

Short-term effects on outcomes related to the mechanism of intervention and physiological outcomes but insufficient evidence of clinical benefits for breathing control: a systematic review

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Questions: What is the volume, quality, consistency, and generalisability of the evidence for breathing control? What is the effect on outcomes related to the target and mechanism of breathing control, as well as physiological and clinical outcomes? **Design:** Systematic review with meta-analysis. **Participants:** People with chronic respiratory disease, post-surgical, or asymptomatic individuals. **Intervention:** Breathing control (relaxed basal, diaphragmatic, or abdominal breathing) as the sole intervention. **Outcome measures:** All outcome measures providing continuous data. **Results:** Twenty studies were included within the meta-analysis. A beneficial effect was found for abdominal movement (SMD 1.36, 95% CI 0.42 to 2.31), diaphragm excursion (SMD 1.39, 95% CI 1.00 to 1.77), respiratory rate (SMD -0.84, 95% CI -1.09 to -0.60), tidal volume (SMD 0.98, 95% CI 0.71 to 1.25), arterial oxygen saturation (SMD 0.63, 95% CI 0.25 to 1.02) and percutaneous oxygen (SMD 1.48, 95% CI 0.85 to 2.11). Breathing control had a detrimental effect on the work of breathing (SMD 1.06, 95% CI 0.52 to 1.60) and dyspnoea (SMD 1.47, 95% CI 0.88 to 2.05). **Conclusion:** When used as a sole intervention, there was a beneficial effect on outcomes related to the mechanism of breathing control as well as on short-term physiological outcomes. In people with severe respiratory disease, breathing control resulted in a detrimental effect on dyspnoea and work of breathing. There was no clear evidence of an effect on ventilation or long-term physiological outcomes related to gas exchange or the energy cost of breathing. Overall, evidence was satisfactory with studies demonstrating poor consistency, good generalisability, and satisfactory volume and quality. [Lewis LK, Williams MT, Olds T (2007) Short-term effects on the mechanism of intervention and physiological outcomes but insufficient evidence of clinical benefits for breathing control: a systematic review. *Australian Journal of Physiotherapy* 53: 219–227]

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Introduction

Breathing control is a therapeutic technique commonly used in physiotherapy practice to manage breathlessness. This technique is also known as relaxed basal breathing, diaphragmatic breathing, and abdominal breathing. Breathing control is defined as tidal breathing using the lower chest with relaxation of the upper chest and shoulders (Pryor et al 2002). During breathing control, the patient is encouraged to predominantly move the abdominal wall during inspiration while simultaneously reducing upper rib cage motion and accessory muscle use (Gosselink 2004).

The reported therapeutic goals of breathing control include correcting abnormal chest motion, reducing the work of breathing and sensation of dyspnoea, improving the efficiency of breathing, and altering the distribution of ventilation (Miller 1954, Cahalin et al 2002). A variety of mechanisms have been suggested as the means by which breathing control achieves these goals. The simplest of these proposes that increases in tidal volume result in a reduction in respiratory rate (Tucker and Jenkins 1996, Cahalin et al 2002). In people with chronic obstructive pulmonary disease (COPD), Gosselink (2004) proposed that breathing exercises sought to relieve dyspnoea by increasing the strength and endurance of the respiratory muscles, optimising the thoracoabdominal respiratory pattern and reducing dynamic hyperinflation of the rib cage, and ultimately improving gas

exchange. Breathing control has also been advocated as a technique to improve excursion of the diaphragm (Blaney and Sawyer 1997) and thus potentially alter the distribution of ventilation and lung volumes following abdominal surgery. Previous literature regarding breathing control has raised questions about the effect of the technique (Cahalin et al 2002). A number of authors report improvements in tidal volume and respiratory rate with breathing control (Miller 1954, Sackner et al 1974, Sackner et al 1984a) while other studies have reported detrimental effects (Gosselink et al 1995, Vitacca et al 1998).

The difficulty in determining which outcomes should be considered in reviewing the literature associated with a technique such as breathing control stems from the ambiguity surrounding procedural, physiological, and clinical outcomes. Clinically, a therapist-guided change in breathing is thought to alter respiratory mechanics and associated physiology, leading to a beneficial effect on symptoms. It is likely that studies investigating the effect of interventions such as breathing control report specific outcomes at one or more points in this sequence. For this review the effect of breathing control was examined for outcomes related to the target and mechanism of the intervention, as well as for physiological and clinical outcomes (Figure 1).

