INTRODUCTION

"In breathing there are two kinds of blessings: inhaling the air and exhaling it; the former is oppressive, the latter refreshing, so strangely is Life mingled. Thank God when He lays a burden on thee and thank Him when He takes it off."—Goethe.

It is a daily event in anaesthesia, and not uncommon in other spheres of medical practice, to meet with the patient who is in need of relief from the burden of inhaling; and one means of relieving such a patient of his burden is the application of positive pressure ventilation.*

The safe application of positive pressure ventilation is, however, no easy matter: for much time, thought, and energy, must be expended if the disadvantages of the method are to be surmounted. If they cannot be surmounted—and the problems they give rise to increase in difficulty as the period of ventilation lengthens—complications develop, and, in the long term medical case, it is then very soon apparent that the resort to positive pressure ventilation has failed: that it has resulted in the conversion of what should have been a rapidly fatal condition into a lingering and exceptionally distressing terminal illness.

One of the disadvantages of positive pressure ventilation is that the method is not only incapable of assisting the patient in exhaling but also capable of causing exhalation to be prolonged: for it is practicable only when the diaphragm and the muscles of the chest wall are atonic; and when these muscles are atonic the lungs are apt to deflate slowly, particularly if they are fibrotic or oedematous or if the chest wall has lost its natural resilience. Now if exhalation is unduly prolonged the patient may still be exhaling at the time the inflation pressure is applied; and in that event exhalation will be cut short, and the resting volume of the lungs will increase.

This increase can produce complications in the circulatory, respiratory, and muscular systems. It may raise the mean intrapleural pressure and so impede the venous return to the heart. It will bring about an increase in the volume of the deadspace air and so impair the efficiency of ventilation. Moreover, by holding the chest partly expanded in the intervals between inflations, it will prevent the diaphragm and the muscles of the chest wall from regaining their normal resting lengths, and may thereby jeopardize the functional recovery of the internal intercostal muscles;
for these muscles will be held under tension all the time that the chest is held expanded—and muscle which has been stretched whilst in the paralysed state either takes a long time to recover its tone or fails to recover it at all. Even the relatively short applications of positive pressure ventilation used in anaesthesia may, by their effect of continually relaxing the external intercostals and continually stretching the internal intercostals, result in the former muscles recovering their tone before the latter; a circumstance that would interfere with the concerted action of these muscles in the immediate post-operative period and delay the return of the patient’s full expulsive powers.

Another disadvantage of positive pressure ventilation is that it subjects the chest to unidirectional stresses when the muscles are paralysed, be borne by the bone, cartilage and fibrous tissue, of the chest wall. These tissues are malleable or deformable; and it is because of that, and because the ventilating pressure, though a small enough force in itself, is applied to them at the rate of some 20,000 applications each 24 hours, that it is undesirable that the stress produced by the pressure should be unidirectional.

Just how long the chest can withstand the stresses of positive or negative, pressure ventilation (for the latter too produces unidirectional stress) is uncertain. It has, however, been the writer’s experience that a “ballooning out” of the upper and anterior parts of the chest wall is often to be detected by the end of the third or the fourth week of ventilation—and that the appearance of the chest by the end of the third or the fourth month of ventilation is not infrequently such as would justify its being described as “blown”.

That the deformity appears first in the upper and anterior parts of the chest is probably because there the internal thrust of the intrathoracic pressure is assisted by the external pull of the accessory muscles of respiration. It always heralds the approach of a rapid deterioration in the effectiveness of the pulmonary exchange, and it should not, of course, be permitted to develop—but how it is to be prevented from developing is another question, and one that is likely to remain unanswered until the orthopaedic implications of artificial ventilation are better understood than they are today.

It is not improbable, however, that the structural changes produced by ventilation vary as the mean value of the ventilating pressure varies, and that they would be reduced to nil if the mean value of the ventilating pressure could be reduced to zero. But the mean value of the pressure cannot be reduced to zero when positive pressures are used alone: it can be so reduced only when two pressures of opposite sign are at work—when, for example, a positive inflation pressure alternates with a negative expiratory pressure. To put the matter in another way; an expansion strain requires for its correction a compression strain; and a negative expiratory pressure will create a compression strain by causing the chest to be compressed by the atmospheric pressure in the intervals between inflations. Another means of limiting the structural changes produced by ventilation would be to limit the expansion of the chest by the application of some form of support or “splint” to the chest wall.
To return for a moment to the source of some of these problems—namely, the prolongation of exhalation. That it is possible for exhalation to become dangerously prolonged follows from the fact that positive pressure ventilation plays no part in the production or in the control of pulmonary deflation; that it is a form of ventilation in which only one phase, usually less than one-half, of the ventilatory cycle is under the anaesthetist's full control.

