Muscle afferent and central command contributions to the cardiovascular response to isometric exercise of postural muscle in patients with mild chronic heart failure

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ABSTRACT

The roles of muscle afferent activity and central drive in controlling the compromised cardiovascular system of patients with mild chronic heart failure (CHF) during isometric exercise were examined. Blood pressure and heart rate responses were recorded in eight stable CHF patients (ejection fraction 20–40%; age 62 ± 11 years) and in nine healthy age-matched controls during voluntary and electrically evoked isometric plantar flexion and subsequent post-exercise circulatory occlusion (PECO). During voluntary contraction, control subjects had a greater mean increase in systolic blood pressure than patients (42.4 ± 19.2 and 23.0 ± 10.9 mmHg respectively; P < 0.01), but this was not the case during PECO. During electrically evoked contraction, but not during PECO, the CHF group had smaller (P < 0.05) mean increases in both systolic and diastolic blood pressure than controls (13.0 ± 5.3 compared with 25.4 ± 14.0 mmHg and 7.6 ± 3.0 compared with 12.9 ± 7.2 mmHg respectively). Intra-group comparison between responses to voluntary and electrically evoked contractions revealed greater (P < 0.05) mean increases in systolic and diastolic blood pressure during the voluntary contraction in both the patients and the control subjects. These data suggest that muscle afferent drive to the pressor response from the triceps surae is low in this age group, both in control subjects and in CHF patients. Additionally, the patients may have a relatively desensitized muscle mechanoreceptor reflex.

INTRODUCTION

In chronic heart failure (CHF), deconditioning occurs in skeletal muscle. This results in muscle fibre atrophy, possibly a relative increase in type IIb fibre area [1], a decrease in oxidative capacity [2] and an increased reliance on anaerobic metabolism during exercise [3–7]. The metabolic products of anaerobic exercise, including the accumulation of hydrogen ions in the active muscle, are known to stimulate chemosensitive muscle afferents and may sensitize mechanoreceptive afferents [8]. However, training of the active muscle under anaerobic conditions can attenuate the pressor response [9]. Thus muscle composition and training status may influence the magnitude of the muscle afferent drive during exercise in CHF patients, as they are thought to in healthy subjects.

Key words: afferent, exercise, heart failure, muscles, reflex.

Abbreviations: ACE, angiotensin-converting enzyme; CHF, chronic heart failure; DBP, diastolic blood pressure; MVC, maximal voluntary contraction; PECO, post-exercise circulatory occlusion; SBP, systolic blood pressure.

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This has important implications for cardiovascular and respiratory control in CHF, since the training status of the active muscle, and the sensitivity of the muscle afferents to metabolic and mechanical stimuli, would hold the key to the exercise capability of the patient, within the limits set by the central circulation [11]. There is now firm evidence from studies of healthy subjects that static and dynamic training of forearm or calf muscles, under anaerobic conditions, may reduce the peripheral reflex input from that muscle during exercise [9,12–14]. There is also some evidence from studies of forearm exercise to suggest that it is possible for such adaptation to take place in CHF patients. This may occur as a result of the habitual, albeit limited, exercise taken by some CHF patients [14], or during specific supervised training of previously untrained muscles [15]. However, the role of skeletal muscle afferents in controlling the cardiovascular response to exercise in patients with CHF is still incomplete, particularly with respect to the slow postural muscles of the lower leg.

To date, investigation into the involvement of muscle afferents in the exercise responses of CHF patients has been based upon the well-established technique [16] of observing cardiovascular and respiratory responses to post-exercise circulatory occlusion (PECO) following voluntary isometric exercise [14,15]. This indicates the involvement of muscle chemoreceptor drive. During voluntary exercise, however, cardiovascular responses may be due to both central command and muscle afferent information. The cardiovascular responses to exercise in the absence of central command can be studied if electrically evoked exercise is used. Furthermore, using a period of PECO after the electrically evoked contraction, it is possible to separate the effects of the muscle chemoreceptor and mechanoreceptor elements of the peripheral reflex. Finally, comparison of the cardiovascular changes during voluntary and electrically evoked contraction performed at the same force level may indicate the role of central drive in the voluntary response [12]. The present study was designed to examine the roles of muscle afferent activity and central drive in controlling the cardiovascular system during voluntary and electrically evoked exercise of the calf muscles of patients with stable CHF.