To date, reviews of the literature concerning breathing control have based their conclusions on findings presented

Box 1: LOW* critical appraisal tool.

| | |
|--|--|
| 1. Did the study address a clearly focused issue? <ul style="list-style-type: none"> • the population studied • the intervention / outcome studied • whether the study tried to detect a beneficial or harmful effect | Yes No Can't tell |
| 2. Were the participants recruited in an acceptable way? <ul style="list-style-type: none"> • Were the eligibility criteria clearly specified so that the participant recruitment could be repeated? • Were the participants representative of a defined population? • How likely was the recruitment process to introduce bias? (participants likely to respond positively or negatively to intervention) | Yes No Can't tell |
| 3. Was there a sufficient number of participants selected? <ul style="list-style-type: none"> • Was there a power calculation? • Did the authors provide any justification for the sample size? | Yes No Can't tell |
| 4. Was there a separate control group? | Yes (go to 5) No (go to 6) |
| 5. Separate control group: <ul style="list-style-type: none"> • Was there equal chance of participants being allocated to either group? • Were the controls representative of the intervention group (similar age, gender and variables other than the variable of interest)? • Were the eligibility criteria clearly specified so that the recruitment of the controls could be repeated? • Was there a sufficient number of controls selected? Go to 7 | Yes No Can't tell |
| 6. Baseline measures for participants acting as their own controls: Where appropriate: <ul style="list-style-type: none"> • Was the baseline stable (how confident are you that the pre measures were stable, was there a run-in period?) • Was the order of interventions randomised? • Was the washout period between intervention/control acceptable? | Yes No Can't tell |
| 7. Were the outcomes measured accurately to minimise bias? <ul style="list-style-type: none"> • Are there references to support the use of outcome measures? (details, reliability and validity of measures) • Were the measurement methods similar / the same in participants and controls? | Yes No Can't tell |
| 8. Have the confounding factors been accounted for? <ul style="list-style-type: none"> • Do the authors state potential confounding variables? • Do the authors discuss and refute the impact of potential confounding variables? • Have the authors taken account of the potential confounding factors in the design, results and / or in the analyses? | Yes No Can't tell |
| 9. Results a. Were the results presented so the effect size was shown or could be calculated? <ul style="list-style-type: none"> • Are mean / SD (or the raw data) available to allow calculation of effect size • Size of the <i>p</i> value • Size of the confidence intervals • Are data for participant attrition /withdrawal presented? b. Do you subjectively believe the results? <ul style="list-style-type: none"> • NOT do you accept the results? • What are the bottom line results? • Was the analysis appropriate to the design? | Yes No Can't tell Yes No Can't tell |
| Final score | / 9 |

*LOW = Lewis, Olds, Williams. Yes = 1, No = 0, Can't tell = 0

in previous narrative reviews, descriptive papers, and uncontrolled experimental studies. Conventionally, systematic reviews of effect pose a 'population-intervention-comparator-outcome (PICO)' question, which specifies and limits the context of the evidence. This systematic review sought to globally review all primary data from experimental studies of breathing control prospectively in order to assess the body of evidence for the intervention. A modified body of evidence matrix proposed by the National Health and Medical Research Council (2005) was used to assess and summarise the volume, quality, effectiveness, and consistency of the evidence underpinning breathing control. The primary questions of this systematic review were:

1. What is the volume, quality, consistency, and generalisability of the evidence for breathing control?
2. What is the effect on outcomes related to the target and mechanism of breathing control as well as on physiological and clinical outcomes?

Method

Identification and selection of studies

Searches were conducted of AMED (1985 to October 2006), CINAHL (1982 to October week 1 2006), MEDLINE (1966 to September week 4 2006), Scopus (to October 2006), Web of Science (all citation databases 1993 to 2006) and the Cochrane Library (all of the Cochrane Library). The search strategy (Appendix 1, see eAddenda) included all commonly used terms for the intervention of breathing control and a reference to the type of therapy. Titles and abstracts (where available) were displayed and screened to identify relevant studies. Full paper copies of studies were obtained and their reference lists screened. The reference lists of any additional studies were searched again manually to ensure completeness. Researchers involved in the area were contacted for additional studies.

Initially citations were identified which indicated explicitly that at least one treatment group had received the sole intervention of breathing control (or appropriate alternative terms) within the title or abstract. One reviewer (LL) screened the initial search results for potentially eligible studies, and two reviewers (LL, MW) independently reviewed these search results for eligibility. The investigators resolved disagreements by consensus. All identified studies were then retrieved in full, as were any studies where the abstract was unavailable or where ambiguity existed. To be included in the review, studies were required to use an experimental design, report primary original data concerning the therapeutic technique of breathing control, and be published in English. There were no publication year limits.

Description of studies

Quality and volume of evidence: Study quality was assessed using a custom-made tool (Box 1). The presence or absence of nine methodological criteria was assessed by two independent reviewers (LL, MW) with a maximum possible score of nine. Criteria were assessed using only the documentation provided in the publication. Disagreements were resolved by a third independent reviewer (TO). Following critical appraisal, two reviewers then independently allocated each study to a level of evidence using the hierarchy proposed by Lloyd-Smith (1997) (Box 2).

