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CENTRAL COMMAND INFLUENCES CARDIORESPIRATORY RESPONSE TO DYNAMIC EXERCISE IN HUMANS WITH UNILATERAL WEAKNESS

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SUMMARY

1. Changes in ventilation and cardiovascular variables which occur during exercise may be partly due to 'radiation' of activity in central neurones innervating exercising muscles to the respiratory and cardiovascular control areas. To test this hypothesis, we compared ventilatory and cardiovascular responses to two levels of steady-state exercise with each leg separately, in subjects with painless unilateral leg weakness. We assumed that exercise with a weak leg would require more central neural drive than the same level of exercise with the normal leg.

2. Ventilation during exercise with the weak leg was greater than with the normal leg ($P < 0{\cdot}02$). This was a result of greater tidal volume (V_t; $P < 0{\cdot}005$). There was a greater increase in heart rate ($P < 0{\cdot}005$), and systolic ($P = 0{\cdot}001$) and diastolic ($P < 0{\cdot}02$) blood pressures during exercise with the weak leg compared to exercise with the normal leg. The increases in stroke volume and cardiac output during exercise were not different with the two legs.

3. These results support the hypothesis that ventilation, blood pressure and heart rate are influenced by the central neural drive to exercise.

INTRODUCTION

Johansson (1893) suggested that the cardiovascular response to exercise was, in part, caused by neurological activation of the heart in parallel with the central neural drive to the exercising muscles. Krogh & Lindhard (1913) extended this hypothesis to include the idea that increased ventilation during dynamic exercise was due to 'cortical irradiation' of the medullary respiratory neurones by the descending corticospinal outflow to the limb muscles.

The strength of this 'central command' influence on ventilation and circulation has been evaluated using neuromuscular blockade (Asmussen, Johansen, Jørgensen & Nielsen, 1965; Gandevia & McCloskey, 1977; Hobbs, 1982; Galbo, Kjær & Secher, 1987), epidural anaesthesia (Mitchell, Reeves, Rogers & Secher, 1989; Fernandes, Galbo, Kjær, Mitchell, Secher & Thomas, 1990) and peripheral sensory anaesthetic blockade (Gandevia & McCloskey, 1977; Lassen, Mitchell, Reeves, Rogers & Secher,

1989) to manipulate the relationship between the central drive to exercise on the one hand, and the work achieved or the corresponding afferent sensory traffic on the other.

The purpose of this study was to examine, in a group of subjects with painless unilateral leg weakness (from medical causes or deliberately induced by anaesthetic nerve blocks), the cardiorespiratory responses to exercising with a weak leg compared to exercise with the contralateral normal leg. In this way we planned to dissociate, within subjects, the central neurological drive to exercise from the work achieved. Specifically we intended to compare responses to exercise at matched submaximal metabolic rates using the weak limb (central drive should be enhanced) and the normal limb (normal central command intensity). The hypothesis was that if 'cortical irradiation' were an important determinant of cardiorespiratory responses, then these responses should be exaggerated out of proportion to metabolic rate when increased central command is required for exercise (weak limb). An advantage of this design is that the cardiovascular and respiratory apparatus of the subjects is constant in the two exercise situations, i.e. the subjects are their own 'internal controls'. A necessary preliminary step was to demonstrate, in normal subjects, that single-leg exercise normally results in comparable cardiorespiratory responses with the right and left legs.

METHODS

Equipment for single-leg exercise

A cycle ergometer was adapted to enable the subject to cycle with one leg while keeping the rest of the body relatively relaxed (Fig. 1). The subject sat in a firm but comfortable chair with a head-rest, and with a belt tightly round the hips to minimize involvement of postural muscles during cycling. One foot was strapped to the pedal of the cycle ergometer, and the subject was instructed to keep the other leg fully relaxed throughout. Two springs (unstressed length 31 cm, spring rate 0·55 cm/N) were attached to the other pedal of the ergometer, arranged in such a way as to return the leg to the bent-knee position. The angle and strength of these springs was optimized to give both maximum efficiency (lowest oxygen consumption/Watt) and a smooth, continuous and comfortable cycling motion.

Normal responses to single-leg exercise

Nine healthy volunteers were recruited (Table 1*A*). Isometric quadriceps strength was first assessed for each leg separately. The cardiovascular and ventilatory responses to two levels of steady-state single-leg exercise (35 and 50 W) were then measured. These two levels of exercise were chosen to be below the anaerobic threshold as judged by (a) respiratory measurements (Wasserman, 1984) and (b) measurement of arterialized venous lactate concentration. The exercise was repeated, after a recovery period of 1 h using the contralateral leg. Right and left legs were studied in random order.