### METHODS

#### Subjects

A total of eight patients were studied (six men) with mild stable CHF due to ischaemic heart disease. All patients had New York Heart Association (NYHA) class II–III heart failure, with a mean duration of 2.3 years (S.D. 2.2 years; range 0.5–7 years). All had left ventricular dysfunction, with an echocardiographic ejection fraction of ≤ 40% (range 20–40%). Physical and clinical characteristics are given in Table 1. Patients continued their medication during the study. None had diabetes, neuro-muscular disorders, chronic lung disease or significant valvular heart disease. Exclusion criteria included atrial fibrillation, peripheral vascular disease and β-blockade. Nine age- and sex-matched control subjects (seven men) took part in the study. These were all healthy, normotensive subjects who had no history of heart disease, peripheral vascular disease or musculoskeletal disease, and who were not taking any prescribed medication. The physical characteristics of the control subjects are also given in Table 1. The local ethical committee approved experimental procedures, and the subjects gave written informed consent before participation in the study.

#### Protocol

Established methods were used to measure muscle contractile character, and evoked and voluntary pressor responses [12,17,18]. The subject was seated with the preferred leg clamped in the dynamometer. The thigh was horizontal and the ankle joint was at 1.48 rad (85°). Upward force generated by the calf muscles was transmitted to a transducer. Output from the transducer was amplified and transmitted to an analogue-to-digital converter (Cambridge Electronic Design 1401 plus) and was displayed on a personal computer (Vale Platinum TX) and a chart recorder.

Muscle contractile character was described by twitch time to peak tension. Stimulating electrodes were placed so that the anode lay over the heads of the gastrocnemius and the cathode lay over the soleus, and twitch responses were evoked using single square-wave pulses of 50 µs. Three maximal responses were recorded, from which the mean twitch time to peak tension was calculated.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CHF patients</th>
<th>Control subjects</th>
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<tr>
<td>n</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>6/2</td>
<td>7/2</td>
</tr>
<tr>
<td>Age (years)</td>
<td>62.4 ± 11.2</td>
<td>60.7 ± 9.04</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>76.4 ± 16.1</td>
<td>80.7 ± 17.5</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170.6 ± 9.3</td>
<td>170.2 ± 9.7</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>26.4 ± 7.1</td>
<td>—</td>
</tr>
<tr>
<td>NYHA class (n)</td>
<td>II (7), III (1)</td>
<td>—</td>
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<tr>
<td>Medication (n)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diuretics</td>
<td>7</td>
<td>—</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>7</td>
<td>—</td>
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<tr>
<td>Warfarin</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>Nitrates</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>Aspirin</td>
<td>6</td>
<td>—</td>
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<td>Digoxin</td>
<td>3</td>
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Maximal voluntary contraction (MVC) force was determined as being the best of three attempts. The twitch interpolation technique was used to ascertain that maximal activation of the muscle was achieved [19], and 30% MVC was calculated and displayed on a chart recorder. For voluntary contractions, subjects maintained the required force by matching the deflection produced on the chart recorder to the pre-determined 30% MVC line. For involuntary contractions, tetanic stimulation at 20 Hz (50 μs pulse width) was used, and small adjustments of stimulus current (Digitimer D56) maintained a force output of 30% MVC. These levels of electrical stimulation are not painful, and were tolerated easily by the subjects. Blood pressure was recorded from the middle finger using a Finapres device (Ohmeda), and heart rate was recorded using a three-lead ECG and heart rate monitor (Cardiorater CR7; Cardiac Records Ltd). Analogue blood pressure and analogue ECG signals were transmitted to an analogue-to-digital converter (Cambridge Electronic Design 1401 plus). For each signal, the sampling frequency of analogue-to-digital conversion was 1000 Hz. Blood pressure and heart rate waveform data were displayed and analysed on a personal computer (Vale Platinum TX).

