Chest Physiotherapy in the Intensive Care Unit

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Preface

It is quite apparent, from even a casual conversation with physicians or other personnel involved in respiratory care management, that there is a large spectrum of differing treatments termed by their users as "chest physiotherapy." The literature is not helpful in specifying what chest physiotherapy is intended to include. Is the inhalation of bronchodilating or mucolytic agents part of chest physiotherapy? In tracheally intubated patients, is manual hyperinflation of the lungs an inclusive part of chest physiotherapy? Many centers would use these therapies, others would not. All would claim to be treating the patient with chest physiotherapy. It is not surprising, therefore, that there are many contradictory opinions concerning the effects of chest physiotherapy. Because of these variations, if an improvement does occur, it is likely to be difficult to determine the beneficial component.

A homogeneous patient population treated in a similar manner by the same personnel over a number of years gives a useful clinical experience that frequently cannot be duplicated. At the Maryland Institute for Emergency Medical Services Systems (MIEMSS) in Baltimore, Maryland, there is a unique and homogeneous population of traumatized patients. Year after year, the admission statistics confirm the similarities in the patients, and their injuries, and in morbidity and mortality. The population is unique because about 60% of the 1,200 or more patients admitted each year come directly from the scene of their accident and about 75% of these patients come to the Institute by helicopter.

For the past 7 years, chest physiotherapy has been used in the critical care and intensive care units to treat patients with lung secretion retention. The physical therapists providing the therapy have a remarkable record of service. The three physical therapists who have contributed to this book have had between them 18 years of work at MIEMSS since 1973. The therapy they have provided encompasses five maneuvers: 1) postural drainage, 2) chest wall percussion and vibration, 3) coughing, 4) suctioning of the loosened secretions, and 5) breathing exercises in the spontaneously breathing patient. In addition, mobilization is used whenever possible.

Besides the similarities in patient population, personnel, and therapy, the mechanical ventilatory support was standardized at the Institute between 1973 and 1978 with the use of only one type of volume-preset ventilator. Controlled mechanical ventilatory support was employed for resuscitation, for anesthesia and throughout recovery, providing humidification at all times. From October 1978 on, intermittent mandatory ventilation was occasionally used instead of controlled mechanical ventilation. No intermittent positive pressure breathing (IPPB) machines were used to deliver bronchodilator or mucolytic agents. No inhaled drugs, other than water vapor, were given in the critical or intensive care units. Tracheal lavage was rarely employed. The "bag squeezing" technique of chest physiotherapy, in which the lung is hyperinflated and the chest vibrated during expiration, was not used. No spontaneously breathing patients were treated with the aid of blow bottles or incentive inspiratory spirometers. Nasotracheal suctioning was seldom used or attempted. Tracheal suctioning was only carried out in intubated patients. Because these other respiratory maneuvers were excluded, the effect of chest physiotherapy alone was determined.

As with any book directed at diverse groups, such as critical care specialists, anesthesiologists, surgeons, internists, chest physiotherapists, nurse intensivists, and respiratory therapists, some areas of the text are more relevant than others to each group. For the physician, the changes that take place with therapy and the ag-

gressive approach taken at MIEMSS are complemented by a considerable quantity of data and many case histories. To the physical therapist working in the intensive care unit, this book provides complete coverage of the specialty of chest physiotherapy. For the nurse intensivist and respiratory therapist, a practical approach to the respiratory management of the multiply-monitored intensive care unit patient is combined with a reference guide to the literature on chest physiotherapy. This book presents our experience with chest physiotherapy in the management of acute lung pathology in patients with previously normal and abnormal lungs. Over the 7 years (1973–80), a homogeneous patient population of over 3,000 intensive care unit patients was treated. The mechanical ventilation and physiotherapy techniques were standardized, and the medical and physical therapy personnel managing the respiratory care were constant. It is hoped that this book will provide others with a well-tested, practical approach to chest physiotherapy for intensive care patients.

C.F.M.
February 1981

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For the photographs, we thank Colin Mackenzie, M.B., Ch.B., F.F.A.R.C.S., and Dick Register for taking them; and Frank Ciesla, MIEMSS and University of Maryland Hospital Illustrative Services, for printing them. For illustrations, we thank Chris McCullough-Green; and for proofreading, Barbara Eerligh and Beverley Sopp. Jeremy Hallisey, M.B., B.S., and David Clark helped with data analysis, proofreading and reference checking. Our thanks go to Marlene Wheeler and Kate McWilliams, who typed the final drafts, and to Sandy Bond-Lillicropp, who organized the typing of the earlier drafts.

