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CARDIOVASCULAR AND VENTILATORY RESPONSES TO DYNAMIC EXERCISE DURING EPIDURAL ANAESTHESIA IN MAN

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SUMMARY

1. In order to evaluate the importance of afferent neural feedback from the working muscles for cardiovascular and ventilatory responses to dynamic exercise, epidural anaesthesia was induced at L3–L4. Six healthy males cycled for 20 min at 57% of maximum oxygen uptake and for 8–12 min at increasing work intensities until exhaustion at 238 ± 30 W without as well as with epidural anaesthesia.

2. Presence of afferent neural blockade was verified by cutaneous sensory analgesia below T10–T11 and attenuated post-exercise ischaemic pressor response (45 ± 8–24 ± 6 mmHg). Efferent sympathetic nerves appear to be intact since basal heart rate and blood pressure as well as the cardiovascular responses to a Valsalva manoeuvre and to a cold pressor test were unchanged.

3. During dynamic exercise with epidural anaesthesia, blood pressure was lower than in control experiments; however, ventilation and heart rate were not affected.

4. The results indicate that afferent neural activity from the working muscles is important for blood pressure regulation during dynamic exercise in man but may not be necessary for eliciting the ventilatory and heart rate responses.

INTRODUCTION

During exercise the cardiovascular and respiratory responses increase with the intensity of physical activity and both a central neural mechanism (feedforward control called 'central command') as well as reflex neural mechanisms (feedback control) from exercising muscles have been proposed to be involved in the regulation of these responses (Mitchell, 1985). In human subjects, experiments with partial neuromuscular blockade have provided evidence that cardiovascular and ventilatory responses to dynamic exercise are dominated by the actual activity performed by the working muscles as expressed by their oxygen uptake rather than by the intended effort (Galbo, Kjær & Secher, 1987; Kjær, Secher, Bach & Galbo, 1987). The view

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that feedback control may be of major importance for cardiovascular and respiratory regulation during dynamic exercise in man is also supported by similar cardiovascular and ventilatory responses to low-intensity leg exercise whether performed voluntarily or produced by direct electrical muscle stimulation (Krogh & Lindhard, 1917; Asmussen, Nielsen & Wieth-Petersen, 1943; Adams, Garlick, Guz, Murphy & Semple, 1984). Also in agreement with a reflex control of the cardiovascular responses to exercise originating from working muscle is the finding that putative chemical stimulation of afferent nerves in the working muscles by occlusion of their blood supply after exercise results in augmented heart rate and blood pressure (Alam & Smirk, 1937, 1938*a*; Rowell, Hermansen & Blackmon, 1976; Freund, Rowell, Murphy, Hobbs & Butler, 1979).

In light of the above described evidence in favour of a reflex neural mechanism primarily determining the cardiovascular and ventilatory responses to dynamic exercise and the fact that epidural anaesthesia has been shown to diminish the heart rate and blood pressure responses to static exercise as compared to neuromuscular blockade (Mitchell, Reeves, Rogers & Secher, 1989; Kjær, Secher, Reeves, Mitchell, Bach & Galbo, 1989*b*), it is surprising that the cardiovascular as well as ventilatory responses to dynamic exercise are not diminished in subjects studied during epidural anaesthesia (Hornbein, Sørensen & Parks, 1969; Freund *et al.* 1979). One explanation could be the extent of the motor blockade associated with the epidural anaesthesia in those experiments. If the subjects became so weak that pedalling the cycle began to resemble intermittent 'static' exercise, then similar heart rate and blood pressure responses should be expected with and without epidural anaesthesia (Mitchell *et al.* 1989; Kjær *et al.* 1989*b*). Consequently, we reinvestigated the influence of epidural anaesthesia on the cardiovascular and ventilatory responses to dynamic exercise attempting to obtain a minimum of motor weakness.