Subjects were habituated to the procedures during preliminary visits to the laboratory. On experimental days, two protocols of 8 min each were performed, involving either voluntary or electrically evoked isometric plantar flexion. Before the start of the protocol, the subject rested for 10 min, positioned in the dynamometer, in order to attain a stable basal circulatory state. A 2 min control period was followed immediately by 2 min of voluntary or electrically evoked contraction. A thigh cuff was inflated to 200 mmHg just prior to the start of the contraction, and this was maintained throughout the 2 min contraction. At the end of the contraction, the thigh cuff remained inflated at 200 mmHg for 2 min of PECO, and was then released for a 2 min recovery period. Heart rate and blood pressure were recorded continuously throughout the 8 min protocol. At the end of the experiment the subject rested, out of the dynamometer, for at least 20 min before the second protocol was carried out. Subjects repeated the experiments on one further occasion, when the order in which the voluntary and involuntary contractions were performed was reversed. At each time point, average values for the two experiments performed under each experimental condition were calculated for every subject.

Statistics
Data are reported as group means (± S.D) unless otherwise stated. To compare data between and within groups during each phase of the experiment, summary measures were calculated (analogous to area under the curve [20]). Comparisons of the data at key time points were made using the Mann–Whitney U-test for between-group analysis and the Wilcoxon signed rank test for within-group analysis. The criterion for statistical significance was \( P < 0.05 \).

RESULTS

Contractile responses
The group mean twitch time to peak tension of the plantar flexors was not significantly different between CHF patients and control subjects (146 ± 11 and 144 ± 15 ms respectively). There was no significant difference between groups in the MVC force produced prior to the 8 min protocol under either experimental condition (patients, 824 ± 256 N; controls, 822 ± 197 N). Similarly, within groups, the MVC forces produced before voluntary and electrically evoked contractions were not significantly different.

Cardiovascular responses

Voluntary contraction
The mean resting heart rate was not significantly different between groups prior to voluntary contraction (Table 2). During voluntary contraction, both groups produced a progressive rise in heart rate (Figure 1, top panel). On cessation of exercise, the heart rate responses of both the control and CHF groups showed the expected rapid fall to basal levels. Although the heart rate response appeared greater in the patients than the control group during the contraction (Figure 1, top panel), this difference was not significant (Table 3). There were no significant differences between the heart rate responses of the patient and control groups during the PECO or recovery phases (Figure 1, top panel).

The mean resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) of the CHF group prior to voluntary contraction were significantly lower (\( P < 0.05 \)) than those of the control group (Table 2). Both groups produced a progressive rise in blood pressure during the voluntary contraction (Figure 1, middle and bottom panels). When the contraction phase as a whole was compared between groups, the mean average SBP response was significantly higher (\( P < 0.01 \)) in the control group (Figure 1, middle panel), but the mean average DBP was not significantly different between the groups (Figure 1, bottom panel). At the end of the contraction period, the mean change in SBP for the control subjects was significantly greater (\( P < 0.01 \)) than that of the CHF patients, while the mean changes in DBP at the end of contraction were not significantly different between groups (Table 3).

During the PECO phase, on cessation of the contraction, both groups showed an immediate fall in blood pressure, but to a level significantly higher than that at rest. Blood pressure was maintained during the PECO...
Table 2  Group mean resting values for blood pressure and heart rate prior to electrically evoked and voluntary contractions

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Electrically evoked</th>
<th>Voluntary</th>
</tr>
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<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>Control</td>
<td>CHF</td>
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</tbody>
</table>
|           | 133 ± 6 | 114 ± 8 | 128 ± 7 | 107 ± 7*
| DBP (mmHg) | 72 ± 3 | 61 ± 4* | 70 ± 4 | 57 ± 3*
| Heart rate (beats·min⁻¹) | 75 ± 3 | 76 ± 7 | 75 ± 3 | 77 ± 7 |

Electrically evoked contraction

There was no significant difference between CHF patients and control subjects in the resting heart rate prior to evoked contraction (Table 2). Heart rate rose during the electrically evoked contraction and showed a prompt return to basal levels at the end of exercise (Figure 2, top panel). The mean change in heart rate was not significantly different between groups at any stage of the contraction (Table 3), PECO or recovery phases.

When the contraction phase as a whole was compared between groups, average changes in SBP and DBP (Figure 2, middle and bottom panels) were not significantly different. However, at the end of contraction the mean changes in both SBP and DBP were significantly smaller (P < 0.05) in the CHF group when compared with the control group (Table 3).