Finally, we thank experts in the field of chest physiotherapy on both sides of the Atlantic who have read and criticized the manuscript at various stages. However, the final result should not be blamed on them. Rather, the end product is the result of our determination to keep some parts, such as the patient population data and the sections on special patients and mobilization which do not relate strictly to "chest therapy" or "intensive care." The reviewers included Margaret Branthwaite, M.R.C.P., F.F.A.R.C.S., and Barbara Webber of the Brompton Hospital, London, England; Anthony Clement, M.B., B.S., F.F.A.R.C.S., of St. Thomas' Hospital, London; John Hedley-Whyte, M.D., and Cynthia Zadai of Beth Israel Hospital and Harvard Medical School, Boston, Massachusetts; T. Crawford McAslan, M.D., of Baltimore City Hospitals and The Johns Hopkins Medical School; Iain L. Mackenzie, M.D., of York Hospital, York, Pennsylvania, and Baekhyo Shin, M.D., Lucille Ann Mostello, M.D., and Martin Helrich, M.D., all of the University of Maryland Hospital and Medical School, Baltimore. Particular thanks are due Martin Helrich M.D., Chairman, Department of Anesthesiology for his support and encouragement.

Contents

CHAPTER 1

History and Literature Review of Chest Physiotherapy, Chest Physiotherapy Program, Patient Population and Respiratory Care at MIEMSS

Colin F. Mackenzie, M.B., Ch.B., F.F.A.R.C.S.

As an introduction to this book, Chapter 1 is intended to put chest physiotherapy into historical perspective and provide a review of the literature. A brief description of the authors' understanding of chest physiotherapy is followed by some comments on the variations found in the literature concerning indications, type of therapy, and duration of chest physiotherapy. The patient population treated and studied at MIEMSS is described, and the chest physiotherapy program and techniques for respiratory management of these patients are discussed.

HISTORICAL SUMMARY AND LITERATURE REVIEW

1901 William Ewart described the beneficial effects of postural drainage in the treatment of bronchiectasis.

1908 Pasteur delivered the Bradshaw Lecture on massive collapse of the lung.

1910 Pasteur reported on the finding of acute lobar collapse as a complication of abdominal surgery.

1915 MacMahon described the use of breathing and physical exercises in patients with lung, diaphragm and pleural injuries sustained in World War I.

1918 Bushnell used postural drainage for patients with pulmonary tuberculosis.

1919 MacMahon used breathing exercises for patients recovering from gunshot wounds of the chest.

1933 Jackson and Jackson wrote on the benefits of pulmonary drainage and coughing.

1934 Winifred Linton, a physiotherapist at the Brompton Hospital, London, England, introduced "localized breathing exercises" for the thoracic surgical patient (Gaskell and Webber, 1973).

1934 Nelson recommended bronchial drainage for management of bronchiectasis in children. He emphasized the use of physical and radiological examination to locate the specific position of the lung lesion and to determine patient positioning for drainage.

1938 Knies recommended bronchial drainage following thoracic surgery.

1950 Temple and Evans defined bronchopulmonary segments to identify areas of the lung needing resection.

1950 Felson and Felson used the silhouette sign to localize intrathoracic lesions radiographically.

1952–1953 Kane described pulmonary segmental localization on posteroanterior chest X-rays. He also noted that the more accurately gravity was applied to the draining bronchus, the more effective was the postural drainage.

Many reports of symptomatic and physiological benifits from breathing exercises and postural drainage appeared up to 1945 (Heckscher). However, until the 1950's there was little change in the incidence of atelectasis from that reported by Pasteur, despite the advances in surgery, anesthesia, and antibiotic usage. Pioneering work on the effects of chest physiotherapy was published by Palmer and Sellick in 1953. They described the use of breathing exercises, postural drainage with vibratory and clapping percussion, and inhalation of isoprenaline before and after surgery. This regime was significantly more effective than breathing exercises alone in reducing pulmonary atelectasis in 180 patients operated upon for hernia repair or partial gastrectomy. They also found that isoprenaline combined with postural drainage and vibratory and clapping percussion prevented atelectasis, but that neither intervention alone prevented it. These studies included controls but were based on the somewhat-subjective data of atelectasis, as judged by clinical examination and chest X-ray. Thoren (1954), using diaphragmatic breathing and deep breathing while sidelying, postural drainage, and coughing, showed that without the use of inhalation therapy it was also possible to produce a significant reduction in pulmonary complications after cholecystectomy. Atelectasis developed in 11 of 101 patients treated before and after cholecystectomy, in 18 of 70 patients (25.7%) treated with chest physiotherapy only after cholecystectomy, and in 68 of the 172 patients (35.9%) who were not given chest physiotherapy after surgery.

