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# SOME PHYSIOLOGICAL AND CLINICAL ASPECTS OF CHEST PHYSIOTHERAPY

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The ciliated epithelium which lines the airways is responsible for continuous flow of mucus over the airway surface to the upper respiratory tract. This mechanism becomes ineffective in bronchopulmonary disease which is characterized by excessive production of mucus, impaired mucociliary clearance and, eventually, pulmonary failure. This may be a chronic disorder, as in bronchitis and cystic fibrosis, or an acute problem occurring in patients following anaesthesia, mechanical ventilation and intensive therapy. This review examines some of the physiological mechanisms involved in clearance of excessive bronchial mucus in these circumstances and the role of various physical therapies designed to accelerate this process. Chest physiotherapy, in the form of postural drainage, percussion and vibration (PDPV), "coughing exercises", and the "Forced Expiratory Technique" (FET) are discussed. The problems of physiotherapy-induced bronchospasm and hypoxaemia are also noted.

## PHYSIOLOGY OF FLOW IN LIQUID LINED AIRWAYS

### *Two-Phase Flow*

Flow of air through the tracheobronchial tree and its interaction with the mucus lining is complex because of branching geometry of the airways, collapsible airway walls, constantly changing velocity of air flow and varying viscoelastic properties of mucus (fig. 1). Simple models of flow in the airways often assume laminar conditions. This may be true in small airways and it implies that the velocity of flow at the airway wall is virtually zero and that there is no interaction between air and liquid lining the wall. A more realistic model, particularly for the large airways,

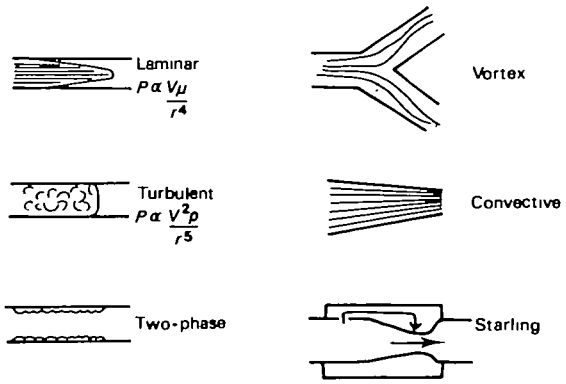


FIG. 1. Different types of flow in the airways. $P$ = pressure decrease in the airway; $\dot{V}$ = flow; $r$ = airway radius; $\mu$, $\rho$ = gas viscosity and density, respectively. Flow through junctions induces vortices, the intensity of which depends upon the angle of branching as well as the velocity of flow. Airway collapse, as in the Starling resistor, induces strong interaction between gas flow and liquid lining the wall.

is turbulent flow where the velocity of gas is high at the wall, with strong interaction between air flow and the mucus lining the wall. This type of gas–liquid interaction is termed two-phase flow, studied originally in models of the trachea and bronchi by Clarke, Jones and Oliver [11] and, more recently, by Sackner and Kim [51]. It is of crucial importance in removal of excessive mucus in endobronchial disease.

The normal human bronchial tree is lined by a thin (5 μm) layer of mucus which is moved over the airway surface by the mucociliary escalator. However, in endobronchial disease this may exceed 5 mm in thickness and ciliary clearance becomes ineffective. Two-phase flow now becomes an important mechanism of clearance, and at a particular combination of air flow, mucus

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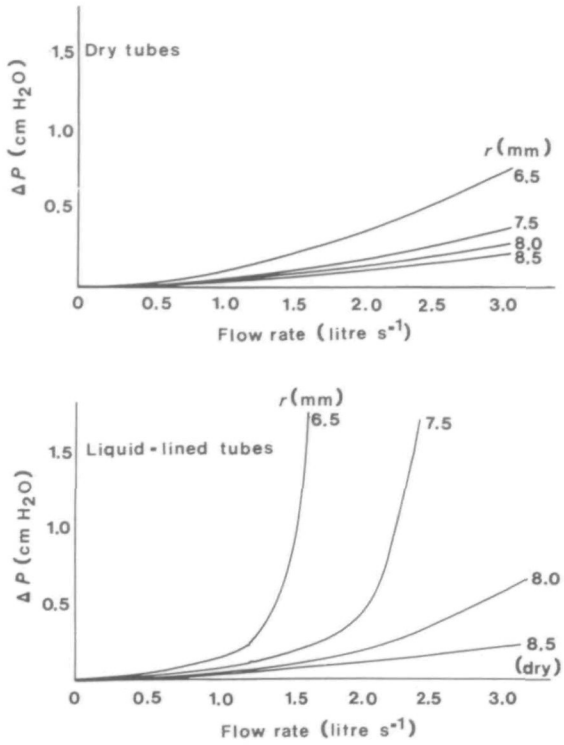


FIG. 2. Effect of a liquid layer on the pressure–flow relationships in a tube similar to the trachea. The predicted increase in pressure change by narrowing the tube radius ($r$) by 0.5, 1 and 2 mm is shown in the upper panel. The actual pressure decrease in the same tube narrowed the same amount by a liquid layer is shown in the lower panel (Redrawn, with permission, from reference [11].)

