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# RESPIRATORY RESPONSES TO SUSTAINED ISOMETRIC MUSCLE CONTRACTIONS IN MAN: THE EFFECT OF MUSCLE MASS

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SUMMARY

1. Respiratory responses to sustained isometric contractions of a small mass of muscle (the finger flexors) during handgripping, and of a larger mass of muscle (the quadriceps) during extension of the leg at the knee, have been studied in man.

2. For both masses of muscle the increases of ventilation and of oxygen consumption were greater for contractions at 40% maximum voluntary contraction (MVC) than for contractions at 20% MVC.

3. The increase of ventilation was not related to the mass of muscle involved.

4. At 20% MVC oxygen consumption during contraction of the quadriceps was greater than that during handgripping. At 40% MVC the oxygen consumptions were similar. The oxygen debts following both handgrip and knee extensor contractions at 20% MVC were negligible. Following 40% MVC contractions of the quadriceps a significant oxygen debt was recorded but no debt was apparent following 40% MVC contractions of the finger flexors.

5. The increases of ventilation during isometric exercise were generally inappropriately high for the increases of gas exchange. This led to reductions of the end-tidal carbon dioxide pressure ($P_{ET,CO_2}$), especially towards the end of exercise.

6. Following 40% MVC handgripping hyperventilation continued despite the reduced alveolar $P_{CO_2}$. By contrast, following 40% MVC knee extension $P_{ET,CO_2}$ transiently rose above the resting level, but did not stimulate ventilation.

7. It appears that following fatiguing isometric contractions hyperventilation continues and appears to be independent of alveolar $P_{CO_2}$. It is suggested that stimuli which increase ventilation during exercise may continue to act during the early phase of recovery.

## INTRODUCTION

The cardiovascular responses to sustained, isometric muscle contractions have been well documented, and include elevation of heart rate and mean arterial blood pressure, with a modest increase in cardiac output and little change in total peripheral resistance. For a particular muscle, or group of muscles, the cardiovascular changes are related to the force of contraction. The mass of muscle involved has less

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effect on the blood pressure and heart rate responses (Lind, Taylor, Humphreys, Kennelly & Donald, 1964; Lind & McNicol, 1967*a*; McCloskey & Streatfield, 1975; Mitchell, Payne, Saltin & Schibye, 1980). The cardiovascular changes are mediated largely by the sympathetic nervous system which is activated by both central command to the autonomic system and by reflexes arising within the contracting muscle (Leonard, Mitchell, Mizuno, Rube, Saltin & Secher, 1985).

The respiratory responses to isometric exercise have been less well investigated. The studies have mainly been on handgrip contractions and represent responses to contractions of a small mass of muscle. The ventilatory responses are variable and the increases are related to the force of contraction. Some subjects hyperventilate inappropriately which leads to marked falls in alveolar partial pressure of $CO_2$ ($P_{CO_2}$) (Wiley & Lind, 1971; Duncan, Johnson & Lambie, 1981; Muza, Lee, Wiley, McDonald & Zechman, 1983). Poole, Ward & Whipp (1988) also observed variable ventilatory responses to two-leg lower body thrust and, although it is difficult to determine whether the ventilatory responses are greater than those observed by other workers during handgripping, the oxygen consumption was higher.

The present studies aim to compare directly the respiratory responses to contraction of a small mass of muscle (the finger flexors) and of a larger mass, the quadriceps examined during extension of the leg at the knee. We will test the hypothesis that the increase of ventilation during sustained isometric contractions is independent of the mass of the muscle involved, although oxygen consumption may rise with increasing active muscle mass. The reduction of alveolar $P_{CO_2}$, resulting from hyperventilation, should be less with a larger muscle mass as carbon dioxide production will be increased. Preliminary accounts of this work have already been communicated to the Physiological Society (Imms & Mehta, 1987, 1988, 1989).

## METHODS

The subjects were eight healthy male students aged 19–26 years, who gave their informed consent to participate in studies approved by the Ethical Committee of Guy's Hospital and Medical School. All had previously used respiratory monitoring equipment, although not during isometric exercise. Measurements were performed with the subjects seated in a knee extension dynamometer (Doré, Hackett, Imms & Prestidge, 1977) with their right hand and forearm resting on the handgrip dynamometer. The output of the strain-gauge amplifiers was recorded (Fig. 1) to give markers of the onset and end of exercise and to monitor that the subjects maintained the pre-set tension for the duration of the exercise. A low-resistance valve was placed in the mouth and inspired volume was measured with a dry-gas meter. Airway carbon dioxide was sampled at the mouth through a narrow bore catheter to a rapid response carbon dioxide meter (Leybold-Heraus). Expired air was conducted through a short length of wide-bore tubing to a mixing chamber, at which site the fractional concentrations of oxygen and carbon dioxide were measured (Fig. 1). Arterial blood pressure was recorded from the non-exercising arm using an automatic sphygmomanometer (Bosomat), and heart rate was counted from a precordial electrocardiogram.

When all monitoring equipment was in place, the subjects were asked to perform maximal handgrip or quadriceps contractions, the latter with the knee joint flexed to 68 deg. They were verbally instructed on technique and exhorted to make maximum efforts. The best of three efforts was taken as the maximum. Twenty or forty per cent of the maximum was then calculated and displayed digitally to the subject, who practised holding this effort for a few seconds. Each subject attended the laboratory on two separate days. On one day they performed handgrip and

quadriceps exercises at 20% maximal voluntary effort (MVC) for 5 min, and on the other day at 40% MVC for 2 min. The order of exercises was randomized. Each contraction was preceded by a 10 min control period and recovery was monitored for 5 min. A rest of 30 min was allowed between contractions.