Therefore, over 25 years ago, there appeared to be a specific indication for chest physiotherapy in the prevention of pulmonary complications after surgery. Since then, there has been an explosion in the apparent indications for chest physiotherapy. It is the application of chest physiotherapy with lack of specific indications that has rightly promoted adverse commentary. The original popularity of chest physiotherapy arose because of the benefits produced in patients with retained lung secretions. However, to our knowledge, no one has shown that treatment with chest physiotherapy has altered the morbidity or mortality of patients with chronic lung disease, whereas chest physiotherapy for acute lung pathology in a previously normal lung may produce a more favorable outcome.

The advent of controlled mechanical ventilation in 1953 (Crampton Smith et al., 1954) as a means of treating acute respiratory insufficiency also gave rise to the realization that during artificial respiration there is a special liability to pulmonary complications. Opie and Spalding (1958) produced a review of some of the physiological changes that occurred during chest physiotherapy and controlled mechanical ventilation, using intermittent positive pressure with a negative expiratory phase. The negative phase was then popular because this improved venous return to the heart. Advocates of intermittent mandatory ventilation make a similar claim about the spontaneous breath with this mode of ventilation.

Opie and Spalding noted that the rate of air flow in and out of the lungs was dependent upon the esophagotracheal pressure gradient, and that a rise in esophageal pressure with chest compression accelerated expiration (Fig. 1.1). Application of chest compression late in expiration caused only a very slight alteration in air flow. Two mechanisms for the action of chest physiotherapy were postulated. 1) By raising intrathoracic pressure as a whole, air was rapidly expelled from the lungs, carrying secretions with it, as in a cough. The paper, however, argues quite successfully against this mechanism. 2) By local compression of the lung underneath the physiotherapist's hands, secretions were pushed from the more peripheral air-

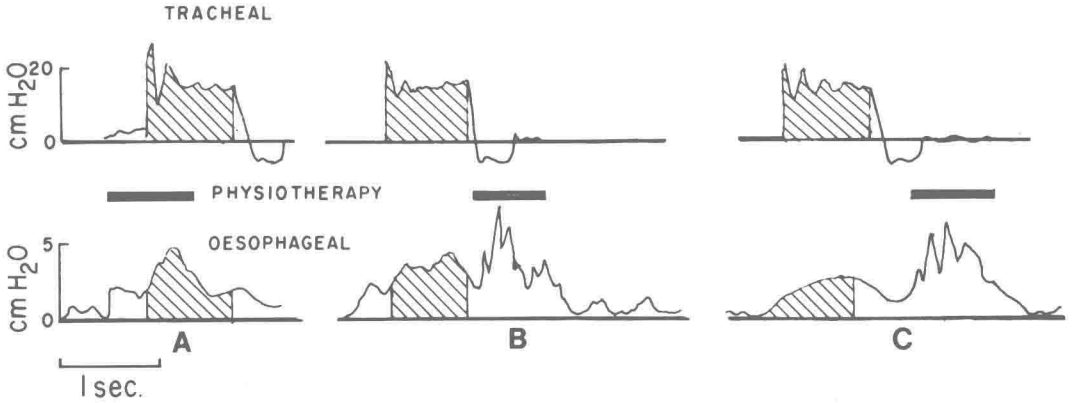

Figure 1.1. The effect of chest physiotherapy on tracheal and esophageal pressure in a patient ventilated with intermittent positive/negative pressure ventilation. *Hatched area,* inspiratory period. *A.* Chest vibration during inspiration. *B.* Chest vibration during the inspiratory-expiratory junction. *C.* Chest vibration during the middle of the expiratory period. Note the greatest tracheoesophageal pressure differences occur with *B* and *C* which are the normally used methods of applying chest vibration. (Tracing reproduced with permission from L. H. Opie and J. M. K. Spalding: *Lancet* 2:671–674, 1958.)

ways into the main bronchi. This has since been disputed as the mode of action (Laws and McIntyre, 1969).