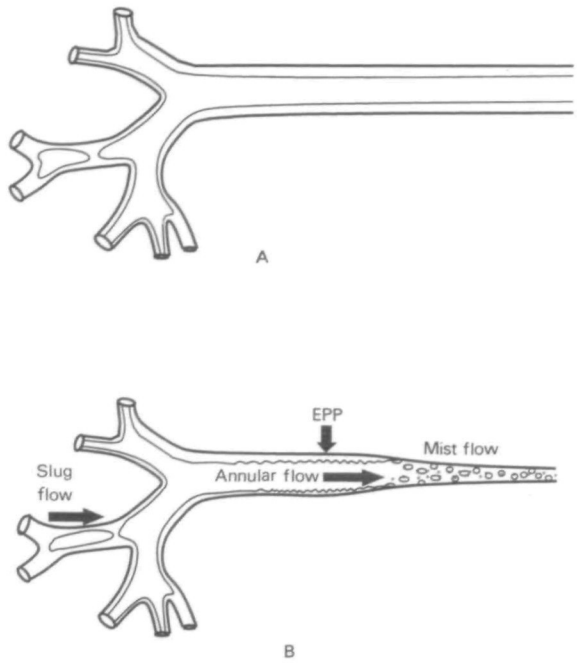


FIG. 3. A: Large airways lined with a thick layer of mucus. B: During forced expiration, different types of gas–liquid flow may be seen in different parts of the airway. In the narrowed downstream segment there is high gas velocity, strong gas–liquid interaction and *mist flow*. Upstream there is less strong gas–liquid interaction and *annular flow*, with *slug flow* in smaller airways further upstream. EPP = equal pressure point which, during forced expiration, moves upstream ahead of the narrowed downstream segment.

viscosity and thickness there is a very strong gas–liquid interaction which first exacerbates the pressure decrease then detaches liquid from the airway wall.

Narrowing the lumen of a tube with a layer of fluid causes a much greater resistance than that of a dry walled tube of the same internal diameter (fig. 2). For example at a flow rate of 1.5 litre $s^{-1}$, above the transition to turbulent flow, the pressure decrease in a trachea lined by a 2-mm thick layer of fluid was more than 10 times greater than expected. Thus two-phase flow may cause considerable increase in airway resistance just before detachment of mucus from the wall. However, if the critical flow rate for mucus detachment cannot be achieved (e.g. if mucus viscosity is too great) then respiratory muscle fatigue may ensue. Therefore, two-phase flow occurs after the transition from laminar to turbulent flow, and Reynold's number ($Re$) may be used to describe the flow rates needed for gas–liquid interaction. $Re$ is a function of velocity of flow ($v$) and tube diameter. An $Re$ of 2000 is considered usually to be the critical value for transition from laminar to turbulent flow in a tube (although it may vary widely) and this value is achieved readily by tidal breathing in the human trachea. We found that gas–liquid interaction would occur with an $Re$ of 3000 with a 1-mm thick layer of mucus in a tube 8 mm in diameter, but at a much lower $Re$ in branched tubes [11]. Higher $Re$ values are needed for thinner layers of mucus, but quite low $Re$ values may be sufficient to detach mucus in smaller airways.

There are three basic patterns of gas–liquid flow which are relevant to mucus clearance from the lung: slug flow, annular flow and mist flow (fig. 3). *Slug flow* occurs when large bubbles of air pass at a velocity ($v$) of 60–1000 cm $s^{-1}$ through airways filled partially with mucus. *Annular flow*

