SHORT COMMUNICATION

Autonomic nervous control of the heart rate during dynamic exercise in normal man


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Summary

1. The relative contribution of the efferent components of the sympathetic and parasympathetic nervous systems to the heart rate (HR) response to dynamic physical exercise was evaluated in 23 normal males.

2. The dynamic exercise was performed on a bicycle ergometer at work loads of 25, 50 and 100 W, before and after pharmacological blockade with atropine (13 individuals) or propranolol (10 individuals).

3. Parasympathetic blockade significantly depressed the rapid HR response at the beginning of the exercise period at all levels of intensity, whereas sympathetic blockade only affected the slow-response phase (1–4 min), especially at the highest level of effort.

4. The present results suggest that the tachycardia evoked by dynamic exercise is mediated by a biphasic mechanism initially depending on rapid vagal release, which increases progressively with increasing effort. An increased sympathetic activity manifests itself in a more delayed manner, especially at the higher levels of activity.

5. Continuous monitoring of HR during the entire period of activity at different levels of intensity permits the utilization of dynamic exercise as a simple and non-invasive method for the functional evaluation of the two components of the autonomic nervous system of the heart.

Key words: atropine, autonomic nervous system, exertion, heart rate, propranolol.

Abbreviation: HR, heart rate.

Introduction

When dynamic exercise starts, a rapid elevation in heart rate (HR) is observed. The rapid onset of this tachycardia suggests a neurogenic mechanism of regulation [1–3], which in the efferent response appears to be mediated by a reduction of vagal tonus on the sinus node [4, 5]. After this initial tachycardia, HR changes more slowly. The relative contribution of the efferent autonomic components during this phase of exercise has not yet been fully established. Some previous observations have suggested that at non-intense levels of exercise the steady-state HR response may be predominantly mediated by reduction of parasympathetic activity, whereas at more intense levels of effort the sympathetic stimulation acquires increasing and predominant importance [6, 7].

This study was undertaken to evaluate the participation of the sympathetic and parasympathetic nervous systems in the regulation of HR response to dynamic exercise using the method of continuous monitoring of the chronotropic response during physical activity, and selective pharmacological blockade of the autonomic nervous system.

Methods

Twenty-three normal male subjects aged 25–35 years (mean age 30 years) were included in the study. None of the volunteers showed any evidence of cardiocirculatory abnormalities, as determined by clinical examination and laboratory tests.

The dynamic exercise was performed in a sitting position on a bicycle ergometer (Godart, N.V.) of the Lanoy type at work loads corresponding to 25, 50 and 100 W, over a period of 4 min. A varying period of rest was allowed between the different levels of effort, so that HR could return to control
levels. Pedalling speed was maintained at 60 rotations per min. The electrocardiogram and instant HR were recorded continuously from 30 s before to 1 min after the effort with a four-channel 7754A Hewlett-Packard recording unit.

All volunteers participated in a first experimental session in which the dynamic exercise was performed in the absence of pharmacological blockade. Thirteen of them were retested after peripheral intravenous administration of atropine at the dose of 0.04 mg/kg body weight injected over a period of 90 s. The 10 remaining individuals were also retested after peripheral intravenous administration of propranolol at the dose of 0.2 mg/kg body weight injected over a period of 2 min. The drugs were administered with the subject in a supine position, after a 20 min period of rest. HR was monitored throughout this period up to 5 min after infusion of the drugs.

The HR values are expressed as means ± SEM calculated at 10 s intervals during the dynamic exercise and at 30 s intervals during administration of atropine and propranolol. The data were analysed statistically by Student's t-test for paired samples, with the level of significance set at 5%.

Results

Effects of parasympathetic blockade on HR response to dynamic exercise

Pharmacological blockade with atropine markedly changed the initial chronotropic response evoked by isotonic exercise. The mean increases observed during the first 10 s of effort, under control conditions and after blockade, were: 16 ± 2.0 and 2 ± 0.8 beats/min ($P < 0.001$) at 25 W, 18 ± 1.2 and 3 ± 1.2 beats/min ($P < 0.001$) at 50 W, and 22 ± 1.8 and 2 ± 1.2 beats/min ($P < 0.001$) at 100 W, respectively. At the end of the first 30 s of activity, the increase in HR reached an average of 22 ± 1.5 and 5 ± 0.6 beats/min ($P < 0.001$) at 25 W, 25 ± 1.7 and 9 ± 0.7 beats/min ($P < 0.001$) at 50 W and 34 ± 1.9 and 15 ± 1.0 beats/min ($P < 0.001$) at 100 W, respectively (Fig. 1). Thus, parasympathetic blockade markedly depressed the magnitude of the tachycardia which rapidly occurred at the beginning of the effort, with this effect being more marked during the first 10 s of activity at all levels of exercise studied.

