

Ventilatory effects of neurophysiological facilitation and passive movement in patients with neurological injury

Angela Chang¹, Jennifer Paratz^{1,2} and Julia Rollston^{1,3}

¹The University of Queensland ²La Trobe University ³Derbyshire Royal Infirmary, UK

Thirteen intubated, high dependency patients with neurological injuries were studied in order to investigate the short term respiratory effects of neurophysiological facilitation and passive movement on tidal volume (V_T), minute ventilation (V_E), respiratory rate (V_R) and oxygen saturation (SpO_2). The subjects were studied under four conditions: no intervention (control) and during periods of neurophysiological facilitation, passive movement and sensory stimulation. All periods were standardised to three minutes duration and all parameters were recorded before and after each intervention. Neurophysiological facilitation produced significant increases ($p < 0.01$) in V_E and SpO_2 ($p < 0.05$) when compared with control values, with an overall mean increase in V_E of 14.6%. Similarly, passive movement increased V_E ($p < 0.01$) by an average of 9.8% and also increased SpO_2 ($p < 0.01$). In contrast, sensory stimulation produced significant increases ($p < 0.01$) in SpO_2 with control levels, with no significant change in V_T or V_E . There was no significant difference in V_R with all treatments. This study provides preliminary evidence of improved short term ventilatory function following neurophysiological facilitation, independent of generalised sensory stimulation, which has not been previously examined in the literature, supporting its use in the management of high dependency neurological patients. [Chang A, Paratz J and Rollston J (2002): Ventilatory effects of neurophysiological facilitation and passive movement in patients with neurological injury. *Australian Journal of Physiotherapy* 48: 305-309]

Key words: Head Injuries, Closed; Physical Stimulation; Physical Therapy; Respiratory Mechanics

Introduction

Neurological insults including head injury and cerebrovascular accidents are a common cause of long-term dysfunction. To optimise long-term function, physiotherapy management is instituted in the acute stage, aiming to assist the musculoskeletal, neurological and respiratory system.

In the immediate post-injury period, patients with severe neurological injuries are usually intubated, ventilated, given vasoactive support, sedated and often paralysed in order to prevent secondary brain damage from inadequate gas exchange (Gruen and Liu 1998, Prough and Lang 1997). Respiratory complications are common (Demling and Riessen 1990), both occurring concomitantly with the neurological insult, as in aspiration pneumonia, thoracic trauma or neurogenic pulmonary oedema, or as a result of mechanical ventilation and immobilisation. As a consequence, respiratory physiotherapy forms an essential component of early management (Ada et al 1990).

As the patient's condition stabilises, they are able to breathe by themselves via either a normal airway or a tracheotomy. However, there is still a high risk of respiratory complications that may result in readmission to intensive care. Respiratory complications are associated with further neurological deterioration (Chen et al 1998). Commonly utilised techniques in this period include secretion

mobilising and removal techniques of percussion, vibration and suction (Ciesla 1996). Techniques to increase depth of ventilation include positioning, intermittent positive pressure ventilation and manual hyperinflation (Ciesla 1996; Webber and Pryor 1993). If the patient has a low Glasgow Coma Score, is not co-operative with treatment and does not have an artificial airway, increasing depth of breathing and removing secretions can be difficult.

Bethune (1975) described a number of neurophysiological facilitation techniques reported to increase the depth of breathing, decrease respiratory rate and increase arousal in patients with a decreased level of consciousness. These neurophysiological facilitation techniques have not been objectively tested to determine whether they are capable of increasing the depth of breathing (Pryor and Webber 2002).

Passive movements are used widely in the management of semi-conscious patients. It has been hypothesised that passive movement can increase ventilation (Guyton 1991) and arousal (Brimouille 1997) but this has not been formally studied.

In order to establish whether an increase in lung volumes does occur following neurophysiological facilitation and passive movement, the techniques of neurophysiological facilitation and passive movement were compared with general tactile and sensory stimulation and a control period of no stimulation in sub-acute patients following a neurological insult. Sensory stimulation was included in

Table 1. Subject characteristics.