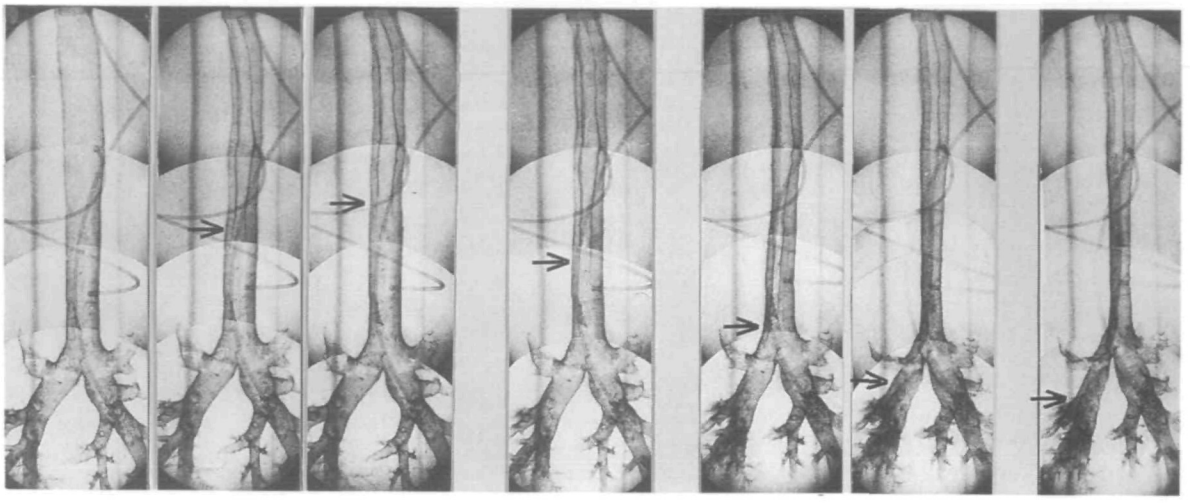


FIG. 4. Frames from a cine tracheobronchogram to show dynamic compression of the trachea extending upstream beyond the carina. A = Just after the start of a cough manoeuvre and before peak flow is achieved. The arrow indicates the upstream end of the compressed segment moving upstream during the cough as lung volume reduces from C to G (residual volume).

takes place when air flows at 2000–2500 cm $s^{-1}$ through tubes lined with a continuous layer of mucus. *Mist flow* occurs at higher flow rates, > 2500 cm $s^{-1}$, which detach mucus from the wall. The fact that the airways are collapsible obviously creates ideal conditions for this reaction and enhances the efficiency of the clearance mechanism.

The layer of mucus may vary in viscosity from 10 to more than 1000 mPa·s and this is determined largely by the water content, the viscosity of water being 1 mPa·s. However, mucus may also show marked thixotropy, so that its viscosity may decrease 100-fold at high sheer rates. Thus the combination of cough and airway narrowing leads to high *Re* values and sheer rates which reduce mucus viscosity and aid clearing of the airways by annular and mist flow.

### *Dynamic Compression of Airways and Mucus Clearance*

During a cough, the upper end of the trachea suddenly narrows and, with diminishing lung volume, this narrowed segment moves rapidly upstream past the carina into the small bronchi. This moving segment, or throat, is the site of maximum gas velocity with high *Re* and maximum gas–liquid interaction which clears mucus from the airway wall (fig. 4). Jones, Fraser and Nadel [28] were the first to show that the maximum expiratory flow could be calculated from the compliance of the compressed segment and elucidated the mechanism whereby this segment maintains a constant flow independent of driving pressure (see Appendix).

Measuring the pressures across the airway walls shows where the pressures inside and outside the tracheobronchial tree are equal—the Equal Pressure Points (EPP) [28]. During a cough, the EPP moves rapidly ahead of the compressed segment along the airway from the trachea into progressively smaller airways. This is important because, upstream of the EPP, the pressure inside the lumen is greater than outside; thus the airways between the alveoli and the EPP are fully patent. This results in relatively low sheer rates between gas and mucus, with poor clearance. Downstream of the EPP, the airway is compressed and has a very high gas velocity, giving mist flow and rapid clearance of airway mucus (fig. 3). This is the normal mechanism whereby cough clears mucus from the walls of the central airways, but it may not be effective if mucus viscosity is very high, > 10000 mPa·s, when the mucus may behave more like a solid than a liquid.

### *High Frequency Chest Wall Oscillation (HFCWO)*

More recently, the principle of two-phase flow has been re-examined to see if a high frequency

oscillation applied to either the airway or the chest wall may achieve a greater degree of mucus clearance than is achieved by cough. Cough is likely to exert a greater effect in the larger central airways than in the smaller peripheral airways, and high frequency chest wall oscillation (HFCWO) was proposed as a mechanism for enhancing peripheral airway clearance via a two-phase flow effect [10, 30]. HFCWO is achieved by means of a modified double arterial pressure cuff wrapped around the thorax and oscillated at 3–17 Hz with peak pressures up to 100 cm $H_2O$. This achieves tracheal airflow of 1–3 litre $s^{-1}$ and it has been shown in animal experiments that 30 min of HFCWO significantly enhanced mucus clearance from the trachea and from more peripheral zones.

