

MECHANICS OF THE TRACHEA AND BEHAVIOUR OF ITS SLOWLY ADAPTING STRETCH RECEPTORS

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SUMMARY

1. The trachea is constructed by a series of U-shaped cartilaginous rings supporting a membranous posterior wall. We have studied separately the pressure–volume relationships of the two components.

2. The motion of the membranous posterior wall contributes most to the tracheal volume change caused by any given transmural pressure change; the cartilaginous rings provide a semi-rigid support to the posterior wall and have a far greater compliance with negative than with positive transmural pressure.

3. The response of tracheal stretch receptors to transmural pressure can be explained by the mechanical coupling between cartilages and posterior wall. They respond both to positive and negative transmural pressure, they are active at zero transmural pressure and have a point of least activity with small negative transmural pressures.

4. The stress–strain relationship of the posterior wall has been studied in static and dynamic conditions in control situations and after removal of either the tunica fibrosa or the trachealis muscle. Each of these two components contributes to the stiffness of the posterior wall, with the trachealis muscle providing most of its viscosity.

5. The response of tracheal stretch receptors to transverse traction of the posterior membranous wall has been studied in both static and dynamic conditions before and after removal of the tunica fibrosa. The behaviour of these receptors reflects the visco-elastic properties of the trachealis muscle in which they have been localized.

INTRODUCTION

In most mammals, including man, the trachea is constructed of a series of U-shaped cartilages which support a membranous posterior wall. This heterogenous structure is also present in the main stem bronchi and in the extrapulmonary portions of the lobar bronchi (Vanpeperstraete, 1973).

The tracheal slowly adapting stretch receptors have been found to be located solely in the posterior membranous portion and to be activated by a transverse traction of this structure (Bartlett, Jeffery, Sant'Ambrogio & Wise, 1976*a*). In the membranous wall there are two components: its most dorsal layer is the musculus transversus tracheae (trachealis muscle) which is covered by the tunica fibrosa. The tracheal stretch receptors are located only within the trachealis muscle (Bartlett *et al.* 1976*a*).

These receptors can be stimulated both by a distending and a collapsing transmural

pressure (Widdicombe, 1954; Bartlett *et al.* 1976*a*; Bartlett, Sant' Ambrogio & Wise, 1976*b*). This suggests that their response to pressure is mediated through an increase in tension of the posterior wall supported by the cartilaginous rings.

In this study we have analysed the mechanical coupling between tracheal horseshoe cartilages and membranous posterior wall, as this determines the tension in the trachealis muscle and hence the activity of tracheal stretch receptors. Furthermore, we have studied the mechanical properties of the membranous posterior wall and the response of stretch receptors to its transverse stretching. This analysis has provided some further insight into the interrelations between a receptor's behaviour and its mechanical environment.

METHODS

The mechanical properties of the trachea and its components were studied mainly in excised specimens from dogs, cats and rabbits. The responses of tracheal stretch receptors to either transmural pressure or transverse stretching of the membranous posterior wall were studied in segments of dog trachea isolated *in situ*.

Mechanical properties of the trachea and its components. The specimens of trachea were obtained from three dogs, three cats and two rabbits; all were anaesthetized with sodium pentobarbitone. The extrathoracic portion of the trachea was used in most instances in dogs, while in rabbits and cats the excised specimens came from the whole trachea.

Each end of the tracheal segment was cannulated with a plastic tube of appropriate size and secured with a clamp to a rigid support. The length of the tracheal segment corresponded to its resting value (L_0), i.e. its length when no forces were applied. One of the plastic cannulas was connected to a Statham pressure transducer and the other to a graduated syringe. The pressure-volume relationship was determined for both inflations and deflations in steps of known volume. The volumes attained at different pressure were corrected according to Boyle's law.

It has been shown that after the trachea has been either inflated or deflated its transmural pressure progressively changes for up to 45 min, but an almost steady state condition can be reached after a period of oscillatory inflations and deflations (Martin & Proctor, 1958). We have found that after *ca.* 30 oscillations the P - V curve of the trachea, obtained as described above, reaches a steady condition and becomes very reproducible.

The anterior-posterior diameter of the trachea, corresponding to the posterior membranous wall, and its lateral-lateral diameter, corresponding to the cartilaginous rings, have been measured by two isotonic force transducers, one side of each diameter having been fixed.

The tension-length relationships of the membranous posterior wall of canine trachea have been studied in eight isolated fragments either *in vitro* or *in situ*. The fragment of posterior wall was obtained from a segment of a dog's trachea longitudinally cut along its anterior aspect. The portions of two or three cartilages nearer to the posterior wall were clamped and fixed to a rigid support connected to an isometric force transducer on one side, and to a movable support connected to an isotonic lever, which could measure its displacement, on the other. The motion of this latter support could be provided either through a motor-driven cam or manually. The transverse dimension of the posterior wall at zero transmural pressure was found to be approximately 12.00 mm, twice its resting length (l_0). The tension-length relationship was obtained both in quasi-static and dynamic conditions starting from its l_0 . The same parameters have been measured after resection of the tunica fibrosa. The force and displacement were simultaneously recorded on a Visicorder oscillograph and displayed on a Tektronix oscilloscope.

