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ORIGINAL ARTICLE

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Effects of isokinetic, isotonic and isometric submaximal exercise on heart rate and blood pressure

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Abstract The purpose of the present study was to compare arterial pressure (AP) and heart rate (HR) responses to submaximal isokinetic, isotonic and isometric exercises currently employed in physical rehabilitation therapy in terms of both magnitude and time-course. To this aim AP and HR were continuously and noninvasively measured in ten healthy subjects performing isokinetic, isotonic and isometric exercises at the same relative intensity. Isokinetic and isotonic exercises consisted of 30 knee extension/flexion repetitions at 40% of maximal effort. Isokinetic speed was set at $180° \cdot s^{-1}$. Isometric exercise consisted of a 60-s knee extension at 40% maximal voluntary contraction. The AP showed a rapid and marked increase from the onset of all types of exercise progressing throughout the exercises. Peak systolic (SAP) and diastolic (DAP) arterial pressure were 190.7 (SEM 8.9) and 121.6 (SEM 7.8) mmHg during isokinetic and 197.6 (SEM 11.2) and 128.3 (SEM 7.7) mmHg during isotonic exercise, respectively. During isometric exercise peak SAP and DAP were 168.1 (SEM 6.3) and 102.1 (SEM 3.7) mmHg, respectively [both lower compared to isokinetic and isotonic exercise ($P < 0.05$)]. The HR rose abruptly and after five isokinetic and isotonic repetitions it had already increased by about 30 beats $\cdot$ min^{-1}, continuing to rise throughout the exercises. The HR response to isometric exercise was significantly less ($P < 0.05$) at all times. An immediate fall in AP, undershooting resting levels, was observed at the cessation of all types of exercise, being more marked after isokinetic and isotonic exercise. These results indicate that submaximal exercise of a dynamic type induces greater AP responses than intensity-matched isometric exercise and that even submaximal endurance-type rehabilitation exercise yields an elevated functional stress on the cardiovascular system which could precipitate hazardous events particularly in subjects with unrecognized cardiac diseases.

Key words Rehabilitation therapy · Cardiovascular hazards · Noninvasive cardiorespiratory monitoring

Introduction

Isokinetic exercise has recently gained a growing popularity in physical therapy after orthopaedic knee injuries (Morrisey 1987; Sherman et al. 1982; Thomee et al. 1987) and in recreational activities for muscle conditioning. Notwithstanding its widespread diffusion the cardiocirculatory responses to isokinetic exercise regimens have not been investigated in detail. In fact, the few studies dealing with the cardiovascular adjustments to isokinetic exercise (Hannel et al. 1992; Negus et al. 1987) have examined only the responses to maximal effort, while the cardiorespiratory responses to submaximal isokinetic protocols have not been documented. Even though isokinetic exercise has been designed to be performed at maximal effort, in clinical practice it is also employed at submaximal intensities, depending on the time elapsed from orthopaedic injury, with the aims of reducing patellofemoral joint stress, enhancing articular lubrication and producing a gradual stress on a neo-ligament after reconstructive surgery.

Thus, knowledge of the cardiocirculatory responses to submaximal isokinetic exercise protocols currently employed in physical therapy is of noteworthy clinical, besides physiological, relevance. Subjects undergoing a rehabilitation programme after orthopaedic injuries are frequently previously untrained and deconditioned from inactivity and/or may suffer from known or unrecognized cardiovascular diseases (e.g. hypertension,

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silent myocardial ischaemia, arrhythmias, etc.) which could precipitate acute dangerous events during the functional overload of the cardiovascular system imposed by not even maximal isokinetic or other exercise training regimens, such as those involving isotonic or isometric muscle contractions.

Even though isokinetic and isotonic muscle contractions are both dynamic in nature, they are different at the level of the muscle. In fact, during an isotonic contraction the resistance offered to the muscle by a load is influenced by the properties of the limb lever system, so that muscle tension varies throughout the range of motion, being maximal for only a brief time (i.e. at the extremities of the range of motion). In contrast it has been found that during an isokinetic contraction the resistance offered to the muscle is maximal during the full range of motion and, consequently, it has been stated that muscle tension would be maximal during the full range of motion (Hislop and Perrine 1967). This difference might produce arterial pressure and heart rate responses differing in magnitude, an issue which has not been addressed to date for isokinetic and isotonic exercises involving muscle masses of the same size, exercising at the same level of intensity.

The purpose of the present study was, therefore, to investigate the cardiorespiratory responses to a submaximal isokinetic protocol, in terms of both magnitude and time-course, in comparison with those induced by isotonic exercise performed at the same relative intensity. Furthermore, both types of dynamic exercise have been contrasted with isometric exercise performed at the same relative intensity, because this latter form of exercise is also currently employed in rehabilitation programmes.