During the first 30 s of PECO, SBP and DBP fell to significantly lower levels (P < 0.05) in the control group than in the CHF group. However, during the remainder of the PECO phase, blood pressure increased gradually in the control group, so that by the end point of PECO, the change in DBP of 6 ± 4 mmHg was significantly higher (P < 0.05) than the change of 4 ± 2 mmHg seen in the CHF group (Figures 2, middle and bottom panels). When the PECO phase as a whole was compared between groups, average changes in SBP and DBP were not significantly different. During recovery, blood pressure responses were not significantly different between groups.

Comparison between voluntary and electrically evoked contractions

In the CHF patient group, the differences in the change in heart rate seen during voluntary and electrically evoked contractions failed to reach conventional significance.
Table 3  Group mean changes in cardiovascular responses at the end of electrically evoked and voluntary contractions  
Values are means ± S.D. Significant differences: *P < 0.05 compared with control group; †P < 0.05; ††P < 0.01 compared with electrically evoked value.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Electrically evoked</th>
<th>Voluntary</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>Control: 25.4 ± 14.0</td>
<td>CHF: 13.0 ± 5.3*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control: 42.4 ± 19.2††</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>Control: 12.9 ± 7.2</td>
<td>CHF: 7.4 ± 3.0*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control: 20.9 ± 9.0††</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>Control: 3.5 ± 5.0</td>
<td>CHF: 5.0 ± 3.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control: 6.7 ± 7.8</td>
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</tbody>
</table>

Figure 2  Comparison between CHF patients (■) and control subjects (□) for the changes in heart rate (HR) (top), SBP (middle) and DBP (bottom) during electrically evoked contraction  
Values are means ± S.E.M.

Figure 3  Comparison of the changes in DBP during voluntary (■) and electrically evoked (□) contraction in CHF patients  
Values are means ± S.E.M.

levels either at the end point of exercise (240 s; P = 0.09) or over the entire exercise period (P = 0.06).

When blood pressure responses to voluntary and electrically evoked contractions were compared in the CHF group, the mean changes in SBP and in DBP (Figure 3) were significantly greater (P < 0.05) both during and at the end of voluntary contraction when compared with electrically evoked contraction (Table 3), but not during the occlusion or recovery phases.

In the control group, the averaged change in heart rate over the contraction phase was significantly higher (P < 0.05) during the voluntary contraction. However, at the end point of contraction there was no significant difference in the heart rate response between the voluntary and electrically evoked contractions (Table 3). When the PECO and recovery phases were examined, there were no significant differences in heart rate between the voluntary and electrically evoked contraction conditions.

During and at the end of the voluntary contraction, the changes in SBP and DBP in the control group were significantly greater (P < 0.05) than in the evoked contraction experiment (Table 3). During PECO as a whole, average changes in SBP and DBP were also significantly greater (P < 0.01) in the voluntary contraction experiment (Figure 4). However, by the end of the occlusion phase (i.e. 360 s), blood pressure responses were the
DISCUSSION

In patients with CHF, skeletal muscle afferents may play a key role in controlling the compromised cardiovascular system during exercise. Previous studies on CHF patients have concentrated on the role of muscle afferents during voluntary forearm exercise [14,15]. Since humans are bipedal animals, and the locomotor effort intolerance of CHF patients is well documented, study of a weight-bearing muscle group seems appropriate. Until now, there has not been an investigation into the involvement of muscle afferents in the cardiovascular response to exercise of the large postural muscles of the lower leg in such patients. Furthermore, to date there has been no attempt to separate the influence of muscle mechano-receptor stimulation from that of chemoreceptor stimulation. Therefore the aim of the present investigation was to record cardiovascular responses to both voluntary and involuntary isometric exercise of the triceps surae. The purpose of the study was to attempt to unravel the relative contributions made by the muscle chemoreflex, mechanoreflex and central command in the pressor response to exercise of this important muscle group in CHF patients.

The effector pathway of much muscle afferent activity is sympatho-excitation [21], which, via muscle sympathetic nerve activity, can alter vascular resistance and thereby may affect total peripheral resistance. Since a change in DBP is a more accurate reflection of a change in total peripheral resistance than are changes in either SBP or mean arterial pressure, and since such a change is independent of baseline blood pressure [22], it is apparent that, in the present experiments, attention should be focused on changes in DBP. SBP, and thus mean blood pressure, responses seen in CHF patients must be greatly influenced by their reduced output due to myocardial weakness, and these responses are therefore difficult to compare with normal responses.