METHODS

Six healthy males (mean age 30 years (range, 20–42 years), weight 80 kg (range, 65–95 kg) and height 179 cm (range, 173–186 cm)) gave their informed consent to participate in the study which was approved by the Municipal Ethical Committee of Copenhagen. Each subject appeared in the laboratory on three occasions separated by 4–20 days. Each time he was studied semi-supine behind a Krogh cycle ergometer, the upper part of the body forming an angle of 45 deg with the couch. The feet were placed in shoes fastened to the pedals in order to keep tight contact and the subjects were able to watch their feet move during the experiment. On the first experimental day, graded exercise to exhaustion was carried out to establish the relationship between oxygen uptake and work load. The maximum oxygen uptake determined during semi-supine bicycling was 3·25 l min^{-1} (range, 2·68–4·63 l min^{-1}). This type of semi-supine cycling has been found to yield a maximum oxygen uptake corresponding to $91 \pm 3\%$ (mean and stand error of the mean(S.E.M)) of the value obtained during ordinary upright cycling (Galbo *et al.* 1987). Following the first study, the subject returned in random order for a control experiment on one day and for an experiment with epidural anaesthesia on another day.

On the day of the control study, the subject arrived postabsorptive (10 h fasting) at 8 a.m. No tobacco and only low-intensity exercise were allowed on the day prior to the study. Subjects cycled in the semi-supine position for 20 min at a work load predicted to be 60% of their maximum oxygen uptake in this position. Subsequently, after 10 min of rest, they performed graded exercise to exhaustion on the cycle ergometer with work loads being increased by 50 W every 2 min to 252 ± 26 W, and exhaustion being reached within $10{\cdot}8 \pm 0{\cdot}5$ min. A metronome gave a pedalling frequency of 60 cycles min^{-1} and the number of revolutions completed was recorded. Work intensity in Watts was calculated from the applied weight (in kilograms) and the number of

revolutions per minute. During exercise the intensity of effort was quantified by a rating of perceived exertion on the Borg scale (Borg, 1970). Before and after exercise, brief maximum static muscle contractions were performed by both the right and left quadriceps muscle with a 90 deg knee angle. Knee extension strength was taken as the highest of three measurements made by a strain gauge connected to a Peekel measuring bridge.

On the day of the study with anaesthesia, the same procedures were performed and, in addition, a 20 gauge spinal needle was inserted in the epidural space through vertebral interspace L3–L4 employing the loss of resistance technique. Epidural anaesthesia was induced by injection of 24 ml 0·25% bupivacaine (Marcain, Astra). Sensory loss was carefully tested on both sides of the body by pin prick before and after exercise. 30 ± 6 min after drug administration sensory loss was present and the subjects began cycling for 20 min. The work load in this 20 min period was similar to that in experiments without epidural anaesthesia. After a 10 min rest, graded exercise to exhaustion was carried out by increasing work load by 50 W every 2 min to 238 ± 30 W and exhaustion was reached in $9{\cdot}9 \pm 0{\cdot}8$ min.

In order to evaluate the degree of sensory blockade obtained by administration of bupivacaine, post-exercise pressor response to muscle ischaemia was measured at the end of the experimental procedure. Rapid inflation of a pneumatic cuff to 300 mmHg was performed 10 s before the end of graded exercise to exhaustion and occlusion was maintained for 2 min post-exercise. This was done both during epidural anaesthesia and during control experiments in a total of six subjects (three of the subjects involved in the main study and three of the subjects recruited for supplementary studies (see below)).

In addition to the six subjects involved in the main study, four other healthy male subjects (mean age 26 years (range, 23–42 years), weight, 75 kg (range, 70–82 kg) and height 181 cm (range, 176–187 cm)) were studied for evaluation of the effect of epidural anaesthesia on the responsiveness of the cardiovascular system during a Valsalva manoeuvre and a cold pressor test of one hand. These subjects received the same anaesthesia given by the same anaesthesiologist as the subjects in the main study. Both with and without epidural anaesthesia they had the quadriceps muscle strength tested and the dermatomal level of analgesia evaluated. During the cold pressor test the subject kept one hand totally immersed in ice-water (0–2 °C) for 5 min while blood pressure and heart rate were recorded. After 20 min of rest a Valsalva manoeuvre was carried out by blowing in a face mask for 20 s at an expiratory pressure of 30 mmHg from total lung capacity. This was done three times separated by 3–5 min, and in each subject the average arterial pressure and heart rate were calculated from these attempts.

