ANAESTHESIA WITH CONTROLLED POSITIVE AND NEGATIVE PRESSURE RESPIRATION

PART II: REVIEW OF CARDIORESPIRATORY FUNCTION AND ACID-BASE HOMEOSTASIS

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In clinical anesthesia the incidence of respiratory acidosis when the pleural cavity has been invaded for pulmonary or cardiopulmonary surgery remains the most serious problem (Beecher, 1950; Gibbon, 1950; Taylor, 1950; Miller, 1950, 1952; Maier, 1951; Gabbard, 1952; Etsten, 1953; and others). This problem can become even more exaggerated by using multiple anaesthetic agents (Ellison et al., 1955). In the presence of an open pleura the provision of adequate pulmonary ventilation is a harassing problem to the anaesthetist, for no anaesthetic procedure ties the clinician so closely to the rebreathing bag of the anesthesia machine (Gibbon et al., 1955; Dobkin et al., 1955). During nonthoracic operations, with the patient in the supine position, the arterial pH and pCO₂ are a close function of the total ventilation (Buckley et al., 1953; Carpenter et al., 1953). On the other hand, patients during intrathoracic operations apparently require a much larger minute volume of ventilation than under normal awake conditions, as deduced from data of Gabbard (1952), Stead (1953), Allbritten (1954), Martin (1955), and others.

This primary allegiance to the patient’s pulmonary ventilation produces a serious threat to life if difficulty develops due to rapid blood loss, excessive tracheobronchial secretions, cardiovascular depression from intermittent positive pressure respiration or inability to inflate or deflate the lungs adequately. Numerous advantages have been suggested by those who advocate the use of a mechanical respirator to take over this very urgent task of the anaesthetist. By hyperventilating the patient mechanically, the efforts on the part of the anaesthetist to provide adequate alveolar ventilation are greatly spared. Hyperventilation will also assist in overcoming the respiratory impediments of surgical pneumothorax, of operative posture (lateral and prone), and of retractors and packs; in overcoming resistance in anesthesia equipment (Orkin et al., 1954, 1956); and in coping with inadequate carbon dioxide absorbers (Lund et al., 1956).

Elam and Brown (1956) have shown clearly that if the carbon dioxide absorber is not completely efficient, an elevated level of carbon dioxide in the inspired gas
results. Then very high rates of pulmonary ventilation using controlled breathing cannot completely clear the alveolar carbon dioxide. Hence respiratory acidosis is inevitable. Comparing expired alveolar carbon dioxide levels with closed and semiclosed systems and using up to 10 and 15 litres of anaesthetic gases per minute inflow rates and assisted or controlled breathing, they found that even this pulmonary ventilation was not sufficient to prevent carbon dioxide accumulation in some patients. In our study, where fresh soda lime was employed for each operation, the elevations of carbon dioxide which we observed could always be explained by other technical factors (anaesthetic management) or physiological factors (surgical pneumothorax, lung disease and intrathoracic manipulation and incision of bronchi and large vessels). Hyperventilation of these patients certainly assisted in maintaining near-normal levels of carbon dioxide.

The primary effects of hyperventilation are quite harmless (Brown, 1953) and its use should not be deterred from by the occasional clinician who still holds to the "need" for carbon dioxide or its usefulness in clinical anaesthesia (Kemp, 1954). No case of the chronic hyperventilation syndrome (Lewis, 1954) has ever been reported following use of hyperventilation during anaesthesia.

When hyperventilation is mechanically provided with a balanced anaesthetic technique employing analgesic, hypnotic and relaxant drugs it has been said that the surgeon can work in a quieter operative field, skeletal muscle tone and reflex pain responses are reduced, capillary oozing and oxygen consumption seem to be reduced, the doses of hypnotic drugs (thiopentone) and relaxant drugs are reduced (Dundee, 1955) and post-anaesthetic complications such as nausea, vomiting and hypotension occur less frequently (Dobkin et al., 1955; Bjork et al., 1956). Moreover, many clinicians have observed that it is difficult to eliminate carbon dioxide effectively under ordinary positive pressure ventilation and to avoid severe acidosis when abnormal postures or the open thorax are necessary for the surgical operation.