Wiklander and Norlin (1957) compared 100 patients who, following laparotomy, received chest physiotherapy, with 100 who did not. Chest physiotherapy was given before and after surgery, for usually 3 days, or as long as sputum was obtained. The incidence of atelectasis was 13% in those who received chest physiotherapy, and 24% in those who were asked to turn from side to side in bed and given instruction and help in coughing.

In a frequently quoted study on the value of lung physiotherapy in treatment of acute exacerbations in chronic bronchitis, Anthonisen and his colleagues (1964) compared conventional treatment and chest physiotherapy to conventional treatment alone. No differences in outcome were found between the groups randomly treated with and without chest physiotherapy. During an acute flare-up of chronic bronchitis, chest physiotherapy did not seem beneficial, but they did not exclude the possibility of benefit in lobar atelectasis.

In 1966, Holloway and his colleagues reported that chest physiotherapy appeared to cause a fall in arterial oxygenation when applied to neonates with tetanus. This preliminary observation was followed by the publication of a study on 22 patients with tetanus who received chest physiotherapy (Holloway et al., 1969). These patients were compared to a matched group of 14 spontaneously breathing patients and a group of 15 neonates receiving mechanical ventilation but not chest therapy. Chest physiotherapy, which took the form of clapping and compression, percussion and vibration, was followed by suctioning. A fall in PaO_2 occurred after chest physiotherapy, but it is doubtful if the changes of PaO_2 50.6 ± 6.4 to 47.0 ± 6.4 torr (mean ± SD) were clinically significant, although it was apparently statistically so. The control group was ventilated but did not receive chest physiotherapy and was not turned to the same positions. Therefore, simple $\dot{V}/\dot{Q}$ changes cannot be excluded as the cause for the fall in PaO_2 in the treated patients.

Further information about chest physiotherapy was published in 1969 when Laws and McIntyre described changes in gas exchange and cardiac output associated with chest physiotherapy in six patients in respiratory failure. All were ventilated with volume ventilators and a tidal volume (V_t) of 10–13 ml/kg. Cardiac output was measured with the dye dilution technique. Since this was before the era of flow-directed pulmonary artery catheters, neither pulmonary artery pressure nor mixed venous gases were monitored. However, mixed expired and inspired gases were analyzed. Alveolar-to-arterial tension gradients for both O_2 and CO_2 were

derived and used to measure the efficiency of gas exchange before, during, and after chest physiotherapy. The procedures performed included postural drainage with percussion, shaking, and vibration. Artificial coughs were given in the supine position, and both lateral positions, and chest compression was performed during expiration. These procedures were followed by lung hyperinflation (V_t, 20–25 ml/kg). The patients were then suctioned; none had large amounts of sputum. This factor appeared to be crucial, as these authors were unable to show any improvement in gas exchange. They also cast doubt on the hypothesis that external chest compression squeezes secretions from completely occluded airways by direct lung compression. They suggest that the amount of compression required to do this would produce areas of collapse. As an alternative hypothesis, they put forward the idea that since clearance of the airway requires some expansion of lung units distal to airway to be cleared, indirect ventilation of these distal airways may be achieved by collateral channels. The more proximal airways are then cleared by increased expiratory flows, generated by the physiotherapists, from the distal airways. Our explanation of the mechanism of chest physiotherapy action is similar and is described in Chapter 7.

In the patients studied by Laws and McIntyre during physiotherapy, cardiac output varied up to 50% from the levels obtained before physiotherapy. These variations persisted for as long as 30 minutes. The greatest variation occurred during the artificial cough with inflation pressures of 60–100 cm H_2O. In some patients, cardiac output fell due to impaired venous return during this maneuver. In those who were conscious, the procedure was also found to be extremely unpleasant. During resistance to lung hyperinflation, and with patient apprehension, cardiac output rose. These hyperinflations, although causing such changes in the cardiovascular system, were not able to produce any lasting benefit to pulmonary gas exchange. These are some of the reasons for the omission of lung hyperinflation from physiotherapy treatment at MIEMSS.