Studies in subjects with unilateral leg weakness

Subjects. Three groups of subjects with painless unilateral leg weakness took part in this study (Table 1*B*):

(i) Six patients recovering from orthopaedic disorders (fractures, ligamentous or tendon injuries), whose weakness was secondary to immobilization of the limb. These patients had no loss of sensation in the weak leg.

(ii) Two patients with neurological disorders. One had suffered a cerebrovascular accident causing a mild hemiparesis with dysaesthesia in the affected leg and the other had an idiopathic neuropathy leading to lower motor neurone weakness and a patch of diminished sensation over the outer aspect of the calf and ankle.

(iii) Eight normal subjects in whom temporary weakness was induced by percutaneous local

anaesthetic block of the femoral nerve just below the inguinal ligament (8–10 ml of 1·5–2% lignocaine).

All three groups had normal hearts and lungs, judged by clinical examination and chest X-ray. Patients were excluded if they developed pain on exercise, were unable to cycle smoothly for the required period, or if they were taking drugs acting on the cardiovascular or respiratory systems. Subjects gave informed consent, and ethical approval for the study was given by the Ethical Sub-Committee of Charing Cross Hospital.

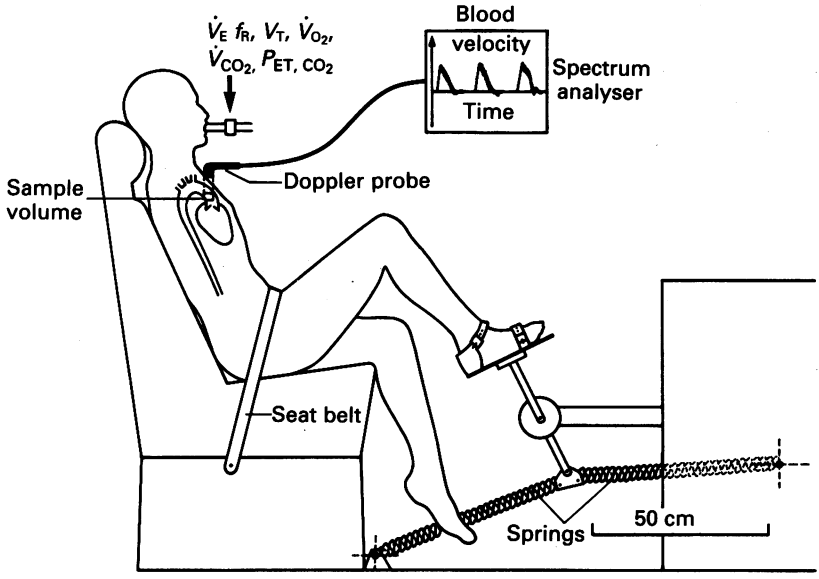

Fig. 1. Scale drawing of the exercise cycle adapted for one-leg cycling. Subject breathes through a pneumotachograph and the flow signals together with gas sampling yield: $\dot{V}_E$ = ventilation, f_R = respiratory rate, V_T = tidal volume, $\dot{V}_{O_2}$ = oxygen consumption, $\dot{V}_{CO_2}$ = carbon dioxide production and P_{ET,CO_2} = end-tidal carbon dioxide.

Protocols. The procedures used were:

(i) Assessment of isometric quadriceps strength in each leg.

(ii) Determination of anaerobic threshold (using ventilatory criteria: Wasserman, 1984) during single-leg exercise, by performing maximal 1 min incremental-cycle exercise tests to exhaustion, once for each leg, separated by at least 1 h rest. Increments were chosen according to the fitness of the subject and varied between 10 and 25 W. The purpose was to determine appropriate sub-anaerobic workloads at which to perform the definitive steady-state measurements.

(iii) Measurement of the ventilatory and cardiovascular responses to two levels of steady-state sub-anaerobic single-leg exercise. The exercise levels were matched by choosing (on the basis of data from the incremental tests) workloads which gave the same oxygen consumption for each leg i.e. the workloads were not necessarily the same but the oxygen consumption was deliberately matched. After resting for 4 min, the subject cycled with one leg for 4 min at each of the chosen exercise levels. This procedure was repeated with the other leg after 1 h rest. Weak and normal legs were studied in random order. Incremental and steady-state tests were performed at the same time of day on separate days within 1 week, with the exception of one subject whose second test was carried out after an interval of 1 month; this subject had chronic stable leg weakness. The subjects did not change their level of activity between the two sets of tests and the isometric strength was measured on both days to ensure that no change in strength had occurred.