Generalisability of evidence: Studies including people with chronic respiratory conditions where breathlessness

was likely to be a feature were considered to represent the population where breathing control is commonly indicated. Generalisability of the body of evidence for breathing control was determined by calculating the percentage of studies that included participants with chronic respiratory diseases. The generalisability was classified as good if 75% of the studies included participants with chronic pulmonary conditions, fair (50–75%), and poor (< 50%).

Participants: There were no restrictions placed upon the age range of participants (children or adults) or symptoms (asymptomatic and participants with acute or chronic medical, surgical or traumatic conditions). Animal studies were excluded.

Intervention: Breathing control was required to be the sole intervention rather than performed in conjunction with other therapeutic techniques. Possible comparisons included breathing control with a control or another intervention.

Outcome measures: All outcomes were recorded.

Data analysis

The relevant details were extracted from the included studies by two reviewers (LL and MW). Information about the method (design, participants, intervention, outcome measures) and results (sample size, means and standard deviations) were extracted. Data for all outcome measures reported by authors or subsequently calculated by the reviewers from published data were extracted.

Outcome measures which used continuous scales of measurement (interval, ratio) only were included within this review. Data were entered into the Cochrane Collaboration's Review Manager Software^(a) to enable calculation of pooled estimates of the effect of breathing control using a fixed effects model.

Where studies reported similar domains of measurement but used different measurement instruments (eg, breathlessness measured by Borg's perceived rate of exertion scale, or visual analogue scale for breathlessness), data were grouped via domain to permit calculation of pooled estimates. The overall effect of the intervention for each outcome was reported as SMD (95% CI) since different metrics were used for some outcome variables. Heterogeneity of studies was determined for each outcome measure using the chi-squared statistic. Where significant heterogeneity existed ($p < 0.05$), studies and data were reviewed by the research team in order to identify possible sources of variation.

Results

Identification and selection of studies

Fifty-seven studies were retrieved from the search. No extra studies were identified through contacting experts in the cardiorespiratory physiotherapy field. Twenty studies met the inclusion criteria (see Table 1 on the eAddenda for details of the studies). All of these studies were published in full with publication dates ranging from 1954 to 2003. Among the 20 included studies, 285 participants were involved, with sample sizes in individual studies ranging from six to 33 participants.

Thirty-seven studies were excluded from the review. Of these, 18 were narrative reviews, three were descriptive papers, three were letters, two were guidelines, one was a case report, and one a consensus statement. Four studies

Box 2. Hierarchy of evidence ranking system (Lloyd-Smith 1997).

- | | |
|----|---|
| 1a | Meta-analysis of randomised controlled trials |
| 1b | One individual randomised controlled trial |
| 2a | One well-designed, non-randomised controlled study |
| 2b | Well-designed quasi-experimental study |
| 3 | Non-experimental descriptive studies – comparative/case studies |
| 4 | Respectable opinion |

were excluded because breathing control was performed in conjunction with other interventions. Five studies were experimental in design but reported insufficient data to enable the calculation of means and standard deviations, and subsequently allow for meta-analysis (Cole et al 1962, Hughes 1979, Sackner et al 1984b, Girodo et al 1992, Bell and Saltikov 2000).

Description of studies

Volume and quality of evidence: The volume of the evidence was assessed using the Lloyd-Smith (1997) hierarchy of evidence ranking system (Box 2). No studies fulfilled the criterion for the highest level of evidence (1a). Two studies were classified as Level 1b (McNeill and McKenzie 1955, Wiens et al 1999) and three as Level 2a (Sackner et al 1974, Brach et al 1977, Sackner et al 1984a). The remaining 15 studies were classified as Level 2b.

Methodological quality of the studies ranged from 3 (Campbell and Friend 1955) to 8 points (Blaney and Sawyer 1997, Wiens et al 1999, Jones et al 2003) as shown in Table 2 (note: a maximum score of nine was possible only for studies with a separate control group). The most common methodological issues were failure to report eligibility criteria so that participant recruitment could be repeated (14 studies), failure to justify sample size (18 studies), absence of a separate control group (15 studies), and of the five studies that did have a separate control group only one provided suitable justification for participant allocation to that group (Wiens et al 1999). Ten of the 15 studies without a separate control group did not report whether the order of interventions was randomised or provide information regarding duration of the washout period or stability of baseline measures.

Generalisability of evidence: Eighty per cent of the included studies included participants with chronic respiratory conditions likely to feature breathlessness. Of these studies, 56% (n = 9) recruited participants with COPD of severe or moderate severity. The remaining 20% of studies included participants with conditions that were not likely to feature breathlessness. Two of these studies recruited people following surgery (cholecystectomy, upper abdominal surgery), one recruited people asymptomatic for respiratory conditions, and one recruited people with chronic progressive multiple sclerosis. Therefore the generalisability was classified as good, with 80% of the included studies carried out on chronic respiratory conditions (emphysema, COPD, asthma, bronchitis, bronchiectasis, pulmonary fibrosis) (see Table 1 on the eAddenda).