Lorin and Denning (1971) found that postural drainage produced more than twice the volume of sputum as an equal period of cough alone in 17 patients with cystic fibrosis. Postural drainage lasted 20 minutes and included positioning for the right middle lobe, lingula, and some basilar segments of the lower lobe. The patients received percussion and vibration in each position. The volume of sputum produced when compared with the volume produced by the same patient in the sitting position, coughing every 5 minutes for 20 minutes, averaged 3.4 cc and was significantly greater than that of the control of 1.6 cc ($p < 0.0001$).

Gormezano and Branthwaite (1972a) reported the effects of chest physiotherapy on 42 adults receiving intermittent positive pressure ventilation. The patients were considered in three groups: Group I included 18 patients with no cardiac disability; Group II, 13 patients with cardiovascular disability; and Group III, 11 patients with respiratory failure. Chest therapy included hyperinflation to 20 cm H_2O above previous ventilator settings, manual chest compression, and tracheal suctioning. Duration of treatment was from 7 to 20 minutes, depending on whether copious secretions were mobilized. Arterial blood gases were sampled before and at 5 minutes, 15 minutes, and 30 minutes after cessation of therapy. Patients in Groups I and III did not show any change; Group II showed a maximum fall in PaO_2 of 14.9 ± 4.5 torr (SE) 5 minutes after therapy. Within 30 minutes this had returned to the levels obtained before therapy. Hyperinflation caused a rise in PaO_2 in all groups. $PaCO_2$ increased in all groups, but a rebreathing circuit was used during manual chest compression. The authors postulated that during chest physiotherapy, 1) cardiac output fell; therefore, $P\bar{V}O_2$ fell; and, therefore, PaO_2 fell; 2) there was an increase in intrapulmonary shunt; and 3) there was increased oxygen consumption. Because no indications for chest physiotherapy were given, it is not known whether treatment was performed prophylactically or for a specific indication. Since the patients were turned on both left and right sides but were not apparently posturally drained with the affected lobe or segment uppermost, it is not certain whether chest physiotherapy produced these changes or whether they were due to changes in posture.

Gormezano and Branthwaite (1972b) also studied patients treated with chest physiotherapy and intermittent positive

pressure breathing (IPPB). Thirty-two chronic bronchitic patients with airway obstruction and sputum production were divided into three groups: those with reversible airway obstruction (Group I), those with profuse secretions (Group II), and those with respiratory failure (Group III). Mean PaO_2 fell 4–6 mm Hg in all groups, but the fall was greatest in Group II. The fall was thought to be due to increased intrapulmonary shunt. However, no specific cause of the increased shunt was identified, although several were hypothesized, such as decreased pulmonary artery pressure with rest after therapy, increased pulmonary artery pressure due to increased cardiac output, and abolition of hypoxic vasoconstriction.

In 1973, Clarke and his colleagues reported the effects of sputum on pulmonary function. Patients with copious sputum production and airway obstruction (forced expired volume in 1 second (FEV_1)/forced vital capacity (FVC) $< 70\%$ predicted) improved in all measured parameters, particularly specific airway conductance, following sputum removal. There was, however, no relationship between the volume of sputum produced and the improvement of pulmonary function. They concluded that although sputum volume production is important, its distribution within the bronchial tree and its viscoelastic properties may be of greater importance.

A report in which removal of inhaled radioactive tracers was used to measure pulmonary mucociliary clearance in cystic fibrosis appeared in 1973 (Sanchis et al.). Despite previous beliefs, mucociliary transport in 13 children with cystic fibrosis was found to take place at a similar rate to that found in normal adults. The theory that the viscid secretions found in cystic fibrosis (or mucoviscidosis) were inadequately cleared, resulting in blocked airways, stasis, and resultant infection, appeared to be considerably set back by this finding (Waring, 1973). However, one problem with the technique used was that the particle size of 3 μm was perhaps too large and, therefore, the radioactive particles did not penetrate the lung effectively. More central penetration occurred in children than in adults. Because mucociliary clearance is faster from larger airways than from smaller airways, the children may only appear to have normal mucus clearance. The radioactive tracer clearance technique, however, is now the accepted model for further investigation in a controlled environment such as the laboratory.

Campbell and his colleagues (1975) reported that bronchoconstriction, as measured by a fall in FEV_1, occurred in seven patients with exacerbation of chronic bronchitis following chest percussion or vibration. They found that bronchoconstriction was particularly noticeable in patients who did not have copious sputum production. The fall in FEV_1 was not confirmed by other studies of chest physiotherapy and chronic bronchitis (Cochrane et al., 1977; Newton and Stephenson, 1978; May and Munt, 1979).