*Statistical analysis*

Control data were analysed by parametric statistics including two-way analysis of variance and Tukey's test to determine significance of differences (Green & Margerison, 1978). The data collected

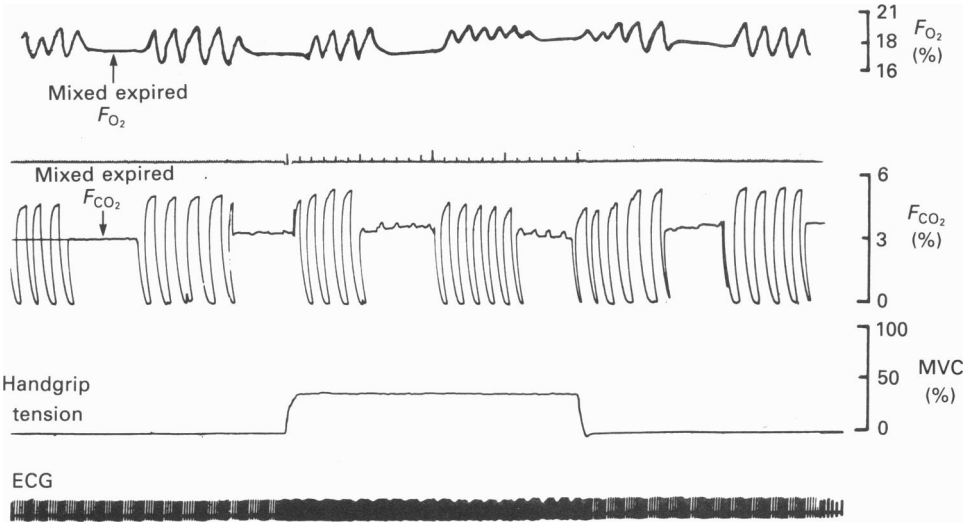


Fig. 1. Responses to 40% MVC handgrip contraction sustained for 2 min. The oxygen and carbon dioxide analysers were periodically switched from sampling mixed expired air and gas from the airway. $F_{O_2}$ and $F_{CO_2}$ are the fractional concentrations of oxygen and carbon dioxide, respectively. The traces from above downwards are: $F_{O_2}$ (the oscillations do not represent true inspiratory and end-tidal levels because of slow response time of analyser); time (5 s); $F_{CO_2}$ (oscillations represent inspired $F_{CO_2}$ ($\simeq 0\%$) and end-tidal $F_{CO_2}$); tension exerted on handgrip dynamometer (% maximum); precordial ECG.

during contractions were often not normally distributed and non-parametric statistics have been employed. Median values were used to represent group responses and differences were determined by the binomial test or by Fisher's exact test (Siegel, 1956). Multiple regression analysis was used to investigate relationships between some apparently normally distributed variables (Wonnacott & Wonnacott, 1977).

## RESULTS

### *Control periods*

All subjects were normotensive, and heart rate and respiratory variables were within normal limits (Table 1). Resting oxygen consumption and carbon dioxide production differed between subjects, but this was eliminated by expressing the volumes per kilogram body weight. The between-subjects variation of ventilation persisted when it was expressed on a body weight basis. The control data did not differ between experiments.

TABLE 1. Control data for the periods preceding each of four isometric contractions. Each point is the mean ±S.D. for eight subjects

| | 20% MVC | | 40% MVC | | *F* Variation between experiments (3 and 21 d.f.) | *F* Variation between subjects (7 and 21 d.f.) |
|---|---|---|---|---|---|---|
| | Handgrip | Leg extension | Handgrip | Leg extension | | |
| Heart rate (beats $min^{-1}$) | 68·3 ±8·6 | 65·5 ±7·2 | 67·7 ±13·9 | 70·3 ±9·8 | 0·54 | 6·40** |
| Mean arterial blood pressure (mmHg) | 85·0 ±7·2 | 86·5 ±3·9 | 89·2 ±3·3 | 91·2 ±2·9 | 2·18 | 3·15* |
| Ventilation (l $min^{-1}$) | 8·2 ±1·0 | 7·9 ±1·4 | 8·0 ±1·2 | 8·4 ±2·9 | 1·11 | 19·5*** |
| Oxygen consumption (ml $min^{-1}$) | 265 ±47 | 269 ±36 | 277 ±19 | 299 ±22 | 2·52† | 2·99* |
| $CO_2$ elimination (ml $min^{-1}$) | 225 ±32 | 230 ±30 | 228 ±27 | 250 ±28 | 2·44† | 5·53** |
| *R* | 0·859 ±0·118 | 0·856 ±0·055 | 0·822 ±0·084 | 0·850 ±0·049 | 0·28 | 1·14 |
| Ventilatory index (l air l $O_2^{-1}$ | 30·6 ±5·2 | 28·7 ±3·7 | 27·8 ±2·3 | 27·2 ±2·8 | 1·49 | 1·64 |
| End-tidal $P_{CO_2}$ (mmHg) | 38·5 ±3·3 | 39·7 ±2·0 | 39·8 ±3·0 | 39·4 ±2·2 | 0·47 | 1·59 |

Two-way analysis of variance for eight subjects, each performing four experiments. †$P < 0{\cdot}10 < 0{\cdot}05$; *$P < 0{\cdot}05$; **$P < 0{\cdot}01$; ***$P < 0{\cdot}001$.