Respiratory acidosis during such surgery is unquestionably harmful (Altschule and Sulzbach, 1947; Orton, 1952; Miller et al., 1952; Campbell et al., 1953; Wilson et al., 1954; Merriman, 1955; Spurrell, 1955; Dripps and Severinghaus, 1955). It predisposes to shock and troublesome reflexes; it increases the requirements for anaesthetic agents; and it delays post-operative recovery. Thus most of the arguments for mechanical respirators are undoubtedly valid.

On the other hand, in clinical anaesthesia, one major and indisputable advantage of attentive manual assistance or control of the patient's breathing during open chest operations is that the anaesthetist can provide the surgeon with almost ideal working conditions. By remaining in constant contact with the patient's cardiorespiratory status the anaesthetist may be of more immediate benefit to the patient under difficult surgical circumstances than by attempting to provide the above named advantages with controlled breathing from a mechanical respirator.

The open chest requires an acute sense of awareness by the ordinary senses on
the part of the anaesthetist: the slight changes of the shades of blood in the wound; the throbbing light and dark pink of the lung surface with each heart beat and with each inflation; the varying shades of skin colour; the warm, dry skin which may insidiously change to a moist cold and clammy surface; and the rhythmic soft blowing sound of gases moving back and forth through the rebreathing tube, which may gradually develop slight crepitations or suddenly reveal bubbling râles. These must all be felt by the anaesthetist. It is therefore essential that his eyes remain on the operating field to provide the major guide to the administration of rhythmic compression of the rebreathing bag.

In the writers’ opinion, this type of management provides the anaesthetist with a closer awareness of momentary changes in the patient’s total condition than the use of a mechanical respirator, which almost always invites distraction of the anaesthetist’s attention from the operation. Only when satisfactory lung deflation is a troublesome problem is a mechanical respirator invaluable. This is particularly so in the patient with parenchymatous lung disease which has destroyed the elasticity of the lungs. For some of these patients a subatmospheric phase in the cycles of the mechanical respirator may assist in providing better pulmonary ventilation and aid in the clearing of carbon dioxide where other means have been unsuccessful.

In planning the assessment of a mechanical respirator three foremost points of controversy must be considered. First are the possible benefits to the patient, as discussed above. Second are the recommended essential requirements of a mechanical respirator. Third are the major objections which surgeons and anaesthetists may have against such a device.

No mechanical respirator presently available satisfies the varying requirements of the individual patient, whether the surgical procedure involves a patient with cardiac or respiratory dysfunction or whether operation in the peritoneal or pleural cavity is being done. The surgeon is frequently harassed because the respirator causes rhythmic bulging of the diaphragm when he is working in the upper abdomen, or because of the balloon ing of the lung when he is attempting a difficult hilar dissection. When the patient is incompletely relaxed or curarized these movements may be quite jerky. The anaesthetist is frequently harassed by the mechanical failings of the respirator, for in a moment of inattention he may miss a rapid development of overinflation, or a complete deflation of the reservoir. There is no signal to inform him that the relaxant drug has worn off until the surgeon complains of irregularities in respiration and jerky movements in the operative field.

Manually assisted or controlled respiration helps the skilled anaesthetist to provide the required amount of relaxant, analgesic and hypnotic drugs which will maintain smoothly the desired level of anaesthesia (“the educated hand”). The respirator removes this fine method of adjustment to individual “needs.” The exhaustion of the Hering Breuer reflex or removal of some unknown visceral function of the lung producing a state of prolonged apnoea was less frequent and less
persistent until mechanical respirators came into use. This apnoea may be so intense that sometimes the patient is wide awake immediately postoperatively, but must be continuously ventilated or urged to breathe. It has been suggested that nikethamide is helpful in restarting spontaneous respiratory efforts in these cases (Wyant, 1953).