Recording from tracheal stretch receptors. Experiments were carried out on seven dogs anaesthetized with an initial i.v. injection of sodium pentobarbitone (30 mg/kg). A catheter was then inserted into the right femoral vein for further administration of the anaesthetic. A trachea cannula was inserted immediately below the cricoid cartilage. Both vagus nerves were cut in their middle cervical portions. Subsequently, the peripheral cut end of the right vagus nerve was placed through a lateral slit on a dissecting tray and flooded with mineral oil. Under a Zeiss binocular microscope, thin filaments were dissected out of the vagus nerve with microforceps and a pair of iridectomy scissors. Single fibres coming from slowly adapting stretch receptors located in the extrathoracic trachea were identified by probing this portion of the airway with

a Foley catheter whose cuff could be inflated, thus producing an outward stretch on the tracheal wall. The action potentials from the vagal fibres were amplified through an AC coupled amplifier, displayed on a Tektronix oscilloscope and recorded with a Visicorder oscillograph.

The response of fourteen extrathoracic stretch receptors to both positive and negative transmural pressure was measured before and after the cartilaginous ring adjacent to the receptor had been clamped with a brace which allowed the length of the posterior wall to be fixed. The restraint fixed the cartilage at a position corresponding to a transmural pressure of *ca.* -35 cm H_2O . At this pressure the distance between the cartilaginous tips was *ca.* 6.00 mm, i.e. equal to the l_0 of the posterior wall and, therefore, the cartilages could not exert a direct stretch on the posterior membranous wall. Then the extrathoracic trachea was cut longitudinally along its ventral aspect and a tracheal cannula inserted at the thoracic inlet. The site of the receptor was once again directly established by local probing of the posterior membranous wall. Above and below the site of the receptor a transverse section was cut between the cartilaginous rings to the membranous posterior wall. The two cartilaginous rings corresponding to the receptor's site were fixed to a clamp on the right side and attached to a movable arm on the left side. The movable arm was then attached to an isotonic lever which could measure the displacement which was monitored on the oscilloscope and recorded on the Visicorder together with the action potentials. A transverse stretch was exerted on the portion of the muscular wall containing the receptor by a motor moving a cam on which the moving arm was fixed. Generally the posterior wall was stretched to twice its length at zero transmural pressure. This elongation is far in excess of the length of the posterior wall at $+40$ cm H_2O (13 mm). The motor provided a sinusoidal motion whose frequency could be varied between 8 and 180 c/min (Fig. 1).

RESULTS

Pressure-volume relationship. We measured the volume contributed by the cartilaginous rings and the posterior wall at different tracheal pressures with the same theoretical approach that Konno & Mead (1967) used to define the volume contributions of the rib cage and the abdomen-diaphragm within the chest wall. We assumed that the two mechanical components of the trachea could be considered as separate 'parts', i.e. by changing the volume of the trachea each one of them could move as a whole, without any intrinsic deformation. Then, at a given tracheal volume, any reduction of one of the two parts should determine the expansion of the other, or, according to the terminology of Konno & Mead (1967), at each volume the trachea should have only one degree of freedom.

To prove whether our assumption was correct we plotted at different tracheal volumes the tracheal anterior-posterior (A-P) diameter, corresponding to the posterior wall, against the lateral-lateral (L-L) diameter, corresponding to the cartilaginous rings. At different volumes we reduced either the L-L or the A-P diameter, and provided that the transmural pressure did not increase markedly causing some deformation within either of the two parts, the relationship between the two diameters was described by a family of parallel iso-volume lines, approximately equidistant for equal volume increments (Fig. 2A).

These results indicate that the trachea can be considered, with little approximation, to be a system with only two parts closely interdependent at different tracheal volumes. At the points where each of the parallel iso-volume lines cross the ordinate the volume change of the trachea is entirely contributed by its posterior wall, with the cartilages fixed at their position of minimum volume. The top iso-volume line represents the maximum volume considered and, therefore, the contribution of the posterior wall is taken as 100% . On this basis in Fig. 2B the volume contribution of each of the two components has been plotted as percent of the range of volumes

investigated; therefore, for each volume we could calculate the volumes contributed separately by either the cartilaginous rings or the posterior wall and, hence, the P-V relationship for each of the two parts.