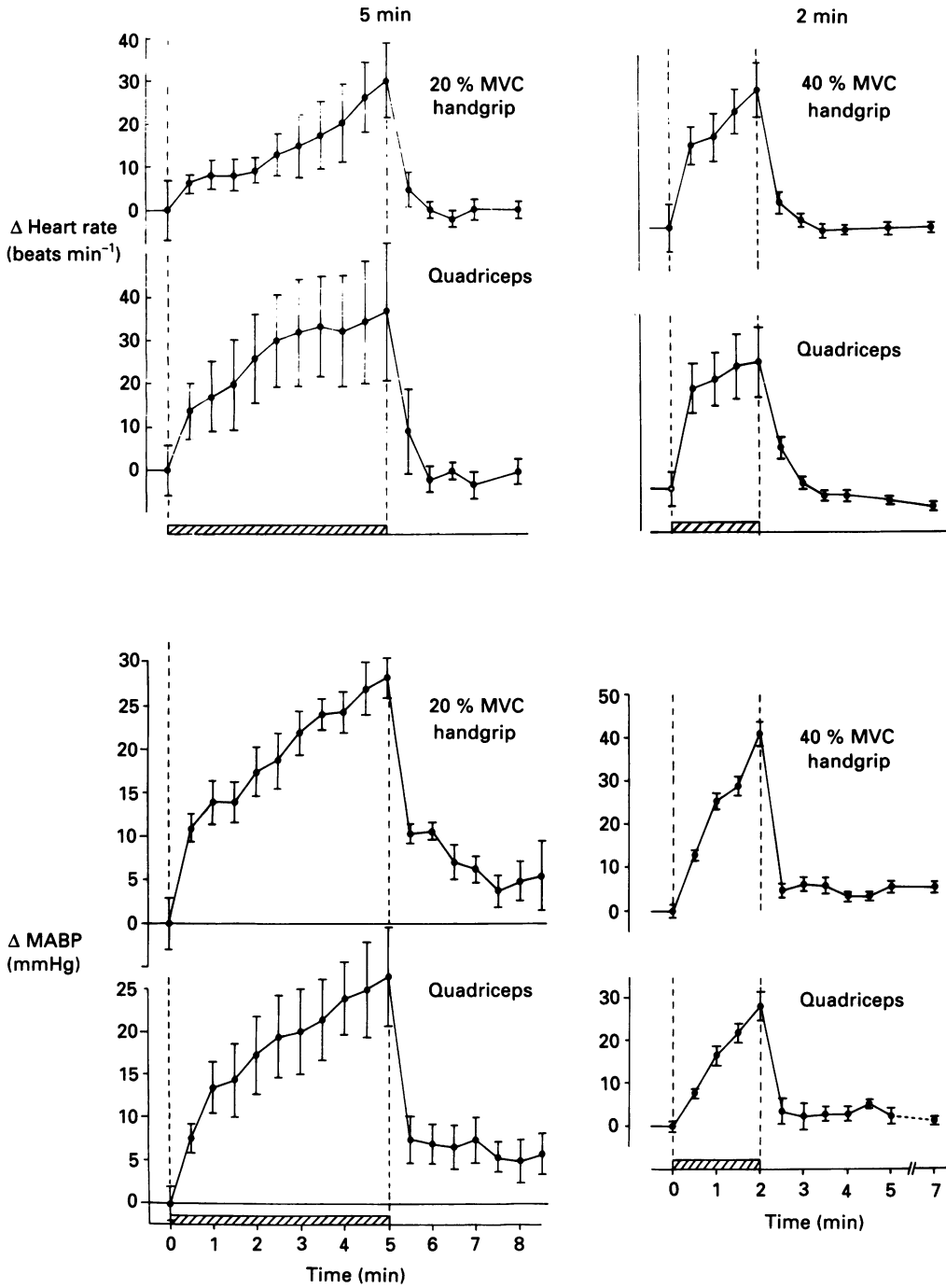


Fig. 2. Heart rate and blood pressure responses during and following 20% MVC handgrip and quadriceps contractions for 5 min, and 40% MVC handgrip and quadriceps contractions for 2 min. All points are means ± S.E.M. Bar indicates period of contraction.

*Cardiovascular responses to isometric exercise*

The changes of both heart rate and mean arterial blood pressure (MABP) which occurred during contractions (Fig. 2) were typical of those expected during isometric exercise.