Even with the most efficient mechanical devices it sometimes requires continuous readjustment of the mechanics of the respirator, whether by a fixed volume or by a fixed pressure, to provide efficient pulmonary ventilation. It thus follows that the varying surgical and anaesthetic conditions in an individual patient together with the wide variety of ventilation requirements cannot be satisfied by a fixed setting on a mechanical respirator. When the control of respiration has been removed by relaxants, the machine does nothing to assist the anaesthetist in relating pulmonary ventilation to the metabolic requirements or metabolic derangements which may be developing. Only where prolonged respiratory paralysis exists can such aids as the Radford nomogram or arterial blood sampling for pH, pCO₂ and pO₂ determinations be of practical assistance (Whittenberger, 1955). Even at present the practical problems of measuring ventilation and alveolar carbon dioxide during anaesthesia require highly sensitive gas analysers and pressure recorders, whose interpretation must be carefully considered until simpler devices can be developed and tested (Elam et al., 1955; Eckenhoff et al., 1956).

Several anaesthetists have considered the essential requirements of a mechanical respirator (Musgrove, 1952; Mørch, 1954; Schultz et al., 1954; Pask, 1955a; Rollason, 1956). Of first importance is the need for a visible measure of the tidal volume which is delivered to the patient. This should be rapidly adjustable and reproducible by the respirator. The inspiratory pressures must be variable up to a maximum of 30 mm Hg, which is controlled mechanically and released accurately at the desired peak pressure by an efficient blow-off valve. Inspiration and expiration together with a rest phase (expiratory pause) at atmospheric pressure should be variable so as to simulate the normal respiratory pattern, with an accelerating initial inflow followed by a quick release to atmospheric pressure on expiration (Cournand et al., 1952). When necessary, the use of subatmospheric pressure in the airway to assist the deflation of abnormal lungs should be sensitive enough to cut off when resistance to outflow has produced bronchiolar collapse.

The only possible indication for providing a lowered mean intrathoracic pressure during clinical anaesthesia (which would require a subatmospheric pressure phase) is in the patient with major cardiovascular disease who responds to intermittent positive pressure breathing with hypotension. This is most likely where an extensive abdominal operation is contemplated in a patient who is also suffering from a low circulating blood volume. Only when the lungs do not deflate well due to emphysema and fibrosis is the subatmospheric phase of value for intrathoracic operations. Surgical operations facilitated by abnormal postures on the operating table that may interfere with breathing or with the circulation may also be considered a relative indication for a low mean airway.
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pressure. With careful pre-operative preparation and continuous vigilance during the anaesthesia for such patients the real indication for a subatmospheric pressure phase in the breathing cycle should be uncommon (see figs. 11 and 12).

![Diagram](https://example.com/diagram.png)

**Fig. 11**
Effect of posture on standard lung volumes and functional residual capacity (FRC).

The mechanical respirator should provide simple adjustments for producing a period of controlled lung inflation or breath-holding, and a small positive pressure at the end of expiration (if desired) for the occasional management of the last stages of chest closure (usually unnecessary); for expelling air from the pleural space; or for treating acute pulmonary oedema.

When spontaneous but shallow breathing is present while the pleura is closed, the respirator should be able to augment the volume of ventilation to the predetermined “normal needs” of the patient. This requires continuous control sensitivity on the respirator, just as manual assistance to respiration provides proper ventilation of the patient who is partially depressed. The mechanism of a respirator should be explosion-proof and not be wasteful of gases where the energy is derived from the respired gases or from compressed air. It is more important that each function of the respirator should have a separate mechanism which is easy to understand and to adjust than to have a machine which appears simple but in which a single mechanism performs several functions (Pask, 1955a).