And it was to provide the anaesthetist with a mean of controlling both inflation and exhalation, and of assisting the patient in exhaling, that the respirator shown in figure 1 was designed.

**THE RESPIRATOR**

This, the Fazakerley respirator, provides positive inflation pressures of 10 to 30 cm H$_2$O, and provides for the control of the frequency and the phases of the ventilatory cycle. And it can also be made to develop a negative pressure in the airway during the exhaling or expiratory phase.

For its motive power, the respirator depends on compressed air: the supply requirements being a flow of 20 to 30 litres a minute and a supply pressure of 100 to 360 mm Hg. In the absence of a suitable air supply, oxygen may be used, but in the interests of economy the gas is usually diluted with air before it is delivered to the machine, its admixture with air being readily effected by means of an air-entraining device that will be described later.

The principal parts of the respirator are "arrowed" in figure 1. They are: an intermittent positive pressure, or I.P.P., unit (3); an expiratory pressure control, or E.P.C., unit (2); a glass pressure bottle (1); a manometer (6); a corrugated flexible connecting tube (4); and an air lead (5).

Of these components, the I.P.P. and the E.P.C. units are those responsible for the production of the positive and the negative pressures. They also provide the innovative features of the respirator, and each unit will therefore be described in detail.

Before they are described it must, however, first be explained how the machine works as a whole, although a short explanation is all that will be required, because the general principles on which the pressure-cycled type of respirator works are by now well known. Briefly then, the Fazakerley respirator operates as follows:

![Diagram of the Fazakerley respirator](https://academic.oup.com/bja/article-abstract/28/4/176/243660)
Inside the glass pressure bottle is a one-gallon rubber bag. This is connected with and employed as the rebreathing bag of the CO₂ absorption apparatus that happens to be in use, when compression and expansion of the bag will produce inflation and deflation of the patient’s lungs. The necessary compression and expansion of the bag is brought about by alternately raising and lowering the air pressure in the bottle: the I.P.P. unit producing a positive pressure in the bottle, and the E.P.C. unit a negative pressure (or an atmospheric pressure), turn about. By generating and applying these pressures the respirator acts as a pneumatic hand and serves as a mechanical means of ventilating the lungs.

The I.P.P. Unit (fig. 2).

The air supplied to the respirator passes through the air-lead (A) to reach and enter the E.P.C. unit, which unit should, for the present, be regarded as a plain L-shaped connecting piece that has been fitted with a side-tube (S).

From the E.P.C. unit the air is free to flow through the corrugated connecting tube (E), the distributor tube (D), the short flexible connection (F), and the cylinder (C), to an exhaust port (e). It is also free to flow into the glass pressure bottle by way of the mount (M).

The port (e) is opened and closed by a mechanically operated valve (V), with the result that the air admitted to the apparatus is alternately able and unable to escape from the apparatus to the atmosphere; the period during which the port is open constituting the expiratory phase, and the period during which it is closed the inflation phase.

The Inflation Phase of the Ventilatory Cycle (figs. 2, 3, 4).

When the exhaust port is closed, the air supplied to the respirator flows into a closed system. It therefore creates pressure.

This pressure is applied to the bag in the bottle and, through a one-way valve (U), to a bellows (B).

The bag in the bottle is compressed by the pressure and its contents are insufflated into the patient’s lungs, which inflate.

The bellows too inflate in response to the pressure—but not until the pressure in the system is great enough to overcome the tension of a spring (T).
This spring extends from the horizontal arm of a lever (L) to a hook located on the underside of the cylinder (C). Its action is to pull the lefthand end of the cylinder downwards against the bellows. The bellows therefore cannot expand until the expansion force of the air pressure inside them exceeds the compression force of the spring.