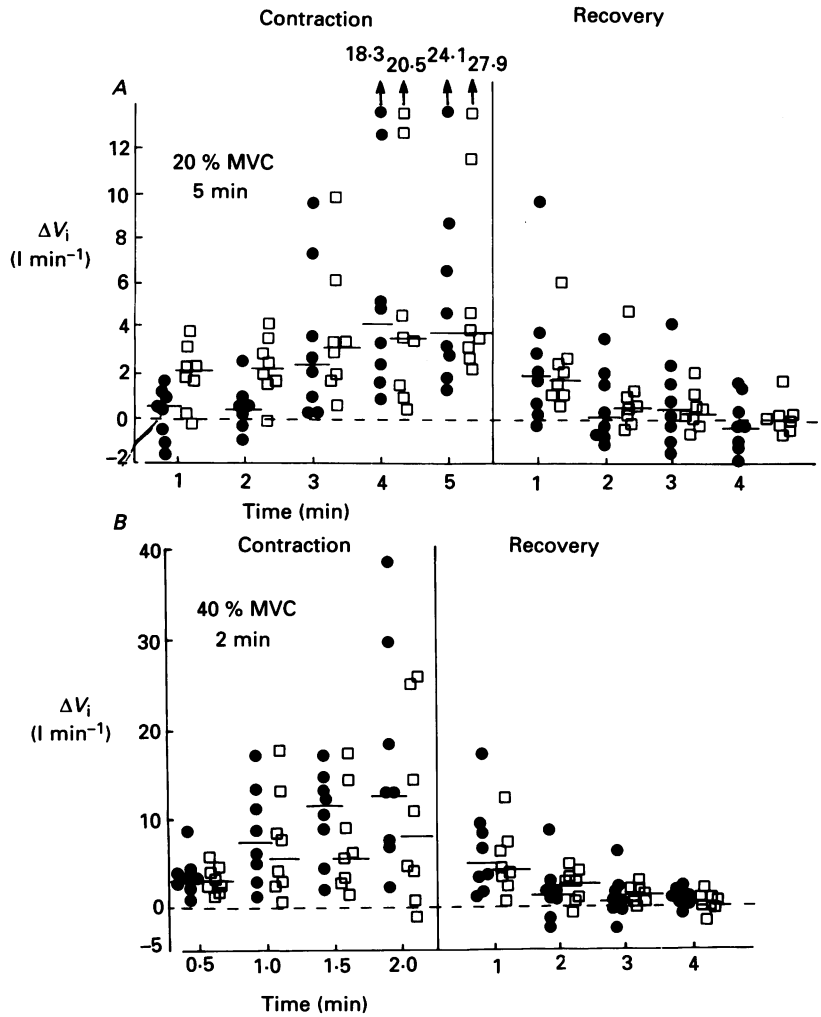


Fig. 3. Changes of ventilation ($\Delta V_I$) during and following sustained contractions at 20% MVC for 5 min (*A*) and 40% MVC for 2 min (*B*). ●, handgrip; □, quadriceps contraction. Horizontal bars represent the *median* responses.

*Respiratory responses to isometric exercise*

The increases of ventilation during both handgrip and knee extension exercise were variable (Fig. 3). Contractions performed at 40% MVC increased ventilation more than those at 20% MVC. During the second minute of handgripping at 20% MVC the median increase of ventilation was 0·4 l min$^{-1}$ compared with 11·5 l min$^{-1}$

during a 40% MVC contraction ($P = 0{\cdot}0004$); during the second minute of quadriceps contraction the respective values were 2·2 and 6·1 l min$^{-1}$ ($P = 0{\cdot}004$). Muscle mass had no consistent effect on ventilation. Handgrip contraction at 20% MVC did not increase ventilation during the first 2 min of effort, whilst the larger muscle mass

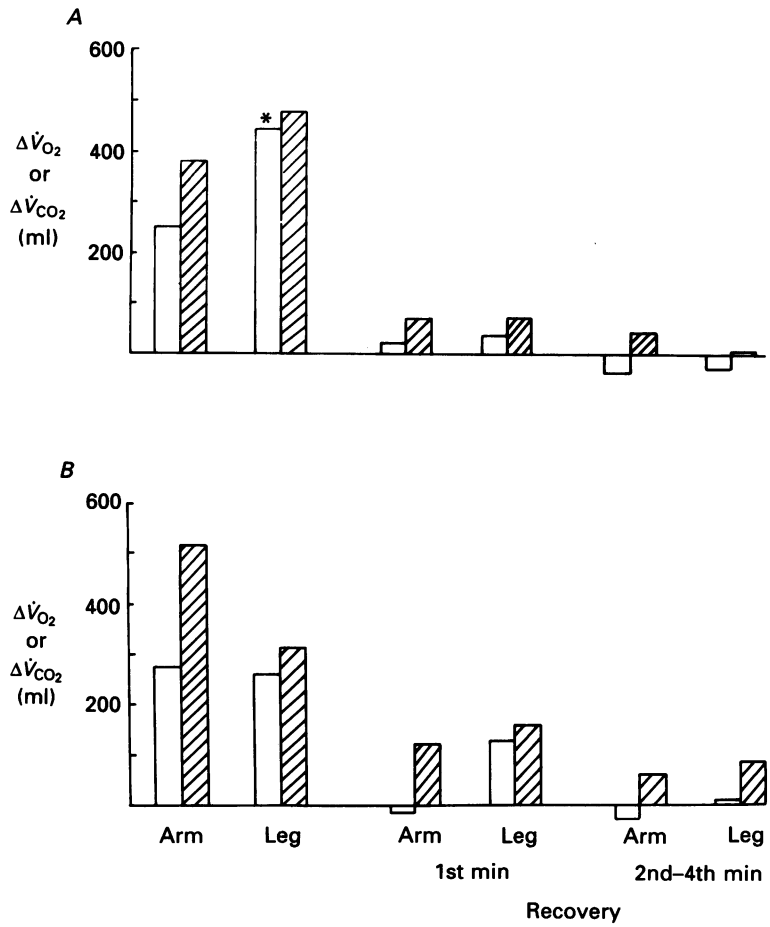


Fig. 4. Changes in oxygen consumption ($\Delta\dot{V}_{O_2}$, open columns) and carbon dioxide production ($\Delta\dot{V}_{CO_2}$, hatched columns) during and following contractions at 20% MVC for 5 min (*A*) and at 40% MVC for 2 min (*B*). * Significant difference ($P = 0{\cdot}004$) arm *vs.* leg.

raised ventilation by around 2 l min$^{-1}$ ($P = 0{\cdot}035$), but during the third to fifth minutes the responses were similar (Fig. 3). During the first minute of contraction at 40% MVC ventilation was similar for the two muscle masses, but during the second minute the response to handgrip contraction (median 11·5 l min$^{-1}$) was greater than for quadriceps contraction (6·1 l min$^{-1}$; $P = 0{\cdot}035$).