Oxygen uptake and ventilation were measured using an Ergo Oxyscreen® (Jaeger Instruments) apparatus which previously had been found to yield the same values as obtained by collecting air in Douglas bags and analysing it with O_2 and CO_2 analysers (Secher, Ruberg-Larsen, Binkhorst & Bonde-Petersen, 1974). An arterial cannula (1·0 mm internal diameter) was inserted in the left radial artery for recording of blood pressure by a Bentley transducer positioned at heart level and connected to a Simonsen and Weel machine and an Elema recorder. The electrocardiogram was recorded from precordial electrodes. Blood lactate was measured in arterial blood by an enzymatic fluorometric method (Hohorst, 1970).

Mean arterial blood pressure was calculated as one-third of the systolic plus two-thirds of the diastolic pressure. Friedman's test was used to test if changes occurred with time (Siegel, 1956) and such changes were then located by the multiple comparison procedure. The Wilcoxon ranking test for paired data (Siegel, 1956) was used to evaluate differences between epidural and control experiments. A P value of 0·05 (two-tailed testing) was considered significant.

RESULTS

Effect of epidural anaesthesia

In the basal state all variables were similar on the two experimental days. Heart rate and mean arterial pressure were 71 ± 7 beats min^{-1} and 94 ± 5 mmHg (mean and S.E.M), respectively, on control days and 69 ± 5 beats min^{-1} and 93 ± 6 mmHg immediately before epidural anaesthesia ($P > 0{\cdot}05$). Epidural anaesthesia resulted in a sensory block below T10–T11 (range), attenuated the post-exercise ischaemic

pressor response from 45±8 to 24±6 mmHg (mean and S.E.M. of values 1 and 2 min post-exercise) (Fig. 1) and reduced muscle strength to 80±5% of the control value ($P < 0.05$) (Table 1). However, epidural anaesthesia neither changed the blood pressure response to the cold pressor test nor to a Valsalva manoeuvre (Fig. 2), nor did it change resting values for heart rate, blood pressure or ventilation ($P > 0.05$).

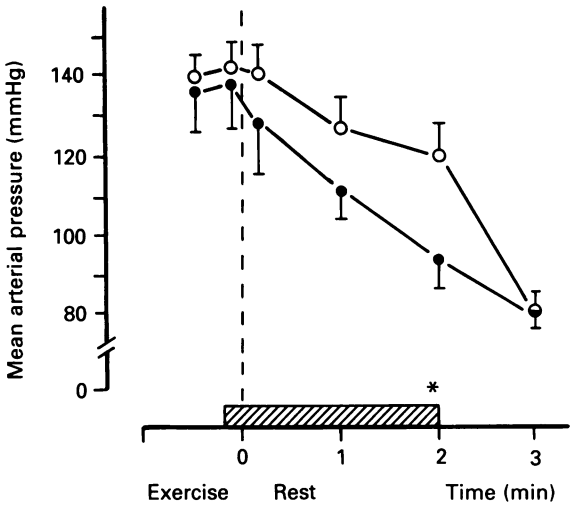

Fig. 1. Mean arterial blood pressure (mmHg) during post-exercise muscle ischaemia. A cuff was inflated around one of the exercising legs (500 mmHg) for 10 s before and 2 min after stop of maximum exercise (hatched period). Blood pressure value before cuff inflation is obtained at the end of performance of graded maximum exercise where subjects were encouraged to continue their exercise for 15–20 s. Exercise intensity in this period was not necessarily maximum. Measurements were obtained in experiments both with (●) and without (○) epidural anaesthesia, and mean±S.E.M. ($n = 6$) values are given. * Difference ($P < 0.05$) between control and epidural anaesthesia experiments.

