DECREASED PREMATURE VENTRICULAR CONTRACTIONS THROUGH USE OF THE RELAXATION RESPONSE IN PATIENTS WITH STABLE ISCHÆMIC HEART-DISEASE

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Summary To determine whether decreased sympathetic-nervous-system activity achieved by the relaxation response could decrease premature ventricular contractions (P.V.C.S), eleven ambulatory patients with proven, stable ischæmic heart-disease and P.V.C.S were investigated. The patients, who were taking no medication for the P.V.C.S, were trained to elicit regularly the relaxation response through a non-cultic psychological technique. The frequency of the P.V.C.S was measured by computer analysis of Holter monitor tapes for 2 complete days before learning the technique, which was learned in approximately 5 minutes after the second day. Patients were instructed to evoke the response for 20 minutes twice daily thereafter. After 4 weeks, a reduced frequency of P.V.C.S was documented in eight of the eleven patients. This effect was especially striking during the sleeping hours and less so during the entire monitoring session. The relaxation response is a simple, no cost, non-pharmacological mechanism without side-effects which seemed to decrease the frequency of P.V.C.S in most patients with ischæmic heart-disease.

Introduction

The relaxation response is believed to be an integrated hypothalamic response associated with decreased sympathetic-nervous-system activity1-3 and the counterpart of the emergency reaction described by Cannon.4 The relaxation response may be elicited by a non-cultic, simple psychological technique,5 or by yogic, religious, and secular techniques.1-3,6,8 The clinical usefulness of the response has been established in hypertension.6,10

Frequent premature ventricular contractions (P.V.C.S) are associated with an increased mortality in ambulatory patients with ischæmic heart-disease.11,12 Since increased sympathetic-nervous-system activity is believed to lead to an increased number of P.V.C.S,13 the hypothesised decreased sympathetic-nervous-system activity associated with the relaxation response should theoretically lead to decreased P.V.C.S and, perhaps, decreased mortality. We have demonstrated the efficacy of the relaxation response in the reduction of P.V.C.S in ambulatory patients with proven, stable ischemic heart-disease.

Methods

Eleven patients were selected from the population of the Lahey Clinic Foundation. Each was ambulatory and had established ischemic heart-disease of at least one year's duration, as manifested by a history of proven myocardial infarction or unequivocal angina pectoris with an exercise test strongly positive for ischemic heart-disease. Each patient had documented P.V.C.S, and none was taking drugs for these P.V.C.S. Written informed consent was obtained from each patient before the investigation.

The frequency of the P.V.C.S was measured by Holter monitoring for two 24-hour periods. In the morning of the second 24-hour period, a standard, multistage exercise test was performed14 while the monitoring continued. After the second day of monitoring was completed, the patient was verbally instructed how to elicit the relaxation response by using the following non-cultic technique:

1. Sit quietly in a comfortable position.
2. Close your eyes.
3. Deeply relax all your muscles, beginning at your feet and progressing up to your face. Keep them deeply relaxed.
4. Breathe through your nose. Become aware of your breathing. As you breathe out, say the word “one” silently to yourself. For example, breathe in . . . out, “one”; in . . . out, “one”; &c.
5. Continue for 20 minutes. You may open your eyes to check the time, but do not use an alarm. When you finish, sit quietly for several minutes at first with closed eyes and later with opened eyes.
6. Do not worry about whether you are successful in achieving a deep level of relaxation. Maintain a passive attitude and permit relaxation to occur at its own pace. When distracting thoughts occur, ignore them and continue repeating “one”. With practice, the response should come with little effort. Practice the technique twice daily, but not within 2 hours after any meal, since the digestive processes seem to interfere with the elicitation of anticipated changes.

This process of instruction required approximately 5 minutes. Each patient was given a written copy of the instructions and a diary to record the frequency of prac-

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>P.V.C.S/hr.</th>
<th>% change</th>
<th>P.V.C.S/1000 heart beats</th>
<th>% change</th>
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<tbody>
<tr>
<td></td>
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<td>Before</td>
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<tr>
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Mean ± s.d. 57.3 ± 7.9  151.5 ± 252.2  131.7 ± 307.2  30.6 ± 53.2  29.5 ± 65.8
After 4 weeks of regularly evoking the relaxation response in their own homes for 10–20 minutes twice daily under their own supervision, the patients returned to repeat the two 24-hour periods of Holter monitoring and the exercise test. During monitoring periods, the patients recorded the hours of sleep in the diary.

Each P.V.C.S was counted by a computerised detection system. The accuracy of the system was verified by randomised manual counting of the P.V.C.S in each patient. The data obtained from exercise tests were manually interpreted by review of the continuous E.C.G. strip chart. Comparisons were made of the frequency of P.V.C.S before and after the 4 weeks of the regular elicitation of the relaxation response by use of the paired t test.

Results

The mean age of the patients was 57.3 years (S.D. ± 7.9). There were nine males and two females (table I). After 4 weeks of regularly eliciting the relaxation response, a reduced frequency of P.V.C.S was documented in eight of the eleven patients, and this was especially striking in patients 2, 3, 4, 5, and 9 (table I). Before regular elicitation of the relaxation response, the P.V.C.S per hour per patient for the entire group of patients averaged 151.5 ± 252.8 for the entire monitoring session. After 4 weeks of evocation of the relaxation response the average had fallen to 131.7 ± 307.2. Before using the response, the number of P.V.C.S per hour during sleeping hours averaged 125.3 ± 221.4, while after regular elicitation of the response the P.V.C.S during sleep decreased to 87.9 ± 200.5 (P < 0.05) (table II). When the P.V.C.S were expressed per 1000 heart beats for the entire group, there was a significant decrease only during the sleeping hours from 29.0 ± 52.5 to 21.1 ± 50.0 (P < 0.05).