Ventilation was also highly variable during recovery (Fig. 3). In the first minute following 20% MVC contractions the median value for ventilation was about 2 l min$^{-1}$ greater than the pre-exercise control, but subsequently the medians were indistinguishable from zero, some subjects ventilating less than before the contraction. The total debt over 4 min following a 20% MVC handgrip contraction

ranged from 16·3 down to −2·2 l, and following a quadriceps contraction 14·4 to 0·3 l. A similar pattern of ventilation followed 2 min contractions at 40% MVC. These contractions were followed by significantly greater debts over 4 min (medians for arm and leg 7·6 and 8·1 l, respectively) than after efforts at 20% MVC (arm and leg 0·3 and 3·1 l, respectively; $P = 0{\cdot}035$). Debts were not related to mass of muscle.

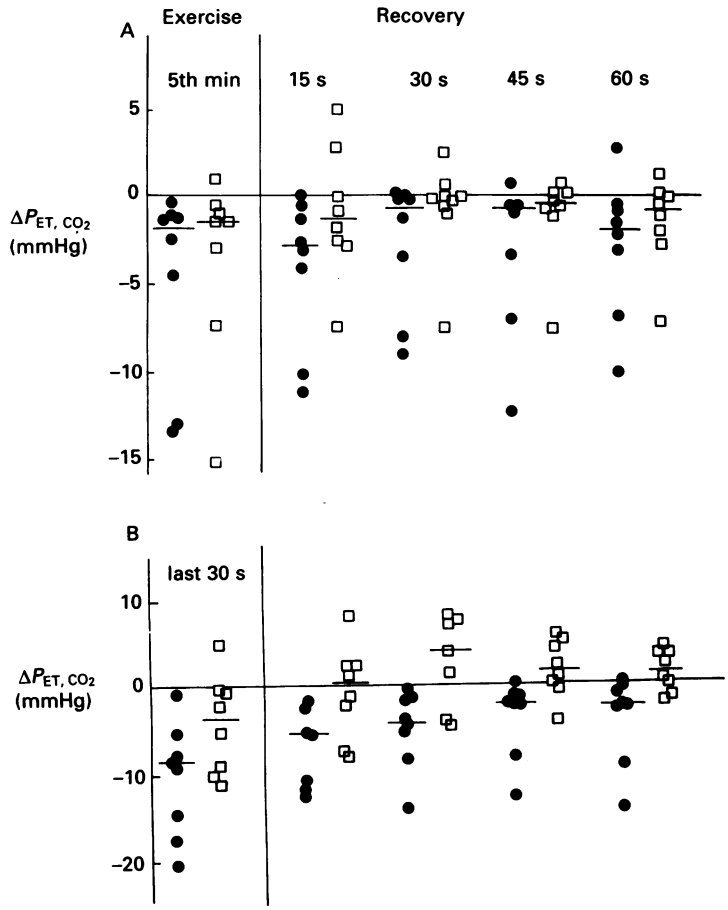


Fig. 5. Changes of end-tidal $P_{CO_2}$ during and following 20% MVC contractions for 5 min (*A*), and 40% MVC contractions for 2 min (*B*). ●, handgrip; □, quadriceps contraction. Horizontal bars represent the *median* responses.

The total increase of oxygen consumption during 5 min of contraction of the knee extensor muscles at 20% MVC (median 445 ml; range 145–853) exceeded that for the handgrip (253 ml; range 68–540 ml; $P = 0{\cdot}004$). The oxygen debt was negligible (Fig. 4). The increase of oxygen consumed during 2 min of contraction at 40% MVC was similar for both the forearm (median 275 ml) and the thigh (265 ml). There was a significant oxygen debt (median 113 ml; $P = 0{\cdot}034$) following quadriceps contractions but not following handgripping.

In most subjects the increment of carbon dioxide elimination during handgrip exercise exceeded the increase of oxygen intake, suggesting that carbon dioxide stores were being reduced. During knee extension exercise carbon dioxide elimination

from the body was not significantly greater than oxygen uptake. Some subjects had large increases of ventilatory equivalent (87 l $l O_2^{-1}$), whereas in others the volume was unchanged. The largest change was during 40% MVC handgrip contractions. During the last 30 s of this effort, the ventilatory equivalent increased by a median value of 21·5 l $l O_2^{-1}$, a value greater than during knee extension (median 8·4; $P = 0{\cdot}022$).

