THE CARDIOPULMONARY EFFECT OF PASSIVE MOVEMENT

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ABSTRACT: *Eleven articles were reviewed on the cardiopulmonary effects of passive movements. These included two articles on the neurological effects of passive movements. Of the eleven articles, four were considered to have level II evidence in accordance with Sackett's rules of evidence. There was little consensus regarding the rate or duration of passive movements. There were some suggestions that upper limb movement produces a greater ventilatory response than lower limb movement. There was a statistically significant increase (p< 0.05) in minute ventilation when the movement was done at a rate of 40 repetitions per minute or more, but this change may not be clinically significant. Passive movements were not detrimental to neurosurgical patients with a normal or slightly elevated intracranial pressure, although the values of the intracranial pressure were not stated. The studies were limited in that eight of the eleven had small sample sizes and most studies were conducted using normal subjects. Further studies with higher levels of evidence need to be conducted to verify any results reported to date in the literature. Studies that are relevant to clinical practice also need to be conducted in populations such as sedated intensive care patients.*

KEY WORDS: PASSIVE MOVEMENT, CARDIOPULMONARY, PHYSIOTHERAPY.

INTRODUCTION

Passive movements are a routine treatment technique employed by physiotherapists for sedated and/or paralysed patients such as intensive care patients, neurosurgical and neurological patients. Passive movements are defined according to Stedman's medical dictionary (Felsher, 1993) as "a joint affected by the hand of another person, or by mechanical means, without the participation of the subject himself". Hollis (1981) defines passive movement as "anatomical movements performed by a therapist for a patient. They are performed at single joints or at several joints in sequence covering any or all of the joint movements and maintaining muscle length."

Passive movements are used for patients who are unable to move actively and are based on the premise that the movement maintains joint range and improves circulation. The purpose of this literature review was to evaluate the existing evidence in the literature of how the circulatory and ventilatory systems are affected by passive movements. The primary objective of passive movements is to maintain joint range, however it would be useful to know if other benefits or harm occur. If no cardiopulmonary effects are obtained it is reasonable to assume that the passive movements can then be conducted at any rate that does not damage the joint. The standard rate of passive movements done in clinical practice is not documented, but from clinical experience the rate is about 10-20 repetitions per minute.

Positive respiratory effects of passive movements have definite clinical relevance. An increase in tidal volume would decrease the risk of alveolar atelectasis and secondary pulmonary complications. Medical management procedures are in place for mechanically ventilated patients to prevent atelectasis, but little can be done to increase tidal volume in comatose or paralysed patients who are breathing spontaneously.

A literature search was conducted using the Medline database (National Library of Medicine). All English articles were extracted from 1970 onwards. The keywords chosen were passive movements, physiotherapy, physical therapy, cardiopulmonary, ventilation and pulmonary function. References from the articles retrieved were checked for any further relevant articles. The articles retrieved were surveyed for articles relating to passive movements in terms of the definition of passive movements above. The eleven most clinically relevant articles were evaluated. The effect of passive movements on muscle and joint integrity are beyond the scope of this review, but it would be a worthwhile subject for further review.

The objectives of this paper are to determine:
- What rate of passive movements is required to get a cardiopulmonary benefit for patients
- How many repetitions need to be done to see a cardiopulmonary change
- Which joint or joints produce the best cardiopulmonary effects
- If there are any other effects of passive movements of which therapists should be aware.

Each article is evaluated according to Sackett's rules of evidence as cited by Pong Wong (1999) for quality of research and strength of evidence.

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Table 1: A summary of Sackett's rules of evidence

Level	Evidence
I	Randomized control trial with a low false positive (α) and a low false-negative (β)
II	Randomized control trial with a high false positive (α) and/or false-negative (β)
III	Non-randomised concurrent group comparison to contemporaneous patients
IV	Non-randomised historical group comparisons to current groups
V	Case series without controls

Each article is reviewed according to the design of the study, the population, sample size, method of passive movement, rate of passive movement and the number of repetitions.