Age	Sex	Diagnosis	Ventilation	GCS*
29	Male	Motor vehicle accident (MVA), hypoxic brain injury and subarachnoid haemorrhage and CSF infection	Spontaneous	3
37	Male	MVA with hypoxic brain injury	Spontaneous	11
66	Female	Ruptured internal carotid and posterior communicating artery aneurysm.	Spontaneous	11
32	Female	Spontaneous subarachnoid haemorrhage	Spontaneous	10
35	Male	Basilar artery occlusion	CPAP	8
36	Male	Subglial haemorrhage of unknown cause	Spontaneous	7
34	Male	(L) subdural haemorrhage following alcohol intoxication	Spontaneous	11
37	Male	MVA with pontine haemorrhage	Spontaneous	4
65	Male	(R) subdural haemorrhage	Spontaneous	11
17	Female	MVA with bilateral frontal lobe contusions	Spontaneous	7
37	Male	MVA with pontine haemorrhage	Spontaneous	4
28	Male	MVA with diffuse axonal injury	Spontaneous	11
62	Female	(R) subdural haemorrhage	Spontaneous	6

*Glasgow Coma Score. (CSF, cerebrospinal fluid. CPAP, continuous positive airway pressure.

Table 2. Summary of study results [mean (SD)].

		V _T (ml)	V _E (L/min)	RR (resp/min)	SpO ₂ (%)
Control	Initial	314 (105)	9.7 (2.1)	34 (13)	96.2 (2.1)
	Final	329 (102)	9.7 (2.5)	32 (9)	96.7 (2.0)
NPF	Initial	304 (106)	9.4 (2.2)	31 (7)	95.9 (1.7)
	Final	376 (132) *	10.7 (2.7) **	31 (9)	97.0 (1.8)**
PM	Initial	303 (81)	9.1 (2.2)	30 (9)	96.4 (1.6)
	Final	333 (116)	10.0 (2.1)**	33 (10)	97.5 (1.4) **
SS	Initial	307 (94)	9.4 (2.2)	32 (9)	96.2 (1.3)
	Final	353 (102)	10.2 (2.4)	31 (9)	97.4 (1.5) **

* denotes $p < 0.05$ compared with initial values

** denotes $p < 0.0125$ compared with initial values

NPF, neurophysiological facilitation. PM, passive movement. SS, sensory stimulation.

order to clarify whether any effect from neurophysiological facilitation or passive movement was due to the techniques themselves rather than the tactile stimulation that would inevitably accompany neurophysiological facilitation and passive movement.

The null hypothesis of the study was that there would be no difference in change in tidal volume (V_T), respiratory rate (V_R), minute ventilation (V_E) and oxygen saturation (SpO_2) following the four interventions of control, sensory stimulation, passive movement or neurophysiological facilitation.

Methods

This study was completed in the Neurosurgical Unit of the Royal Brisbane Hospital. Ethical clearance had been granted by University of Queensland and Royal Brisbane Hospital ethics committees and informed consent was gained from the subjects' next of kin. Subjects were included if they had a Glasgow Coma Score of less than 11, were breathing spontaneously (either on increased inspired oxygen or continuous positive airway pressure) had a tracheostomy, and were haemodynamically stable, defined as no change in heart rate above 30 beats per minute and