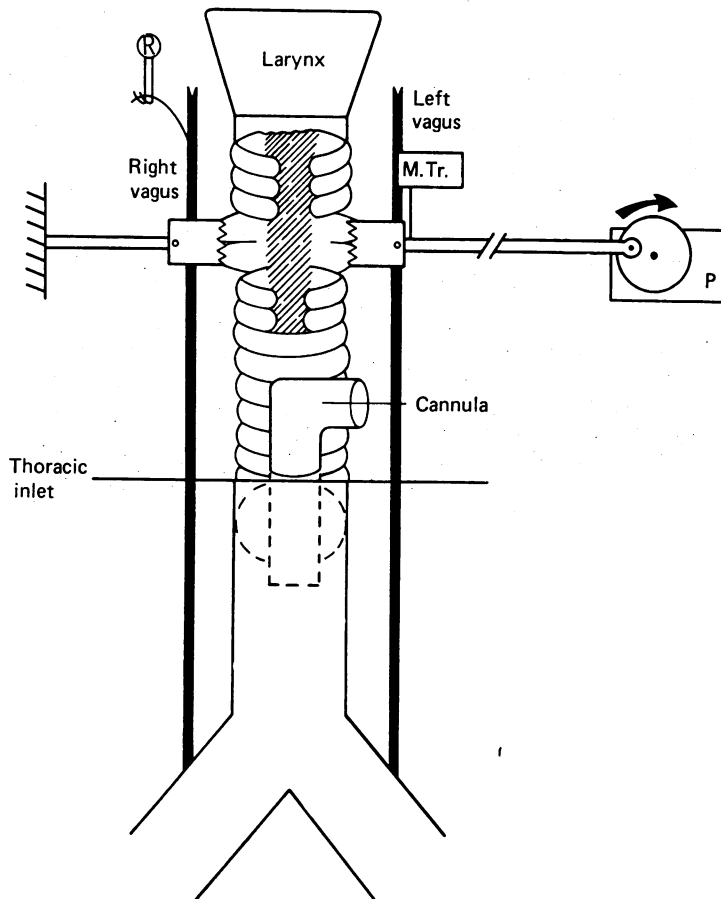


Fig. 1. Diagrammatic representation of the experimental arrangements. The portion of extrathoracic trachea, where the stretch receptor has been localized, is cut longitudinally along its anterior aspect and its posterior wall (dashed area) exposed; the dog breathes spontaneously through a lower cervical cannula. The portion of the posterior wall where the receptor is located is stretched transversally by the moving arm attached to the motor (P). This provides a sinusoidal motion at variable frequencies. The other side of the fragment is fixed by clamping the tip of the cartilage. The displacement of the moving arm is recorded by an isotonic force transducer (M.Tr.). R = recording electrodes.

The P-V curve of each of the two components could also be constructed by keeping the other part fixed in the position it occupied at the lowest tracheal volume considered, i.e. at -40 cm H_2O transmural pressure. In this condition the increase in volume was only contributed by the part free to expand and, therefore, the resulting P-V curve is related only to that component.

Actually, since the two parts are mechanically very different, it was hardly possible to increase the volume after having 'fixed' the A-P diameter because the trachea,

thus constrained, was so rigid that the pressure changes necessary to expand it would have caused it to expand with considerable deformation (i.e. at each volume the system would have more than one degree of freedom) and our assumption would no longer have been valid.

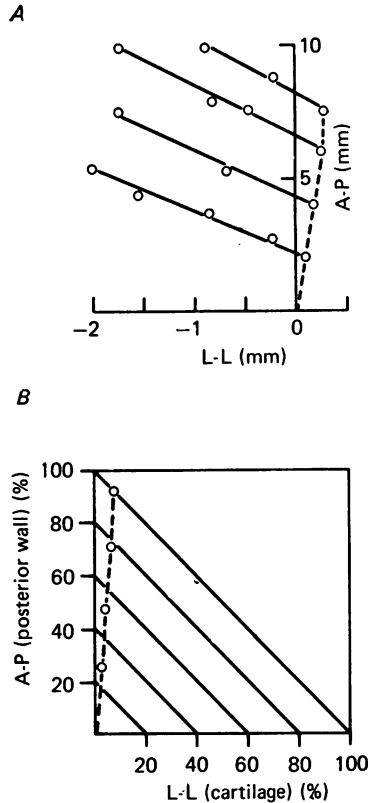


Fig. 2. *A*, A-P (posterior wall), L-L (cartilages) relationship at different tracheal volumes (dashed lines). At different volumes the restraining of the cartilages determines a corresponding expansion of the posterior wall (continuous lines). The iso-volume lines constructed in this way are parallel and almost equidistant for equal volumes increments. *B*, the volume contributions of each of the two components (posterior wall and cartilages) is represented as percentage of the maximum volume considered, as derived from *A* (see text). The dashed line represents the volume partitioning of the two components. Each parallel line corresponds to a different volume of the trachea, which each of the two components may contribute from 0 to 100% (iso-volume manoeuvre).

Therefore, it is only possible to keep the L-L diameter fixed at its lowest position (reached at a transmural pressure of *ca.* -40 cm H_2O) so that any increase in volume of the trachea represents the volume contributed only by its posterior wall. The P-V relationship of the posterior wall constructed in this way corresponds exactly to that obtained by measuring at each point of the P-V relationship of the trachea the relative volume contributed by the posterior wall, as described above (Fig. 2*B*).

Therefore, in order to study separately the volume contributions of the posterior wall and of the cartilaginous rings, we measured the P-V curve of the unrestrained

trachea and afterwards, having fixed the cartilaginous rings at their lowest position (i.e. at the lowest tracheal volume considered), we repeated the manoeuvre, obtaining the P-V relationship of the posterior wall. Subtracting this function from that of the whole trachea we could obtain the P-V curve of the cartilaginous rings.