Participants: Of the nine studies that investigated breathing control in people with COPD, seven reported disease

severity as a percentage of the forced expiratory volume in one second (FEV₁) in accordance with The American Thoracic Society COPD severity classification (2004): mild ≥ 80 , moderate 50–80, severe 30–50, very severe < 30 . The severity of COPD in participants in five of the studies was classified as severe (Willeput et al 1983, Gosselink et al 1995, Vitacca et al 1998, Ito et al 1999, Jones et al 2003), and in two studies as moderate (Grimby et al 1975, Sackner et al 1984a).

Intervention: Eight of the included studies investigated the impact of breathing control programs with training durations ranging between three and 12 weeks (Becklake et al 1954, Miller 1954, Campbell and Friend 1955, McNeill and McKenzie 1955, McKinley et al 1961, Williams et al 1982, Gosselink et al 1995, Wiens et al 1999). The remaining 12 studies investigated breathing control compared with natural or spontaneous breathing (See Table 1 on the eAddenda).

Outcome measures: A total of 20 outcome measures provided sufficient data to permit meta-analysis. The most commonly reported outcomes were respiratory rate (n = 10), tidal volume (n = 10), and ventilation (n = 8). The results have been grouped according to whether outcome measures reflect key targets or mechanisms (short and long term) of breathing control, or physiological (energy cost/gas exchange) and clinical outcomes (reduce breathlessness). Effect estimates are summarised in Figure 1 and Table 3 (see eAddenda for Table 3).

Effect on outcomes related to the target of intervention

Abdominal excursion: One study reported the impact of breathing control on abdominal excursion (Sackner et al 1974) in 11 participants with chronic respiratory disease. Breathing control resulted in a significant increase in abdominal excursion when compared with natural breathing (SMD 1.36, 95% CI 0.42 to 2.31).

Diaphragm excursion: Four studies involving 72 participants reported diaphragm excursion during the intervention of breathing control (Miller 1954, Sinclair 1955, Chuter et al 1990, Blaney and Sawyer 1997). Breathing control resulted in a significant increase in diaphragm excursion (SMD 1.39, 95% CI 1.00 to 1.77) with the studies demonstrating significant heterogeneity ($p < 0.0001$). Two of these studies (Chuter et al 1990, Blaney and Sawyer 1997) were conducted on post surgical patients while the remaining two studies were on patients with chronic pulmonary disease. When considered as two separate groups (post surgical and chronic pulmonary disease) for the analyses, the studies were still shown to be heterogeneous ($p < 0.05$). This could be explained by the diverse patient populations, intervention type, and duration (short term breathing control versus prolonged training programs) in the different studies.

Short-term effect on outcomes related to mechanism of intervention

Respiratory rate: Ten studies involving 151 participants reported respiratory rate (Miller 1954, Campbell and Friend 1955, Sackner et al 1974, Grimby et al 1975, Willeput et al 1983, Sackner et al 1984a, Gosselink et al 1995, Vitacca et al 1998, Ito et al 1999, Jones et al 2003). The intervention significantly decreased respiratory rate (SMD -0.84 , 95% CI -1.09 to -0.60). The 10 studies were significantly heterogeneous ($p = 0.002$). When one low quality (3 out

Table 2. Methodological quality of the 20 included studies based on the LOW critical appraisal tool.

| Study | LOW item | | | | | | | | | | Final score |
|-------------------------|----------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9a | 9b | |
| Becklake et al 1954 | yes | | | yes | | | yes | yes | yes | yes | 6 |
| Blaney & Sawyer 1997 | yes | yes | yes | | | yes | yes | yes | yes | yes | 8 |
| Brach et al 1977 | yes | | | yes | | | yes | yes | yes | yes | 6 |
| Campbell & Friend 1955 | yes | | | | | | yes | | yes | | 3 |
| Chuter et al 1990 | yes | | | | | | yes | yes | yes | yes | 5 |
| Gosselink et al 1995 | yes | yes | | | | yes | yes | yes | yes | yes | 7 |
| Grimby et al 1975 | yes | | | | | | yes | yes | yes | yes | 5 |
| Ito et al 1999 | yes | yes | | | | yes | yes | yes | yes | yes | 7 |
| Jones et al 2003 | yes | yes | yes | | | yes | yes | yes | yes | yes | 8 |
| McKinley et al 1961 | yes | | | | | yes | yes | yes | yes | yes | 6 |
| McNeill & McKenzie 1955 | yes | | | yes | | | yes | yes | yes | yes | 6 |
| Miller 1954 | yes | | | | | | yes | yes | yes | yes | 5 |
| Sackner et al 1974 | yes | | | | | | yes | | yes | yes | 4 |
| Sackner et al 1984a | yes | | | yes | | | yes | yes | yes | yes | 6 |
| Shearer et al 1972 | yes | | | | | | yes | yes | yes | yes | 5 |
| Sinclair 1955 | yes | | | | | | yes | | yes | yes | 4 |
| Vitacca et al 1998 | yes | yes | | | | | yes | yes | yes | yes | 6 |
| Wiens et al 1999 | yes | yes | | yes | yes | | yes | yes | yes | yes | 8 |
| Williams et al 1982 | yes | | | | | | yes | yes | yes | yes | 5 |
| Willeput et al 1983 | yes | | | | | | yes | | yes | yes | 4 |