In using a mechanical respirator, is it more important to provide a fixed minute volume of respiration by regulation of rate and tidal volume, or should the pulmonary ventilation be regulated by particular pressure changes determined in the airway?

It is, in our opinion, necessary that both factors be subject to regulation for controlled respiration, or at least that both the tidal volume and the phase pressures be known at any time during the administration of the anaesthetic. Whichever is used as the primary means of control, the other should have a reliable preset blow-off valve that releases when dangerous levels develop in the course of attaining the desired level of lung inflation.

If subatmospheric pressure is employed is the suction on the airway actually transmitted to the pulmonary alveoli? If so, does effective pulmonary ventilation continue even when tracheobronchial and alveolar secretions are present in abnormal amounts? Further, does a subatmospheric pressure phase in the respiratory cycle decrease the compliance of the lung, and require periodic hyperinflation (Radford et al., 1954)? In clinical practice, where there is a prolonged expiration time or
Compliance–resistance curves in health and disease with chest wall intact and with pleura open.

**Compliance (C)**

\[ C = \frac{\text{Volume of air entry (in litres)}}{\text{Pressure for air entry expressed in mm Hg}} \]

**Resistance (R)**

\[ R = \frac{\text{Volume of air entry per second}}{\text{Pressure for air entry}} \]

1. When chest wall is in resting position, lung holds 4 litres.
2. Compliance of lung is an S-shaped curve, usually most steep in 2–4 litre range. Lung volume changes 120 ml per second for each mm Hg positive pressure when functional residual capacity (FRC) is normal, e.g. when normal individual is standing (R=1–3 mm Hg).
3. Lung volume changes less for each mm Hg positive pressure when FRC is below normal, e.g. when normal individual is supine (R is increased).
4. Patients with emphysema or obese patients whose lungs are at normal FRC, compliance is much reduced.
5. If such patients as in (4) are placed in lateral or face-down position on operating table (FRC is reduced) at the end of expiration much less volume of air is in the lung and C-R curve is shifted to the left and flattened. Thus obtain much less change in alveolar ventilation with changes in airway pressure.

**Note.** (a) In normal lung, pressure change of 4 mm Hg provides tidal volume of 500 ml. (b) Airway pressure changes below atmospheric pressure ("negative") produce less change in airflow per mm Hg than above atmospheric pressure ("positive"). (c) As lung volume extends to inspiratory reserve volume all volume curves flatten, thus requiring ever increasing pressures for small volume changes. (d) In patients under anaesthesia with diaphragm paralysed by curarizing drugs the airway pressure is zero at end of expiration, but lung still holds 3–4 litres (1): 1.5 litres residual volume which cannot be exhaled, and 2.5 litres expiratory reserve volume which can be exhaled. When pleura is opened, major part of residual air can be expired or sucked out. (e) Cough, hiccough, laryngeal stridor, upper respiratory obstruction, bronchial secretions, bronchoconstriction, all increase resistance to airflow; while pulmonary vascular engorgement and pulmonary oedema stiffen the lung (decrease compliance) and resist lung inflation. These shift C-R curve to right and flatten the flow rise, as noted in curves (4) and (5).
be defined in terms of the economy of oxygen and carbon dioxide tension existing in the blood and alveolar air, as pointed out by Tenney (1956). For practical purposes, simple devices which indicate the arterial oxygen saturation and the carbon dioxide in the inspired and expired gas should be employed during all major anaesthetic procedures. An added consideration in long anaesthetics is the volume of nitrous oxide which has dissolved in the circulation and tissues. This quantity may be such that it would take many minutes to remove the major part of the nitrous oxide from the circulation. It is essential, therefore, that pulmonary ventilation be supported with a higher oxygen tension during the early post-operative period in order to avoid "diffusion anoxia" (Fink, 1955). Another way to avoid this condition is to gradually "wean" the patient off controlled respiration and discontinue the nitrous oxide during the last 10-20 minutes of an operation.