MANUAL CONTACTS/ MECHANICAL CONTACTS

Passive movements are performed in a number of ways. The most common is manual contact, however mechanical methods are also used (Hollis, 1981).

Narain et al (2001) investigated the cardiopulmonary effects of manual passive movements done at the ankle joint in sixty normal subjects aged 18-30 years. Three procedures were conducted in random order in each subject: passive movement, no movement and cutaneous contact to the foot but no movement at the ankle. There was a significant increase in the respiratory rate from the resting rate in both the cutaneous contact and passive movement trials. However there was no significant difference between the cutaneous contact and the passive movement trial. There was a statistically significant change of 40ml ($p<0.05$) in tidal volume between the cutaneous stimulation and passive movement trial. A ninety-millilitre change in tidal volume was found between the control group and the passive movements trial. It is questionable that such a small change (8% increase) in tidal volume would equate to a clinically significant difference. A physiological sigh is approximately 50% greater than tidal volume and decreases microatelectasis in normal active people (Pierce, 1995). An assumption can then be made that a similar increase would be required in immobile patients. Although the study merits a level I according to Sackett's rules (Pong Wong, 1999), the extrapolation of the statistical significance of the tidal volume is inappropriate to clinical practice. The study was conducted on the ankle of normal awake subjects and therefore the 40ml and 90ml changes may be from voluntary input rather than from the passive movements.

Table 2: Summary of studies and levels of evidence

Author	Sample size (n)	Subjects Age (yrs)	Limb and joint involvement	Position of subject	Rate (rpm)	Duration	Manual or induced	Level of evidence
Ishida et al (1994)	7	Healthy 19-31	UL, LL, combo	Sitting	±60	5 reps, 6 ex	Ropes	IV
Waisbren et al (1990)	9	males, 21-25, healthy	LL	leg ergometer	60	5 min	bike	IV
Nakazona (1985)	4	males healthy	LL	bike, ergometer	30/90/30	4 min	bike	IV
Smith (1976)	17	healthy	UL	supine	±20/min	10 reps/ movement	manual	IV
Gozal et al (2000)	6	CCHS children	LL	sitting	Varied	2-3 min	bike	II
Ishida et al (1993)	5	healthy, asleep	LL knee	supine	±60/min	8 sec	manual	II
Brice et al (1988)	12	healthy	LL	seated	40/min	3 min	Electrical	IV
Brimioulle et al (1997)	65	ICU pts, N and ↑ICP	sh flex, hip flex	supine	20 in 2 min	2min	Manual	IV
Gozal et al (1996)	6	CCHS children	LL	sitting	40-50/min	2-3min	manual/ foot plate	II
Narain et al (2001)	60	healthy	ankle	supine	60/min	2 min	manual	II
Koch et al (1996)	12	6 mech ventilated, 6 spont	all joints	supine	20 or 10 sessions	7 min	manual	IV

CCHS- congenital central hypoventilation syndrome, ICU - intensive care unit, ICP - intracranial pressure, mech - mechanically, spont - spontaneously, UL - upper limb, LL - lower limb, sh - shoulder, flex - flexion

ACTIVE PARTICIPATION

It is difficult to identify the effects of pure passive movements, as most studies used normal subjects (Ishida et al 1994, Waisbren et al 1990, Nakazono 1985, Smith 1976, Brice et al 1988, Narain et al 2001) and some active muscle recruitment may have occurred.

Seven of the eleven studies considered the response to passive movement in normal subjects. In order to establish if cardiopulmonary effects are from passive movements, it is necessary to determine which authors included measures that identify any voluntary movement and eliminate the voluntary component. Three of the studies (Gozal 2000, Ishida et al 1993, Ishida et al 1994) considered whether active movement was minimized or eliminated. For example, Ishida et al (1994) used electromyography to check that minimal muscle contraction of the relevant muscles had occurred.

In the study conducted by Waisbren et al (1990) the authors recognized that there was an active element since subjects were required to stabilize themselves on a cycle ergometer without back support, while their legs were rotated at a rate of 60 rpm.