Starting from the 30 s time point, the effects of atropine on the tachycardia induced by dynamic effort were less sharp when compared with those obtained under control conditions. Between 30 s and 1 min of exercise, the following mean variations in HR were observed: 1 ± 1.2 and 2 ± 0.7 beats/min at 25 W, 3 ± 0.8 and 5 ± 1.0 beats/min at 50 W and 8 ± 1.6 and 11 ± 1.3 beats/min at 100 W, before and after blockade, respectively. These variations before and after atropine were not statistically significant (Fig. 1). Similarly, the further changes in HR observed between 1 and 4 min of exercise were superimposable, i.e. −2 ± 1.3 and 1 ± 1.2 beats/min at 25 W, 5 ± 1.6 and 5 ± 1.2 beats/min at 50 W and 17 ± 2.8 and 14 ± 1.8 beats/min at 100 W, before and after atropine, respectively.

Effects of sympathetic blockade on HR response to dynamic exercise

Sympathetic blockade had no statistically significant effect on the initial tachycardia at any level of
exercise. During the first 10 s, a mean respective increase of 16 ± 1.9 and 16 ± 2.3 beats/min at 25 W, 13 ± 2.3 and 14 ± 0.9 at 50 W, and 19 ± 2.2 and 16 ± 1.5 beats/min at 100 W, before and after blockade, respectively, was observed. During the exercise. During the first 10 s, a mean respective

min W, 13 ± 2.3 and 14 ± 0.9 at 50 W, and 19 ± 2.2 and

calated, before and after propranolol, respectively

observed under conditions of /?-adrenergic block-

beats/&, propranolol: 7

beats/&, propranolol: 12

were equivalent (control: 12

P < 0.025) level. At the 100 W level, these increases were equivalent (control: 12 ± 1.2 beats/min, and propranolol: 12 ± 1.2 beats/min).

Propranolol significantly reduced the tachycardia observed between 1 and 4 min of exercise with the 100 W work load (control: 22 ± 2.4 beats/min; propranolol: 12 ± 1.5 beats/min; P < 0.005). The effect of the drug was not perceptible at 50 W (control: 5 ± 1.3 beats/min; propranolol: 4 ± 1.1 beats/min). Between 1 and 4 min of exercise at the 25 W level, the mean variation in HR observed after proprano-

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(P < 0.025) from that observed under control condi-

tions (2 ± 1.3 beats/min).

Discussion

In the present investigation, HR was monitored continuously throughout the period of the exercise. This approach permitted us to verify that atropine practically abolished the tachycardia occurring during the initial 10 s of activity at all levels of exercise, whereas propranolol showed no interference with this initial response. The initial tachycardia ana-

lysed up to 30 s was clearly depressed under conditions of parasympathetic blockade. Sympathetic blockade did not modify appreciably the HR response during the first 30 s. These results indicate that the tachycardia during the first 10 s of exercise almost exclusively depends on the release of vagal influence on the sinus node and that this mechanism predominates in the regulation of the tachycardiac response up to 30 s of effort. These data agree with the few reports available in the literature in which an analogous approach was used [4, 5]. During the phase of slow HR variation (from 30 s on) no appreciable change in response could be observed in the presence of atropine, since the time course of the chronotropic response was parallel to that observed in the control situation at all levels of effort. In contrast, during this phase sympathetic

blockade appreciably depressed the increase in HR between 1 and 4 min of exercise at 100 W, but had no effect on the response to effort at 50 W. A slight but significant decrease in tachycardiac response occurred between the first and fourth minute of exercise at 25 W in the presence of /?-adrenergic blockade. These results indicate the lack of appreciable participation of the parasympathetic compo-

nent in the regulation of HR response during the phase of slow HR changes. In contrast, an impor-

tant sympathetic contribution to the mediation of the tachycardiac response was quite evident between 1 and 4 min. In addition, at less intense levels of exercise (25 and 50 W), the sympathetic contribution appears to be minimal, whereas during the period of most intense effort the activation of the sympathetic nervous system acquires an impor-

tant role in the regulation of tachycardia induced by dynamic exercise.

The pattern of autonomic regulation of HR dur-

ing exercise established on the basis of the present results is in perfect agreement with experimental evidence indicating that the two branches of the autonomic nervous system, when stimulated, exhibit a marked difference in the speed at which they modify HR [8, 9]. The vagal responses are extremely rapid, with a time delay of less than 1 s, whereas the sympathetic responses are slower [10].

In parallel, it should be pointed out that continu-

ous monitoring of HR behaviour throughout the period of exercise at different levels of effort represents a simple and non-invasive method of functional evaluation of the two autonomic components which could be useful to assess in man the func-

tional conditions in which autonomic activity may be altered. This is particularly relevant to the sympathetic system, because dynamic exercise, at incremental work loads, can elicit progressive grades of increase in sympathetic activity, a feature not shown by other tests of sympathetic control of the HR.

References

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