Tecklin and Holsclaw (1975) found that following postural drainage, percussion, vibration and coughing in 26 patients with cystic fibrosis, significant increases occurred in peak expiratory flow rate, FVC, expiratory reserve volume and inspiratory reserve capacity. Larger airways appeared to be the sites of this beneficial action. There was no indication that these benefits lasted beyond 5 minutes after treatment had ceased. Cystic fibrosis is one of the few chronic lung diseases for which the benefits of chest physiotherapy are documented. A conference in Europe, published in 1977, summarized the state of the art (Baran and Van Bogaert, 1977).

Objective evidence of change in the lungs following sputum removal by chest physiotherapy in mechanically ventilated patients was reported by Winning and colleagues (1975). They estimated alveolar pressure by means of a retard mechanism applied to the lungs at end expiration. A significant fall in "alveolar pressure" was noted to occur after chest physiotherapy in 17 patients. Unfortunately the adjustment of the ventilator necessary to produce the "alveolar pressure" alters the characteristics of the lung under study. Therefore, it is difficult to determine whether the changes found were due to chest physiotherapy or ventilator manipulation.

The additional effect of only a mucolytic agent, or a bronchodilator and a mucolytic agent, on arterial oxygenation following chest physiotherapy was compared to the therapy alone. No differences were found (Brock-Utne et al., 1975). A similar finding was reported in which clearance of inhaled polystyrene particles tagged with

technetium-99m was used to assess removal of lung secretions in a double-blind crossover trial in 16 patients with chronic bronchitis (Thomson et al., 1975). There was no significant difference in weight or radioactive content of sputum expectorated between the patients who were given *S*-carboxymethylcysteine, a mucolytic agent, and those who were not. Ventilatory capacity as assessed by dry spirometry was not changed, nor was there subjective improvement noted by the patients. Roper and colleagues (1976) found right upper lobe atelectasis occurred after tracheal extubation in 18 of 188 newborn infants. This was thought to result from the anatomical positions of the right upper lobe bronchus and damage from suction catheters. The atelectasis could usually be expanded by chest physiotherapy. If it was unresponsive, an orotracheal tube was inserted, and the lungs manually hyperinflated and suctioned until all secretions were mobilized. Following this the trachea was immediately extubated. This regime resulted in the elimination of recurrent atelectasis as a major problem after extubation. Tecklin and Holsclaw (1976) found that *N*-acetylcysteine (Mucomyst) and bronchial drainage and coughing produced the same changes in respiratory function in 20 patients with cystic fibrosis, that occurred without the use of the mucolytic agent. In fact, maximal midexpiratory flow rate worsened, showing significant decrease with the inhalation. This was thought to be due to reflex small airway constriction and edema following *N*-acetylcysteine or due to coughing. Using technetium-99m, Pavia et al. (1976) found that a mechanically vibrating pad did not significantly alter clearance of sputum when compared in 10 patients who had histories of productive cough and difficulty expectorating phlegm; however, postural drainage was not used.

Martin et al. (1976) investigated the ability of unilateral breathing exercises to alter distribution of ventilation and blood flow in patients undergoing bronchospirometry. In no instance was the distribution of ventilation or blood flow altered to the side that was supposed to be limited. However, although these patients had active tuberculosis, there was no indication that the pathological lung was the target of the therapy, since both sides of the chest were tested. All patients had less than 15% involvement of the lung fields by chest X-ray, respiratory function tests were all normal, and only one subject was thought to have moderately advanced disease. Therefore, it is possible that in major lung pathology or in patients with chest splinting due to pain, breathing exercises may have a different effect, when large differences in lung/thorax compliance occur.

Removal of sputum by chest physiotherapy produced an improvement in specific airway conductance in 17 of 23 patients with chronic cough, airway obstruction, and at least a 30-ml sputum production per day (Cochrane et al., 1977). This improvement did not occur in 4 normal subjects, nor in 8 of the study patients who, on the following day, were given 150 mg isoprenaline base by inhalation instead of physiotherapy. Cochrane and his colleagues reiterated their belief that the distribution of sputum throughout the airways appeared to be more relevant than sputum volume, viscosity, or character. No correlation was found between changes in specific airway conductance and sputum volume produced by chest physiotherapy.