In contrast to these findings, the application of High Frequency Oscillation (HFO) to the airway opening reduced the rate of mucus clearance from peripheral airways [34]. These interesting results lend some support to the practice by physiotherapists of external chest vibration as a method of clearing peripheral airways. However, they have yet to be shown to be of any proven value in clearing peripheral airways in man.

Meanwhile, the most plausible mechanism of clearing airways in man is by inducing gas–liquid interaction during a simulated cough manoeuvre. There are sound physiological reasons why this method should be utilized in the physiotherapy of patients with chest disease, but more attention should be given to the viscoelastic and thixotropic properties of mucus [30] and to the possibility that chest wall oscillation has an important role to play in clearance of lung mucus.

## CLINICAL ASPECTS OF CHEST PHYSIOTHERAPY

### *Management of Ward Patients with Respiratory Disease*

Previous authors have attempted to clarify the role of chest physiotherapy in patients with both acute and chronic respiratory problems [31, 53, 58], and copious production of sputum has been shown to be a sound indication for its use [2, 12, 39, 59]; the benefits are reflected by improved lung function tests [12, 59] and enhanced clearance of sputum [2, 39]. In contrast, physiotherapy in patients with acute exacerbation of chronic bronchitis but without copious sputum either produced no improvement in lung function tests and blood-gas tensions [1, 41] or even caused a reduction in forced expiratory volume in 1 s [8, 65], which was preventable by prior administration of a bronchodilator [8].

Coughing exercises have been compared also with PDPV in patients with cystic fibrosis, and found to be equally effective in increasing sputum production [16]. We have already discussed the "two-phase gas–liquid flow" mechanism by which cough effects sputum movement. However, the high transmural pressures produced during coughing lead to dynamic compression of the airways, which may inhibit mucociliary clearance upstream of the EPP [54]. Therefore, a procedure termed the "forced expiratory technique" (FET) was introduced to circumvent this problem, and promotes a higher rate of airflow in smaller airways by moving the EPP further upstream. This technique involves expiring forcefully from mid to low lung volumes whilst maintaining an open glottis ("huffing" exercises). Studies using FET [47, 56, 57] have been encouraging, and it was shown to be superior to both directed coughing [57] and PDPV [47, 56] in enhancing removal of sputum. Sutton and colleagues [56, 57] evaluated this technique in chronic bronchitics with copious sputum using an inhaled radioaerosol method. They found that FET produced greater clearance of inhaled radiolabelled particles than both regimented coughing [57] and percussion and vibratory exercises [56]. However, the amount of sputum obtained was increased further when FET was combined with postural drainage compared with FET alone. Pryor and colleagues [47] also compared FET and postural drainage with PDPV in subjects with cystic fibrosis and confirmed that this regimen cleared more sputum and in less time than conventional physiotherapy. This is of particular importance to these patients as it enables them to practise an effective method without having to rely on others for help.

Several studies have been performed also to assess the effects of PDPV, cough and FET on peripheral lung clearance. In 1979, Bateman and colleagues [2] used a radioaerosol method to define the parts of the lungs affected by PDPV in patients with chronic bronchitis with copious sputum. The authors concluded that PDPV had improved radioaerosol particle clearance from all compartments of the lung, including the periphery. However, their use of the term lung periphery was misleading, as it referred to the lateral 40% of the lung in terms of area on an

isotope scan, and included parts of the main bronchi. Also, their computer pictures of lung radioactivity showed that the 5-μm particles were confined mainly to the central airways. Wollmer and colleagues [65] used a radioaerosol technique similar to that of Bateman's group [2], but found that chest percussion did not enhance particle clearance from either central or peripheral regions. The explanation for these contrasting results is that Wollmer's patients were given coughing exercises, whereas Bateman's control patients were asked to refrain from coughing during the monitoring period. Bateman's group, therefore, repeated their earlier study of patients with chronic bronchitis [2], and compared coughing exercises with PDPV [3]. They found that both therapies produced equal central lung clearance, but that only PDPV had any effect on the periphery. However, the same misleading criteria for interpreting the term "lung periphery" were used as in their previous study.