In the subjects with femoral nerve blocks it was necessary to use a simplified protocol because the induced weakness was relatively short-lived. The subjects therefore did not carry out maximal

exercise. Responses to two levels of steady-state exercise (25 and 50 W) were measured. The study was started within approximately half an hour of administration of the anaesthetic, which was the time when the induced weakness was greatest, as judged by repeated isometric strength measurements.

Measurements

Isometric leg strength. This was measured as the maximal force generated in attempted extension of the flexed knee against a strain gauge (Strainstall) with the subject in a sitting position (Edwards, Young, Hosking & Jones, 1977).

Ventilatory measurements. The subject breathed through a pneumotachograph (Fleisch No.3,) by which means ventilation, tidal volume and respiratory rate were measured, and simultaneous sampling of expired gas enabled on-line calculation of oxygen consumption ($\dot{V}_{O_2}$) for each 30 s period (Fenyves and Gut, Ergostar, Basel, Switzerland). End-tidal carbon dioxide concentration (P_{ET,CO_2}) was also monitored (infra-red principle; Beckman LB2, Fullerton, USA) using a probe close to the mouth.

Cardiovascular measurements. During the last minute of each 4 min period of steady-state rest or exercise, cardiac output (Innes, Mills, Noble, Murphy, Pugh, Shore & Guz, 1987) was calculated using spectral analysis (Doptek, Chichester) of pulsed Doppler ultrasound measurements of ascending aortic blood velocity (Pedof, Vingmed, Norway), and multiplying by a separate measurement of cross-sectional aortic area (Irex, Ramsey, NJ, USA, 2-D echocardiograph). Subjects were excluded if technically adequate Doppler signals (Innes, 1987) could not be obtained. Cardiac output was not measured during maximal exercise. Blood pressure was measured every 2 min (Narco automatic sphygmomanometer, Houston, TX, USA) throughout both maximal and steady-state exercise. The electrocardiogram was monitored continuously and used to derive heart rate.

Effort. During the last half-minute at each level of steady-state exercise, the subject was asked to score on a category scale (adapted from Borg, 1970) between 1 and 15 in answer to the question 'how hard are you having to try to do this work?'. A score of 1 meant not hard at all and a score of 15 meant as hard as the subject could possibly try. By turning a dial, the subject could indicate the score on an illuminated display. The standard Borg scale for perceived exertion was not used because we wished to obtain a measure of the subjects' perception of effort to exercise untainted by their perception of the amount of work being achieved.

Arterialized venous lactate. Venous blood, arterialized by warming the hand (Forster, Dempsey, Thomson, Vidruk & DoPico, 1972) was sampled every 2 min from an indwelling butterfly cannula in the back of the hand and lactate concentration determined using a polarographic enzyme electrode (Yellow Springs Instrument Co., Yellow Springs, OH, USA). This was done during both maximal and steady-state exercise tests. The sampling time coincided with the time of both blood pressure and cardiac output measurements.

Data analysis

For the normal subjects with no leg weakness, data for each leg, averaged in the last minute at each of the exercise levels, were compared by two-way analysis of variance.

To determine whether or not the effect of unilateral leg weakness on cardiovascular and ventilatory variables was the same for the patients and the normal subjects with femoral nerve block, data were compared from the two groups using three-way analysis of variance (ANOVA). This procedure requires equal numbers of subjects and patients, but occasionally data for one variable for one subject was incomplete. In this case the ANOVA for this variable was performed after excluding a subject at random from the other group to equalize the numbers. If the three-way ANOVA showed no difference between results from patients and subjects, two-way analysis of variance (ANOVA) was performed between weak and normal-leg data using the pooled results from all subjects in both groups. Effort scores were compared using Wilcoxon's matched-pairs signed rank test. Quadriceps strength was compared for left and right legs (normal volunteers) and for weak and strong legs (patients and subjects with unilateral femoral nerve block) also by Wilcoxon's matched-pairs signed ranks test.