Submaximum exercise for 20 min

Similar exercise intensities were performed in control (133±18 W) and epidural anaesthesia experiments (134±18 W), also requiring a similar oxygen uptake in the two experimental situations (1·86±0·26 (control) *vs.* 1·84±0·27 l min^{-1} (epidural anaesthesia)). Work intensity expressed relative to 'actual' work capacity (oxygen uptake as percentage of maximum oxygen uptake found during graded exercise to exhaustion at the end of the same experiment) was also similar in control and epidural experiments. Muscle strength was reduced to 71–87% of control (range of four mean values, each mean value is the mean of six individual strength values determined during one-leg contractions) during epidural anaesthesia ($P < 0.05$) and rating of perceived exertion was slightly higher in epidural anaesthesia than in control experiments ($P < 0.05$; Fig. 3). Ventilation and heart rate during exercise were similar with and without epidural anaesthesia: for ventilation 48±6 (control) *vs.* 53±8 l min^{-1} (epidural anaesthesia) and for heart rate 138±3 *vs.* 140±4 beats min^{-1} (epidural anaesthesia) (Figs 3 and 4). In contrast, during exercise blood pressure was reduced during epidural anaesthesia (120±9 mmHg) compared to control experiments (139±10 mmHg) ($P < 0.05$; Fig. 4).

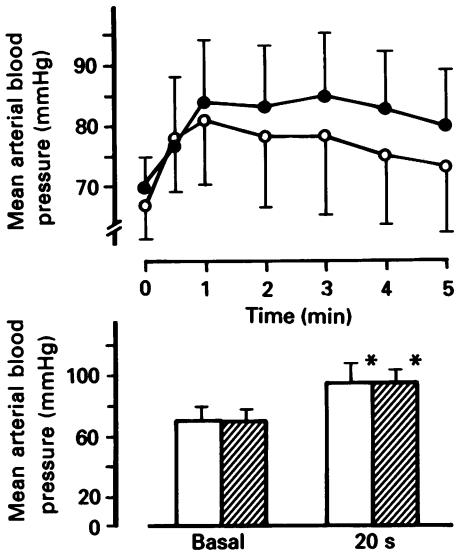

Fig. 2. Mean arterial blood pressure (mmHg) during a cold pressor test (one hand in ice water for 5 min) (upper part of the figure), and a Valsalva manoeuvre (blowing in a rubber face mask with an expiratory pressure of 30 mmHg for 20 s) (lower part of the figure). Four subjects were studied both with (● and hatched bars) and without (○ and open bars) epidural anaesthesia. Epidural anaesthesia resulted in a sensory block below T10–T11 (range) and reduced muscle strength to 73+8% of the control value. Values given are mean±S.E.M. Valsalva manoeuvre was repeated three times and in each individual the average of blood pressure changes calculated. *Significant difference ($P < 0.05$) from basal value.

TABLE 1. Sensory and motor functions during dynamic exercise performed without (control) and with lumbar epidural anaesthesia (epidural)

		Dermatomal level of analgesia		Leg strength (% of maximum control)	
		Left	Right	Left	Right
Control	Before	—	—	100	100
	After submaximum exercise	—	—	102±4	97±2
	After maximum exercise	—	—	98±2	99±3
Epidural	Before	T10–T11	T10–T11	71±6	77±5
	After submaximum exercise	T10–T12	T11–L1	74±6	87±10
	After maximum exercise	T11–L1	T11–L1	83±10	84±8

Values are range (dermatomal level) or mean±S.E.M. (leg strength) in six healthy subjects. Test before exercise in the epidural experiments was done after administration of epidural anaesthesia (30±6 min) and immediately prior to the exercise period. Spinal sensory segment projection to cutaneous sensation was tested by pin prick.

During epidural anaesthesia the increase of blood lactate concentration in response to exercise was larger (0·72±0·10 mmol l^{-1} (rest) to 3·68±0·71 mmol l^{-1} (20 min)) compared to the increase observed during exercise without epidural anaesthesia (0·72±0·10 (rest) to 1·87±0·42 (20 min)) ($P < 0·05$).

