normal range on quiet breathing might not necessarily indicate spasm of bronchial muscle as the rise might be due to air trapping alone, though air trapping should be reflected in an increased excursion of the oesophageal pressure tracing. Of course the oesophageal pressure excursion would also increase if all the obstruction to breathing were due to contraction of bronchial muscle. There seem to be three possible ways of overcoming this difficulty.

1 If the rise of bronchial squeeze pressure above the normal were greater in an individual patient than the rise in oesophageal pressure, this would suggest that much of the increased endomural pressure was due to bronchial contraction. That is to say, an increased ratio of balloon to oesophageal pressure suggests spasm of bronchial muscle.

2 If, for instance in the recovery after deep breathing, the oesophageal pressure dropped markedly while the bronchial squeeze pressure remained raised, then clearly little of this pressure could be due to air trapping. Such a change has been illustrated in Figure 8.

3 If antispasmodic drugs reduced balloon pressure much more than oesophageal pressure, then the raised balloon pressure would probably have been due mainly to contraction of bronchial muscle.

The technique we have outlined has now been used to investigate a number of patients in addition to those with whom this paper deals. Our preliminary experience suggests that these measurements may prove of value in investigating the contribution of bronchial contraction to airway obstruction in patients with chronic bronchitis. It has also been possible to demonstrate the effect of antispasmodic drugs. We hope to develop the technique further by the use of more sophisticated apparatus and the simultaneous measurement of tidal air.

SUMMARY

The possible factors affecting bronchial squeeze pressure are discussed. A method is described for the simultaneous measurement, in a basal segmental bronchus, of bronchial squeeze pressure and of the pressure in the bronchial lumen. Intra-oesophageal pressure, as a measure of intrapleural pressure, was recorded at the same time.

The range of bronchial endomural ('squeeze') pressure, bronchial intraluminal pressure, and intra-oesophageal pressure at each breath was in general larger with a deep respiration and smaller with a shallow respiration. As would be expected, the three pressures tended to increase or decrease together.

Nevertheless this close correlation was not always found. The variation of intra-oesophageal pressure with each breath was usually much greater than that of the endomural pressure, but on occasion, especially in the recovery phase after voluntary deep breathing, the intra-oesophageal pressure was lower than the endomural. There was sometimes a similar lack of correlation between endomural and intraluminal pressures. It seems unlikely, therefore, that the bronchial squeeze pressure depends directly or entirely on either the intrapleural or the intraluminal pressure.

The possibility that the bronchial squeeze pressure depends on an interaction between intraluminal and intra-oesophageal pressures was also examined. The data were not consistent with this hypothesis.

We think that the endomural bronchial ('squeeze') pressure, at least in a segmental bronchus, is partly, if not mainly, influenced by contraction of bronchial muscle. This might not hold true if there were much air trapping. The problem of differentiating the effect of air trapping from that of bronchial muscular contraction is discussed.

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