RESULTS

Normal responses to single-leg exercise

There was no difference between the quadriceps strength of the left and right legs ($P > 0.05$), and the strongest leg was not consistently that innervated by the dominant hemisphere (Table 1*A*). There were no differences (two-way ANOVA)

TABLE 1. Anthropometric and leg strength data for all subjects

Subject	Sex	Age (years)	Weight (kg)	Dominant leg	MVC (kg) Right	MVC (kg) Left	Diagnosis
(A) Subjects without leg weakness							
1	M	33	83·3	R	34·8	37·2	Normal
2	M	26	65·0	R	28·6	25·1	
3	M	23	81·0	R	46·8	39·6	
4	M	33	80·0	R	43·0	41·3	
5	M	39	70·0	R	45·8	38·6	
6	M	27	71·5	R	32·9	27·4	
7	F	26	58·5	R	35·4	30·5	
8	F	26	53·4	R	22·9	13·6	
9	M	27	67·0	R	32·4	40·0	
(B) Subjects with one weak leg							
Orthopaedic weakness							
10	M	26	72·4	R	42·6	16·2	Ruptured quads tendon
11	M	55	72·0	R	16·2	40·2	Meniscus injury
12	F	32	51·4	R	26·7	16·0	Fractured fibula
13	M	40	74·0	R	24·7	4·2	Fractured hip
14	F	36	44·8	R	6·7	51·2	Cruciate ligament tear
15	M	48	82·0	R	30·2	20·4	Cruciate ligament tear
Neurological weakness							
16	M	32	90·2	R	40·9	51·2	Idiopathic neuropathy
17	M	58	81·0	L	26·4	14·2	Cerebrovasc. accident
Femoral nerve block							
1	M	34	83·3	R	39·2	12·1	Normal
5	M	39	70·0	R	35·6	48·3	
6	M	27	71·5	R	1·1	37·2	
7	F	27	58·5	R	33·0	18·1	
9	M	27	65·5	R	41·3	8·0	
18	F	27	59·0	R	11·6	25·3	
19	F	26	79·5	R	23·0	10·2	
20	F	22	73·0	R	9·0	37·7	

between left and right leg exercise responses for $\dot{V}_{O_2}$ ($P = 0.71$) and arterialized venous lactate level ($P = 0.73$). Effort scores were higher during exercise with the left leg (Wilcoxon's test; $P < 0.05$, Fig. 2, upper panel). Comparing left and right leg responses (two-way, ANOVA; Fig. 3, upper panel), there were no differences for ventilation ($\dot{V}_E$; $P = 0.61$), tidal volume (V_T; $P = 0.20$) and P_{ET,CO_2} ($P = 0.51$); however respiratory rate was greater during exercise with the left leg ($P < 0.05$). Similar comparison of cardiovascular variables (Fig. 4, upper panel) showed no differences for cardiac output ($P = 0.65$), stroke volume (SV; $P = 0.70$) and heart rate ($P = 0.87$).