Methods

Subjects

Ten healthy male volunteers [aged 22–42 years, mean height 178.4 (SEM 6.3) cm, bodymass 77.8 (SEM 9.9) kg] participated in this study. All the subjects were normotensives (sitting blood pressure < 140/90 mmHg), taking no medication and free from cardiovascular or other diseases based on medical history, physical examination and electrocardiograms (ECG) at the time of the study. All the subjects were non-smokers. Each participant gave informed consent after receiving full details of the protocol and of any known risks involved.

Experiment protocol

The experimental protocol involved three types of exercise: (1) isokinetic, (2) isotonic and (3) isometric. All the types of exercise were performed in random order on a computer-based multifunctional dynamometer apparatus (REV 9000, Technogym, Gambettola, Forli', Italy) in the seated position with the trunk supported by the chair back of the dynamometer. The subjects were familiarized with the apparatus and exercise protocols in preliminary sessions.

Isokinetic exercise

The exercise protocol consisted of 30 submaximal concentric knee extension/flexion at a speed of $180° \cdot s^{-1}$. The submaximal effort for each contraction was set at 40% of peak torque previously determined as the highest value achieved in five maximal repetitions tests performed at $180° \cdot s^{-1}$ with the dominant leg. During the exercise the subject was aided in maintaining constant as far as possible the required force at each repetition by a visual feed-back appearing on the screen of the ergometer's personal computer placed in front of him. The range of motion was 90°. The axis of the knee joint was aligned with the axis of rotation of the lever arm of the dynamometer and the inferior third of the leg was attached to the distal end of the lever arm. The subject was held in position by restraining belts around the trunk and thigh and asked to relax all the muscles not primarily involved in contraction as much as possible.

Isotonic exercise

Isotonic exercise consisted of 30 submaximal concentric contractions of knee extension/flexion each one performed at 40% of maximal effort as for the isokinetic exercise. The range of motion was 90°. The set-up configuration of the dynamometer with respect to the knee joint was the same as in the isokinetic exercise as well as the subject's position. A visual feed-back as described for the isokinetic exercise aided the subject in maintaining constant the required force for each extension/flexion repetition.

Isometric exercise

Isometric exercise consisted in 60-s sustained knee extension, with the dynamometer velocity selector set at $0° \cdot s^{-1}$, at 40% of maximal voluntary contraction (MVC). The MVC had been previously determined as the highest force developed by the subject in three trials. The knee angle was fixed at 80° (full extension considered 0°). The muscle tension at 40% MVC during the whole period of static contraction was held constant with the aid of a visual feedback as described for the isokinetic exercise.

For the isokinetic and isotonic protocols the exercising limb was weighed before starting exercise by the automatic limb weighing system of the dynamometer (REV 9000) to correct the gravitational effect on torque.

At the beginning of each experiment the subject underwent a warm-up period, consisting of 5 min of unloaded cycling and leg stretching. The determination of maximal effort was done at the beginning of each experiment after the warm-up; 5 min were allowed between the maximal strength tests.

The three exercise protocols were separated by 15-min recovery during which the subjects were allowed to move freely and perform recovery stretching.

Recorded variables

The force × time integral (i.e. the area under each tension curve) generated in each extension and flexion movement during isokinetic and isotonic exercises and during the whole period of sustained contraction during isometric exercise was evaluated. This was done to compare the amount of muscle activity in the three types of exercise, since both isokinetic and isotonic exercise produce external work, owing to their dynamic nature, while during isometric exercise only tension (force) is produced.

Systolic (SAP), diastolic (DAP), mean (MAP) arterial pressure and heart rate (HR) were continuously and non-invasively measured from the third finger of the nondominant hand using the plethysmograph method of the unloaded arterial wall (Penaz 1973) (FINAPRES, Ohmeda 2300 NIBP monitor, Englewood, Co., USA). This device has been shown to provide excellent estimates of changes of intra-arterial pressure during laboratory, including ex-

ercise, tests (Parati et al. 1989) and it has been widely used in different laboratories during exercise of different types, intensity and size of muscle masses involved (Potts et al. 1993; Iellamo et al. 1994; Papelier et al. 1994). During the data collection periods the servo-reset mechanism of Finapres was turned off to permit continuous data acquisition. Pressure traces from Finapres were visually inspected for artefacts.

The arm with the instrumented finger was held extended at heart level by means of a pulley arrangement with rubber slings supporting the arm at the wrist. This was done to avoid the effect of hydrostatic pressure on arterial pressure readings. The elbow was also held by a padded support, while the forearm was free. The subjects were not allowed to grasp any part of the apparatus with the instrumented hand.

Three ECG electrodes were also placed to monitor heart rhythm and HR on the oscilloscope of a defibrillator (HP 43120A)

Continuous monitoring of gas exchange and ventilation was performed as has been described before with the aid of a computer-based breath-by-breath system which included a mass spectrometer and two pneumotachograph (Fleisch no. 3)-pressure transducer sets (HP 47304A) connected to a mouth face-mask two-way non-rebreathing valve assembly (Hans Rudolph, model 7921) (Iellamo et al. 1992). Flowmeter and mass spectrometer calibrations were always done before each experiment using a calibrated syringe and gas mixtures of known concentrations. For the purpose of the present study only oxygen uptake ($\dot{V}O_2$) and minute ventilation ($\dot{V}_E$) are reported.