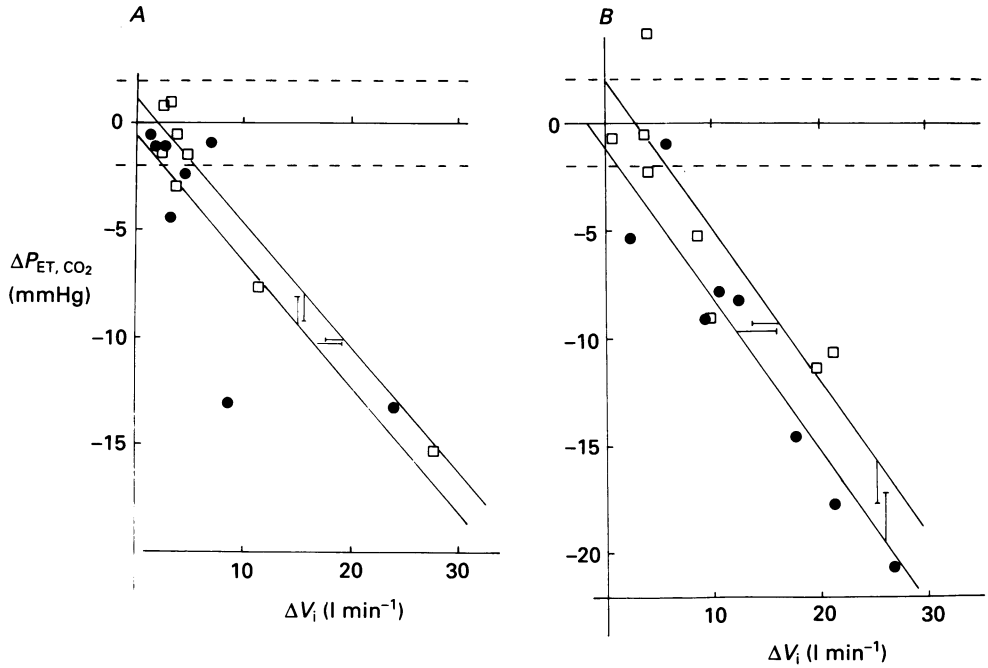


Fig. 6. Relationship of changes of end-tidal $P_{CO_2}$ to changes of ventilation during the final minute of 20% MVC contractions for 5 min (*A*) and the final 30 s of 40% MVC contractions lasting 2 min (*B*). ●, handgrip; □, quadriceps contractions. The dashed lines represent ±2 mmHg from pre-contraction controls, the limits within which values might be expected to fall if carbon dioxide homeostasis were maintained. Vertical and horizontal bars represent 95% fiducial limits of the multiple repression lines.

*Alveolar* $P_{CO_2}$

Inappropriately high ventilation reduces alveolar $P_{CO_2}$. During the final minute of contractions at 20% MVC the median fall in end-tidal carbon dioxide pressure $P_{ET,CO_2}$ was only 1·6 mmHg, although in two subjects it was much greater (Fig. 5). In most subjects ventilation may be therefore considered eucapnic whilst two subjects, in whom ventilation was high despite a low $P_{ET,CO_2}$, were hyperventilating (Fig. 6). During the final 30 s of knee extensor contractions at 40% MVC, $P_{ET,CO_2}$ fell by a median value of 3·7 mmHg, whilst following handgripping the fall was greater (8·7 mmHg; $P = 0{\cdot}035$). The magnitude of the falls in $P_{ET,CO_2}$ were clearly related to the increases in ventilation. Multiple repression lines have been fitted to the data (Fig. 6) to demonstrate that at a given ventilation, $P_{ET,CO_2}$ was lower during arm contractions than during leg exercise. Following exercise, hyperventilation persisted