In reviewing current data available on ventilatory changes with mechanical respiration the following factors should be borne in mind in designing fixed pressure or fixed volume respirators. In the paralysed patient with healthy lungs the tidal air is proportional to the inspiratory pressure within the limits of 0.275 to 1.1 litres of tidal air and a peak pressure of 9 to 25 cm of water. Duration of inspiration influences tidal air optimally at about 1.5 seconds. At a peak pressure of 15 cm of water 0.48 litre of air enters the lungs in the first 0.7 seconds, and 0.19 litres enter in the next 0.7 seconds (Spalding, 1955). This is true only when the source of gas is supplied at an airflow of 40-60 litres per minute (Pask, 1955b). In order to circumvent this latter requirement in a practical respirator application of suction during the expiratory phase may be essential. The average curarized patient should therefore receive an adequate tidal volume with an inspiratory phase lasting between 1 and 1.5 seconds, and an adequate alveolar minute volume when the rate is 18 per minute. Lucas and Milne (1955) found that varying the relative lengths of the different phases of the respiratory cycle made no difference to carbon dioxide elimination, and if a pause were made between each breath the rate could not be increased sufficiently to maintain effective ventilation. This was not found in our clinical studies.

Nealon et al. (1955) showed that at a respiration rate of 20 per minute and a positive-negative mechanical respiration of +10 to +15 cm of water pressure, minute volumes of 15 litres per minute were easy to attain. It is the unusual patient who cannot be maintained at normal or slight alkalotic blood pH and pCO₂ levels with this ventilation, if the carbon dioxide absorber is operating efficiently. In our studies, where practically no pressure was lost between the respirator reservoir bag and the endotracheal tube through the use of semidistensible and nondistensible (tygon) tubes, it was evident that the lungs of almost all patients were adequately inflated and deflated by the pressures employed in positive-negative pressure respiration. This clinical observation was verified by the arterial blood pH, pCO₂ and oxygen content, and is logical to assume from airway pressure-gas flow curves of lung compliance (Fry et al., 1954; Brown et al.,
In Nealon's study it was noted that ventilation was adequate and operating conditions were good with pressures of +15 mm Hg and −5 mm Hg, phases 1: 2 and mean endotracheal pressures of 5 mm Hg, which produced a minute ventilation of 10 litres per minute. When the mean orotracheal pressure was 3 mm Hg, the surgeon observed that the lung was partially collapsed, and when the orotracheal pressure was 7 mm Hg, the surgeon considered that the lung was too tense or over-distended. When the phases were set at a 2: 1 ratio, ventilation was much better though the lung was too tense with +15 mm Hg/−5 mm Hg. The lungs were satisfactory with pressures of +10 mm Hg/−5 mm Hg, giving 14 litres per minute and with pressures of +10 mm Hg/−10 mm Hg, giving 15 litres per minute. They noted that the greatest ventilation occurred when the ratio was 1: 1 using +15 mm Hg/−5 mm Hg. Apparently acid-base homeostasis was well maintained with these three patterns using inflation and deflation of the lungs. They also found that the pCO₂ was raised in 85 per cent of cases during induction (17 out of 20 patients). The pCO₂ was markedly elevated at the time of intubation in 6 of 20 patients and fell to near-normal levels with the respirator in 18 of the 20 cases. This latter observation was also made with manually controlled respiration without the chest open (Buckley et al., 1953).

Bjork and Engstrom (1955) and Elam (1955c) favour a fixed volume delivery. Elam contends that during anaesthesia and surgery the compliance of the lung changes, and therefore a fixed pressure is not suitable for delivering an adequate tidal volume. However, the compliance may either increase or decrease, perhaps indicating that the lung “needs” more or less alveolar ventilation. With the anaesthetic technique used in our study, the compliance probably increased markedly. This might indicate a much greater “need” for alveolar ventilation. However, the changes in either direction do not necessarily imply that a set volume should be delivered at all times under varying anaesthetic and surgical conditions.