The pressure-volume relationships for the tracheal segments of three dogs, three cats and two rabbits are represented in Fig. 3 (top panels). The volume (ordinate) is expressed as percent of its change within the pressure range considered (from -40 to $+40$ cm H₂O). The zero percent volume does not indicate a complete collapse of these tracheal segments. The separate relative volume contributions of the posterior membranous wall (PW) and of the cartilages (CR), are represented in the middle and bottom panels for each species.

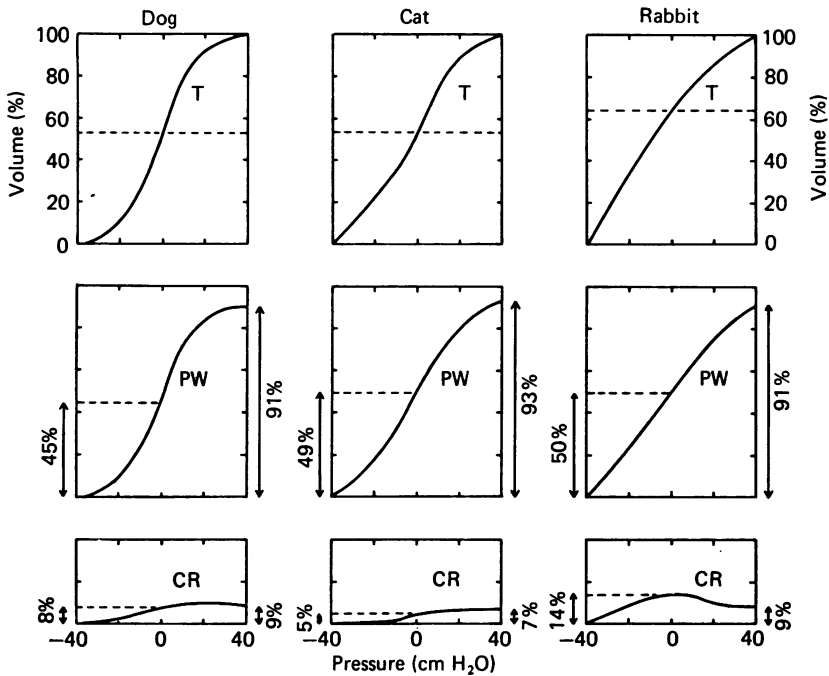


Fig. 3. Pressure volume relationships of a tracheal segment (T, panels at the top), of its posterior wall (PW, panels in the middle) and of the cartilaginous rings (CR, panels at the bottom) for a dog (left), a cat (middle) and a rabbit (right). One hundred per cent volume represents the volume displaced by the trachea within the pressure range considered (± 40 cm H₂O, abscissa). The curve of the cartilage is the difference between the first two relationships.

The compliance of the trachea appears to be lower with positive than with negative pressures and the general pattern of the P-V curves is similar to that found in dogs *in vitro* by other authors (Martin & Proctor, 1958). The posterior wall appears to contribute most of the volume changes of the trachea in all the species considered: 91% in dogs, 93% in cats and 91% in rabbits (middle panels of Fig. 3). The cartilages provide most of their small contribution at negative pressures: 8 out of 9% in dogs, 5 out of 7% in cats, and in rabbits their contribution with negative pressure (14%) is even greater than their total contribution (9%) (bottom panels of Fig. 3). This

indicates that in rabbits with distending pressures the outward bulging of the posterior wall pulls the cartilages inward.

Tension-length relationships of the posterior wall. A representative tension-length relationship of a fragment of posterior wall of the trachea is represented in Fig. 4 (top left panel, heavy continuous line, filled circles). The general pattern is in