During the recovery periods among exercises, while subjects were free to move and stretch the Finapres finger cuff was left in place.

Data analysis and statistics

Reported data on arterial pressure and HR at rest represent the average of all beat-by-beat values recorded during 3.5 min before the onset of the exercise. Resting respiratory and gas exchange data are given as three-breath moving average values for the same period.

Exercise data are reported as maximal changes from rest (Δ) or as maximal absolute values attained during the exercise. For time-course analysis of MAP and HR responses, the maximal values of these variables every 5 repetitions or at each 10-s period (for isometric exercise) from the onset up to the end of exercises were calculated. For time-course analysis of the recovery phase calculations were made of the minimal MAP and HR values at each 10-s period during 60 s immediately following the end of exercises.

The significance of the differences in

1. The MAP and HR among rest, exercise and recovery within each type of exercise and
2. The rest and cardiorespiratory response values among the three types of exercise were evaluated by repeated-measures analysis of variance.

The Neuman-Keuls test was used to determine differences between means for significant main effects. Differences were considered statistically significant when P was less than 0.05.

Results

The force × time integral was significantly greater ($P < 0.05$ by ANOVA) during isometric [5353.0 (SEM 465.0) $N \cdot s$] than during both isokinetic and isotonic exercise [2406.4 (SEM 103.5) and 2616.0 (SEM 194.8) $N \cdot s$, respectively], the values for the latter being the sum of the areas under the tension curves produced by both the extension and flexion movements in the 30 repetitions. The force × time integral was not significantly different between isokinetic and isotonic exercise.

Cardiovascular and respiratory responses to exercise

All types of exercise produced significant increases in SAP, DAP and HR (Table 1). The maximal increases in SAP were 59.6 (SEM 9.1), 70.2 (SEM 10.6) and 37.2 (SEM 5.0) mmHg during isokinetic, isotonic, and isometric exercise, respectively. The DAP increases were 39.4 (SEM 6.6), 49.6 (SEM 6.4) and 23.1 (SEM 2.6) mmHg, respectively. Increases in SAP and DAP were not significantly different between isokinetic and isotonic exercise while both were significantly greater than those attained during isometric exercise ($P < 0.05$). The HR increased by 54.1 (SEM 3.8) and 58.1 (SEM 4.6) beats $\cdot$ min^{-1} during isokinetic and isotonic exercise respectively and by 25.9 (SEM 3.8) beats $\cdot$ min^{-1} during isometric exercise ($P < 0.05$ vs both isokinetic and isotonic). There were no significant differences in resting

Table 1 Cardiovascular and respiratory responses to isokinetic, isotonic and isometric exercise ($n = 10$ subjects). *SAP* Systolic arterial pressure, *DAP* diastolic arterial pressure, *HR* heart rate, $\dot{V}O_2$ oxygen uptake, $\dot{V}_E$ minute ventilation

	Isokinetic				Isotonic				Isometric			
	Rest		Exercise		Rest		Exercise		Rest		Exercise	
	mean	SEM	mean	SEM	mean	SEM	mean	SEM	mean	SEM	mean	SEM
SAP (mmHg)	131.0	3.4	190.7	8.9[a,b]	127.4	3.4	197.6	11.2[a,b]	130.9	3.7	168.1	6.3[a]
DAP (mmHg)	82.8	4.2	121.6	6.8[a,b]	78.7	3.0	128.3	7.7[a,b]	79.4	2.7	102.1	3.7[a]
HR (beats $\cdot$ min^{-1})	78.2	3.9	132.3	8.8[a,b]	79.9	4.8	138.0	6.6[a,b]	77.2	3.7	103.1	6.3[a]
$\dot{V}O_2$ (ml $\cdot$ kg^{-1} $\cdot$ min^{-1})	4.1	0.1	21.1	0.9[a,b]	3.7	0.2	20.2	0.9[a,b]	3.9	0.2	7.5	0.5[a]
$\dot{V}_E$ (l $\cdot$ min^{-1})	10.8	0.9	46.2	4.3[a,b]	9.8	0.7	44.9	5.3[a,b]	9.2	0.7	20.3	2.0[a]