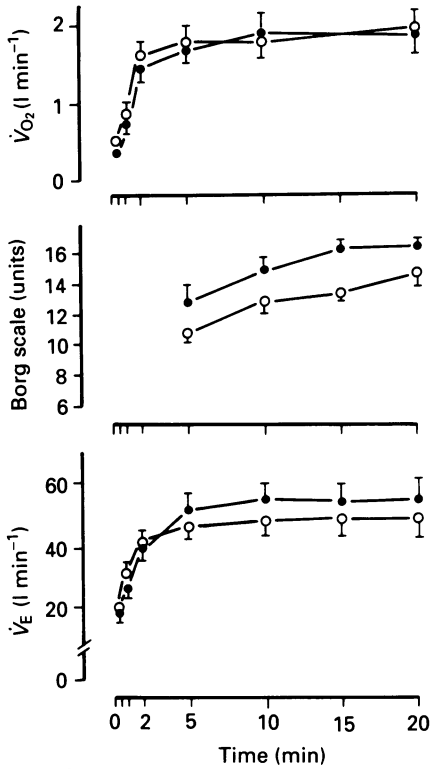

Fig. 3. Oxygen uptake ($\dot{V}_{O_2}$), rating of perceived exertion (Borg scale), and ventilation ($\dot{V}_E$) during 20 min of submaximum dynamic exercise (134±18 W) in six healthy subjects. Rating of perceived exercise was evaluated on a Borg scale ranging from 6 to 20. Values are mean±S.E.M. ($n = 6$) obtained during exercise with (●) as well as without (○) epidural anaesthesia.

Graded exercise to exhaustion

In control compared to epidural anaesthesia experiments oxygen uptake attained similar values (3·25±0·32 (control) *vs.* 3·13±0·41 mmol l^{-1} (epidural anaesthesia) being 98±4% (control) of values obtained during the preliminary graded exercise ($P < 0·05$). Muscle strength was reduced 82±5% in experiments with epidural anaesthesia. However, at a given oxygen uptake, rate of perceived exertion was similar in control and epidural anaesthesia experiments ($P > 0·05$) and, furthermore, at the point of exertion maximum values were obtained in both experiments (Fig. 5). Both ventilation and heart rate increased with oxygen uptake, identically in control experiments and experiments with epidural anaesthesia ($P > 0·05$; Figs 5 and 6). Thus, the peak ventilatory and heart rate responses obtained in control experiments could also be reached with epidural anaesthesia (ventilation: 128±13 (control) *vs.*

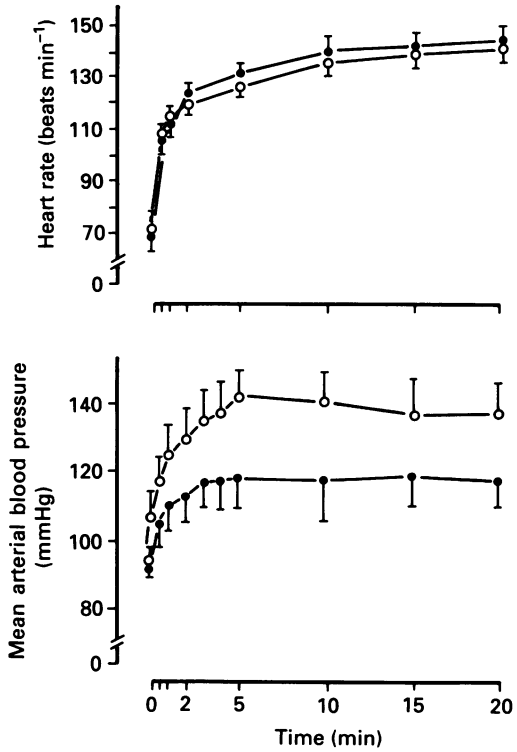

Fig. 4. Heart rate and mean arterial pressure responses to 20 min submaximum exercise (57% maximum oxygen uptake) with (●) as well as without (○) epidural anaesthesia in six heathy subjects. Values are mean ± S.E.M. Resting values given for epidural anaesthesia experiments were obtained 30 ± 6 min after administration of epidural anaesthesia and immediately before onset of exercise.

116 ± 11 l min^{-1} (epidural anaesthesia) and heart rate 179 ± 2 (control) *vs.* 177 ± 1 beats min^{-1} (epidural anaesthesia)) ($P > 0{\cdot}05$; Figs 5 and 6). However, mean arterial blood pressure was at a given submaximum as well as maximum exercise always lower during epidural anaesthesia (maximum blood pressure: 132 ± 10 mmHg) than in control experiments (maximum blood pressure: 159 ± 13 mmHg) ($P < 0{\cdot}05$; Fig. 6).

DISCUSSION

The principle finding in this study is that the arterial pressure response to prolonged submaximum as well as to graded dynamic exercise to exhaustion is lower in experiments with epidural anaesthesia than in control studies (Figs 4 and 6). This finding indicates that afferent neural feedback from the working muscles is important for the normal blood pressure response to dynamic exercise in man.