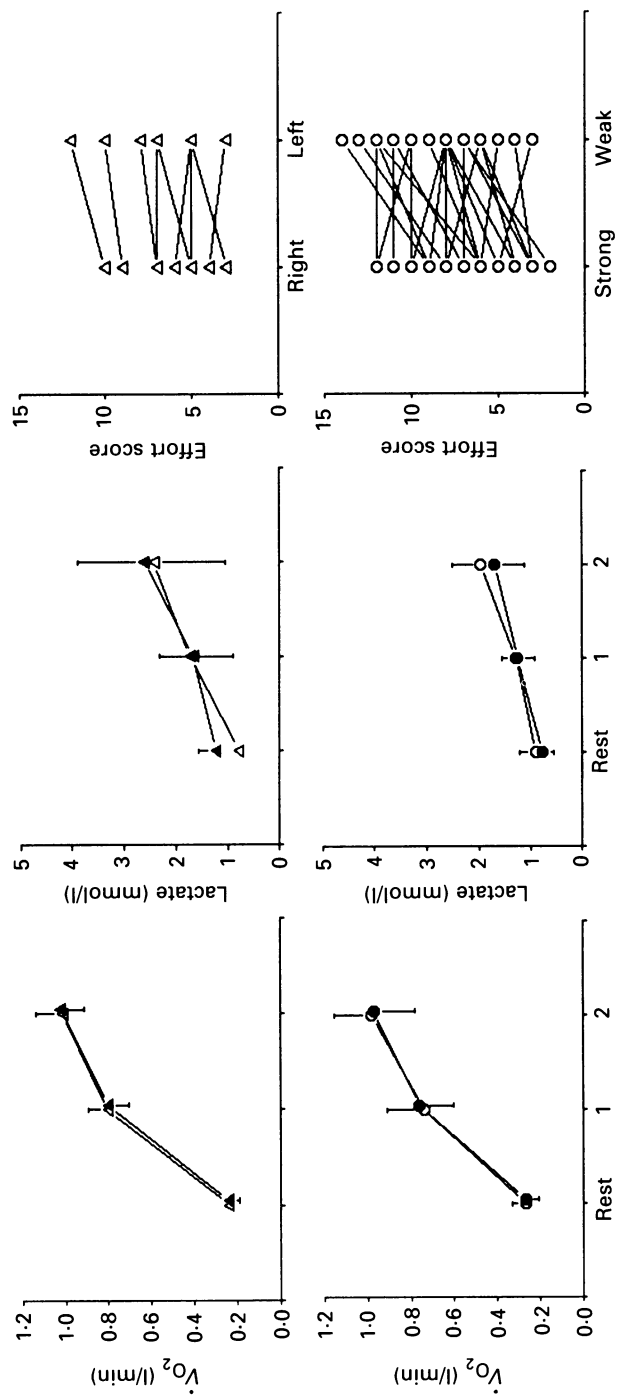

Fig. 2. Metabolic data. Upper panel (subjects without leg weakness), mean (±S.D.) $\dot{V}_{O_2}$ and arterialized venous lactate and individual effort scores in subjects at rest and exercising at two workloads matched for O_2 consumption (see text) with the right (△——△) and left (▲——▲) legs. The maximum voluntary isometric contraction of the quadriceps of each leg is shown in Table 1*A*. The right leg is invariably innervated by the dominant hemisphere. Lower panel (subjects with unilateral leg weakness), mean (±S.D.) of O_2 consumption and arterialized venous lactate and individual effort scores of subjects with unilateral leg weakness due to orthopaedic or neurological disorders or femoral nerve block (Table 1*B*) at rest and during exercise at two workloads matched for O_2 consumption with the weak (○——○) and strong (●——●) legs.

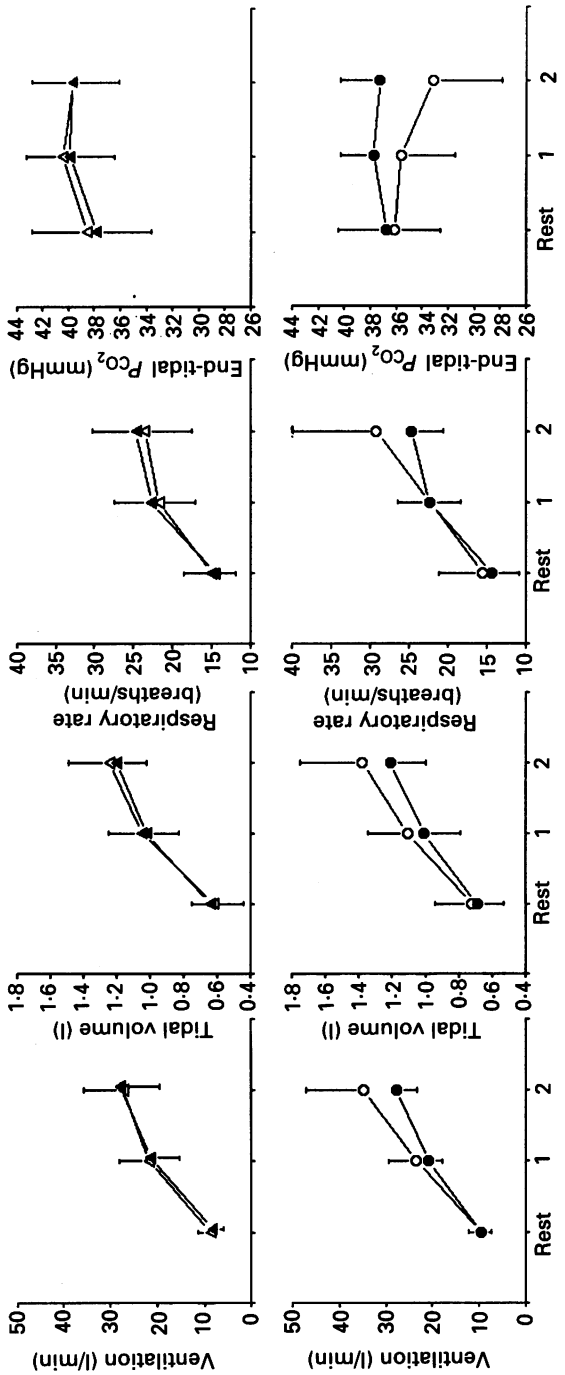

Fig. 3. Ventilatory data. Upper panel (subjects with no weakness), mean (±S.D.) ventilatory variables during rest and two levels of steady-state exercise with matched O_2 consumption with the right (△——△) and left (▲——▲) legs (see text). Lower panel (subjects with unilateral leg weakness), mean (±S.D.) ventilatory variables during rest and two levels of exercise with matched O_2 consumption with the weak (○——○) and normal (●——●) legs (see text).