[a] $P < 0.05$ vs Rest, [b] $P < 0.05$ vs isometric

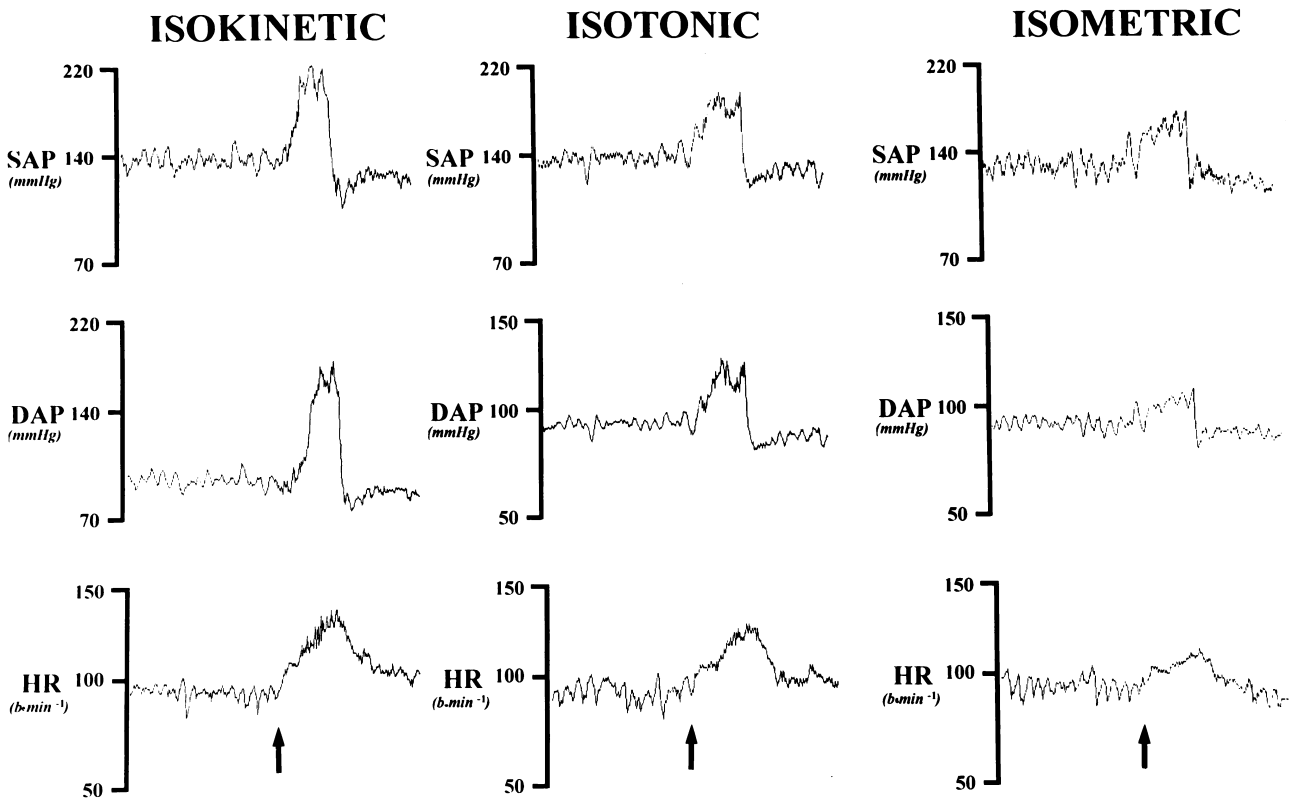

Fig. 1 Original recording from one subject showing arterial pressure and heart rate (*HR*) responses to isokinetic, isotonic and isometric exercise at 40% of maximal effort. Tracings, from *top to bottom*, are systolic (*SAP*) and diastolic (*DAP*) arterial pressure and *HR*. *Arrows* indicate start of exercise

SAP, DAP and HR values among the three types of exercise. In Fig. 1 an original recording from one representative subject of the cardiovascular responses to the three types of exercise is shown for illustrative purpose.

During isokinetic and isotonic exercise SAP attained values equal or greater than 200 mmHg in four and six out of ten subjects, respectively, with values up to 240 and 260 mmHg achieved by two different subjects during the two types of exercise. Only one subject achieved a SAP value of 200 mmHg during isometric exercise.

The increase in arterial pressure and HR during the three types of exercise was accompained by a significant increase in $\dot{V}O_2$ and $\dot{V}_E$ (Table 1, Fig. 2). The $\dot{V}O_2$ increased by 16.9 (SEM 1.0) and 16.3 (SEM 0.8) $ml \cdot kg^{-1} \cdot min^{-1}$ during isokinetic and isotonic exercise respectively. During isometric exercise the increase in $\dot{V}O_2$ was, as expected, significantly less ($P < 0.05$) than in the other two types of muscle contraction [3.8 (SEM 0.4)$ml \cdot kg^{-1} \cdot min^{-1}$]. The increase in $\dot{V}_E$ was closely similar during isokinetic and isotonic exercise [35.4 (SEM 4.1) and 35.1 (SEM 4.9) $l \cdot min^{-1}$, respectively] and it was significantly greater ($P < 0.05$) than during isometric exercise [11.1 (SEM 1.4) $l \cdot min^{-1}$].

Time-course analysis

Arterial pressure showed a rapid and marked increase from the onset of all types of exercise (Fig. 3). During both isokinetic and isotonic protocols MAP had increased by more than 25 mmHg after the completion of the first 15 repetitions and continued to rise with a less steep slope up to the end of exercise. Isometric exercise also resulted in an abrupt and progressive increase in MAP which was similar in magnitude, during the first 20 s to that observed during the first 10 isokinetic and isotonic repetitions. Thereafter the MAP response was significantly less than that observed during the two types of dynamic muscle contraction.