The degree of sensory blockade in our subjects is demonstrated by the finding of cutaneous analgesia below dermatomal level T10–L1 (Table 1). Furthermore, the

post-exercise ischaemic pressor response was diminished after exercise with epidural anaesthesia as compared to control experiments without sensory blockade (Fig. 1), a finding which previously has been found to result from epidural anaesthesia (Freund *et al.* 1979). The applied blockade has been shown to diminish the ACTH and

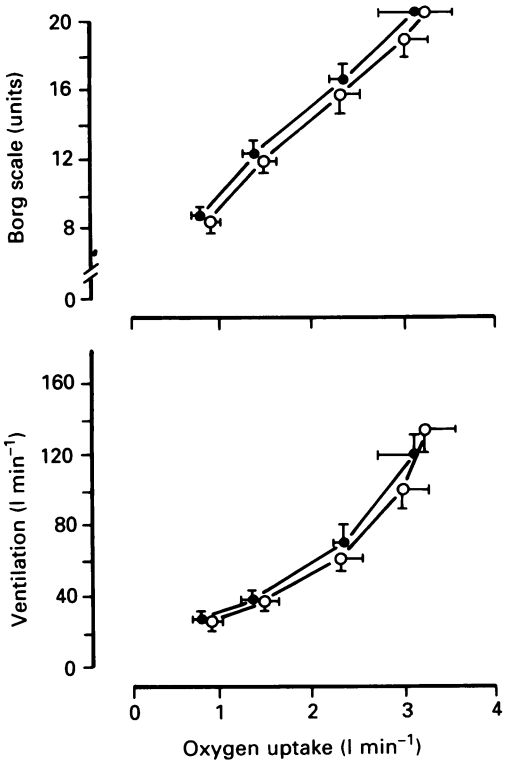

Fig. 5. Rating of perceived exertion (Borg scale) and ventilation ($\dot{V}_E$) during graded exercise without (○) as well as with (●) epidural anaesthesia in six healthy subjects. Exercise work load was increased every 2nd min by 50 W (up to 252±26 (control) and 238±30 W (epidural anaesthesia)) until exhaustion (after 10·8±0·5 (control) and 9·9±0·8 min (epidural anaesthesia)) ($P > 0·05$). Rating of perceived exertion was evaluated on Borg scale ranging from 6 to 20. Values are mean±S.E.M.

β-endorphin responses to exercise (Kjær, Secher, Bach, Sheikh & Galbo, 1989*a*). Efferent sympathetic nerves appear to be intact since basal heart rate and blood pressure as well as the cardiovascular responses to a Valsalva manoeuvre and to a cold pressor test were unchanged. Furthermore, the applied type of epidural anaesthesia does not effect plasma catecholamine values neither at rest nor during dynamic exercise (Kjær *et al.* 1989*a*).

In contrast to the conclusion stated by Freund *et al.* (1979), that during mild dynamic exercise small sensory fibres are not essential for the normal pressor response during exercise, we find that during submaximum exercise, the pressor response is markedly diminished when exercise is performed during sensory blockade. The disagreement might be due to experimental design and to interpretation of the data in the two studies. Data presented by Freund *et al.* (1979) include results from

two subjects exercising for a total of five periods at 50 W. In their experiment, epidural anaesthesia resulted in a dramatic and asymmetric reduction in leg strength, never allowing the weaker of the two legs to obtain more than 36% of the control strength. Because of this, the subjects were not able to work at 100 W. In this