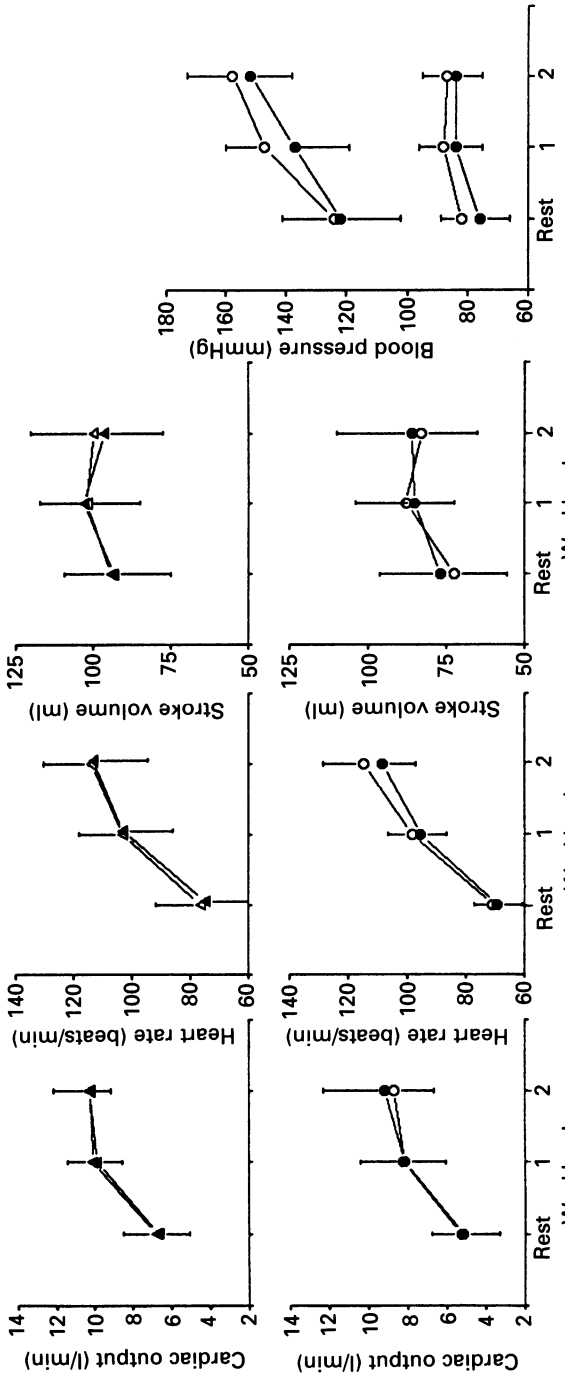

Fig. 4. Cardiovascular data. Upper panel (subjects with no weakness), mean (±S.D.) cardiovascular variables at rest and during two levels of steady-state exercise with matched O_2 consumption with the right (△——△) and left (▲——▲) legs (see text). Lower panel (subjects with unilateral weakness), mean (±S.D.) in the weak (○——○) and normal (●——●) legs during two levels of steady-state exercise with matched O_2 consumption in subjects with unilateral leg weakness (see text). Blood pressure data were obtained only in subjects with leg weakness.

Subjects with one weak leg

The strength of the weak legs of the neurological and orthopaedic patients varied between 17 and 80% of that of the normal leg (Table 1*B*). Percutaneous lignocaine femoral nerve block resulted in variable quadriceps weakness with minimum strength ranging from 4 to 73% of the strength of the normal leg (Table 1*B*) and a varying degree of numbness to light touch and pin-prick in the area of the cutaneous distribution of the femoral nerve. Both weakness and numbness began to appear after 5–10 min, were maximal at 20–30 min and subsided almost completely by 90 min following injection. The knee-jerk reflex was abolished in all eight subjects.