The use of a fixed volume respirator may be a logical approach where bronchus blockers and endobronchial tubes are used to conduct gas to and from the normal parts of the lung. Otherwise provision for reinflation at increased pressures must be available to ventilate areas of lung with partial atelectasis which are discovered on opening the pleura, or which have been permitted to develop in an attempt to facilitate the surgical dissection. In this study, it was always possible to provide the volume of ventilation as predetermined and as found desirable during the pulmonary operation, without exceeding safe inflation pressures. Moreover, the Radford nomogram figure was greatly exceeded in all the cases in these studies. Theoretically, this assured a normal pH and pCO₂ as well as satisfactory oxygenation, even if varying degrees of pulmonary congestion with or without intra-alveolar or interstitial oedema decreased lung compliance. Even then, undesirable changes in pH sometimes occurred. These could usually be explained.

Normal oxygen figures found by Lucas and Milne (1955) when minute volumes as low as 1.5 litres, as sometimes encountered in patients in the supine posture for abdominal surgery, were doubtless due to
the "spike" flow of gases (Comroe, 1953). Kergin et al. (1948) found quite the reverse when the chest was opened. In patients with advanced pulmonary disease oxygen content of the blood is usually decreased, and when anaesthesia and thoracic surgery are superimposed the provision of increased oxygen tension in the inspired air appears to be essential if a near-normal tension in the blood is to be maintained. Some of the reasons for these disturbances are revealed by Rappaport's (1954) postulation of disturbed visceral function of the lung caused by pulmonary disease; Johnson's (1951) and Eckenhoff's (1955) findings on the effect of premedication with narcotics; the effect of the artificial pneumothorax and posture as found by Beecher (1950) and Etsten (1953); and the effects of circulating anaesthetic drugs as described by Watrous (1950), Taylor (1950), Gabbard (1952) and Buckley (1953).

It appears evident that to assure adequate alveolar ventilation, normal acid-base balance, and normal cardiovascular function, the surgeon must accommodate himself to a pressure-volume curve for lung inflation which extended experience proves best for the patient; and the anaesthetist must learn to adjust the respirator to the various needs of his patients and to varying operating conditions. The anaesthetist must also impress the surgeon working in the open thorax with two facts: First, in reducing the movement in the operative field to facilitate his operation, the surgeon must recognize that low oxygen levels and rising CO₂ tensions may be produced. The suboxygenation can usually be corrected rapidly, but high CO₂ levels may be most difficult to clear. Reduction of pressures which allow segmental and lobar collapse permit surface tension increases which may become almost insurmountable with short periods of high inflation—as evidenced by the necessity for prolonged efforts to re-expand the lungs. These efforts undoubtedly can cause extensive damage to the normal portions of the lung. Second, the surgeon should also recognize that opening a major bronchus and permitting massive leak from the airway must be of minimum duration, since here oxygenation must be maintained by insufflation. This permits considerable levels of carbon dioxide retention to develop (Whitehead, et al., 1949).

Physical principles which play an important role in the provision of optimum pulmonary ventilation are now well known to the anaesthetist. These include the necessity of providing an airway system which will maintain the lowest possible resistance to breathing that is mechanically possible (Orkin et al., 1954, 1956) and which circumvents the normal resistance encountered in the oropharynx (tongue, epiglottis, larynx and salivary secretions). To provide this requires the design of an anaesthetic gas machine with valves of very low resistance, gas flows which satisfy a full range of requirements for the instantaneous rate of inspiration and the avoidance of corrugations and sharp curves in the inflow limb of the ventilation circuit. For intrathoracic operations it is helpful to use nondistensible tubing from respirator to the anaesthesia machine and semidistensible tubing from the anaesthesia machine to the patient in order to avoid loss of volume or pressure in the mechanical circuit. The anaesthetic
machine should be joined to the largest size endotracheal tube which can be inserted without trauma. These factors remove the loss of a substantial volume of ventilation due to alternate ballooning and collapsing of the oropharynx. Thus, adequate ventilation may be provided at lower respirator pressures and volumes.