JOINT INVOLVEMENT

Ishida et al (1994) suggested that movement of the upper limbs at the shoulder might produce a greater ventilatory response due to the attachments of the respiratory accessory muscles. It would therefore be of value to determine if a different response is obtained when different joints are mobilized. If only the proximal joints produce a cardiopulmonary response, then it would be necessary to increase the number of repetitions or the frequency of the passive movements to the relevant joints.

Ishida et al (1994) compared the initial cardiopulmonary response of passive and active movement in the upper and lower limb in seven healthy males (19 - 31 years). Six different exercises were repeated randomly five times, with a three-minute interval between exercises. The six exercises were: active bilateral knee extension and relaxation, active bilateral elbow flexion and extension, active combined leg and arm movement, passive bilateral knee extension and

flexion, passive bilateral elbow flexion and extension, passive combined leg and arm movement. All exercises were done at an estimated rate of sixty repetitions per minute. During the passive movements, an electromyographic electrode was attached to the relevant muscle to ensure that there was minimal muscle contraction.

Minute ventilation (V_e), tidal volume (V_t), end tidal carbon dioxide partial pressure ($P_{et}CO_2$), partial pressure of oxygen (PaO_2) and cardiac output were measured. The authors found that V_e, V_t and expired carbon dioxide increased significantly more with active exercise compared to passive movement. The change in respiratory rate was not significant in active and passive movements, however the results showed that there was a greater response in minute ventilation to arm movements than to leg movements with both passive (legs, 3.1 ± 0.73l/min, arms, 4.57 ± 0.94l/min) and active movement (legs, 5.19 ± 0.79l/min, arms 7.75 ± 1.32l/min), however none were statistically significant. All parameters were monitored for the first four breaths once the movement was initiated and no follow-up measurements were taken after the movements were completed, therefore no subsequent effects were measured. Due to the small sample size (n = 7), the results need further verification by studies with larger samples. They concluded that further studies needed to be conducted to determine whether the upper limb produces a greater cardiopulmonary response compared to the lower limb. The initial ventilatory response to upper limb movement is greater than lower limb movement.

RATE OF MOVEMENT AND NUMBER OF REPETITIONS

There was no uniformity in the rate of passive movement in the studies reviewed and not all investigators included accurate measurements of the rate. The rate varied from ten repetitions per minute to 90 rpm.

The purpose of the study by Smith (1976) was to determine the cardiopulmonary effects of passive movement as done by physiotherapists in clinical practice. Smith (1976) measured the

response of heart rate (HR), respiratory rate (RR) and tidal volume (V_t) in seventeen healthy adults (21-30 yrs), when passive movements were performed to the right upper limb. Ten full range repetitions were carried out on each joint of the right upper limb, at an estimated rate of twenty per minute, and thumb opposition was repeated once with no reason why this was done. There was no significant change in heart rate, respiratory rate or tidal volume either during or after the movements. The results can therefore only measure the total response and not the response of movement at each joint. It is important to note that passive movements done at a slow rate may not produce any cardiopulmonary effect after all the joints have been moved. A faster rate of passive movements or done at a single joint may be indicated to produce a cardiopulmonary effect.

In a study of paediatric patients, Gozal et al (1996) found that passive movement at a rate of 40 rpm or more produced a significant increase in minute ventilation by increasing the respiratory rate (p < 0.05), oxygen consumption (p< 0.02) and expired carbon dioxide in subjects (p < 0.005). This study was done on six children (8-15 years) with congenital central hypoventilation syndrome with a matched control in terms of age and gender. A small convenience sample was used according to the availability of subjects. The method was well described and controlled (level II). An adapted, motorized cycle ergometer was used that was able to rotate at different frequencies up to 60 rpm. Subjects were well supported in the sitting position and were able to familiarise themselves with the equipment. The cycle ergometer was rotated backwards to decrease the active workload. Each subject underwent passive movement of the lower limbs at a rate of 6, 18, 30, 42 and 60 rpm for two to three minutes per level. This was done in a random order and each rate was performed twice. Minute ventilation, V_t, RR, oxygen consumption (VO_2), CO_2, carbon dioxide production (Vco_2), and HR were measured. In both the control and experimental group the V_e remained constant at lower rates of ergometry, while at 40 rpm and above, the