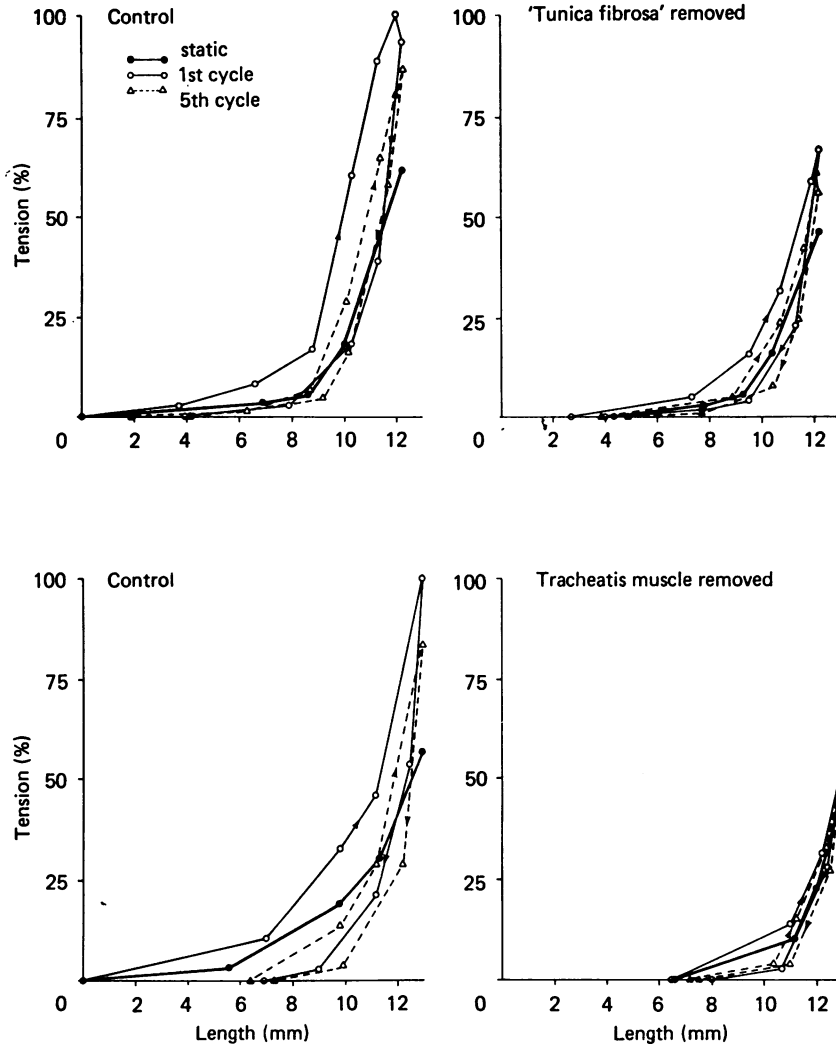


Fig. 4. Tension-length relationships of the posterior wall of a representative tracheal segment before (control, left upper panels) and after (right upper panel) removal of the 'tunica fibrosa'; the same relationships are shown for another sample of posterior wall before (control, left lower panel) and after (right lower panel) resection of the trachealis muscle. In each panel are represented the static relationships (heavy continuous lines) and the dynamic relationships for the first (thin continuous lines) and the fifth oscillations (dashed lines). The tensions (ordinate) are represented as percentage of the maximum tension of the corresponding control situation. The trachealis muscle alone (right upper panel) still shows most of the viscous properties of the posterior wall, while the 'tunica fibrosa' by itself (right lower panel) makes only a small contribution to the viscous properties of the posterior wall.

agreement with that described by Stephens (1970). This relationship was markedly modified in all the eight samples by the removal of the tunica fibrosa (Fig. 4, top right panel) which appears to account for a considerable fraction of the stiffness of the membranous posterior wall.

When the same preparation was subjected to oscillatory stretching of constant amplitude at a frequency of 38 c/min, there was a progressive decrease in tension which reached an approximately steady value only after 30 oscillations.

The tension-length relationships of the posterior wall for the first and the fifth oscillations are represented in Fig. 4 before (top left panel) and after (top right panel) resection of the tunica fibrosa and before (bottom left panel) and after (bottom right panel) resection of the trachealis muscle. There was a marked hysteresis, as

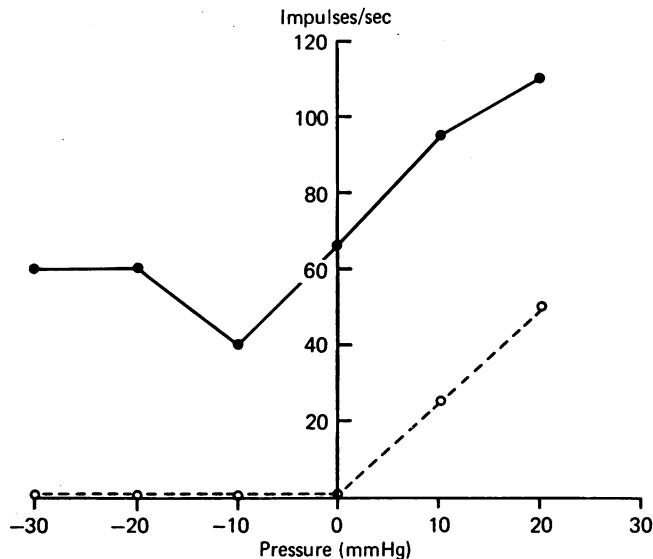


Fig. 5. Response of a tracheal stretch receptor (impulse/sec) to a maintained positive and negative pressure without (filled circles, continuous line) and with the transverse compression of the corresponding cartilaginous ring (open circles, dashed line). In this latter situation the response of the receptor is less.

indicated by the width of the tension-length loops both in control situations and after the removal of the tunica fibrosa, especially evident during the first oscillation. In contrast, after removal of the trachealis muscle (bottom right panel) the hysteresis of the posterior wall was greatly reduced. These findings indicate that the hysteretical properties of the posterior wall are predominantly those of the trachealis muscle. Similarly Hughes, May & Widdicombe (1959) provided evidence that the hysteretical properties of the tracheal segment isolated *in situ* depend on its smooth muscle.

Response of the tracheal stretch receptors to transmural pressure

Fig. 5 represents the response of a tracheal stretch receptor to maintained distending and collapsing pressures (filled circles, continuous line). This behaviour, which is representative of most of the tracheal stretch receptors, show that the receptor was active at zero transmural pressure, i.e. at the resting volume. This

activity disappeared when the cartilaginous ring, corresponding to the receptor site, was fixed in its position at -40 cm H_2O transmural pressure (open circles, dashed line). The response to positive pressure after the cartilage had been restrained (open circles, dashed line) was less than in the control situation. The response to negative pressure was entirely abolished by the restraint and this may indicate that the posterior wall was, in this condition, completely supported by the cartilages. With a negative pressure corresponding or in excess to that necessary to narrow the cartilages at their restrained position the receptor's activation did not differ from that in the unrestrained condition.