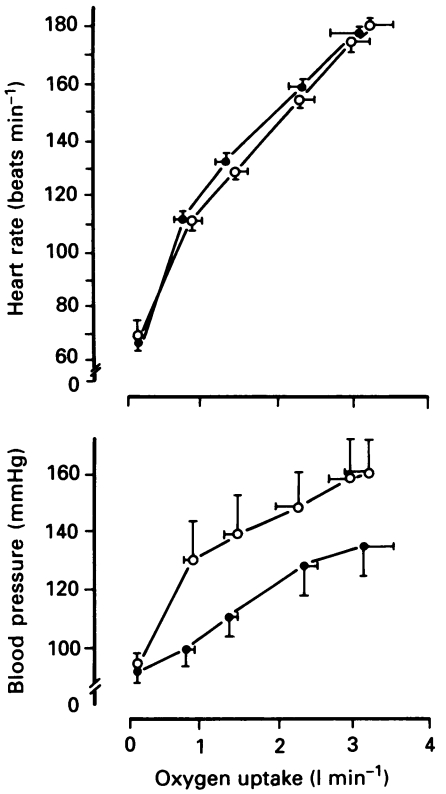

Fig. 6. Heart rate and mean arterial blood pressure responses to graded exercise to exhaustion in six healthy subjects. Exercise was performed both without (○) and with (●) epidural anaesthesia. Values are mean ± S.E.M. Values given for epidural anaesthesia experiments at rest are values obtained (60 ± 6 min) after administration of epidural anaesthesia and immediately before start of exercise. For further explanation see Fig. 5.

situation, central command is probably markedly enhanced during exercise compared to control exercise of the same intensity. An enhanced central command may have compensated for the blood pressure-lowering effect of epidural anaesthesia. During epidural anaesthesia our subjects had only a small and almost symmetric force reduction (to approximately 80% of control strength). Accordingly, they were able to work at 134 W for 20 min with only a slightly elevated rate of perceived exertion (Borg scale). Thus, in our experiments using a bupivacaine solution with a very low concentration, epidural anaesthesia probably did not require a marked increase in central command. Furthermore, reducing muscle strength as dramatically as done by Freund *et al.* (1979) might change the mode of exercise. In order to overcome a certain work load intermittent static contractions might be involved,

and static exercise is known to be accompanied by relative large increases in blood pressure (Leonard, Mitchell, Mizuno, Rube, Saltin & Secher, 1985). Also, it is likely that during cycle exercise with weak muscles, the subject used accessory muscles to accomplish the external work. In the study by Freund *et al.* (1979), oxygen uptake

TABLE 2. Blood pressure responses to 5 min of dynamic exercise without and with epidural anaesthesia

	Leg strength (% of maximum control)		Mean arterial blood pressure (mmHg)		
	Left	Right	Rest	5 min exercise 134±18 W	Δ
Control	100	100	94±5	141±8	47
Epidural	71±6	77±5	92±4	118±8	26
Data from Freund *et al.* (1979)	Left	Right	Rest	2–5 min exercise 50 W	Δ
Control	100	100	98	116	18
Epidural 1	6	20	101	130	29
Epidural 2	—	—	95	113	18
Epidural 3	—	—	94	103	9

A comparison between own data and data obtained by Freund *et al.* during control exercise and during exercise at different levels of epidural anaesthesia (1979). Own data are mean ± S.E.M. values from six healthy subjects. Data from Freund *et al.* (1979) are obtained from recordings shown in the article. One subject was studied during control exercise and during three exercise periods with epidural anaesthesia. From Epidural 1 to Epidural 3 the drug effect declined and muscle strength was reported to recover, although no data on muscle strength was given for Epidural period 2 and 3.

was not recorded during exercise, but we have provided evidence that a reduction in muscle force by 40–60% (with tubocurarine) does increase oxygen uptake required at a given absolute cycle work load, suggesting recruitment of additional muscle groups (Galbo *et al.* 1987). Thus, although sensory input from the legs to pressure-regulating centres in the brain was reduced in the experiments by Freund *et al.* (1979), overall sensory feedback from working muscles may have been similar.

In the study by Freund *et al.* (1979) it was also found that as muscle strength recovered, while sensory blockade was maintained (subject G.M. was followed in four work periods, a control period and three periods with epidural anaesthesia), results similar to ours were found (Table 2). Thus, although in the first two work periods with blockade the blood pressure response was similar or even higher than that seen in control experiments, the blood pressure response to exercise was diminished in the third period of exercise with epidural anaesthesia compared to control experiments suggesting that the effect of epidural anaesthesia on mean arterial pressure varies with the level of associated motor blockade (Table 2).