Immediately on cessation of both isokinetic and isotonic exercise there was a marked fall in MAP from the elevated levels achieved during exercise which undershot the rest values by 9.5 (SEM 4.8) and 5.8 (SEM 3.4) mmHg, respectively, in the first 20 s of recovery. Pooling together data from all the subjects tended to underestimate this phenomenon. In fact, analysis of individual data showed that in five out of ten subjects the MAP fall undershot rest values by 20.0 (SEM 3.5) mmHg 20 s after isokinetic exercise, this fall being significantly greater than that observed in the other five subjects [−0.2 (SEM 2.1) mmHg; $P < 0.01$ by paired Student's *t*-test]. Similarly, also after isotonic exercise there was a greater MAP fall in five out of ten subjects 20 s after the cessation of exercise [−17.4 (SEM 2.4) vs −2.4 (SEM 3.2) mmHg, respectively $P < 0.01$]. For a better appreciation of this phenomenon, MAP data for

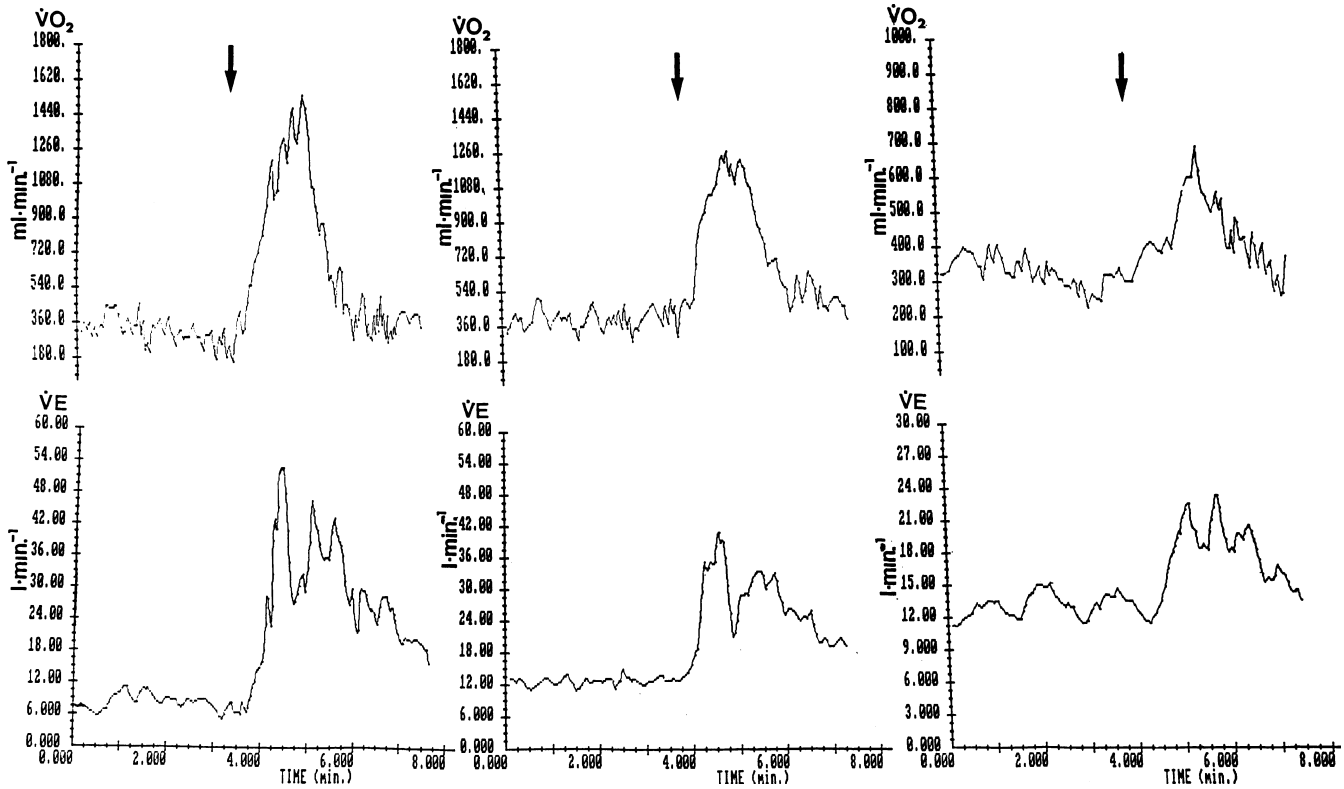

Fig. 2 Original recording from one subject showing oxygen uptake ($\dot{V}O_2$) and ventilation ($\dot{V}_E$) responses to isokinetic, isotonic and isometric exercise at 40% of maximal effort. *Left panel* isokinetic exercise, *middle panel* isotonic exercise, *right panel* isometric exercise. *Arrows indicate* start of exercise. Note the differences in the scale on the ordinates between dynamic (i.e isokinetic and isotonic) and static (i.e. isometric) exercise