Three-way ANOVA revealed that there was no difference between the effect of unilateral leg weakness on cardiovascular or ventilatory variables in the patients and normal subjects with femoral nerve block (P values between 0·27 and 0·95) with the exception of V_T ($P < 0·02$; patients breathed more deeply during both weak and normal leg exercise). The data for all patients and subjects with leg weakness were therefore pooled. Despite the unilateral leg weakness ($P < 0·001$, $n = 16$), there was no difference between the workloads required to obtain matched levels of $\dot{V}_{O_2}$ with both legs, the lower workloads being $23 \pm 7·8$ (mean $\pm$ S.D.) and $25 \pm 7·0$ W, and the higher workloads $41 \pm 12·3$ and $42 \pm 10·9$ W for the weak and strong legs respectively. The aim of comparing cardiorespiratory responses at matched metabolic workloads was achieved; there was no difference between $\dot{V}_{O_2}$ during exercise with the weak and the normal leg (Fig. 2, lower panel, $P = 0·51$, $n = 16$). Blood lactate increased with increasing exercise level (Fig. 2, lower panel); however it stayed below 2 mmol/l. There was no difference between lactate levels during exercise with the weak and normal legs ($P = 0·06$, $n = 14$). Effort score was higher during exercise with the weak leg (Fig. 2, $P = 0·001$, $n = 16$). Ventilation during exercise with the weak leg was also greater than with the normal leg ($P < 0·02$, $n = 16$, Fig. 3, lower panel), and the degree of exaggeration of ventilation was dependent upon the workload ($P < 0·05$). The enhanced ventilatory response to exercise was mainly due to increased V_T during exercise with the weak leg ($P < 0·005$, $n = 16$, Fig. 3, lower panel). P_{ET,CO_2} was correspondingly lower during exercise with the weak leg (Fig. 3, lower panel, $P < 0·01$, $n = 15$), and the difference was dependent upon the work rate ($P < 0·05$). Heart rate during exercise with the weak leg was greater than with the normal leg ($P < 0·005$, $n = 16$, Fig. 4, lower panel); however there was no difference between stroke volume or cardiac output during exercise using the weak and normal legs (Fig. 4, lower panel; $P = 0·78$ and $P = 0·25$, respectively, $n = 13$). Both systolic and diastolic blood pressures were greater during exercise with the weak leg (Fig. 4, lower panel, $P = 0·01$ and $P = 0·02$ respectively, $n = 12$).

DISCUSSION

This study provides evidence that dynamic exercise using a limb with neuromuscular weakness results in ventilatory, blood pressure and heart rate responses which are exaggerated out of proportion to the work achieved and to the responses seen at the same metabolic rate when using the normal (contralateral) limb. If increased central neural drive is required to achieve a given level of work in

the presence of weakness, then these results may be interpreted as evidence for a neurological feedforward mechanism linking the efferent central neural drive to the cardiovascular and respiratory responses. Several important assumptions underlie these conclusions. Clearly we were unable to directly quantify the central neural drive present during unilateral exercise. Although our hypothesis assumes that central neural drive will be increased when weakness is present, it is conceivable that the ability to maximize central neural outflow may depend on intact peripheral nerves and muscles, an intact spinal cord or on intact afferent feedback from the target muscles. Our best measure of the prevailing central neural drive was the score indicated by the subjects in response to the question 'how hard are you having to try to do this work?'. Previous observations in subjects with lesions of varying severity in the pyramidal tracts (Gandevia, 1982) showed that the reported sense of effort with movement was increased in the presence of weakness unless the limb was completely paralysed. This is in agreement with the results of Galbo *et al.* (1987) who used neuromuscular blockade with curare to induce temporary weakness in normal subjects. In the presence of partial unilateral weakness induced by neuromuscular blockade, Gandevia & McCloskey (1977) found that normal subjects overestimated the weight of objects, suggesting that they were rating weight according to the efferent motor drive necessary to perform the tasks and not the afferent feedback from the limb.

In the present study, we found that as a group, the subjects scored that they were having to try significantly harder (at a given metabolic rate) when using the weak limb to exercise, but surprisingly this was not true for every subject (Fig. 2). In addition, the normal subjects reported they were having to try significantly harder to exercise with the left (non-dominant) leg, despite no difference in strength. We therefore regard the subjects' category-scaled effort scores as an imperfect measure of central neural drive, but the best available to us.