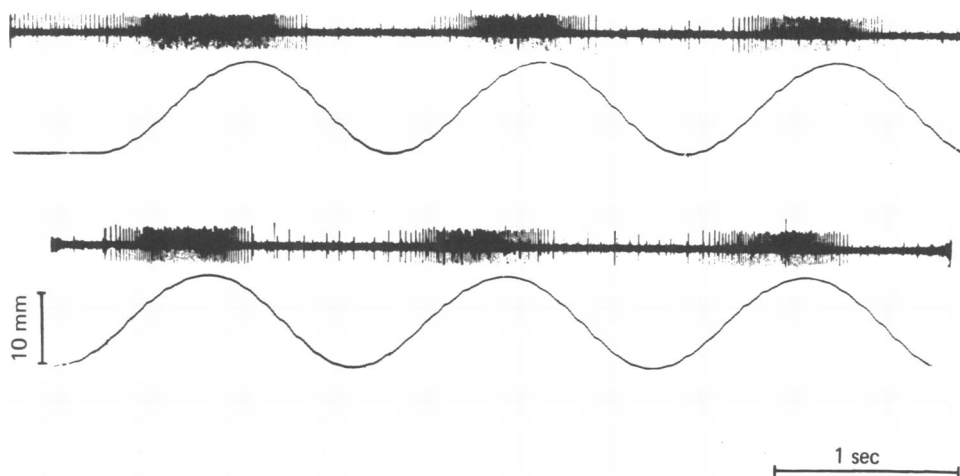


Fig. 6. Pattern of discharge of a tracheal stretch receptor (top tracing) during the first six sinusoidal oscillations of constant amplitude (bottom tracing) at a frequency of 38 c/min; the two records are continuous. The response of the receptor decreases between the first and the successive oscillations.

Response of tracheal stretch receptors to transverse stretching of the posterior wall

The tracheal slowly adapting stretch receptors respond only to transverse stretching of the posterior wall and not to longitudinal tension (Bartlett *et al.* 1976a). Fig. 6 shows the discharge pattern of a tracheal stretch receptor during the first six sinusoidal oscillations of a series. Fig. 7, left panel, represents the response of one of these receptors to maintained transverse stretching of different amplitude (open squares, heavy continuous line) and to the first (open circles, dashed line) and the fifth (filled circles, thin continuous line) sinusoidal oscillations of the same maximal amplitude. By comparing the responses to the two oscillations we can observe that the threshold for the receptor's activation had increased by the fifth cycle and that, more generally, its response to any given elongation had decreased.

In Fig. 7, right panel, the response of the same stretch receptor is represented for the same experimental condition after removal of the overlying tunica fibrosa. There is a marked increase of the receptor's discharge during the stretching leg of the first oscillation. Its response to maintained stretching and to the fifth applied oscillation do not appear to be different.

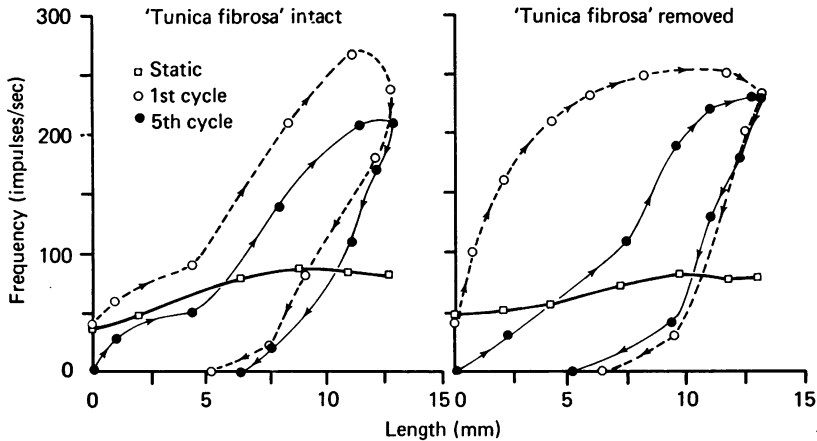


Fig. 7. Response of a tracheal stretch receptor (impulse/sec) to transverse stretching of the posterior wall (l , mm; zero is the length of posterior wall at zero P_{tm}) before (left panel) and after (right panel) removal of the 'tunica fibrosa'. The heavy continuous lines (\square) represent its response to maintained distension, the dashed lines (\circ), its response during the first applied sinusoidal oscillation and the thin continuous lines (\bullet) its response during the fifth sinusoidal stretching.

DISCUSSION

Our results indicate that most of the volume changes of the trachea following a variation in transmural pressure are contributed by the posterior wall. Essentially the cartilages give their contribution only below the resting volume of the trachea. This asymmetrical behaviour could lead to a lower posterior wall tension with negative pressure than with positive pressure. In fact, with negative pressures the radius of curvature of the posterior wall, because of the closing of the cartilaginous rings, decreases more than with positive pressures, and could even cause a reduction of its tension. This fact could explain the asymmetry of the response of the stretch receptors to maintained positive and negative pressures (Fig. 5, continuous line) and confirms that these receptors are stimulated by transverse tension of the tracheal posterior wall.