The value of coughing exercises has been demonstrated in other studies [3, 16, 43], and a radioaerosol method in patients with chronic bronchitis showed that they increased both peripheral and total lung clearance [43]. However, after assessing the effects of directed coughing compared with FET on sputum clearance, Sutton and colleagues [57] observed correctly that they were unable to comment on regional mucus clearance because the 5-μm radioactive particles did not penetrate to the periphery. Therefore, the validity of such results regarding peripheral clearance using inhaled radioparticles is uncertain; Pavia and colleagues [46] have discussed the problems inherent in this methodology.

Alternative evidence for the effects of chest physiotherapy on peripheral lung secretions is available from clinical outcome studies in patients with peripheral lung disease. In 1978, Graham and Bradley [22] assessed patients with acute pneumonia who demonstrated radiographic and clinical evidence of consolidation. The results showed no difference between the PDPV and control groups in earlier resolution of chest x-ray signs, duration of fever, or decreased hospital stay. Britton, Bejstedt and Vedin [7] monitored 171 patients with acute primary pneumonia, and compared the effects of regular PDPV with advice on expectoration and deep breathing. In addition to the lack of benefit found by Graham and Bradley [22], this study demonstrated prolongation in the duration of fever and an increased hospital stay in patients given physiotherapy. The only explanation offered by the authors was that, instead of clearing the infected material, PDPV may have caused it to spread to the surrounding tissue.

Therefore, PDPV, cough and especially FET are beneficial in enhancing clearance of excessive central airway secretions, but there is far less support for their use in patients without copious sputum or with peripheral consolidation.

### *The Role of Chest Physiotherapy in Perioperative Patient Care*

Pulmonary complications are a common cause of postoperative morbidity and mortality; the incidence has been reported to vary between 6% [64] and 80% [32]; it is increased in upper abdominal surgery [32], older patients [13], smokers [64] and patients with pre-existing lung disease [64]. Atelectasis is the most common postoperative complication, especially after upper abdominal surgery, and was noted as early as 1908, by W. Pasteur [45]. Many studies have been performed since to assess the effects of physiotherapy and other treatments on the incidence of postoperative respiratory problems.

In 1953, Palmer and Sellick [44] suggested the following sequence of events in the aetiology of bronchopneumonia:

Various factors → increased secretions → blockage of smaller bronchi → absorption of air distally → atelectasis → bronchopneumonia.

They postulated that, if the bronchi could be kept clear of secretions, subsequent complications would be reduced. They set up two large studies in patients undergoing either inguinal hernia repair or partial gastrectomy. In the first study the control group was given regular breathing exercises, and the treatment group underwent frequent PDPV combined with 6-hourly isoprenaline inhalation. Postoperative atelectasis diagnosed radiographically was reduced from 43% to 9% by this treatment regimen. However, their second study showed that physiotherapy without the isoprenaline inhalations had no effect on postoperative outcome compared with regular breathing exercises. The importance of bronchodilator therapy during PDPV was confirmed in 1975 by Campbell, O'Connor and Wilson [8].

Stein and Cassara [55] evaluated the effects of the patient's preoperative chest condition on postoperative complications. Their patients were classified into a "healthy" control group and a

"poor risk" group with abnormal preoperative lung function tests. Chest physiotherapy was administered to only 50% of the poor risk group and was combined with antibiotics, $\beta_2$-agonists, and humidified gases. An increased incidence of postoperative pulmonary complications was demonstrated only in the untreated poor risk group. However, one cannot deduce from this study the exact benefits of physiotherapy in poor risk patients because of the range of therapy used, including the use of bronchodilators.

Laszlo and colleagues [32] confirmed the observation that "healthy" patients are unlikely to benefit from chest physiotherapy. They studied 86 non-bronchitic patients allocated at random to treatment and control groups. The treatment group was given twice daily PDPV for 5 days after operation, but was found to have the same incidence of respiratory complications, assessed by sputum and radiographic changes, as the no-treatment control group. An inherent problem in such studies is the difficulty in differentiating between chest infection and atelectasis. For example, Morran and colleagues [40] monitored 102 consecutive patients presenting for cholecystectomy. Physiotherapy and control groups were matched well for characteristics likely to affect postoperative respiratory morbidity; the authors concluded that prophylactic physiotherapy reduced the incidence of postoperative chest infection. However, this conclusion was not justified, as the authors' criteria for infection and atelectasis were similar, and there was little difference in the incidence of combined postoperative complications.