Even if central neural drive is increased, the ability to distinguish a 'cortical irradiation' (Krogh & Lindhard, 1913) or feedforward component of the exercise responses will also depend on the fact that we are studying parameters which are subject to intact feedback control mechanisms. Thus disproportionate ventilation will be partly constrained by hypocapnia and respiratory alkalosis, and the observed degree of excess ventilation will thus be a complex function of the presumed central neural drive and the gain of the chemoreceptor feedback loop. Similarly, inappropriate blood pressure responses would be constrained by the arterial baroreceptors, leaving an 'error signal' whose magnitude would depend both on the central neural drive and the baroreceptor sensitivity.

Differences between responses with weak and strong legs may represent differences between the exercising muscles at the structural or biochemical level. Such differences might result in altered blood-borne or afferent neural information from the exercising limb changing the response. The design of the study was intended to avoid the pitfall of comparing a weak limb exercising anaerobically with a control limb exercising aerobically; the arterialized venous lactates demonstrate that the measurements were made at workloads where both legs were using principally aerobic pathways. It remains possible that the fibre composition differed between weak and the normal limbs in the subjects with unilateral weakness leading to differences in aerobic

capacity or afferent neural traffic from the leg. *In vitro* studies in the mouse (Crow & Kushmerick, 1982) and the cat (Folkow & Halicka, 1968) have shown a lower yield of tension per molecule of ATP in fast-twitch compared to slow-twitch fibres. Furthermore, Petrofsky & Lind (1980) showed in the cat gastrocnemius muscle that if fast-twitch fibres are selectively weakened using decamethonium, the strength of the exercise-pressor reflex (a reflex subserved by group III and IV muscle afferents) is diminished more than if the slow-twitch fibres are selectively weakened using tubocurare. This suggests an uneven distribution of afferent endings within the muscle. We have no direct evidence regarding the distribution of fibre types in the present studies, but in subjects who are weak due to immobilization of a limb there is evidence from previous work that atrophy of both slow and fast-twitch fibres occurs in approximately equal proportion (Sargeant, Davies, Edwards, Maunder & Young, 1977).

The influence of sensory feedback

In this study, both neurological cases had mild sensory abnormalities in the weak limb, and the eight subjects with femoral nerve blocks all had diminished sensation in the femoral nerve distribution. The study of Fernandes *et al.* (1990), who studied a group of subjects during cycle exercise before and during epidural anaesthesia, indicates that normal ventilation and heart rate responses can persist despite objective evidence that afferent traffic from the exercising muscles was impaired (attenuated post-exercise pressor response). Blood pressure responses, in contrast, were significantly lower during anaesthesia, which the authors interpreted as dependence of blood pressure on afferent feedback; however, it is also possible that during epidural block these subjects were unable to generate normal vaso-constriction in non-exercising vascular beds. It could be argued that, during epidural block, reduced afferent information reduced ventilatory stimulation but that this effect was masked by increased central neural drive (due to weakness) which stimulated ventilation. The epidural anaesthesia, however, reduced static leg strength by only 20%, while the corresponding sensory loss was profound, so such cancelling effects are unlikely. This apparently small degree of dependence of the responses on peripheral feedback during dynamic exercise contrasts with the earlier studies of Mitchell *et al.* (1989) who studied cardiovascular responses to static exercise during epidural blockade and found that in this situation heart rate and blood pressure responses occurred in proportion to the force achieved rather than the magnitude of the central command. It seems likely that the control of responses to static and dynamic exercise will differ substantially, since the impairment of muscle perfusion by static contraction will promote early tissue hypoxia and acidosis, and the vascular conductance of the exercising muscles will fall during static exercise in contrast to the rapid rise seen in dynamic exercise (De Cort, Innes, Barstow & Guz, 1991).

When afferent feedback is intact but strength is reduced by partial neuromuscular blockade with curare, Asmussen *et al.* (1965) and Galbo *et al.* (1987) found evidence of exaggerated ventilatory responses to dynamic exercise (as in the present study); however blood pressure and heart rate responses were similar for matched metabolic workloads with and without curare. We have no explanation of why Asmussen *et al.*

and Galbo *et al.* found no evidence of an effect of central neural drive on blood pressure and heart rate, whereas this was apparent in the present studies. Klausen, Secher, Clausen, Hartling & Trap-Jensen (1982) found indirect evidence of a central neural influence on heart rate during a one-legged training protocol. Morgan, Raven, Drinkwater & Horvath (1973) also suggested that the increased heart rate observed during hypnotic suggestion of high exercise level was a result of central neural influence.

In conclusion, during dynamic exercise, the present study suggests that the ventilatory, blood pressure and heart rate responses to exercise are influenced by the magnitude of central neural drive; however cardiac output appears to be closely linked to the prevailing metabolic rate.

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