minute ventilation increased ($p<0.005$). This increase in minute ventilation was due to an increase in both respiratory rate and tidal volume. There was a greater rise in respiratory rate in the experimental group. Regardless of the rate at which the ergometer was set, there was no significant change in the $PetCO_2$ of the control group, while the experimental group showed a significant decline ($p<0.001$) in $PetCO_2$. This could be due to the retention of CO_2 from the chronic hypoventilation that was being normalised by activity.

These findings are based on a small number of children aged 8-15 years. The response of passive movements may differ in adults. The findings of the above study suggest that it may be appropriate to set the rate of passive movement greater than 40 rpm, however a rate of 40 rpm or higher may be unrealistic in the clinical setting where the movement is performed manually.

In 1985 Nakazono et al conducted a study on passive movements on four healthy male volunteers (22 - 25 years). Cardiac output (stroke volume and HR), RR, V_t, $PetCO_2$ and $PetO_2$ and V_e were measured while patients' legs were moved on a cycle ergometer. The ergometer was set at a rate of 30 revs/min, 90 revs/min and again at 30 revs/min. Each period of passive movements lasted four minutes. The rate of 30 revs/min was considered as the baseline and 90 revs/min the stimulated rate. The protocol was repeated at least eighteen times with each subject, and a minimum of ten minutes rest between sessions was allowed. The results showed that as soon as the pedaling rate was increased, so did the V_e ($p<0.01$), VO_2 (NS), $P_{et}CO_2$ ($p<0.05$), V_t (NS) and RR ($p<0.05$). The values returned to baseline about one minute after the increased pedaling rate. The cardiac output increased by 11% for the duration of the stimulus.

The above study gives an indication that an increased rate of passive movements may increase the pulmonary response. The subjects were asked not to contribute actively to the leg movement, but this was not confirmed with investigations of muscle activation of the trunk. The subjects were seated on a bicycle where positioning and amount of trunk and arm activation used for stability was not mentioned. There must be some active stabilization occurring in the trunk and arms to stay on a seated bicycle with the legs moving at a rate of 90 revs/min, so these results should be interpreted with caution.

Although the above studies lack information regarding active muscle control while performing passive movements, there is a suggestion that passive movements done at 40 rpm or more may positively change the ventilation.

VENTILATORY AND CARDIAC RESPONSE

The ventilatory response varied from study to study. When the rate was slow (20 per minute) and all joints in the upper limb were involved, no significant changes in the ventilatory or cardiac parameters (RR, V_t and HR) were noted (Smith, 1976).

If the rate, joint moved or method of the movement is altered, a different response is obtained. Volume of expired gas, VO_2 and $P_{et}CO_2$, and HR were monitored in nine healthy male subjects (21 - 25 years) using leg cycle ergometry that was modified to allow for passive movement (Waisbren et al, 1990). The exercise was repeated three times and a mean calculated. A three-minute rest interval was allowed between trials. The ergometer rotated at a rate of 60 revs/min for five minutes followed by three minutes of rest. They found all values were significantly higher ($p<0.05$) during the passive limb movement compared to the resting stage, with the greatest increase in the initial movement.

Ishida et al (1994) found similar results in V_t, $PetCO_2$, PaO_2, cardiac output and respiratory rate when passive movements were done at the same rate to the upper and lower limb, one limb at a time. They noted that the RR was not significantly altered.

It is helpful to compare the response of active exercise to passive movements, to establish the degree of difference. Induced contractions of a muscle still involve contraction of the muscle. Electrically induced leg exercises were performed in twelve volunteers (ten males and two females) seated in a specially designed chair (Brice et al, 1988). Voluntary and induced contractions were produced in the quadriceps muscles, at a rate of forty per minute with the work rate lasting three minutes. The $PaCO_2$ did not change significantly throughout all exercises. The minute ventilation increased with both active and induced exercises, where the frequency contributed more than the tidal volume. This is different to the findings of Ishida et al (1994) where the V_t contributed more to the minute ventilation, but similar to the findings of Ishida et al (1993). The heart rate did not significantly increase in either the active or induced exercises compared to the resting state. The method of Brice et al (1988) was poorly described, unclear and would be difficult to reproduce.