If the cartilaginous ring were a completely rigid support, and the posterior wall, at zero transmural pressure, were approximately at its l_0 (neither of these two assumptions being true, as previously mentioned, see Methods), the tension applied to the posterior wall at different transmural pressure (P_{tm}) could be calculated according to the Laplace's relationship, if the radius of curvature (R) of the posterior wall is known (Fig. 8).

From the diagram of Fig. 8 it is obvious that

$$R = a + k. \quad (1)$$

From Pythagoras' theorem

$$R^2 = h^2 + k^2. \quad (2)$$

A radius drawn at right angles to any chord will bisect that chord. Hence the chord

$$L = 2h. \quad (3)$$

Substituting k and h from (1) and (3) in eqn. (2):

$$R^2 = \frac{L^2}{4} + (R - a)^2.$$

Simplifying,

$$2aR = \frac{L^2}{4} + a^2.$$

Dividing both sides by $2a$,

$$R = \frac{1}{2} \left(\frac{L^2}{4a} + a \right). \quad (4)$$

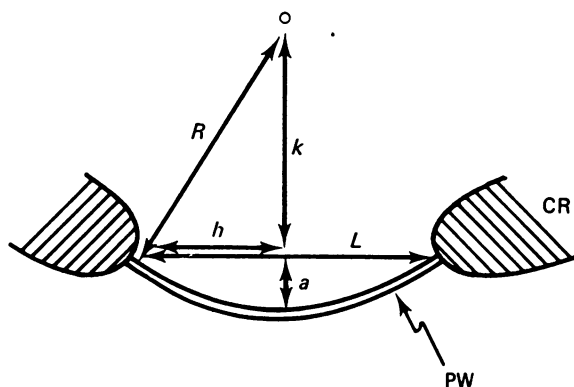


Fig. 8. Diagrammatic representation of a tracheal cross-section of the posterior region. CR = cartilaginous ring; PW = posterior wall; R = radius of curvature of PW which is part of a circle having centre O and a chord L. See text for explanations.

When the cartilaginous rings are kept fixed (see Methods) in eqn. (4) L is constant and a is proportional to the changes of tracheal volume above (positive pressure) and below (negative pressure) the resting volume of the trachea, and its values are obtainable from the data of Fig. 3, middle panel. The radius of curvature calculated in this way multiplied by P_{tm} gives the tension applied to the posterior wall according to the Laplace's relationship ($T = PR$). This tension is represented at different P_{tm} in Fig. 9 (dashed line). The reason why this curve is not perfectly symmetrical is that the compliance of the posterior wall is slightly different with positive and negative pressures (Fig. 3, middle panels).

The mechanical coupling between cartilages and the posterior wall is such that even at zero P_{tm} the posterior wall is stretched above its l_0 and, furthermore, the cartilaginous rings are not a completely rigid support, but move slightly, particularly with negative transmural pressures (Fig. 3, bottom panels), therefore applying on the posterior wall a tension proportional to the distance between the tips of the cartilage, i.e. L . We have measured L at different transmural pressures, within the range -40 to $+40$ cm H_2O , and determined *in vitro* the tension-length relationship for the corresponding lengths. This tension ($T = kL$) is represented at different P_{tm} by the thin continuous line in Fig. 9. This curve is similar to the P - V curve of the cartilage (Fig. 3, bottom panels) because the tension-length relationship of the posterior wall, corresponding to a variation of P_{tm} between -40 and $+40$ cm H_2O , is almost linear.

The total tension applied to the posterior wall of the trachea (heavy continuous line in Fig. 9) has been obtained by adding the two functions $T = PR$ (dashed line) and $T = kL$ (thin continuous line). Actually, with increasing P_{tm} , the tips of the cartilages move out and the radius of curvature of the posterior wall, at any given P_{tm} , should be greater than if the cartilaginous rings were a rigid support, and therefore the tension should be greater. The difference is probably very small because the motion of the cartilage relative to that of the posterior wall is small, and in any case similar results are obtained if in eqn. (4) L is considered either equal to 5 or to 10 mm.