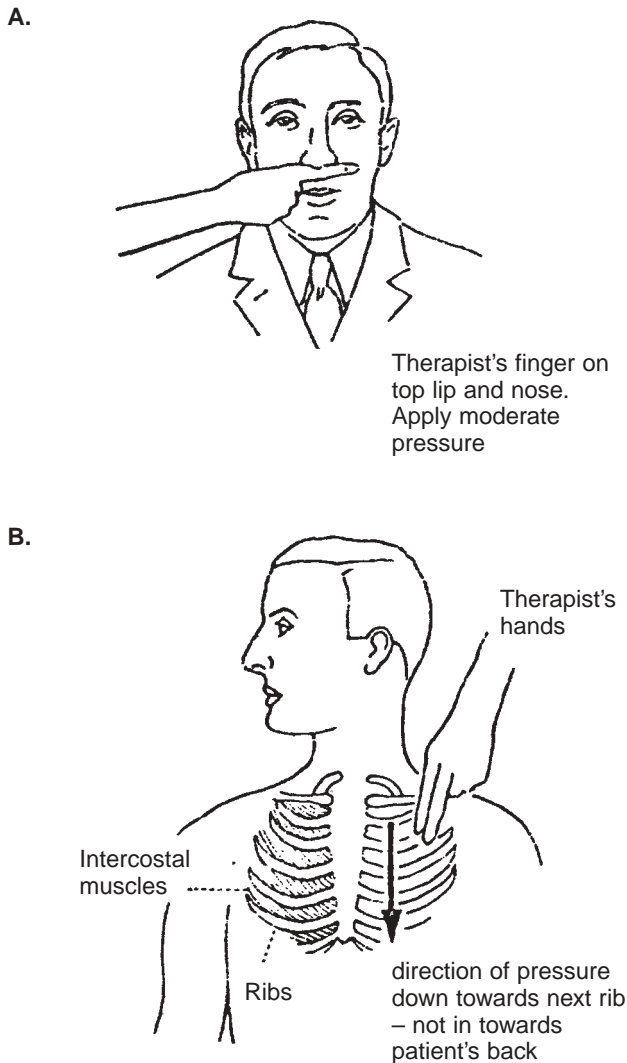


Figure 1. Neurophysiological facilitation. A: Perioral stimulation; B: Intercostal stretching. (From Bethune 1975 with permission from *Physiotherapy Canada*).

blood pressure above 20mmHg with any stimulation. They were excluded from the study if there were facial fractures, fractures of the rib cage or sternum, limb injuries, or suspected or actual deep venous thrombosis.

A total of 13 sub-acute patients with neurological injuries with a mean age of 38 (17-66) years were included in the study. No subject had a proven chest infection or pulmonary infiltrate on chest x-ray. Table 1 lists the characteristics of the subjects.

A pneumotach^(a) was attached to the patient's tracheostomy and the parameters of tidal volume (V_T), respiratory rate (V_R) and minute volume (V_E) were recorded continuously

by a VentrakTM respiratory mechanics monitoring system^(b) and downloaded to a computer. The respiratory mechanics monitor and pulse oximeters were calibrated according to manufacturers instructions. The digital waveforms were recorded for 60 seconds before and immediately after each intervention and waveforms analysed and interpreted using Analysis PlusTM software package. If the subject coughed during the period of measurement, the software was able to identify this and eliminate such periods from the data analysis. The blood oxygen saturation was recorded using pulse oximetry^(c). Values were taken before and immediately following each intervention. Previous studies have shown excellent correlation between SpO_2 and PaO_2 from an arterial blood gas sample (Escourrou et al 1990, Nyarwaya et al 1994).

Subject positioning was standardised to supine flat, limbs positioned in neutral. Prior to testing and following placement of equipment, each patient was left with no intervention for 15 minutes. Between each intervention, the subject was given no stimulation for 15 minutes until the next intervention. If any nursing intervention was required during the study, the subject was rested for 15 minutes after the intervention, to stabilise the subject. One investigator performed all testing, to standardise testing procedures. The order of the four interventions, control, sensory stimulation, passive movement and neurophysiological facilitation, was randomly allocated to each subject.

The control measure involved no handling, auditory or tactile stimulation for three minutes. Neurophysiological facilitation involved perioral stimulation (Figure 1A) applied for 10 seconds followed by intercostal stretch for 20 seconds (Figure 1B) applied bilaterally over anterior ribs 2 and 3. The cycle was repeated for three minutes. Perioral stimulation is a firm maintained pressure on the frenulum using a finger pad and intercostal stretch is a caudal stretch pressure on the upper ribs during expiration (Bethune 1975).

Sensory stimulation involved stroking the upper and lower limbs for 90 seconds at 1 Hz, and verbal stimulation, that is, calling the subject's name and asking them to breathe more deeply. Passive movements required moving the limbs at a rate of 0.5 Hz for 45 seconds each limb. Handling was minimised and standardised to control the amount of tactile stimulation.

Statistical analysis The raw data for each 60s interval were collated and checked to ensure a normal distribution. The data were analysed using repeated measures multivariate analysis of variance to examine the relationship between treatment intervention and time for all four dependent variables (V_E , V_R , V_T and SpO_2). Probabilities of less than 0.05 were considered significant. When a significant interaction between treatment and time was found, the relationship was investigated with univariate repeated measures ANOVA for each variable and post hoc paired *t*-tests, with a modified Bonferroni correction level of 0.0125.