Contribution of muscle afferents to the pressor response during exercise and PECO

Contribution of the muscle chemoreflex
During evoked exercise, the rise in DBP was significantly attenuated in the CHF patients compared with the control subjects. In the absence of central command, this difference would be best explained by a relative reduction in muscle afferent drive during exercise in the CHF patients. Examination of the DBP response to the PECO phase as a whole revealed no significant difference between the responses of the patients and the control subjects following either electrically evoked or voluntary exercise. In the absence of central command or muscle mechanical activity during PECO, only muscle chemoreflex activity remains to generate the response. Therefore, on the basis of this evidence, it is unlikely that the activity of the muscle chemoreflex was significantly reduced during exercise in the CHF patients, when compared with the control subjects that we studied. However, the magnitude of the PECO response in both the control subjects and patients was much lower than that reported previously in young subjects [18]. This observation is compatible with a decreased muscle chemoreflex activation in the older subjects taking part in the present study. Furthermore, attenuation of the pressor response to isometric exercise of the triceps surae in older men has been linked to an age-related change in muscle composition of the gastrocnemius and soleus [10].

Contribution of the mechanoreflex
By a process of elimination, we are left with the possibility of an attenuated muscle mechanoreceptor reflex to explain the diminished DBP response during exercise in the CHF patients. Furthermore, the maximal strength of the CHF patients was not significantly different from that of the control subjects, and both groups performed exercise at 30% of MVC, so the mechanical conditions within the muscles of each group should have been comparable. The attenuated DBP response seen during electrically evoked contractions in the CHF patients may, therefore, indicate a desensitization of the muscle mechanoreflex.

Influence of muscle contractile character on the pressor response
The changes in DBP seen during exercise in the control subjects are in keeping with activation of a predominantly slow-twitch muscle group [23], resulting in relatively weak muscle chemoreflex stimulation. In the present study, the twitch time courses of the patient and control...
groups were not significantly different, and were similar to that expected for their age group [24,25]. This suggests that, in these particular CHF patients, there have been no additional alterations in muscle composition other than those associated with the expected age-related slowing of twitch time. This is in contrast with reports of a significantly quicker twitch time to peak tension in CHF patients than in age-matched controls [26] and with biopsy data indicating a higher proportion of fast-twitch fibres in the gastrocnemius muscle of CHF patients [2].

Our finding that the MVC force was not significantly different between CHF patients and control subjects is in agreement with other studies that have used twitch interpolation or superimposed tetanic stimulation to aid in determining whether maximal muscle activation occurred during the performance of the test [26,27]. Some studies have reported that CHF patients are weaker than normal subjects [28,29] and, where muscle size has been estimated, suggest that this is due to muscle atrophy, since specific tension (force per unit area) is not altered. Clearly, cross-sectional studies such as ours do not allow further comment on the onset and progression of muscle atrophy in CHF without a direct measurement of muscle fibre area.

**Influence of central command on the pressor response**

During voluntary exercise, central command appears to add to, or ‘top up’ [30], the cardiovascular response in the face of low muscle afferent drive in both CHF patients and controls, resulting in the greater rises in DBP seen in both subject groups. It is also clear from our data that the heart rate response was greater during voluntary exercise than during electrically evoked exercise at the same intensity. This difference was significant in the control subjects; however, although the effect appeared to be greater in the CHF patients, it just failed to reach statistical significance. Again, this would be consistent with low muscle afferent drive, particularly mechano-receptor input to the ‘muscle heart reflex’ [31] in the CHF patients. In previous studies of healthy subjects with faster muscles, the muscle afferent drive has been found to be larger, and indeed capable of sustaining the same rises in blood pressure and heart rate as seen in voluntary exercise at the same intensity [18,23]. Nevertheless, recent evidence suggests that muscle afferent input and the influence of central command can be reduced by appropriate local muscle training [9,12]. This may have important implications for exercise tolerance in CHF patients and for the design of cardiac rehabilitation programmes.