By using transcutaneous O_2 monitoring, the effect of chest physiotherapy on PaO_2 in 45 patients who had undergone abdominal surgery was compared to three deep breaths using incentive spirometry, a mechanical lung insufflator, and the blowing up of a paper coil (Hedstrand et al., 1978). Chest physiotherapy produced a greater increase in PaO_2 than did the other maneuvers, though it is doubtful if a 7 mm Hg rise in PaO_2 is clinically any different from the 3 to 4.5 mm Hg obtained with the respiratory therapy devices. The reliability of transcutaneous O_2 monitoring, when used in adults, is also in question. This paper does not record why the patients needed therapy. The respiratory therapy devices may have been used in the recommended manner; however, chest physiotherapy, which apparently consisted of ten deep breaths and a minute of coughing followed by assisted costal breathing in the lateral position, at our institution would be considered inadequate to clear retained secretions.

Two abstracts (Finer et al., 1977; Fox et al., 1977) that described chest physiotherapy for the neonate were published as papers the following year. Finer and Boyd

(1978) studied 20 neonates with a mean weight of 2.07 kg. Seven neonates were mechanically ventilated; all had respiratory failure and were receiving supplemental O_2. Respiratory failure was due to respiratory distress syndrome in 14 neonates, tachypnea in 2, pneumonia in 3, and apnea in 1. Arterial blood gases were analyzed before, and 15 minutes after, postural drainage and suction (10 infants) or postural drainage, percussion and suction (10 infants). The neonates showed a rise in PaO_2 when postural drainage, percussion and suction were used but no significant change with postural drainage and suction alone. The same findings, in a population of a different age and ventilated differently, were reported in the abstract. It is not clear why some patients, whose data appeared in the abstract, were omitted from the paper.

Fox and his colleagues (1978) studied 13 newborns to "determine the benefit/risk ratio of chest physiotherapy." All were intubated, breathing spontaneously with positive airway pressure, and were recovering from respiratory disease (respiratory distress, 10; aspiration, 2; apnea, 1). After a control period, 30 seconds of anterior chest wall vibration was performed by using a mechanical vibrator. The infants were then suctioned and hyperventilated for ten breaths. Since neither postural drainage nor percussion was used, the treatment given was not strictly chest physiotherapy. However, there was a consistent trend in which compliance and functional residual capacity (FRC) increased in parallel throughout all the periods of study. Inspiratory airway resistance was noted to fall significantly following chest vibration and suctioning, but this had returned to control levels within 2 hours. Arterial oxygenation fell significantly following suctioning. This was reversed with hyperventilation. Two hours following therapy, PaO_2 levels did not differ from control. The fall in PaO_2, which was as high as 81 torr in a patient breathing 55% oxygen, was not thought to be due to atelectasis because there was no change in FRC and no fall in lung compliance. It was, perhaps, due to the rise in pleural pressure accompanying coughing and suctioning which may have increased a right-to-left shunt.

Chest physiotherapy produced a different effect on pulmonary function in patients with acute exacerbations of chronic bronchitis (Newton and Stephenson) than in patients with bronchiectasis and cystic fibrosis (Cochrane et al.). The 33 patients studied by Newton and Stephenson, within 4 days of admission for acute exacerbation of chronic bronchitis, had an FEV_1/FVC ratio of <50%, indicating considerable respiratory impairment and airway obstruction. They had less than 15% improvement after use of bronchodilators. Thoracic gas volumes and airway resistance were measured in a body plethysmograph, and specific conductance was derived. FEV_1, vital capacity, and inspiratory capacity were also measured before chest physiotherapy. All tests were repeated 2 times at half-hourly intervals after 15 minutes of physiotherapy. Positioning and chest physiotherapy maneuvers were not adequately described. No more than 5 ml of sputum were produced in any patient. An acute rise in lung volume, FRC and conductance occurred, but there was no change in arterial blood gases. No deterioration occurred in FEV_1. Most patients produced 2 ml of sputum or less; it is not surprising that the authors concluded that their patients did not show any obvious benefit from chest physiotherapy.