of 9 points) study (Campbell and Friend 1955) which included people with emphysema of unknown severity was excluded from the analysis, breathing control still decreased respiratory rate significantly (SMD -0.75 , 95% CI -1.00 to -0.51) and the studies were homogenous ($p = 0.07$).

Tidal volume: Ten studies involving 127 participants reported tidal volume (Miller 1954, Campbell and Friend 1955, Sackner et al 1974, Grimby et al 1975, Brach et al 1977, Willeput et al 1983, Sackner et al 1984a, Gosselink et al 1995, Vitacca et al 1998, Ito et al 1999). Breathing control significantly increased tidal volume (SMD 0.98, 95% CI 0.71 to 1.25) though significant heterogeneity existed between studies ($p = 0.0001$).

Three of these studies used pre/post designs to investigate the impact of prolonged breathing control training (Miller 1954, Campbell and Friend 1955, Gosselink et al 1995), while the remaining studies reported tidal volumes during the breathing control technique compared with spontaneous breathing. When the studies were divided into these two groups, breathing control significantly increased tidal volume following training programs (SMD 1.23, 95% CI 0.75 to 1.70) and when compared to spontaneous breathing (SMD 0.86, 95% CI 0.52 to 1.19). Significant heterogeneity existed between studies that compared tidal volume during breathing control with spontaneous breathing ($p = 0.0002$) while the three studies in the post breathing control training group were homogenous ($p = 0.06$).

Four studies included participants with severe COPD, two studies included participants with moderate disease severity, and in four of the studies the severity of disease was not defined. When studies including people with severe COPD were excluded from the analysis, breathing control increased tidal volume significantly (SMD 0.98, 95% CI

0.61 to 1.34) and the studies were homogenous ($p = 0.09$). Breathing control resulted in significant increases in tidal volume (SMD 0.98, 95% CI 0.57 to 1.39) for the four studies including people with severe COPD, but the studies demonstrated significant heterogeneity ($p < 0.0001$).

Ventilation: Eight studies involving 104 participants reported ventilation (measured in litres per minute) (Miller 1954, Grimby et al 1975, Brach et al 1977, Willeput et al 1983, Sackner et al 1984a, Gosselink et al 1995, Vitacca et al 1998, Ito et al 1999). Breathing control had no significant effect (SMD 0.17, 95% CI -0.11 to 0.44) and studies were homogenous ($p = 0.05$).

Long-term effect on outcomes related to mechanism of intervention

Vital capacity: One study involving 22 participants with emphysema reported the impact of breathing control training over a prolonged period on vital capacity (Sinclair 1955). There was no significant effect of the intervention on vital capacity (SMD 0.26, 95% CI -0.33 to 0.86).

Forced vital capacity (FVC): One study involving eight participants with chronic obstructive bronchitis reported FVC (Williams et al 1982) before and after a three week breathing control program. No significant effect was calculated (SMD 0.18, 95% CI -0.80 to 1.17).

Expiratory flow rate: McNeill and McKenzie (1955) reported expiratory flow rate in 33 pulmonary outpatient participants following a four week breathing control program compared to a separate control group. No significant effect was calculated for expiratory flow rate (SMD 0.27, 95% CI -0.47 to 1.02).