When a cycle ergometer at a rate of 90 revs/min was used for passive movements, minute ventilation and $P_{et}CO_2$ increased significantly, with no significant change in the tidal volume (Nakazono et al 1985). The ventilatory response returned to baseline within one minute of the 90-rpm rate. The cardiac output increased by 11% for the duration of the stimulus.

There appears to be a trend in the respiratory response in that the respiratory rate increases as the rate of the movement increases. The above authors do not seem to find the same result regarding the tidal volume. What can be determined is that the tidal volume will remain the same or increase slightly as found by Narain et al (2001)which was an 8% increase.

The heart rate and cardiac output were the main cardiac parameters that were measured by Waisbren et al (1990), Nakazono et al (1985), Ishida et al (1993). Apart from Smith (1976) where no changes in any parameters were found, there seems to be general consensus that the heart rate increases. Ishida et al (1994) found a significant increase in heart rate in both passive arm and leg movements in the first two breaths, but it had returned to a baseline value within the next two breaths. Once again these values need to be regarded with caution, as they are statistically significant, but not necessarily clinical relevant. If an increase in heart rate is found it could mean that patients who are tachycardic and critically ill, may be compromised by passive

movements. If patients have stagnant hypoxia, a rise in heart rate by passive movements could improve oxygen delivery to the periphery and benefit the patient.

PASSIVE MOVEMENTS DURING SLEEP

Ishida et al (1993) studied the effects of passive movements in sleep and awake states. Five healthy males (19-31 years) had passive movements done to the knee in the supine position, at an estimated frequency of sixty per minute, for about eight seconds (Ishida et al, 1993). The movement was done when subjects were awake, and in deep sleep state. The sleep-state was determined by electro-encephalograph and standard sleep state criteria were met. If the sleep-state altered to a light sleep once the passive movements were initiated, then that set of data was excluded from the study. The aim of the study was to determine if there was an abrupt cardiopulmonary response to passive movements while a person is sleeping, and to correlate the cardiac response to the ventilatory response. Tidal volume, RR, $P_{et}CO_2$ and O_2, CO, HR and stroke volume were measured. They found that the two responses did not correlate and that there was a significant increase in minute ventilation ($p<0.05$) while sleeping, where the respiratory rate contributed more than the tidal volume (both had a $p<0.05$). The tidal volume showed more of an increase in the sleep-state than the awake-state. The $PetCO_2$ did not decrease significantly in the awake or sleep-states. Most patients who require passive movement are sedated or paralysed; therefore it is interesting to note that it is possible that a greater response in tidal volume is obtained from patients who are sleeping.

Gozal et al (2000) studied passive movements in paediatric patients with congenital central hypoventilation syndrome during sleep. A small experimental group of children between the ages of 0.2 and 7 years underwent twelve trials each where they acted as their own controls. The subjects were allowed to fall asleep naturally and no sedation was given. The passive movements were ankle plantar flexion and dorsiflexion moving through a range of approximately

45 degrees. Either the movements were performed manually or using a hydraulic powered footplate that moved the ankle through the range at a rate of 40-50 strokes per minute. No mention is made in the article as to when the foot-plate was used and why. The movements were performed in non-REM sleep. If the subjects were aroused by the experiment then that data set was discarded. Expired carbon dioxide, oxygen saturation, PaO_2, $PaCO_2$ and RR were measured. Sixteen trials needed to be aborted because of EEG changes or behavioural arousal, leaving seventy-four trials for analysis. There was no significant change in the oxygen saturation levels throughout any of the trials, but each subject received supplemental oxygen so no significant change was expected. The respiratory rate increased ($p< 0.0001$) during the passive movements however the authors do not state if any residual effects post movements were monitored. The study did not include the measurement of tidal volume.