The possibility that chest physiotherapy may cause a complication which it is aiming to prevent has been demonstrated in paediatric patients by Reines and colleagues [48]. They monitored 50 patients aged 3 months to 9 yr undergoing cardiac surgery for congenital heart disease. Patients were allocated randomly to routine physiotherapy and control groups, and atelectasis was diagnosed by radiographic interpretation by a radiologist unaware of the treatment each patient had received. The physiotherapy group not only developed atelectasis more frequently than the control group, but also had a more prolonged hospital stay. Explanations proposed by the authors for this unexpected result included: pain induced by physiotherapy, the Trendelenburg position, mucus plugging of larger airways, and the compressive effects of percussion on a compliant chest. Therefore, routine chest physiotherapy without positive indications may be detrimental, but patients with excessive secretions or acute atelectasis caused by sputum blockage of central airways merit treatment and should not be denied physiotherapy.

Chest physiotherapy has been compared also with other forms of perioperative respiratory therapy. Schuppisser, Brandli and Meili [52] studied the postoperative effects of physiotherapy compared with intermittent positive pressure breathing. Although the number of patients in this trial was small, the results showed that neither therapy produced any beneficial change in pulmonary function. When compared with incentive spirometry in patients undergoing upper abdominal surgery, Craven and colleagues [15] found that physiotherapy increased the incidence of postoperative chest problems; 17 of the patients in the physiotherapy group were smokers or had chronic lung disease and 15 developed some degree of collapse or consolidation.

These studies thus indicate that routine perioperative chest physiotherapy in the form of PDPV is not of value in patients with healthy lungs even when undergoing upper abdominal surgery, but it may benefit patients with chronic respiratory disease if combined with bronchodilators. Otherwise, it should be used selectively in patients with positive indications such as copious sputum or acute atelectasis. Furthermore, in view of the poor results from several of the studies described above, therapies other than PDPV merit investigation, and the forced expiratory technique in particular is worthy of future evaluation.

### *The Role of Chest Physiotherapy in Critically Ill Patients*

The studies discussed so far can be used to provide some guidelines on the likely benefits or disadvantages of PDPV in patients in the Intensive Care Unit (ICU). However, the critically ill patient may be at greater risk during physiotherapy because of the severity of the illness (e.g. septicaemia, hypotension or respiratory failure), and the presence of other non-pulmonary injuries or problems (e.g. patients with increased intracranial pressure) [18, 42, 50]. In particular, numerous studies have shown that PDPV may produce short term hypoxaemia in both adult [14, 20, 21, 26, 33, 61] and neonatal [19, 25, 63] patients.

*The problem of physiotherapy-associated hypoxaemia*

In 1980, Connors and Hammon [14] evaluated sputum production as an indicator for chest physiotherapy in critically ill patients with non-surgical pulmonary pathology. Their patients were classified into those with little sputum production and those with moderate to large volumes of sputum. In the first group, they found a decrease in $Pa_{O_2}$ of 2.23 kPa immediately after PDPV, and a further decrease of 0.7 kPa at 30 min. In contrast, there was no change in $Pa_{O_2}$ after physiotherapy in the second group. However, other studies [21, 33] showed that hypoxaemia following PDPV may occur even in patients with profuse secretions. These changes in $Pa_{O_2}$ are unexpected, as increasing sputum clearance should have improved ventilation; studies of neonatal chest physiotherapy may help elucidate the possible mechanisms for this hypoxaemia.

Holloway and colleagues [25] assessed the effects of PDPV and hyperinflation on the $Pa_{O_2}$ of neonates undergoing ventilation for tetanus. Physiotherapy produced a decrease in mean $Pa_{O_2}$ lasting for 1 h after treatment. Hyperventilation was unable to prevent this decrease, but did hasten the return to pre-physiotherapy concentrations of $Pa_{O_2}$. Fox, Schwartz and Shaffer [19] studied neonates in whom the trachea was intubated mainly for respiratory distress syndrome (RDS) and found an alarming reduction in $Pa_{O_2}$ from 9.7 to 5.7 kPa, which lasted for 30 min after physiotherapy. Bradycardia was noted also during tracheal suction in some patients. The main difference between this study and that of Holloway [25], in which reductions in $Pa_{O_2}$ were much less severe, is that these patients were not paralysed. Therefore, one reason for this hypoxaemia is greater neonatal activity, as reflected by increased oesophageal pressure and frequency of ventilation [19]. The need for patient sedation during tracheal suctioning was investigated by Ninan and colleagues [42] in neonates with RDS. Sedation attenuated increases in mean arterial pressure and intracranial pressure during tracheal suctioning, but decreases in $Pa_{O_2}$ occurred in both groups. Walsh and colleagues [63] showed that chest vibration and tracheal suctioning have an additive adverse effect on transcutaneous oxygen tensions ($Ptc_{O_2}$) of premature neonates undergoing ventilation. Furthermore, supplementary oxygen was unable to prevent the severe reductions in $Ptc_{O_2}$, indicating a large shunt, but it did hasten return to baseline values.