Forced expiratory volume in one second (FEV₁): One

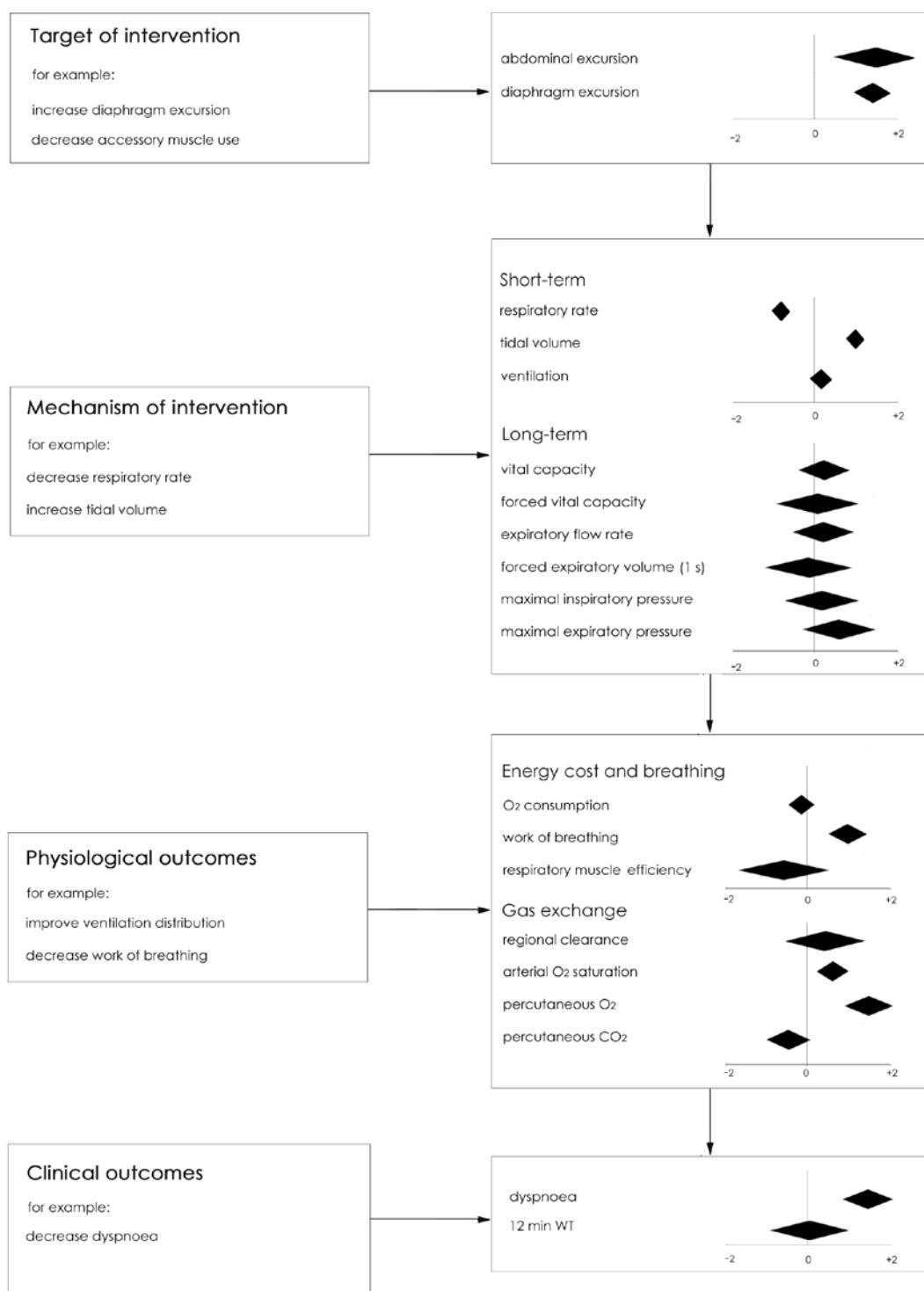


Figure 1. Examination of the effect (SMD, 95% CI) of breathing control on all outcomes by pooling data from all included trials. Effects to the right of zero favour breathing control except for respiratory rate, work of breathing, percutaneous CO₂ and dyspnoea.

study involving eight participants with chronic obstructive bronchitis reported FEV₁ (Williams et al 1982) before and after a three week breathing control program. No significant effect was found (SMD -0.18, 95% CI -1.16 to 0.81).

Respiratory muscle strength: Wiens et al (1999) reported respiratory muscle strength in 19 people with chronic progressive multiple sclerosis following 12 weeks of instruction in music therapy with an emphasis on diaphragmatic breathing compared to a control group. There

was no significant effect in the intervention group compared with the control for either maximum inspiratory pressure (SMD 0.28, 95% CI -0.63 to 1.18) or maximum expiratory pressure (SMD 0.66, 95% CI -0.27 to 1.59).

Effect on physiological outcomes of energy cost and breathing

Oxygen consumption (VO₂): Three studies involving 53 participants with chronic respiratory disease reported oxygen consumption during and/or directly following

breathing control (Gosselink et al 1995, Ito et al 1999, Jones et al 2003). Breathing control had no significant effect on oxygen consumption across the three studies (SMD -0.17, 95% CI -0.56 to 0.21) and the studies were homogenous ($p = 0.14$).

Work of breathing: Two studies involving 31 participants with chronic respiratory disease reported the impact of breathing control on the work of breathing measured in joules per litre (McKinley et al 1961, Vitacca et al 1998). Overall, breathing control significantly increased the work of breathing (SMD 1.06, 95% CI 0.52 to 1.60). Studies were homogenous ($p = 0.11$).

Respiratory muscle efficiency: One study involving seven participants with severe respiratory disease reported respiratory muscle efficiency during spontaneous breathing and breathing control following a three week breathing control program (Gosselink et al 1995). Respiratory muscle efficiency was determined through analysis of measures of minute ventilation, carbon dioxide production, oxygen consumption, and inspiratory and expiratory pressures (Gosselink et al 1995). No significant effect was found (SMD -0.62, 95% CI -1.70 to 0.46).