this group of subjects are shown separately in Fig. 4. After the first 20 s of recovery a tendency for MAP to return toward the rest level was observed but, on average, after 60 s MAP had still not completely recovered, particularly after isotonic exercise (Fig. 4). Also at the end of isometric exercise MAP showed an immediate fall undershooting rest values by 8.4 (SEM 2.1) mmHg. As occurred for both types of dynamic exercise, analysis of individual data revealed that in six subjects the fall in MAP was more marked, with a maximal decrease of 12.8 (SEM 1.9) mmHg at the 30^{th} s of the recovery period. Here again, after 60 s MAP had not regained the rest value.

The HR featured an abrupt and marked increase at the onset of both types of dynamic exercise (Fig. 3). After the first 5 repetitions HR increases were 26.7 (SEM 3.1) and 27.0 (SEM 2.9) beats $\cdot$ min^{-1} and after 10 repetitions HR had increased by 35.7 (SEM 4.5) and 38.5 (SEM 2.0) beats $\cdot$ min^{-1} during isokinetic and isotonic exercise, respectively. As observed for MAP, thereafter HR progressively increased with a less steep slope up to the end of exercise.

At the cessation of both isokinetic and isotonic exercise HR showed a gradual decrease toward rest values and 60 s after the end of exercise HR did not differ significantly from the control values.

The HR increased abruptly during the first 20 s of isometric contraction, showing a further small increase with the progression of exercise. The increase in HR during isometric exercise was significantly less ($P < 0.05$) than that observed during both isokinetic and isotonic exercise at each time (Fig. 3). After the end of isometric exercise HR gradually recovered and after 60 s it did not significantly differ from rest.

After the end of all types of exercise $\dot{V}_E$ and $\dot{V}O_2$ rapidly returned to rest levels.

Discussion

To our knowledge this is the first study in which arterial pressure and HR responses during and in the early recovery from a submaximal isokinetic exercise have been documented and contrasted with other types of exercise currently employed in physical rehabilitation therapy. The isokinetic protocol used in the present study, which utilized prolonged submaximal repetitions at a relatively fast speed, is of the type employed to improve muscle endurance and work capacity.

Isokinetic exercise induced a striking increase in both SAP and DAP and HR. The time-course analysis showed that increases in MAP and HR were marked during the first 10 repetitions and continued up to the end of exercise. Strictly similar arterial pressure and HR responses were observed during isotonic exercise. The