In considering the factors within the lung which affect pulmonary ventilation it is most essential to avoid reduction of the functional residual capacity (FRC) (Whitfield et al., 1950). According to Dubois et al. (1952) the FRC depends on changes in the blood buffers; oxygen and carbon dioxide content of the blood; and on the surface tension in the lungs. Reduced FRC results in more rapid changes in gas concentration in the lung. The changes with inhalation anaesthesia are more rapid, emergence from inhalation anaesthesia is more rapid, induced apnoea produces hypoxaemia more rapidly; while the arterial pH and pCO₂ have wider variations during the breathing cycle. Whitfield et al. (1950) and Fowler et al. (1950) have shown the changes in FRC with alterations of posture (see fig. 11).

The added effects of anaesthesia on FRC are still unknown. However, the anaesthetist must attempt to provide conditions which will allow a maximum volume of gas exchange in the lungs for each unit change in pressure. That is, the compliance of the lungs must not be permitted to fall because of pulmonary vascular engorgement and lung oedema, tracheobronchial secretions, bronchial and bronchiolar constriction, and inadequate muscular relaxation during controlled breathing. Increased compliance may be assured by using adequate amounts of drying agents (atropine or scopolamine), suctioning of the tracheobronchial tree at frequent intervals, by administering bronchodilators, and by maintaining adequate muscular relaxation. The surface tension changes due to small amounts of sticky secretions deep in the tracheobronchial tree can rapidly cause an adequate tidal volume to be distributed to an inadequate fraction of the total number of functioning alveoli. This must be avoided. It is also important to defer pulmonary surgery shortly after pneumoperitoneum, or else withdraw the air from under the diaphragm in order to reduce its ballooning into the operative field and thus compressing the lungs.

As the anaesthetist assists or controls the respiration of these patients and checks the circulatory status every few minutes, he may wonder about the disruption of physicochemical function that has occurred with induction of anaesthesia and production of surgical pneumothorax. Does the function of the lung under normal conditions really involve the intermittent and regular switching on and off of a suction device for inspiration, concurrent with the compression of a bellows (chest wall) and the elastic recoil of a balloon (lung parenchyma) for expiration, while the processes of pulmonary ventilation, gas distribution, gaseous diffusion, and capillary flow are represented as a "physico-mathematical image" (Arnott, 1955)?

Otis et al. (1956) have even considered the lung as a number of parallel pathways, each consisting of a compliance (C) and a resistance (R) in series, and by analogy to an electrical circuit they concluded that the distribution of ventilation would be
uninfluenced by changes in breathing frequency only if the time constants (R × C) of the separate pathways were the same. If the time constants differed, the distribution of ventilation would alter with changes in breathing frequency. This would be accompanied by changes in overall mechanical behaviour of the lungs. Then compliance of the lungs would decrease and resistance in the airway would increase as the breathing frequency increased. Pulmonary compliance drops most and airway resistances increase most with increased breathing frequencies in subjects with bronchospasm or in patients with asthma and emphysema. It therefore appears important during anaesthesia to provide alveolar ventilation by the slowest rate of respiration which will provide adequate tidal exchanges without over-inflating the lung.

Rappaport (1954) is among the few who currently believe that a primary visceral function of the lung exists rather than a physical function only. The manifestations of this are most obvious when parenchymal lung disease is present or a major physiological change has been produced either, by diseases of the heart or by such acute conditions as pulmonary surgery or pulmonary infection. He makes two major assumptions: (1) there exists a special functional activity of the breathing surface structures which serve to adapt the diffusing surface of the lung constantly to the momentary gas exchange requirements of the body; and (2) this activity is of a type involving changes in quality (permeability) as well as in quantity (distribution) of the diffusing surface.