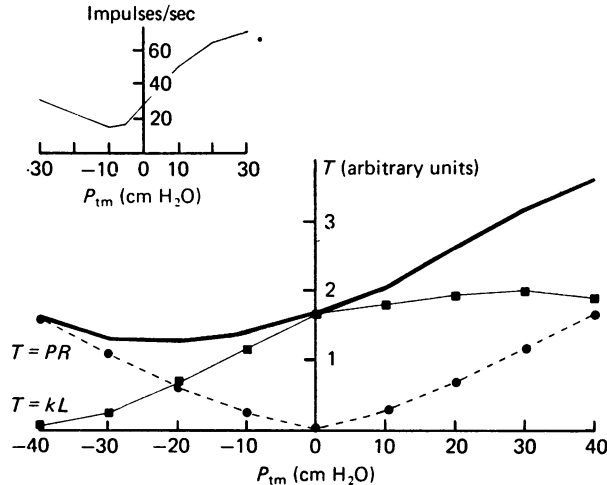


Fig. 9. Relationship between the tension applied to the posterior wall of the trachea (T , arbitrary units) and transmural pressure (P_{tm} cm H₂O). The dashed line represents the tension calculated according to the Laplace relationship ($T = PR$) assuming a completely rigid cartilaginous ring. The thin continuous line represents the tension applied to the posterior wall by the cartilages ($T = kL$), where k is a proportionality constant and L the elongation of the posterior wall. The thick continuous line is the sum of the two relationships and represents the total tension applied to the posterior wall, its shape is very similar to that representing the response of tracheal stretch receptors (impulses/sec) to transmural pressure as shown in the inset obtained from the data of Sant'Ambrogio & Mortola (1977).

In conclusion, the tension applied to the posterior wall at different P_{tm} is very similar to the static response of tracheal stretch receptors, as represented in the inset of Fig. 9. This suggests that the static response of the tracheal stretch receptors to P_{tm} is strictly related to the tension applied to the posterior wall of the trachea, in which they are located, and confirms that their asymmetrical response to positive and negative P_{tm} can be explained by the mechanical coupling between cartilages and posterior wall.

The asymmetry of their response to transmural pressure and their discharge at zero transmural pressure render the slowly adapting stretch receptors of the extrathoracic trachea capable of differentiating between inspiration (their activity decreases) and expiration (their activity increases). Had the supporting cartilaginous rings been completely rigid there would have been an almost symmetrical response to negative and positive transmural pressures. In this case, therefore, equally

increasing signals would have originated during each of the two phases of the respiratory cycle.

The greater compliance of the cartilages with negative transmural pressure could not entirely explain the ability of tracheal stretch receptors to differentiate between positive and negative pressures unless they were active at zero transmural pressure. Indeed, most of the tracheal stretch receptors are active at zero transmural pressure (Miserocchi & Sant'Ambrogio, 1974; Bartlett *et al.* 1976*b*; Sant'Ambrogio & Mortola, 1977). This activity disappears when the corresponding cartilage is restrained (Fig. 5, dashed line); this fact indicates that the cartilaginous rings keep the membranous posterior wall of the trachea stretched even at zero transmural pressure (Macklin, 1929).

These findings suggest that the mechanical coupling between the cartilages and the posterior wall is a key factor in determining the response of these receptors to transmural pressure.

As previously pointed out, these stretch receptors are found solely in the posterior wall and respond only to transverse traction of this structure. Stretch receptors showing a decreasing sensitivity with increasing values of transmural pressure (type 1, Miserocchi & Sant'Ambrogio, 1974) still show a similar pattern of discharge with transverse traction even after the 'tunica fibrosa' has been resected. This finding seems to indicate that if there are structures, in parallel with the receptors, which resist stretching in response to transmural pressure, they are likely to be within the trachealis muscle itself. Another consideration is related to the observation that the maximum frequency of discharge observed with transmural pressure stimulation is always reached with transverse stretching of the posterior wall. This suggests once more that the response to transmural pressure is mediated by transverse traction of the membranous posterior wall.

In Fig. 7 we can compare the response of an ending to a maintained elongation and to a sinusoidal oscillation of the same maximal amplitude. We can see that for any elongation the response is greater when the posterior wall is dynamically stretched. This indicates that the receptor's activity is not only related to the length but also to its rate of change. The presence of a dynamic sensitivity has similarly been demonstrated by Bradley & Scheurmier (1977) for tracheal stretch receptors studied *in vitro*.

The dynamic sensitivity decreases from the first to the fifth cycle and this behaviour parallels the decrease in force with oscillations of constant amplitude as shown in Fig. 4 (top left panel); hence these receptors sense the force applied to the structure in which they are placed. Analogous observations have been made on airways stretch receptors by Davis, Fowler & Lambert (1956) during successive inflations and deflations of the entire tracheo-bronchial tree. The difference between the response of the ending between the successive cycles appears to be greater after the removal of the 'tunica fibrosa' (Fig. 7, right panel) in the majority of the receptors studied.

The dynamic sensitivity of these endings, which appear to be proportional to the amount of hysteresis, seems to be related to the viscous properties of the trachealis muscle in which they have been located (Fig. 4). Similar considerations have been brought forward for the dynamic sensitivity of primary endings of the muscle spindles (Smith, 1966; Andersson & Lennerstrand, 1966). The tunica fibrosa *per se* does not contribute to the viscous properties of the posterior wall (Fig. 4, top two panels) and,

as indicated by the firing pattern of most of the receptors, should actually decrease the intrinsic viscosity of the trachealis muscle. In any event, this possibility is not supported by the tension-length relationship after the removal of the 'tunica fibrosa' which does not indicate any increase in hysteresis.

In conclusion, the activation of tracheal stretch receptors by the transmural pressure can be explained by the change in transverse tension of the posterior wall. Furthermore, the response of these receptors to transverse stretching of the posterior wall is explicable from the visco-elastic properties of the trachealis muscle.

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