Another possible mechanism for this hypoxaemia is atelectasis, as its incidence was shown to increase after both PDPV [48] and tracheal suctioning [6, 49]. In addition, repetitive coughing following intubation was found also to decrease markedly the functional residual capacity in adult surgical patients [4]; it may have a similar effect during chest physiotherapy and contribute to atelectasis. By using a CT scanner and other techniques, Hedenstierna and colleagues [24, 60] showed that general anaesthesia itself induced basal collapse which was potentiated possibly by neuromuscular blocking drugs and was associated with increased alveolar–arterial oxygen difference. Sedated patients who have undergone tracheal intubation are, therefore, already compromised and PDPV or tracheal suctioning may cause further atelectasis and account for the hypoxaemia demonstrated in many studies.

Additional evidence for a link between tracheal suctioning and aetelectasis was demonstrated by Velasquez and Farhi [62] in anaesthetized, paralysed dogs. They showed a strong correlation between the negative intratracheal pressure during tracheal suctioning and both reduced lung compliance and increased venous shunting. Various methods of preventing the hypoxaemia caused by the suctioning have also been studied. Carlon, Fox and Ackerman [9] evaluated a "closed-tracheal suction system" which obviated the need for ventilator disconnection on each occasion that the airways were suctioned. This closed system was compared with conventional open tracheal suctioning; deterioration in $Pa_{O_2}$ occurred only during open suctioning in patients receiving more than 10 cm $H_2O$ of PEEP. The advantage of avoiding ventilator disconnection during tracheal suctioning was shown to be one of the benefits of jet ventilation [29], and a valve attachment for tracheal tubes was designed allowing suction without interruption of conventional ventilation [5].

Therefore, PDPV, tracheal suctioning and ventilator disconnection may contribute to the short term hypoxaemia occurring in many critically ill patients following chest physiotherapy. The most likely mechanism for this hypoxaemia is atelectasis, although stimulation of the patient causing increased oxygen extraction may also be a factor.

*Studies supporting chest physiotherapy in ICU patients*

In contrast with the studies demonstrating physiotherapy-associated hypoxaemia, there is also evidence for beneficial effects of chest physiotherapy in critically ill patients; three studies were performed by the same authors, MacKenzie and Shin, with others [35–37].

In 1978 [37] they assessed 47 patients in their ICU. Most patients had either chest or head injuries, were septicaemic or had spinal cord transection. The indications for PDPV in these patients were secretions detected by auscultation, impaired gas exchange with radiographic changes of atelectasis, or lung contusion. Before each physiotherapy session, patients were assessed clinically and had a radiograph to locate the site of pathology. PDPV was then used until this portion of the lungs was clinically improved. Their mean time for each session was 51 min! The results showed no improvement in post-treatment $Pa_{O_2}$, but there was a 74% success rate in clearing unilobular radiological densities or atelectasis, and a 60% rate of resolving multilobular changes.

In 1980 [36] the same group studied changes in total thoracic compliance following physiotherapy in 42 patients undergoing ventilation for chest trauma. With the exception of excessive secretions, the indications for PDPV in this group were atelectasis (29 patients) and lung contusion (eight). The results showed an increase in total lung + chest wall compliance immediately after and for 2 h after physiotherapy. No other data were recorded in this study and the mean time for PDPV was 57 min! However, in 1985 the same authors [35] conducted a more detailed investigation of cardiorespiratory function before and after chest physiotherapy in patients undergoing ventilation for post-traumatic respiratory failure. The only improvements in cardiorespiratory function were reduced intrapulmonary shunt immediately after PDPV, and increased total thoracic compliance at 2 h after the procedure. Cardiac index (CI) and $Pa_{O_2}$ values were not altered significantly. One reason for the stable CI is that these patients did not undergo hyperventilation during PDPV by either "bag squeezing" or other methods. The significance of these results is questionable, as decreased pulmonary shunt combined with stable CI should have improved $Pa_{O_2}$. Furthermore, as these physiotherapy sessions lasted 67 min, monitoring arterial oxygenation during the procedure would have been both advisable and informative.