The mechanistic or physical concepts of pulmonary function are only now beginning to explain a purposeful correlation between pulmonary ventilation and circulation through all parts of the lungs and a correlation of diffusing capacity with momentary requirements of the body in health and disease (Comroe, 1953, 1955). For patients with pulmonary pathology or those undergoing any intra-thoracic operation the anaesthetist should recognize that much higher rates of alveolar ventilation and much higher tensions of oxygen must be supplied if near-normal oxygenation is to be attained. This is necessary to compensate for the unknown extent of shunting of blood past normal lung, the shuntlike effect due to distribution difficulty in the emphysematous patient, and the shift of the oxygen saturation-tension curve to the right which causes diffusion difficulty when the patient is hyperventilated (Cournand, 1950; Perkins, et al., 1956). The solution of the problem of providing adequate alveolar ventilation when relaxant, hypnotic and analgesic drugs, respiratory paralysis and surgical pneumothorax are added to a disease state must require higher oxygen tensions and increased minute volume of ventilation in order to counterbalance the pathology present and abnormal physiology superimposed by the therapeutic procedure.

SUMMARY

Data which have been gathered during recent years clearly indicate that the management of all general anaesthetics requires assistance to the patient's pulmonary ventilation for all operative procedures. Adequate pulmonary ventilation should imply maintenance of oxygen
saturation of the arterial blood, elimination of the alveolar carbon dioxide to levels which maintain the arterial blood pH within normal range (7.35–7.45), and arterial carbon dioxide tensions between 35 and 45 mm Hg. This ventilation should also sustain or promote normal cardiovascular function as indicated by a stable blood pressure and pulse rate.

It is necessary to control the respiration when the pleura is opened, for otherwise the definition of adequate pulmonary ventilation cannot be fulfilled. The advantages and disadvantages of manual control of respiration have been noted. When diligently carried out with close attention to the patient to balanced anaesthesia, and to the operative procedure, most patients remain in a near-normal physiological state. The use of mechanical respirators is of considerable assistance if the inherent limitations imposed by lowered lung compliance and elevated airway resistance due to cardiopulmonary disease and the mechanical failings of present-day respirators are recognized. Current data contend that both volumes and pressures applied to the patient’s airway must be considered for each individual patient if optimum anaesthetic care and management is to be provided.

CONCLUSIONS

As one views a procession of patients with normal and abnormal lungs, together with their lung function studies, and then subjects them to analgesic drugs and paralysis of respiratory muscles, both before and after surgical pneumothorax, one frequently becomes aware that pulmonary ventilation and respiration involve more than a static and dynamic mechanical phase (or an electric circuit); a physicochemical phase in the lungs and in the pulmonary blood flow; and a simple transport system via the systemic circulation. These may be the simple facts. However, the management of these patients to date has not proved a simple matter which can be reproduced easily by mechanical means. The major success has involved the substitution of another biological factor, namely, the alert hand of the anaesthetist. Until better mechanical devices are developed, the anaesthetist should continue to use manual control until the “hand” is no longer our most “educated” device.

In our position of fading darkness it is urgent for the clinical anaesthetist to stand close to the patient and to the rebreathing bag and view surgical pneumothorax with renewed interest and clinical acumen, for at present the mechanical respirator relegates his thoughts to pure physics, mechanics and mathematics. This furthers the trend described by Einstein and quoted by Rappaport: “Physics has often reduced the biologist into interpreting biological phenomena too primitively.” Until more knowledge is procured, developed and simplified, the anaesthetist should stoop over the head of the operating table and still consider the lung and the physiological changes in cardiorespiratory function and acid-base homeostasis associated with its pathology as “the little black box” of Meneely’s editorial “For Breath Too is Nutriment” (1955)

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