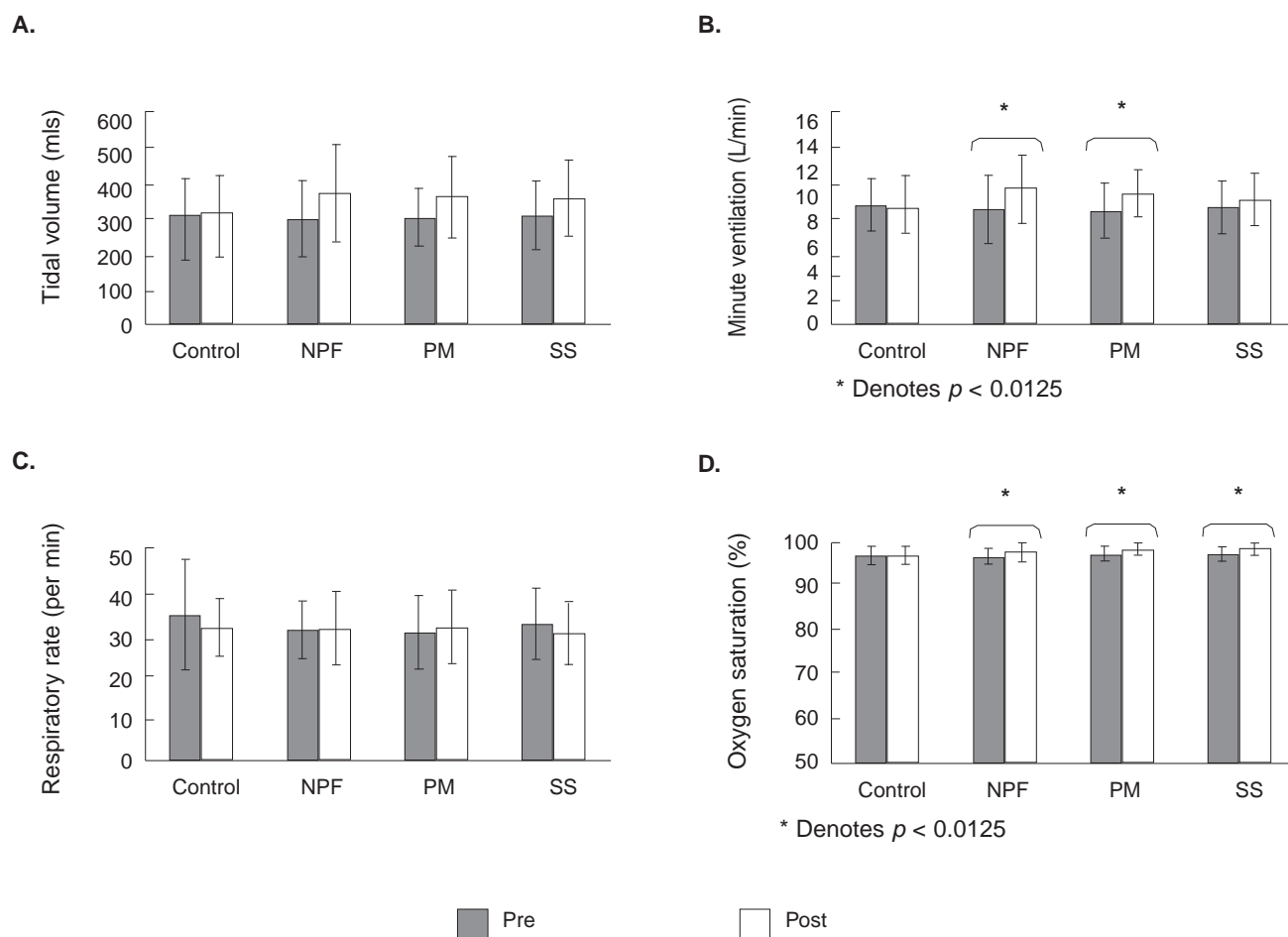


Figure 2. Effect of neurophysiological facilitation, passive movement and sensory stimulation on respiratory variables. A, Tidal volume. B, Minute volume. C, Respiratory rate. D, Oxygen saturation. NPF, neurophysiological stimulation. PM, passive movement. SS, sensory stimulation. Data are means \pm SDs.

Results

A summary of means, standard deviations and significance levels is shown in Table 2 and Figure 2. There was a significant interaction between treatment and time for the multivariate test ($p = 0.04$). With univariate testing, there was a significant interaction between treatment and time for V_E ($F_{(3,36)} = 11.09, p < 0.001$) and SpO_2 ($F_{(3,36)} = 4.77, p = 0.03$), whereas there were no significant interactions with V_R ($F_{(3,36)} = 1.15, p = 0.33$) or V_T ($F_{(3,36)} = 1.43, p = 0.26$).