Effect on physiological outcomes of gas exchange

Ventilation distribution (regional clearance): Shearer et al (1972) reported regional clearance of nitrogen from the lungs in eight participants asymptomatic for respiratory disease. There was no significant effect of breathing control on regional clearance (SMD 0.41, 95% CI -0.58 to 1.41). Three other studies also investigated the impact of breathing control on regional clearance (Sackner et al 1974, Grimby et al 1975, Brach et al 1977) but did not report sufficient data to enable inclusion within the analyses. All of these studies reported no significant difference in regional clearance during the intervention of breathing control.

Arterial oxygen saturation (SaO_2): Three studies involving 55 participants with chronic respiratory disease reported the effect of breathing control on arterial oxygen saturation (Becklake et al 1954, Miller 1954, Vitacca et al 1998). Breathing control resulted in a significant increase in arterial oxygen saturation (SMD 0.63, 95% CI 0.25 to 1.02). Studies were homogenous ($p = 0.39$).

Percutaneous gases: One study involving 25 participants with chronic respiratory disease reported the effect of breathing control and natural breathing on percutaneous oxygen and carbon dioxide (Vitacca et al 1998). A significant increase in percutaneous oxygen (SMD 1.48, 95% CI 0.85 to 2.11) occurred, without a significant effect on percutaneous carbon dioxide (SMD -0.43, 95% CI -0.99 to 0.13).

Effect on clinical outcomes

Dyspnoea: Two studies involving 33 participants with chronic respiratory disease reported the effect of breathing control on dyspnoea (Williams et al 1982, Vitacca et al 1998). Breathing control was shown to increase dyspnoea significantly (SMD 1.47, 95% CI 0.88 to 2.05) but studies were heterogeneous ($p = 0.0002$).

The two studies used different outcome measures to investigate the impact of breathing control on dyspnoea with Vitacca et al (1998) using a visual analogue scale and Williams et al (1982) the Borg Rating of Perceived Exertion

scale. When these studies were analysed separately, dyspnoea increased (deteriorated) during breathing control (SMD 2.30, 95% CI 1.57 to 3.02) in the study by Vitacca et al (1998) and no significant difference was found in dyspnoea following a three week breathing control program compared with pre-training measures (SMD -0.04, 95% CI -1.02 to 0.94) (Williams et al 1982).

One further study by Gosselink et al (1995) measured dyspnoea with the modified Borg scale following a three week breathing control training program. Participants' ratings of dyspnoea were measured both during spontaneous breathing and breathing control post training program. The study contained insufficient data to enable inclusion within the meta-analysis, although the authors reported a significant ($p < 0.05$) increase in dyspnoea during breathing control. Both of these studies (Gosselink et al 1995, Vitacca et al 1998) recruited people with severe pulmonary impairments.

12-minute Walk Test: One study involving eight participants with chronic respiratory disease reported the impact of a three week breathing control program on the distance achieved in the 12-minute Walk Test (Williams et al 1982). There was no significant effect following training (SMD 0.07, 95% CI -0.91 to 1.05).

Discussion

Breathing control was shown to have a significant beneficial effect on abdominal and diaphragm excursion, respiratory rate, tidal volume, arterial oxygen saturation, and percutaneous oxygen. However, breathing control was shown to have a significant detrimental effect on the work of breathing and dyspnoea. There was no clear evidence of an effect on ventilation, longer-term mechanisms such as pulmonary volume or flow, and respiratory muscle strength. Additionally, there was no overall effect on physiological outcomes related to the energy cost of breathing such as oxygen consumption and respiratory muscle efficiency, or outcomes related to gas exchange such as the distribution of ventilation. Only one study investigated exercise capacity (12-minute Walk Test) following three weeks of breathing control training and this showed no significant effect.

Given the range of therapeutic goals reported by previous authors, it was unclear whether the corpus of studies investigating breathing control would assess the ultimate clinical endpoint of the intervention (ie, breathlessness) or earlier stages (mechanism or physiological outcomes) on the assumption that changes at this level would be likely to result in beneficial outcomes. In this systematic review of experimental studies, the impact of breathing control was assessed using a variety of outcome measures. When these outcome measures are allocated within the framework of outcomes related to the targets and mechanisms of breathing control, and physiological and clinical outcomes (Figure 1), the paucity of clinical outcomes becomes apparent. The most common outcomes investigated reflected the target of intervention (diaphragm and abdominal excursion) or mechanism of intervention (respiratory rate, tidal volume, and ventilation). In contrast, there was a distinct lack of studies reporting the impact of breathing control on clinical outcomes such as dyspnoea. Therefore, the majority of the experimental studies investigating the intervention were concerned with the mechanism(s) underpinning the technique rather than the effect of breathing control in the relief of breathlessness. This finding was surprising considering that

Table 4. Level of evidence for volume and consistency of studies, adapted from NHMRC (2005) and modified for the Lloyd-Smith (1997) hierarchy of evidence.