MacKenzie and Shin considered that atelectasis was one of the main indications for chest physiotherapy, and this has been confirmed by other workers [23, 38]. Hammon and Martin [23] reported five case histories involving nine episodes of *acute* lobar collapse, and in every case PDPV was successful in re-expanding collapsed portions of the lung, but they emphasized that patients must be monitored closely, as "some critically ill patients receiving PDPV have a worrisome and unexplained fall in arterial oxygen tension". Marini, Pierson and Hudson [38] compared PDPV with fibreoptic bronchoscopy in 31 patients with acute lobar collapse. Pre- and post-treatment chest x-rays showed that both methods were equally effective in increasing lung volume. However, an additional finding in this study was that the success rate depended on whether or not there was an air bronchogram. If this was present, the resolution rate was only 26%, compared with 86% with no bronchogram. The explanation was that with no air bronchogram there was sputum blockage of a bronchus with secondary collapse, but with a bronchogram there was distal collapse, but no sputum plug. The fact that chest physiotherapy has little effect in clearing secretions from the lung periphery has been demonstrated also in patients with consolidation [7, 22].

In contrast with the adverse effects of neonatal chest physiotherapy observed in previous studies [19, 25, 63], Finer and Boyd [17] found that a specially designed form of chest percussion improved the oxygenation of neonates with respiratory distress. Postural drainage alone had no effect on oxygenation but, when combined with "contact-heel" chest percussion (rhythmic application of the heel of the hand to the neonate's chest wall), mean $Pa_{O_2}$ values improved from 8 to 10 kPa. Because of the special nature of the chest percussion utilized in this unit, these results cannot be compared directly with those from other neonatal studies, and the authors observed correctly that each unit should evaluate its own physiotherapy procedures before accepting them as standard practice.

Thus excessive secretions and acute atelectasis are also sound indications for chest physiotherapy in critically ill patients, as demonstrated by beneficial changes in total thoracic compliance and chest x-ray signs. However, because of the possibility of short term hypoxaemia associated

with physiotherapy, arterial oxygen tensions should be monitored and PDPV must be used with particular care in patients with low baseline oxygenation.

## CONCLUSION

The physiological aspects of mucus clearance have been discussed, and the relative merits of coughing exercises, the forced expiratory technique (FET), and postural drainage, percussion and vibration (PDPV) have been evaluated. All these manoeuvres are of value to patients with copious sputum confined mainly to the central airways, but FET, especially when combined with postural drainage, has been shown to be superior to both cough and PDPV in such patients. Atelectasis caused by sputum blockage of a major airway is also a good indication for PDPV. However, PDPV may induce both bronchospasm and hypoxaemia in many patients, and therefore the indications should be evaluated soundly before its use. This is particularly necessary in critically ill patients with little cardiorespiratory reserve. More research is still required and, in particular, we need to:

(a) verify the effects, and possible benefits of high frequency chest wall oscillation on clearance of peripheral mucus;

(b) determine if FET + postural drainage should replace PDPV in patients breathing spontaneously;

(c) evaluate the role of bronchodilators during physiotherapy;

(d) examine more clearly the reasons for the short term hypoxaemia caused by physiotherapy in many critically ill patients;

(e) assess if regular prophylactic chest physiotherapy in ICU patients undergoing ventilation decreases the incidence of chest complications.

## APPENDIX

Jones, Fraser and Nadel [28] showed that flow ($\dot{V}$) or velocity ($v$) can be predicted from $\rho$, the gas density, the cross sectional area ($A$) and the transmural pressure ($\Delta P$) at the compressed segment:

$$\frac{\dot{V}}{A} = A\sqrt{\frac{2\Delta P}{\rho}} \tag{1}$$

or

$$v = \frac{\dot{V}}{A} = \sqrt{2\Delta\frac{P}{\rho}} \tag{2}$$

The compliance of the compressed airway shows that:

$$\frac{A}{Ao} = \frac{1}{2}\log\Delta P \tag{3}$$

where $Ao$ is the area at full lung inflation.

From equation (3), the specific compliance per unit length is:

$$\frac{1}{Ao}\cdot\frac{dA}{dP} = \frac{1}{2\Delta P}$$

Therefore:

$$\Delta P = \frac{Ao}{2}\cdot\frac{dP}{dA}$$

Substituting into (2):

$$v = \frac{\dot{V}}{A} = \sqrt{\frac{2\Delta P}{\rho}} = \sqrt{\frac{2}{\rho}\cdot\frac{Ao}{2}\cdot\frac{dP}{dA}} = \sqrt{\frac{Ao}{\rho}\cdot\frac{dP}{dA}} \tag{4}$$

Equation (4) is called the *Wavespeed equation*, which has been widely quoted as a unique predictor of maximum flow [27]. Its importance is that it describes the velocity of gas flow at the compressed segment and thus the intensity of gas–liquid interaction.

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