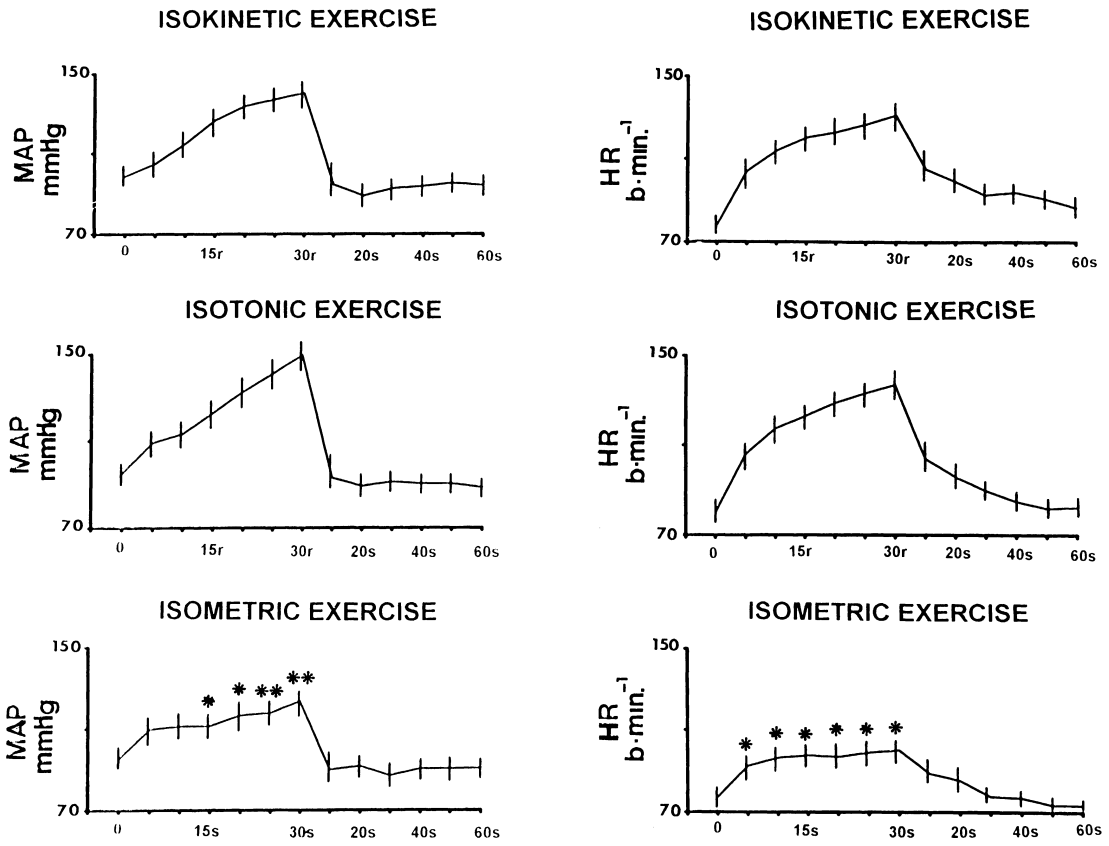

Fig. 3 Time-course analysis of mean arterial pressure (*MAP*) and heart rate (*HR*) during isokinetic, isotonic and isometric exercise and recovery. Data have been plotted for each five repetitions during isokinetic and isotonic exercise and for each 10-s interval during isometric exercise. Letters *r* and *s* beside numbers on the abscissa denote repetitions (for isokinetic and isotonic exercise) and seconds (for isometric exercise, respectively). Recovery data have been plotted for each 10-s interval from exercise cessation. $^{*}P < 0.05$ versus isokinetic and isotonic exercises using ANOVA

lack of significant differences in arterial pressure and HR responses between isokinetic and isotonic exercise could be explained by the submaximal intensity of the protocol used. It could be argued that at submaximal intensities the maximal tension produced by isotonic exercise at the extremities of the range of motion (Hislop and Perrine 1967) would compensate for the maximal tension developed throughout the full range of motion during isokinetic exercise. This suggestion would be supported by the lack of a significant difference in the tension × time integral between isokinetic and isotonic exercise.

Unexpectedly, the arterial pressure response to both types of submaximal dynamic exercise was significantly greater than the pressor response to isometric exercise performed at the same relative intensity. Several mechanisms might have contributed in producing this new and somewhat unexpected finding.

The marked increase in arterial pressure observed in response to both isokinetic and isotonic exercise might have been caused by the mechanical compression of the contracting muscles with the accompanying pressor reflex as reported by Mitchell et al. (1983). Indeed, concentric isokinetic contractions have been reported to produce an elevation in intramuscular pressure of such a magnitude as to restrict blood flow in the contracting muscles (Aratow et al. 1993). Evidence has also been provided that dynamic exercise of moderate to high intensity results in activation of a muscle "chemoreflex" (Joyner et al. 1992; Victor and Seals 1989), with a resultant increase in vasoconstrictor sympathetic outflow to nonexercising (Victor and Seals 1989) and, possibly, also to exercising (Joyner et al. 1992; Rowell 1993) muscle resistance vessels. The involvement of such a mechanism is conceivable taking into consideration the progressive increase in arterial pressure up to the end of the dynamic exercises observed in this study.

It should also be kept in mind that the muscle mass engaged in dynamic exercise was of an intermediate level in this study and thus the metabolically-induced vasodilatation in the active muscles, which normally opposes the sympathetically-induced vasoconstriction, should have had a lesser effect on the total systemic conductance than would have been expected for dynamic exercise of larger muscle masses. Last, and possibly most important, the marked increase in HR, and thus in cardiac output, may have afforded a considerable contribution, in addition to the vasoconstriction, to the progressive increase in arterial pressure. During the isometric exercise the increase in HR was in fact 50% less than during isokinetic and isotonic exercise.

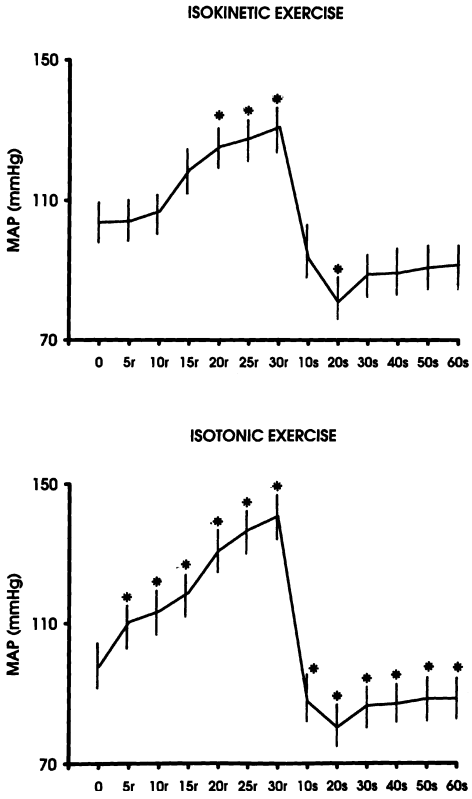

Fig. 4 Time-course analysis of mean arterial pressure (*MAP*) during isokinetic and isotonic exercise and recovery of a group of subjects ($n = 5$) showing a striking fall of arterial pressure at cessation of exercise. $^*P < 0.05$ versus rest using ANOVA. Definitions as in Fig. 2

Obviously, our results should not be generalized in relation to other different combinations of exercise intensity, duration and/or masses of active muscle engaged, all of which have been shown to affect the arterial pressure response to exercise (Mitchell et al. 1980; Rowell 1993).