When the bellows do inflate, they drive the lefthand end of the cylinder upwards, causing the cylinder to tilt on the pivot (X) by means of which it is mounted on the brackets (H).

The slope of the cylinder is thus reversed, and the heavy ball (W) consequently rolls from the left- to the right-hand end of the angled perspex strip that forms the valve (V)—causing the valve to "see-saw" into a position which leaves open the port (e).

With the opening of the exhaust port the inflation phase ends and the expiratory phase begins, but before the expiratory phase is described the means by which the inflation pressure and the length of the inflation phase are controlled must be explained.

The Control of the Inflation Pressure (fig. 2).

The peak inflation pressure is the pressure present in the apparatus at the moment the exhaust port opens. It varies as the tension of the spring (T).
To increase the tension of the spring, and so raise the inflation pressure, the anaesthetist turns the knob (P) clockwise. By doing so he screws a threaded rod to which the knob (P) is attached against the vertical arm of the lever (L), causing that arm of the lever to be pushed to the left and the horizontal arm of the lever to be depressed—when the spring will be further stretched and its tension increased. To lower the inflation pressure the anaesthetist turns the knob anticlockwise.

**The Control of the Length of the Inflation Phase.**

The length of the inflation phase varies as, and is controlled by varying, the rate at which air is supplied to the respirator.

**The Expiratory Phase and its Control (figs. 5 and 6).**

The opening of the exhaust port has the immediate effect of reversing the pressure gradient across the oneway valve (U), and the valve therefore closes. It also has the effect of dispersing the pressure in the bottle, so that the patient's lungs are free to deflate.

The bellows, too, deflate, but to do so they must expel the air they contain through the rubber tube (G), since this tube is the only exit from the bellows. The tube is furnished with a clamp (CL), which the anaesthetist can open and close by turning the knob (R) anticlockwise and clockwise respectively, and the rate at which the bellows deflate depends on the extent to which the clamp obstructs its lumen.

Now the more rapidly that the bellows deflate, the more rapidly will the spring (T) draw the lefthand end of the cylinder downwards again, the sooner will the cylinder regain its original angle of slope, and the sooner will the ball (W) roll back to close the valve and bring the expiratory phase to an end. Contrariwise, the more slowly that the bellows deflate the longer will be the period that elapses before the expiratory phase ends.

The knob (R) therefore controls the length of the expiratory phase of the cycle: to provide an expiratory pause, the anaesthetist has only to turn the knob clockwise until the bellows deflate more slowly than the lungs deflate.

**The Control of the Ventilatory Rate.**

The ventilatory rate (or the frequency of the mechanical cycle) is governed by
the factors shown in the following equation:

\[
\text{Rate} = \frac{60}{\text{Length of inflation phase in seconds} + \text{Length of expiratory phase, including any pause, in seconds}} \text{ cycles/min.}
\]

Anything that reduces the value of the denominator of the equation will increase the rate, and anything that increases the denominator of the equation will reduce the rate. In practice, the anaesthetist usually alters the rate by providing a slightly longer or shorter expiratory phase.

The E.P.C. Unit (fig. 2).

The upper one-third of the E.P.C. unit forms a small air chamber. This chamber has an inlet for the admission of air, the side tube (S), and two exits for its discharge: the annular space (O), and the bore of the jet (J).

The first of these exits, the annular space, is a variable aperture, since clockwise rotation of the knob (K) will drive the cone-shaped point of the jet into the annular space, causing the space to close; whilst anticlockwise rotation of the knob will withdraw the jet, causing the space to enlarge.

The second exit, the bore of the jet is a fixed aperture.

Of the compressed air admitted to the air chamber, some will be discharged from the chamber by way of the bore of the jet valve, and some by way of the annular space; and the way in which the discharge of air is distributed between these two exits determines the pressure in the mount (M). For the air that is discharged from the chamber by the bore of the jet valve is injected into a venturi tube (N), and its entry into the venturi tube will tend to create a negative pressure in the mount; whereas the air that is discharged from the chamber by way of the annular space will enter the mount itself, under pressure—a circumstance that tends to create a positive pressure in the mount.