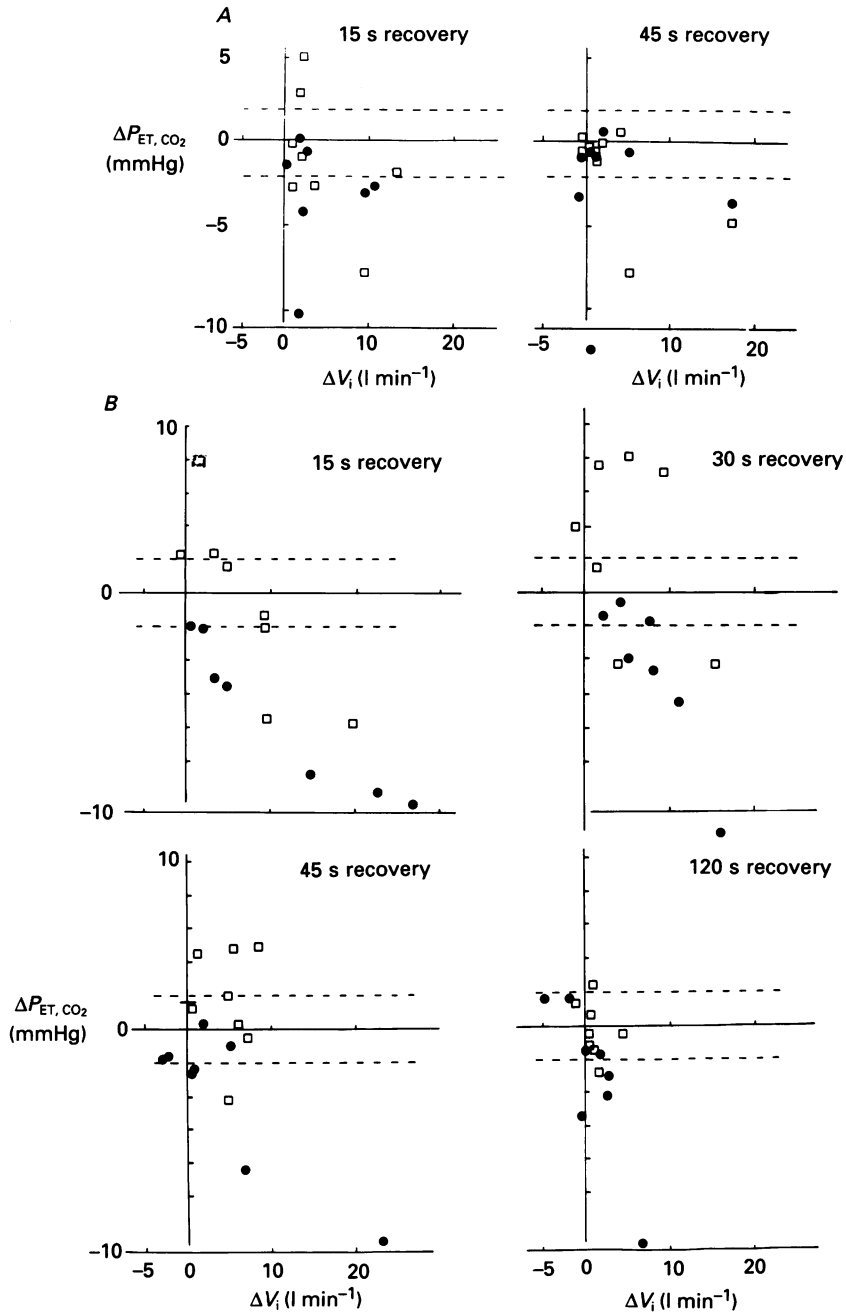


Fig. 7. Relationship of changes of end-tidal $P_{CO_2}$ to changes in ventilation during recovery from 20% MVC contractions for 5 min (*A*) and 40% contractions for 2 min (*B*). ●, handgrip: □, quadriceps contractions. The dashed lines represent ±2 mmHg from pre-contraction controls, the limits within which values might be expected to fall if carbon dioxide homeostasis were maintained.

in some subjects, despite the marked reduction of $P_{ET,CO_2}$ (Fig. 7). In no case was ventilation reduced appreciably below the pre-exercise level. Following exercise at 20% MVC, in all except one subject $P_{ET,CO_2}$ was normal 45 s after the end of contraction. During the first minute following contraction, carbon dioxide excretion was greater than in the control period (median 77 ml for arm; 75 ml for leg; Fig. 4), suggesting that carbon dioxide production was raised since stores were also being replenished. Fifteen seconds after exercise at 40% MVC, $P_{ET,CO_2}$ was reduced by 5 mmHg or more in five subjects following handgrip exercise, but in only two subjects following knee extension. Thirty seconds after quadriceps contractions, $P_{ET,CO_2}$ was clearly elevated in four out of seven subjects, whilst in four subjects who had performed handgrip it was clearly reduced. One minute into recovery from handgrip contraction, $P_{ET,CO_2}$ was still reduced by 9·5 and 14 mmHg in two subjects who continued to hyperventilate.

## DISCUSSION

These experiments have confirmed that isometric contractions of skeletal muscle cause marked increases of ventilation, though changes in oxygen consumption are more modest. As a consequence alveolar $P_{CO_2}$ is decreased. We have also confirmed that during contractions the magnitudes of these responses are related to the percentage maximum voluntary contraction. No clear overall relationship of muscle mass and respiration emerges from the present studies. During the early stages of exercise at 20% MVC the increase of ventilation is greater for quadriceps contractions than handgrip contractions. Although this difference disappears towards the end of contraction, the total increase of ventilation over the whole 5 min of contraction was greater for the quadriceps. At 40% MVC the situation was reversed and the increase of ventilation was higher for arm than leg contractions. For contractions at 20% MVC oxygen consumption was increased by increasing muscle mass, but at 40% MVC oxygen consumption was unaffected by muscle mass. Reductions of $P_{ET,CO_2}$ were not affected by muscle mass during 20% MVC contractions, but during 40% MVC handgrip contractions $P_{ET,CO_2}$ fell more than during leg extension.

There was marked individual variation of both cardiovascular and respiratory responses to exercise, particularly if the contractions were fatiguing. The rank orders of the subjects' ventilatory responses were similar in all four exercise tests (Imms & Mehta, 1989) and, furthermore, subjects with the highest ventilation tended also to have large increases in blood pressure and heart rate.

Contractions at 20% MVC are only mildly fatiguing, blood flow is elevated during the contractions and post-exercise hyperaemia is only transient (Humphreys & Lind, 1963; Lind & McNicol, 1967*b*). The oxygen requirements of the muscle are largely met during exercise which confirms the lack of oxygen debt observed in the present studies. During contractions at 40% MVC, blood flow through the contracting muscle is partially occluded by the intramuscular pressure and post-exercise hyperaemia may be prolonged. A substantial oxygen debt (median 113 ml) followed a 40% quadriceps contraction, whereas no debt was recorded after handgripping.

There is a discrepancy between values for oxygen consumption obtained using whole body respiratory measurements, and those calculated from limb blood flows

and the oxygen saturation of blood draining from the muscle. The former suggest that 20 or 25% MVC handgrip contractions increased whole body oxygen consumption by 40–90 ml $min^{-1}$ and that 40 or 50% contractions raised it by 130 to 270 ml $min^{-1}$ (Fig. 4 and Table 2; Wiley & Lind, 1971; Duncan *et al*, 1981;

TABLE 2. Partitioning of oxygen consumption isometric exercise. The calculated increase of oxygen consumption by the heart (*b*), and for ventilation (*c*) have been subtracted from the observed increase of oxygen consumption by the body during the final minute of exercise (*a*) to give a balance representing the oxygen consumption by the muscle under study and other muscular activity. Data are median values for eight subjects

| | Measured $\Delta\dot{V}_{O_2}$ (ml $min^{-1}$) (*a*) | Calculated $\Delta\dot{V}_{O_2}$ for cardiac work (ml $min^{-1}$) (*b*) | Calculated $\Delta\dot{V}_{O_2}$ for ventilatory work (ml $min^{-1}$) (*c*) | Balance (ml $min^{-1}$) $a-(b+c)$ |
|---|---|---|---|---|
| 20% MVC handgrip | 65·8 | 13·8 | 6·2 | 45·8 |
| 20% MVC leg extension | 105·9 | 13·6 | 19·5 | 72·8 |
| 40% MVC handgrip | 136·9 | 23·0 | 23·7 | 90·2 |
| 40% MVC leg extension | 169·5 | 16·5 | 15·2 | 137·8 |