Graham and Bradley (1978) compared a randomized group of 27 patients, treated with chest physiotherapy and IPPB for 20 minutes, to a control group of 27 similar patients. Both groups had pneumonia, as judged by a compatible clinical history of fever and increased cough, radiological confirmation, and a positive gram stain of sputum and blood cultures (12% positive). They found no difference in duration of fever, extent of radiographic clearing, duration of hospital stay, or mortality between the control and treated groups. As was pointed out in the correspondence following this article, the authors excluded patients with bronchiectasis, lung abscess and cystic fibrosis who might have expected to benefit from chest physiotherapy. The establishment of a diagnosis of pneumonia in the intensive care unit (ICU) is not as simple as was cited by Graham and Bradley. As mentioned in Chapter 2, what appeared to be a pneumonic process was cleared in its early stages with treatment by chest physiotherapy (Case His-

tory 2.1). Restriction of chest physiotherapy to a predetermined time of 20 minutes may not provide sufficient duration to clear secretions, especially when IPPB is also given during the same 20 minutes. The conclusion by Murray (1979), that the use of chest physiotherapy for patients with otherwise-uncomplicated pneumonia should stop, appears quite reasonable, especially if the patients are ambulatory or mobilized. For the mechanically ventilated patient in the ICU this may not be valid, since it is difficult to arrive at the diagnosis of pneumonia unless retained secretions are not cleared or radiological improvement fails to occur following chest physiotherapy.

An editorial in the *Lancet* (1978) scrutinzied the use of chest physiotherapy and noted that surprisingly few studies showing objective assessment were published. Those that were published seemed to have concentrated on areas where physiotherapy is predictably ineffective. Chest physiotherapy was thought to be most useful when copious amounts of very sticky sputum were produced. It was also emphasized that by talking to, touching, and making the patient more comfortable, the physiotherapist provides an important link between the patient and other members of staff.

Newton and Bevans (1978) treated 39 patients with acute exacerbations of chronic bronchitis with antibiotics, bronchodilators and diuretics (standard treatment). These patients were compared with 40 patients treated with IPPB (3 times daily with nebulized saline) and chest physiotherapy for 10–15 minutes, in addition to the standard treatment. Arterial blood gases, sputum volume, FEV_1 and vital capacity were measured among other parameters. Only admission and discharge data were provided. Since discharge is frequently not a clinical but an administrative decision, it would be helpful if some daily data were included. Chest physiotherapy was not adequately described and the results were at times confusing. However, no differences were found in PaO_2, $PaCO_2$, FEV_1, vital capacity or duration of hospital stay between the two groups. The only difference was that men who received chest physiotherapy produced a greater sputum volume than those who did not.

The effects of chest percussion and postural drainage on respiratory function in 35 patients with stable chronic bronchitis were compared to sham treatment with an infrared lamp (May and Munt). The conclusions were similar to the study of Newton and Stephenson on patients with acute exacerbations of chronic bronchitis and suggested that postural drainage and chest percussion did not benefit the patients. This study showed that although chest physiotherapy improved FVC and FEV_1, these also improved following the use of the infrared lamp (sham treatment). Sputum production was greater during percussion and postural drainage than during the infrared warming (5.5 vs. 1.4 ml average) or during coughing (9.0 vs. 3.5 ml average). Bateman and his colleagues (1979) did not use a sham treatment but used a crossover control when they compared clearance of bronchial secretions labeled with technetium-99m from 10 patients with stable chronic airway obstruction and regular sputum production. They found that chest physiotherapy was highly effective in moving bronchial secretions from peripheral to more central lung regions and aided in expectoration.

Feldman et al. (1979) found that postural drainage with chest percussion, vibration and coughing resulted in significant improvement in expiratory flow at 50% and 25% of FVC. In the 19 patients studied who had chronic bronchitis or cystic fibrosis, there was no correlation between volume of sputum produced and changes in lung function.

Oldenburg et al. (1979) studied the effect of postural drainage, exercises and cough in 8 clinically stable patients with chronic bronchitis. They found that cough alone greatly accelerated bronchial clearance of a radioactive tracer deposited in the tracheobronchial tree. Postural drainage without coughing did not alter clearance. The differences in the findings of this study and that of Bateman et al. may be a function of study design and the properties of the radiolabeled aerosols used (Rochester and Goldberg, 1980). The site of deposition of radioactive aerosol showed considerable variation between the subjects in the study by Oldenburg and colleagues. Therefore, the conclusion that cough was effective in improving peripheral airway clearance may not be valid. These findings need confirmation in a larger patient pop-