Neurophysiological facilitation increased V_E ($t_{(12)} = 4.86, p < 0.001$) compared with initial levels, an average of 14.6% compared with control levels. There was also an increase in SpO_2 ($t_{(12)} = 2.98, p = 0.01$) post neurophysiological facilitation, an average increase of less than 1%, however there was no significant effect on V_T ($t_{(12)} = 2.34, p = 0.04$). Similarly, there was an increase in

V_E ($t_{(12)} = 5.33, p < 0.001$) and SpO_2 ($t_{(12)} = 3.34, p = 0.006$) with passive movement. The mean increases in V_E were 9.6% above control levels and a mean increase of 1.1% in SpO_2 . In contrast, following sensory stimulation, there was only an increase in SpO_2 ($t_{(12)} = 3.34, p = 0.006$), a similar mean increase of 1.1% to control levels.

Discussion

The results of this study indicate that neurophysiological facilitation can increase ventilation of patients with decreased consciousness (Figure 2B). The lack of improvement in V_T and V_E following sensory stimulation indicates that the increases in lung volumes following neurophysiological facilitation are from mechanisms independent of the tactile stimulation inherent in the technique.

The significant increase in V_E following the

neurophysiological facilitation found in this study supports Bethune's clinical observations which, to our knowledge, have not previously been objectively tested. Bethune (1975) had proposed that the mechanism of increased respiration with intercostal stretching was by stimulation of a stretch reflex of the intercostal muscle (Eklund et al 1964). The suggested action of perioral stimulation was the stimulation of a primitive sucking reflex which stimulates the respiratory centre (Pieper 1963). The present study, while establishing the effectiveness of this technique, does not explain the mechanism. This could be the basis of further study.

There was an increase in V_E following neurophysiological facilitation with no change in V_R and a non significant increase in V_T . Although there was no statistically significant increase in V_T with neurophysiological facilitation, there was a trend for increasing V_T post neurophysiological facilitation, with a mean increase of 16.3% compared with control values (Figure 2A). In addition, 10 of the 13 subjects demonstrated an increase in V_T post neurophysiological facilitation. However, the significance level for V_T did not reach the Bonferroni adjusted level. This may be a result of the small subject population and variation in subject responses to neurophysiological facilitation.

The mean increase in V_E with neurophysiological facilitation was 14.6% compared with control values post intervention and following passive movement V_E was 9.6% higher compared with control levels. In spirometry analysis, a clinically significant level of improvement is defined as 12% (American Thoracic Society 1995), although similar values for changes in V_E have not been reported to our knowledge. The reported increase in V_E following three minutes of neurophysiological facilitation is above the American Thoracic Society guidelines of 12%, and thus may be of clinical significance.

Passive movements produced an increase in V_E . Although the main aim of passive movements in this population is to prevent muscle contracture (Ada et al 1990), increases in ventilation are also believed to occur (Ishida et al 1994). Guyton (1991) hypothesised that passive movement increases ventilation as peripheral proprioceptive activity stimulates the respiratory control centre. Previous studies have reported an increase in lung volume with passive movement, however these studies were performed on normal subjects during sleep (Ishida et al 1993 and 1994) and may not hold true for other populations or clinical situations. This study demonstrated a significant increase in V_E with passive movement, however, as the mean increase was 9.8%, there is limited clinical significance of these findings, unlike the V_E increase post neurophysiological facilitation.

Oxygen saturation increased during the three interventions of passive movement, neurophysiological facilitation and sensory stimulation. However as the mean increase was from 96.4% to 97.4%, this is of dubious clinical significance. The region of the haemoglobin dissociation

curve from 96% to 100% is nearly flat with limited improvement in O_2 saturation possible, and so these changes in SpO_2 would not have increased the partial pressure of oxygen within the blood by more than 2.5% (Sherwood 1993).

Although the inclusion criteria required subjects to have tracheostomies, this was purely in order to attach the pneumotach for an accurate recording of ventilation. It is expected that these results could be extrapolated to unconscious and semi-conscious patients with no definitive airway. Indeed they would be of great benefit in this population, as the choices of intervention strategies are more limited in a patient with no airway, for example, manual hyperinflation is not possible.