The two tendencies vary in strength as the airflows by which they are produced vary in strength; and since the anaesthetist can, by manipulating the knob (K), increase the strength of either flow, it follows that he can accentuate either tendency, and so produce a positive or a negative pressure in the mount at will. And that he can produce a positive or a negative expiratory pressure—because the pressure produced in the mount will be
propagated to the pressure bottle and reproduced in the rebreathing bag during the expiratory phase. During the inflation phase, the small pressure produced in the mount by the tendencies under discussion is overwhelmed to insignificance by the inflation pressure.

How the anaesthetist adjusts the expiratory pressure in practice is explained below.

To produce a negative expiratory pressure.

The anaesthetist turns the knob (K) clockwise in order to reduce the patency of the annular space and so increase its flow resistance. Once the flow resistance of the space exceeds the flow resistance of the bore of the jet valve, most of the air admitted to the air chamber will leave the chamber by the bore of the valve (i.e. by the easier route), with the result that more air will pass through the venturi tube than will pass into the mount. The dominant tendency will, therefore, be that produced by the venturi tube, and the pressure in the mount will become negative—and increasingly so as the knob continues to be turned. Once the space has been completely closed, no air at all can enter the mount and, the venturi effect being unopposed, the pressure in the mount may fall to as low as minus 10 cm H₂O.

To produce a positive expiratory pressure.

The anaesthetist turns the knob (K) anticlockwise in order to increase the patency of the annular space, and so reduce its flow resistance. Once the flow resistance of the space has fallen below that of the bore of the jet valve, most of the air admitted to the air chamber will leave by way of the annular space. More air will therefore pass into the mount than will pass into the venturi tube and the dominant tendency will be that produced by the expansion of air in the mount. The pressure in the mount accordingly becomes positive. Once the space is fully open, little or no air enters the venturi tube and, the expansion effect of the air entering the mount being unopposed, the pressure in the mount rises to around 5 cm H₂O.

To produce atmospheric expiratory pressure.

The anaesthetist turns the knob (K) one way or the other until the manometer registers atmospheric pressure during the expiratory phase. He will then in effect have matched the flow resistance of the annular space with that of the bore of the jet valve; and equal volumes of air will be passing into the venturi tube and the mount to produce equal but opposite effects.

From the point of view of clinical usage, the E.P.C. unit may be said to function only during the expiratory phase of the cycle. The pressure created in the mount by the interaction of the two forces just described does, however, play a minor role during the inflation phase of the cycle. A negative pressure, for example, then acts as an anti-inflationary force; as is evident by the rise in the peak inflation pressure that occurs when a negative pressure is cancelled, and by the fall in the inflation pressure that occurs when the strength of a negative pressure is increased. Conversely, the provision of a positive expiratory pressure raises, and its cancellation lowers, the peak inflation pressure. These side effects to the use of the E.P.C. unit have one, apparently quite unimportant,
result: that the difference between the maximum and the minimum pressure remains constant for any given setting of the inflation pressure control knob (P) no matter how the expiratory pressure control knob (K) may be set and reset.

**MANOMETER; PRESSURE RELEASE VALVE; AIR-ENTRAINING DEVICE**

There remain to be described: the manometer (fig. 2); the pressure release valve (fig. 2 (RV)); and the air entraining device.

The manometer is connected with the breathing circuit. Because of that, and because it is a water manometer, it serves not only as a pressure indicator but as a safety valve as well, for the height and the depth of the manometer tube will limit the positive and the negative pressures that the machine can apply to the lungs.

The pressure release valve (RV) consists of a rubber flap covering a hole in the bag mount (BM). The valve lifts to allow excess gas to pass from the breathing circuit to the bottle, and thence to the atmosphere during the expiratory phase. It prevents the rebreathing bag from over-distending during use.

The air-entraining device which is not illustrated here saves oxygen when the respirator is worked from a cylinder. Designed to be connected with B.O.C. oxygen valve MS.55, or MS.56, by a short length of stout-walled rubber tubing, and with the respirator by not more than 10 feet of 3/8-inch bore tubing, the device utilizes the injector principle to convert a small volume of oxygen at a high pressure into a larger volume of air and oxygen at a relatively low pressure. It is capable of supplying up to 40 litres of air-oxygen mixture a minute and of delivering the mixture, which contains 30 to 35 per cent oxygen, under a pressure of about 100 mm Hg. With the device in use 100 cubic feet of oxygen will run the respirator for from 6 to 8 hours, according to the depth and the frequency of the ventilation provided.