The results of the above two studies are relevant clinically as most of the patients where passive movements are indicated are sedated. Although the pathology is different, it does give an indication that similar cardiopulmonary responses could occur when a person is sleeping.

NEUROLOGICAL RESPONSE

Two studies were found which investigated the effect of passive movements on intracranial pressure (ICP) in neuro-surgical patients. Identification of any detrimental effects in patients with raised intracranial pressure while passive movements are being performed needs to be established to minimise secondary brain injury from occurring.

Brimioulle et al (1997) studied the effects of passive movements on sixty-five patients in the Neuro-Intensive Care Unit (NICU) with normal to high ICP. Passive shoulder flexion from neutral to 150-180 degrees and hip flexion up to 90-150 degrees were carried out on one upper limb and one lower limb in random order. These were done with all patients who were unable to perform active exercises. Each movement was repeated twenty times in two minutes. The exer-

cises included shoulder flexion, full hip flexion and extension with associated knee flexion and extension, end of range knee extension and small range straight leg raises. The results showed a non-significant increase in heart rate (97bpm to 103bpm) with passive movements and no effect on the systemic arterial blood pressure. The passive movements conducted on subjects with a normal ICP did not significantly affect the ICP. Patients who had an increased ICP showed a decrease in ICP and an increase in the cerebral perfusion pressure during movements. Some patients with high ICP showed a suppression of abnormal intracranial waves and an improvement in their level of consciousness. Passive movement is, therefore, safe with patients who have a normal (below 15 mmHg) or slightly elevated ICP (value not stated).

Koch et al (1996) studied patients (24 - 73 years) with ICP monitors in situ who underwent passive range of movement. Six patients were mechanically ventilated and six patients were breathing spontaneously. The spontaneously breathing patients had ten sessions of passive movement, while the ventilated patients received twenty sessions (the reason for doing this was not stated). Mean arterial pressure, intracranial pressure, heart rate and respiratory rate were measured at one-minute intervals for the duration of the exercise. The passive range of movements included flexion and extension of the hips, knees, ankles, shoulders, elbows and wrists plus abduction and adduction where appropriate and truncal rotation. The subjects included any patients with an ICP less then 20 cmH_2O for 30 minutes prior to the passive movement. The average duration of treatment was approximately seven minutes. The study showed that there were no significant effects on any of the above-mentioned values with ventilated patients. There were non-significant increases in heart rate, mean arterial pressure and cerebral perfusion pressure at the end of the movements for the spontaneously breathing patients.

The movements were poorly described as to the degree of range of movement, the rate at which they were performed and the technique used to perform the passive movements. Without this infor-

mation, it is difficult to determine if effects may have been found if the rate were faster or the range was bigger. The authors concluded that passive movement is safe in stable neurological patients with normal ICP. This is an assumption made from a sample size of six and therefore cannot be conclusive.

The studies conducted on neurologically impaired patients showed that passive movements are safe and not detrimental to patients with normal or slightly elevated ICP. Further investigation is required to determine at what level of ICP it is safe for passive movements to be conducted.

CONCLUSION

Passive movements are a routine physiotherapy intervention. The research to substantiate the claims of improving circulation and ventilation is extremely varied and conclusions are drawn from small sample sizes. No conclusion can be reached regarding the cardiopulmonary effect of passive movements, although there does seem to be a trend of increases in minute ventilation and heart rate. It is questionable as to whether these small increases would be significant clinically. The research does suggest that passive movements conducted at a rate greater than forty repetitions per minute will produce an increase in minute ventilation. There is also a suggestion that the shoulder will result in the best cardiopulmonary benefit. How many repetitions required of each joint has not been established. The highest level of evidence in these studies, according to Sackett's rules of evidenceas cited by Pong Wong (1999), is a level II (see table 1). Randomised controlled trials need to be conducted to clarify some of the ambiguities found in the present studies. Active movements provide a far greater cardiopulmonary response compared to passive movements (Ishida et al 1994) and therefore active movements should always be the movement of choice, if this is possible.

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