Blomquist, Lewis, Taylor & Graham, 1981). It is estimated by the latter method that the resting forearm consumes between 0·15 and 0·5 ml 100 $ml^{-1}$ $min^{-1}$ (Barcroft, Greenwood & Whelan, 1963; Kontos, Richardson & Patterson, 1966; Baker & Mottram, 1973). Oxygen consumption increases by up to 3 ml 100 $ml^{-1}$ $min^{-1}$ during isometric exercise, perhaps 20 ml $min^{-1}$ for the whole forearm. Since the quadriceps is a complex muscle, it is difficult to estimate the volume of muscle actually involved in the extension exercise. It is unlikely to exceed 1.5 l, suggesting a maximal increase in oxygen uptake of around 45 ml $min^{-1}$.

The difference in values from the two approaches represents oxygen consumed at sites other than the specific muscle group. Since stroke volume is essentially unaltered during this type of exercise (Perez-Gonzales, Schiller & Parmley, 1981), then the product of blood pressure and heart rate gives an index of cardiac work. Assuming myocardial oxygen consumption to be 25 ml $min^{-1}$ at rest (Ganong, 1985) the likely increase of cardiac metabolism during the final minute of exercise has been calculated (Table 2). The oxygen cost of the increased ventilation was calculated assuming 1·6 ml oxygen per litre ventilation (Silver, 1963). The median values for these 'known' sites of utilization have been subtracted from the measured whole body oxygen consumption to give a balance. This exceeds the oxygen consumption for the muscle derived from limb blood flow and venous oxygen saturation. This difference probably represents oxygen used in other muscles for postural and fixation work. These calculations would be more precise if muscle mass were estimated for each individual either by anthropometry or by imaging, and if some attempt were made to assess the extent of postural fixation by appropriate surface electromyographic recording.

The absence of appreciable oxygen debts may be for three reasons. Firstly, increases of plasma lactate concentration are small and fall slowly after exercise (Poole *et al.* 1988) and only small quantities of oxygen will be required for metabolism of lactate. Since the proportion of the cardiac output going to active muscle is small, there will be only a small fall of mixed venous oxygen saturation

which will require little additional oxygen to replenish it. Finally, hyperventilation during exercise will raise the alveolar $P_{O_2}$, and create a 'store' of oxygen in the lung.

An important consequence of relative overventilation is reduction of alveolar $P_{CO_2}$. Following handgrip contractions hyperventilation persisted despite the lowered $P_{ET,CO_2}$ which might be expected to depress ventilation. Fifteen or thirty seconds after ceasing contraction, there is an inverse relationship between ventilation and alveolar $P_{CO_2}$ (Fig. 7*B*), rather than the expected positive correlation. By contrast, after contractions of the quadriceps at 40% MVC there was an apparent surge of carbon dioxide from the exercising muscle, resulting in a rapid rise in $P_{ET,CO_2}$ (Fig. 7*B*) similar to that observed by Poole *et al.* (1988). Ventilation was no higher in those subjects with elevated carbon dioxide levels, and the usual relationship between alveolar $P_{CO_2}$ and ventilation was again absent. It is suggested that following a fatiguing contraction ventilation continues to be driven by factors which stimulated it during the exercise. Alveolar $P_{CO_2}$, whether low or high, is apparently ineffective as a controller in this situation. The levels of lactate present in the blood during and after contraction (Poole *et al.* 1988) are insufficient to stimulate ventilation.

What is the nature of the stimulus to ventilation during isometric exercise which continues into the recovery period and apparently destroys, temporarily, the relationship between ventilation and alveolar $P_{CO_2}$? Since cardiovascular changes during isometric exercise are partly stimulated by drive from higher centres to the brain stem in parallel with the voluntary motor activity, and partly by reflexes arising from within the exercising muscle, it is reasonable to suppose that ventilation may be similarly controlled, though additionally a role for the chemoreceptors must be envisaged. Central drive is assumed to cease at the end of contraction, but the reflex drive from the muscle may continue after the contraction has ceased. Alternatively a tide of 'metabolites' (not carbon dioxide or lactate), washed from the muscle after exercise, may stimulate the chemoreceptors.

Further evidence may be gained by occluding the circulation to the exercising limb, during and following a contraction, and trapping metabolites within the muscle. Results from some small series suggest that occlusion had no effect, but Duncan *et al.* (1981) suggested that, if the circulation remains occluded following a 40% contraction to exhaustion, the subject continues to hyperventilate. This suggests that the muscle reflex can sustain increased ventilation in the absence of contraction and without metabolites reaching the chemoreceptors.

We conclude that at relatively low rates of working (20% MVC), when most of the muscles' energy requirements are met from arterial oxygen, then both ventilation and oxygen consumption are related to the bulk of muscle. This hypothesis requires further testing with a wider range of muscle sizes. For stronger contractions which derive much of their energy anaerobically, then oxygen consumption is similar for the two muscle groups and ventilation is significantly higher for forearm contractions. Fatiguing contractions induce inappropriately high ventilation, which persists during recovery and reduces alveolar $P_{CO_2}$. The pattern of recovery of alveolar $P_{CO_2}$ depends upon the mass of muscle involved in the contraction. Much of our present knowledge of respiratory responses comes from studies of handgrip contractions, whilst real life work situations usually involve much greater masses of muscle. Care should be taken in extrapolating to the whole body data obtained with the convenient forearm model.

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