Here it would be as well to point out that the respirator is subject to the law of supply and demand, and that the more work that it is called upon to perform, the more energy in the form of air does it need to be provided with. The correct flow is always that which provides satisfactory ventilation and the type of inflation phase (rapid or slow) that the anaesthetist favours.

**DIRECT INFLATION OF LUNGS**

The lengths of the tubes joining the E.P.C. unit with the I.P.P. unit make the respirator cumbersome and detract from its appearance, but their lengths are a convenience when it is necessary to ventilate a patient with air or with an air-oxygen mixture in an emergency. For the respirator to be used in this way, the E.P.C. unit is detached from the pressure-bottle and connected to the endotracheal or facemask connection: and the long tubes enable this to be done with the minimum of delay.

**CONCLUSION AND APPARATUS FOR STUDIES OF VENTILATION**

In conclusion, this respirator was designed in the hope that the provision of a negative phase would enable some of the complications of positive pressure ventilation to be avoided, but it is emphasized that as yet there is only the most tenuous
evidence that the use of negative expiratory pressures is in any way protective. It should also be mentioned that the findings of a recent investigation suggest that there are occasions when the use of negative expiratory pressures is undesirable—during anaesthesia for intrathoracic procedures for example, when their use may lead to collapse of the contralateral lung, to underventilation with retention of CO₂, and a dangerous reduction in the uptake of oxygen.

In the course of this investigation it became necessary (i) to obtain serial samples of the air present in the trachea during expiration, and (ii) to record the mean ventilating pressure. To this end, two pieces of apparatus suitable for use in conjunction with the respirator were designed. Each is briefly described below.

**Apparatus for the Continual Withdrawal of Samples of the Tracheal Air**

Figure 7 shows the respirator connected with a simple absorption system, in which the volume of the exhaled air may be measured by a meter placed in the expiratory side of the circuit, and in which the fresh anaesthetic gases are admitted at a point that precludes their passing through the meter during the expiratory phase.

The rest of the apparatus is concerned with the withdrawal of air from the region of the tracheal bifurcation during, and only during, the expiratory phase, and with the delivery of the air withdrawn to a collecting bag attached at (S).

(M) and (N) are two bellows linked in such a manner that the expansion of one bellows will result in the compression of the other.

The lefthand bellows (M) are connected with the pressure bottle (P) of the respirator. The righthand bellows (N) are connected with the outlet (S), and with a tube (St) to which is attached a catheter whose tip lies near to the tracheal bifurcation.

During the expiratory phase, the bellows (M) are emptied by the suction then present in the pressure bottle, or (if it is not intended to use a negative expiratory pressure) by means of weights placed on top of the bellows. As the bellows (M) empty, so the bellows (N) expand. And as the bellows expand they fill with air drawn from the trachea.

During the inflation phase, the positive pressure in the pressure bottle expands the bellows (M) and as they expand they compress the bellows (N), driving the tracheal air contained in the latter towards and into the collecting bag attached at (S). Nonreturn valves incorporated in the bellows (N) maintain the necessary unidirectional flow.
A tube leading to an infrared gas analyser is substituted for the collecting bag when a breath-by-breath analysis of the exhaled air is required.

**The Mean Pressure Manometer**

The following method of measuring the mean ventilating pressure is based on the fact that the mean or average pressure in a system is the effective working pressure or the pressure continuously at work in that system.

In figure 8 (A) and (B) are two bottles of large capacity joined together by a syphon tube (T). Each bottle is partly filled with water.

When bottle (A) is connected with the patient’s airway, the mean pressure will, if it is greater than zero, work to displace water from bottle (A) to bottle (B). At first the water level in bottle (B) rises steadily; but very shortly it ceases to rise and remains at a constant height above the water level in bottle (A). The difference in height (H) is the height of the water column that the pressure in the airway can sustain continuously: it is therefore the mean ventilating pressure, or a close approximation to that pressure.

The water levels alter when the mean pressure alters, but they do not oscillate (to any great extent) in response to fluctuation of the inflation pressure. This is because the arrangement allows of the displacement of a large volume of water over a period of minutes, but does not allow of the displacement of more than a few cubic centimetres of water during the second or so that each pressure-phase lasts.

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