**Influence of CHF drug therapy on the pressor response**

Some of the drugs commonly prescribed for CHF [nitrates, angiotensin-converting enzyme (ACE) inhibitors] are vasodilators. Since the patients’ medication was not withdrawn during the study, it could be argued that the attenuated pressor response in the patients was due to their drug therapy, which might interfere with the effectiveness of sympatho-excitation from a baseline level of vasodilatation. However, it has been reported that, in normal subjects during the cold pressor test, blood pressure and muscle sympathetic nerve activity responses were the same before and after administration of oral nitrates [32]. In addition, it is improbable that nitrates, which were taken by only three of the eight patients, would lead to a significant effect in a small population. Furthermore, group mean changes for the three patients on nitrates were similar to those for the five who were not. With regard to ACE-inhibitor therapy, it does not affect the autonomic responses to the cold pressor test. The heart rate, blood pressure and muscle sympathetic nerve activity responses of CHF patients have been reported to be no different from those of healthy control subjects, whether the patients were taking ACE inhibitors [33] or not [34]. Secondly, there is clear evidence from studies during which CHF patient medication was uninterrupted that ACE-inhibitor therapy does not impair the ability of the sympathetic nervous system to increase activity during exercise, and nor does it appear to affect the relative magnitude of the change from baseline. A study of CHF patients who were taking ACE inhibitors, nitrates and diuretics found that the percentage increases in muscle sympathetic nerve activity and skin sympathetic nerve activity were at least as great as those in control subjects during standardized handgrip exercise [35]. In addition, Narkiewicz et al. [33] reported the ability of the sympathetic nerve activity to increase markedly in activity from already high baseline levels in CHF patients who were on a cocktail of drugs (ACE inhibitors, nitrates and diuretics). Finally, the pressor response in patients on ACE-inhibitor therapy has been reported to be equal to [36] or greater than [15] that of control subjects during handgrip exercise. Therefore it seems unlikely that our finding of an attenuated pressor response during calf muscle exercise can be explained by the effects of drugs on sympatho-excitation, since all of the above studies involved patients on similar standard drug therapy to our own.

**Comparison with studies performed using upper-limb muscles**

Our data are in contrast with a study by Piepoli et al. [15], in which untrained CHF patients showed greater increases in blood pressure after 3 min of voluntary handgrip at 50% MVC in comparison with controls. Our findings are also at variance with those of Sterns et al. [14], who found no difference between CHF patients and controls in the blood pressure response to 2 min of voluntary handgrip at 30% MVC. While precise comparison between our own CHF patients and those in
other studies [14,15] is difficult, it seems that the degree of heart failure (judged by duration, ejection fraction and NYHA class) in our CHF group was similar to [15] or slightly less severe than [14] that in the other studies. It is possible, therefore, that much of the apparent discrepancy in the findings may be due to the active muscle group chosen for study and the training status of the active muscle. An isometric contraction of an upper-limb muscle, with a greater preponderance of fast-twitch fibres, is likely to produce a bigger pressor response than that of a slow-twitch, weight-bearing, postural muscle in older subjects [23].

Training and habitual activity pattern
Training of the active muscle under anaerobic conditions can also reduce the pressor response [9]. Piepoli et al. [15] recorded the pressor response to isometric grip from an arm that had been deconditioned by rest and also after it had undergone forearm strength training. This form of training places a considerable anaerobic load on the muscle. They found that, although the blood pressure response of the CHF patients was higher than that of the control group in both untrained and trained conditions, forearm strength training reduced the magnitude of the pressor response to a greater extent in the patient group [15]. Indeed, the pressor response following training in the CHF group was very similar to that of the control group before training. In the present study, the limited cardiac output of the CHF patients would place an anaerobic conditioning stimulus on the triceps surae (which are a predominantly slow-twitch, weight-bearing muscle group) if the patients habitually attempted locomotor activity, which they did. Therefore it is likely that, in terms of the study by Piepoli et al. [15], the postural muscles of the patients included in the present CHF group were already ‘trained’.

In conclusion, we find evidence supporting low chemoreflex activation and mechanoreceptor desensitization in the calf muscle of our patients with mild CHF. This may be due to their habitual activity pattern, which highlights the need to know muscle training status and muscle composition before making a judgement on the contribution of muscle afferents to cardiovascular control in CHF patients.

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Muscle reflexes and central drive in heart failure


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