Marked increase in both SAP and DAP have been reported in other studies in which the cardiovascular responses to strength or endurance maximal isokinetic (Hannel et al. 1992; Negus et al. 1987) and heavy isotonic one-leg exercise (MacDougall et al. 1992) have been investigated. However, in these studies (Hannel et al. 1992; MacDougall et al. 1992; Negus et al. 1987) the arterial pressure response to dynamic exercise has not been found to be significantly different from that observed during isometric exercise of comparable intensity. This discrepancy between the above and the present study could be related to differences in the exercise protocols employed.

Hannel et al. (1992) have compared the arterial pressure response to 20-s maximal isometric contraction of knee extensors with that elicited by the maximal number of maximal isokinetic repetitions that subjects were able to perform in a 20-s period. This isokinetic protocol resulted in a gradual degradation in the peak torque height as repetitions progressed and fatigue ensued (Hannel et al. 1992). This could have resulted in a relative reduction of the pressor response, which is affected by variations in the tension exerted by the contracting muscles. In our protocol an examination of the torque versus angle loops demonstrated a good reproducibility of each repetition for the whole duration of isokinetic exercise with a decline in mean peak torque from the first 3 to the last 3 repetitions of about 5%. Mac Dougall et al. (1992) have examined the arterial pressure response to 30 s of continuous static exercise in comparison to that induced by 10 intermittent, 3-s-duration, weight lifts by the knee extensor muscles, the weight being lowered by assistants after each lift. This protocol resulted in blood pressure peaks and falls with each contraction (MacDougall et al. 1992).

In contrast, in our study both dynamic exercise protocols were continuous, involving knee extension-flexion movements without interposed pauses between contractions. Indeed, we did not observe arterial pressure falls during isokinetic and isotonic exercise.

Compared with the large load on the cardiovascular system, the metabolic cost of isokinetic and isotonic rehabilitation exercises was not high, amounting to about 6 mets (1 met = resting metabolic rate). The relatively low metabolic cost of both dynamic exercises could be due to the single limb being involved in exercise. Compared to whole body exercise, e.g. cycling or running on a treadmill, single limb exercise is associated with greater haemodynamic responses in relation to absolute $\dot{V}O_2$, while the muscle mass being exercised is a major determinant of total metabolic cost of the work performed.

In the immediate recovery from isokinetic and isotonic resistance exercise a fall in blood pressure, undershooting resting level, was observed (about 20 mmHg in half of our subjects). The fall in blood pressure was somewhat less marked at the end of isometric exercise. It has been reported that this immediate fall in blood pressure at the end of exercise is likely to be due to the sudden perfusion of an extremely vasodilated muscle mass as the exercise stimulus abruptly ceases but alteration in the reflex control of the circulation from arterial and/or cardiopulmonary baroreceptors could also contribute (MacDougall et al. 1985).

In contrast to the blood pressure, the HR decline after the cessation of exercise was more gradual and after 1 min it had completely recovered. Also $\dot{V}O_2$ and $\dot{V}_E$ returned rapidly to rest levels.

Clinical implications

There are evident clinical implications ensuing from the present study. It demonstrated that even submaximal endurance-type rehabilitative exercise yields a surprisingly elevated functional stress on the heart. The marked increase in HR observed from just the first repetitions, rapidly reaching values above 100 beats $\cdot$ min^{-1}, has been reported to imply an abrupt and massive increase

in adrenergic outflow to the heart (see Rowell and O′Leary 1990) which may precipitate arrhythmic events. Indeed, we observed isolated ventricular ectopic beats (VEB) in some and one VEB couplet, at peak isokinetic exercise, in one out of our ten healthy and sedentary subjects. These potentially harmful events would obviously be worsened in ischaemic patients, especially if coronary artery disease is unrecognized or silent. Episodes of arrhythmias and hypertensive paroxysms requiring pharmacological interventions have indeed been reported in cardiac patients as well as in healthy subjects during post-traumatic physical rehabilitation therapy (Bianchi et al. 1989).

Furthermore, the immediate fall in blood pressure observed at the end of dynamic exercise may have been responsible for the temporary lipothymic symptoms we have observed in some healthy subjects at the end of maximal isokinetic exercise (unpublished observations) and which have also been reported by others (MacDougall et al. 1985) immediately after heavy lifting. Implications for patients with cerebrovascular disease are evident here.

The findings of the present study thus suggest the need for a careful cardiovascular examination before subjects undergo orthopaedic rehabilitation exercise protocols of even submaximal intensity, particularly for patients of middle or advanced age.

We would recommend that during exercise heart rhythm is monitored at least in the first exercise sessions, particularly in those subjects thought to be at risk of cardiovascular disease and that emergency equipment should be rapidly available on site.

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