| | Excellent | Good | Satisfactory | Poor |
|-------------|--|---|--|--|
| Volume | Several level 1a or 1b studies with a low risk of bias | Level 1b studies with low risk of bias or multiple level 2a or 2b studies with low risk of bias | Level 2a or 2b studies with low risk of bias, or level 1a or 1b studies with moderate risk of bias | Level 3 studies, or level 1-2 studies with high risk of bias |
| Consistency | All studies consistent | Most studies consistent and inconsistency may be explained | Some inconsistency reflecting genuine uncertainty around clinical question | Evidence is inconsistent |

breathing control is commonly recommended for managing breathlessness or dyspnoea in people with chronic respiratory disease (Pryor et al 2002).

This systematic review examined the effect of breathing control in general rather than in specific populations or clinical situations. Accordingly, this review included studies on the basis of pre-determined criteria for design, intervention, and comparisons, but all outcomes were accepted. The likelihood of missing studies was minimised through a comprehensive search strategy and consultation with experts in the field. Additionally, studies were excluded if they investigated breathing control in conjunction with other interventions such as the active cycle of breathing or forced expiration. Therefore the implications of this review may be applied only to situations where breathing control is applied as the sole intervention and should not be extrapolated to regimens which incorporate breathing control as part of a combined intervention.

The effect of breathing control in the included studies was investigated in a variety of populations. The majority of studies recruited people with chronic pulmonary disease likely to feature breathlessness. Only two studies investigated breathing control on post-surgical patients where it was likely that the intervention was targeted at altering the distribution of ventilation rather than at managing breathlessness.

The included studies reported the effect of breathing control in the short term such as in the immediate management of breathlessness ($n = 6$) and altering breathing patterns post surgery ($n = 2$). Surprisingly, the majority of studies included in this review investigated the long-term value of prolonged breathing control training of varying intensity and duration as a means of altering physiological outcomes (ie, pulmonary function). It is clear that there is no high-level evidence to indicate a beneficial effect on lung volume or exercise capacity for long-term training with breathing control as the sole intervention in people with chronic respiratory disease.

The National Health and Medical Research Council (2005) have proposed a process and matrix to assist guideline developers to assess the body of evidence underpinning therapeutic interventions and formulate evidence-based recommendations. A modified version of this matrix was used in this review to assist in classifying the overall volume and consistency of the evidence for breathing control (Table 4). The overall volume of evidence for breathing control was classified as satisfactory. Studies were included in this systemic review if they were classified as between Level 1

and 2b on the Lloyd-Smith hierarchy. However, the majority of studies (75%) were classified as Level 2b. The evidence for breathing control was classified as inconsistent as indicated by the significant heterogeneity across studies in many of the outcomes (Table 3). These inconsistencies most likely result from the diversity of designs, interventions (short versus longer term applications), outcome variables, and disease severities included within the studies.

Implications for clinical practice

Despite breathing control having long been advocated as an intervention to manage breathlessness in people with chronic pulmonary disease, there was insufficient high-level evidence to recommend breathing control on its own as an effective management strategy for dyspnoea. This should not be interpreted as compelling evidence that breathing control does not alleviate breathlessness. Rather, the lack of experimental studies which assess clinical outcomes such as dyspnoea has resulted in insufficient evidence on which to provide a recommendation. Breathing control was found to have a significant short-term effect on a number of outcomes related to the mechanism of intervention and physiological outcomes (respiratory rate, tidal volume, arterial oxygen saturation), though no effect was evident for altering the distribution of ventilation or oxygen consumption. Long-term training of breathing control did not result in any significant improvement in outcomes related to the mechanism of intervention, or to physiological or clinical outcomes. This review clearly indicates that breathing control is likely to have a detrimental effect on dyspnoea and work of breathing in people with severe COPD (FEV_1 %predicted 30–50). Based on this finding, clinicians planning to use breathing control as a short-term strategy to affect outcomes related to the mechanism of intervention or physiological outcomes in people with severe airflow obstruction should proceed cautiously and monitor dyspnoea and work of breathing.

Implications for research

This systematic review has highlighted the lack of high quality research concerning the therapeutic intervention of breathing control. The majority of studies included assessed outcomes related to the mechanisms underpinning breathing control on the assumption that changes in these would directly alter the sensation and sequelae of breathlessness. The most obvious deficiency in the evidence concerning breathing control is the lack of information about clinical outcomes specific to dyspnoea (intensity, quality of life, exercise capacity). The effect of breathing control in people with moderate or mild pulmonary impairment needs to be explored further. While breathing control has been advocated

for altering distribution of ventilation and improving lung volumes following surgery, few studies are available to support this assumption.

Footnotes: ^(a)RevMan 4.1.

eAddenda: Appendix 1, Table 1, and Table 3 available at www.physiotherapy.asn.au/AJP

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