This study demonstrates an improvement in short term ventilation, clinically significant levels for neurophysiological facilitation only, not with sensory stimulation or passive movement. Some limitations of the study include the small sample, ceiling effect of the SpO_2 measurements and the lack of follow-up respiratory measurements to see the duration of the observed increases in V_E . The long-term effects on pulmonary morbidity, prevention of infection and resolution of atelectasis were not addressed in the current study and could be the basis of future trials. In addition, further study is needed to determine the optimal duration of neurophysiological facilitation and passive movement techniques. However, this study demonstrates that neurophysiological facilitation can improve the short term ventilation of patients with reduced consciousness following neurological injury and provides preliminary support to the use of neurophysiological facilitation as a technique to improve short term ventilation.

Footnotes (a) Ventrak™ paediatric/adult flow sensor Model 7222; (b) Ventrak™ Model 1550 Novamatrix Medical Systems Inc. Wallingford, Connecticut USA; (c) Pulse Oximeter, Criticare Systems, Model 503, Hewlett Packard USA.

Correspondence Angela Chang, Physiotherapy Department, The University of Queensland, Queensland 4072. E-mail: a.chang@shrs.uq.edu.au.

References

- Ada L, Canning C and Paratz J (1990): Care of the unconscious head-injured patient. In Ada L and Canning C (Eds): Key Issues in Neurological Physiotherapy. Oxford: Butterworth Heinemann, pp. 249-289.
- American Thoracic Society (1995): Standardization of spirometry 1994 update. *American Journal of Respiratory and Critical Care Medicine* 152: 1107-1136.
- Bethune DD (1975): Neurophysiological facilitation of respiration in the unconscious adult patient. *Physiotherapy Canada* 27: 241-245.
- Brimioule S, Moraine JJ, Norrenberg D and Kahn RJ (1997): Effects of positioning and exercise on intracranial pressure in a neurosurgical intensive care unit. *Physical Therapy* 77: 1682-1689.

- Chen LM, Martin CM, Keenan SP and Sibbald WJ (1998): Patients readmitted to the intensive care unit during the same hospitalisation: clinical features and outcomes. *Critical Care Medicine* 26: 1834-1841.
- Ciesla N (1996): Chest physical therapy for patients in the intensive care unit. *Physical Therapy* 76: 609-625.
- Demling R and Riessen R (1990): Pulmonary dysfunction after cerebral injury. *Critical Care Medicine* 18: 768-774.
- Eklund G, von Euler C and Rutkowski S (1964): Spontaneous and reflex activity of intercostal gamma motoneurons. *Journal of Physiology* 171: 139-163.
- Escourrou P, Delaperche M and Visseaux A (1990): Reliability of pulse oximetry during exercise in pulmonary patients. *Chest* 97: 635-638.
- Gruen P and Liu C (1998): Current trends in the management of head injury. *Emergency Medicine Clinics of North America* 16: 63-83.
- Guyton (1991): *Textbook of Medical Physiology* (8th ed.) Philadelphia: WB Saunders, pp. 525-535.
- Ishida K, Takaishi T and Miyamura M (1994): Ventilatory responses at the onset of passive movement and voluntary exercise with arms and legs. *Acta Physiologica Scandinavica* 151: 343-352.
- Ishida K, Yasuda Y and Miyamura M (1993): Cardiorespiratory response at the onset of passive leg movement during sleep in humans. *European Journal of Applied Physiology* 66: 506-513.
- Nyarwaya JB, Maziot JA and Samii K (1994): Are pulse oximetry and end tidal CO₂ tension monitoring reliable during laparoscopic surgery? *Anaesthesia* 49: 775-778.
- Pieper A (1963): *Cerebral Function in Infancy and Childhood*. New York: Childhood Consultants.
- Prough DS and Lang J (1997): Therapy of patients with head injuries: Key parameters for management. *Journal of Trauma, Infection and Critical Care* 42: S10-S18.
- Pryor JA and Webber BA (2002): Physiotherapy techniques. In Pryor JA and Prasad SA (Eds): *Physiotherapy for Respiratory and Cardiac Disorders*. (3rd ed.) London: Churchill Livingstone, pp. 161-242.
- Sherwood L (1993): *Human Physiology: From Cells to Systems*. (2nd ed.) Minneapolis: West Publishing Company, pp. 418-467.