In order to study the importance of feedback from working muscles, interest has been taken in patients who have a neurological disorder causing a loss in neural feedback from muscle. In patients with congenital sensory neuropathy or syringomyelia, it has been found that the exercise-induced pressor response was identical (Alam & Smirk, 1938*b*; Duncan, Johnson & Lambie, 1981) or reduced (Lind, McNicol, Bruce,

MacDonald & Donald, 1968) compared to responses in healthy control subjects. Furthermore, a patient with Brown–Sequard syndrome was studied during one-leg cycling and it was found that whether he cycled with the weak leg, which had reduced motor function but intact sensory feedback from the working muscles, or with the leg with intact motor function, which had reduced neural feedback, the pressor response to dynamic exercise was the same at a given workload (authors' unpublished observation). However, such studies are difficult to interpret since they do not allow paired observations, it is difficult to evaluate the extent of the neurological damage, and compensatory mechanisms might develop in patients with chronic neural lesions.

Cardiac output has been shown to be determined by the actual workload (Clausen, 1977), and, therefore, was probably the same in experiments with and without epidural anaesthesia. Accordingly, the fact that blood pressure was lower in the former experiments indicates a lower total vascular resistance. Since resistance in working muscles is primarily determined by local factors (Åstrand & Rodahl, 1986), vascular resistance in the splanchnic area and/or inactive muscle was probably lower during exercise in experiments with epidural anaesthesia. In agreement with a redistribution of blood flow away from the exercising muscle, blood lactate levels during exercise were higher in experiments with compared to experiments without epidural anaesthesia. Thus, our findings suggest that in man afferent activity from working muscle is important for the exercise-induced increase in vascular resistance in the splanchnic area and/or non-exercising muscle (Rowell, 1986). In agreement with this hypothesis, animal experiments have clearly shown that an increase in blood pressure can be elicited by activity from afferent nerves in exercising skeletal muscle (Mitchell, Reardon & McCloskey, 1977).

In the present experiments the ventilatory response to both submaximum and maximum dynamic exercise were not altered by epidural anaesthesia indicating that impulses in afferent nerves from working muscles are not necessary for this response. For ventilation this is in accordance with findings by Hornbein *et al.* (1969). As discussed above, use of a high dose of local anaesthetic for lumbar blockade may markedly decrease muscle strength and, in turn, elicit an augmented central command. This may account for the enhancement of ventilation. This interpretation is in accordance with human studies in which central command was increased by partial neuromuscular blockade (Ochwadt, Bücherl, Kreuzer & Loeschcke, 1959; Asmussen, Johansen, Jørgensen & Nielsen, 1965; Galbo *et al.* 1987). In fact studies with neuromuscular blockade have indicated that both central command and reflexes from exercising muscles may enhance ventilation during exercise since ventilation increases with increased central command at a given oxygen uptake and also with increasing oxygen uptake in face of constant central command (Galbo *et al.* 1987). The view that reflex neural mechanisms may influence ventilation during exercise is supported by experiments in cats in which stimulation of afferent nerves from limbs results in an increase in ventilation (McCloskey & Mitchell, 1972; Mitchell *et al.* 1977).

As judged from the present experiments central mechanisms are sufficient also for a normal heart rate response to dynamic exercise. Experiments with partial neuromuscular blockade have indicated a predominant role of the actual exercise

performed (oxygen uptake) for control of heart rate during exercise (Galbo *et al.* 1987). Taken together, the findings support the concept that central and reflex neural mechanisms exert redundant control of heart rate during exercise (Mitchell, 1985; Galbo *et al.* 1987). However, it cannot be excluded that the similar heart rate response in experiments with and without epidural anaesthesia is due to compensatory mechanisms in the former experiments, the reduction in blood pressure diminishing baroreceptor inhibition of heart rate (Bevegaard & Shepard, 1966). Lumbar epidural anaesthesia does not influence the baroreceptors directly as shown by an unaltered relationship between changes in heart rate and blood pressure (Dohi, Tsuchida & Mayumi, 1983).

The findings in this study suggest that impulses in afferent nerves from working muscles are important for the normal blood pressure response to dynamic exercise in man. In contrast the responses of heart rate and ventilation to dynamic exercise are normal during epidural anaesthesia indicating that neural feedback from working muscles is not necessary for eliciting these responses.

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