The average heart-rate before regular elicitation of the relaxation response was 77.2 ± 13.4 beats per minute for the entire monitoring session, while after 4 weeks it was 76.1 ± 13.4. During sleeping hours, the heart-rate averaged 68.2 ± 140 before and 70.0 ± 11.9 after regularly eliciting the relaxation response. There was no consistent relation between these small changes in heart-rate and the frequency of the P.V.C.S.

The results of the exercise testing before and after the regular elicitation of the relaxation response revealed that, in one of the patients, the P.V.C.S were decreased during the exercise period itself, while in three the P.V.C.S were decreased in the recovery period (table II). Seven of the patients demonstrated essentially no change.

No adverse side-effects were noted. In fact, most patients described pleasurable feelings after the elicitation of the relaxation response.

Discussion

The regular elicitation of the relaxation response was associated with decreased P.V.C.S in eight of the eleven ambulatory patients with proven, stable ischemic heart-disease. The response is easily learned in about 5 minutes. Its elicitation involves no cost other than time. It is non-pharmacological and thus without the side-effects of drugs. Its side-effects, if indeed any, are minor; they are essentially those of praying twice daily.

The decreased frequency of P.V.C.S might have been spontaneous or it might have resulted from random variation, rather than resulting directly from the relaxation response. Since the patients had clinically stable ischemic heart-disease of at least one year's duration, a spontaneous decrease is unlikely. The lengthy periods of E.C.G. monitoring during which the P.V.C.S were accurately counted by a computer-detection system minimised sampling bias. Since the follow-up in this investigation was 4 weeks, the relation of the relaxation response to P.V.C.S over longer time periods remains to be assessed.

The decrease in the average P.V.C.S per hour for the entire monitoring session after 4 weeks of regularly eliciting the relaxation response without a corresponding decrease in average P.V.C.S per 1000 heart beats is accounted for primarily by the disproportionate contribution of P.V.C.S to the average by patient 7. This discrepancy between P.V.C.S per hour and P.V.C.S per 1000 heart beats could not be attributed to heart-rate since heart-rate remained essentially unchanged.

The regular elicitation of the relaxation response is presumed to decrease sympathetic-nervous-system activity and may be the mechanism by which P.V.C.S were decreased. This is consistent with the data of Lown et al., who reported a decreased frequency of P.V.C.S during sleep in patients with and without heart-disease. They hypothesised lessened sympathetic tone as the mechanism involved, although others
implicate increased parasympathetic activity.17 Our data not only replicate the finding of decreased P.v.c.s during sleep in the control period, but also document a yet further reduction during sleep after the regular elicitation of the relaxation response.

The long-term, pharmacological therapy of P.v.c.s in ambulatory patients is often toxic or ineffective. Further, sudden death from ischemic heart-disease is common and believed to be related to the frequency of P.v.c.s. This non-pharmacological method associated with decreased P.v.c.s in most patients investigated may therefore prove to be most useful and significant.

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REFERENCES

IMPAIRED CARDIOVASCULAR RESPONSIVENESS IN LIVER DISEASE

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Introduction

CHRONIC liver disease is often associated with changes in the cardiovascular system. Cardiac output may be increased and peripheral vascular resistance lowered.1 Hypotension often develops in patients with hepatic failure, and high blood-pressure is rare in cirrhosis.2,3 Little, however, is known of the neurogenic mechanisms of vascular control in liver disease. We therefore investigated reflex cardiovascular responsiveness in patients with liver disease and found impaired vascular responses to induced postural change in patients with cirrhosis.4 The study has now been extended; cardiovascular responses to different autonomic stimuli and to infusion of adrenaline, noradrenaline, and angiotensin II have been measured in patients with chronic liver disease to determine the possible site of interference with cardiovascular function.

Patients and Methods

Patients

75 patients with chronic liver disease were studied; reflex cardiovascular responses were investigated in 54 patients and vasoactive agents were infused in 21. All patients had evidence of cirrhosis on liver biopsy and were fully ambulant; only 9 patients had clinical evidence of hepatic decompensation. Cryptogenic cirrhosis had been diagnosed in 38 patients, active chronic hepatitis with cirrhosis in 18, primary biliary cirrhosis in 15, secondary biliary cirrhosis in 2, hemochromatosis in 1, and Wilson's disease in 1. Patients with alcoholic cirrhosis, peripheral neuropathy, or diabetes mellitus were excluded. Most were not taking drugs at the time of study; 11 were taking prednisolone and 15 frusamide. A control group for study of reflex cardiovascular responsiveness was drawn from healthy volunteers and inpatients awaiting minor " cold " surgery who were age-matched with the patients who had liver disease. Control data on the effects of infusing vasoactive agents were derived from previous studies. The investigation was approved by the ethics committee of the hospital and informed consent for the study was given by each subject.

Limb Blood-flow

Subjects were tested at rest in the postoperative state in standard laboratory conditions. They lay supine whilst peripheral blood-flow was measured at fifteen-second intervals by venous occlusion plethysmography.

In the study of reflex cardiovascular responses, forearm blood-flow was measured using two mercury-in-rubber strain gauges, placed at standard points on the upper and lower forearm so as to assess the relative changes in forearm muscle and skin blood-flow, respectively.5,6

Hand and forearm flow during the infusion of adrenaline, noradrenaline, and angiotensin II were measured by water-filled temperature-controlled plethysmographs, as in previous studies of circulatory effects of drugs in health and disease.5,6

Pulse-rate was determined from standard lead II of an electrocardiogram.

Experimental procedure.—Subjects lay at rest in the laboratory for at least fifteen minutes before any measurements were made. The plethysmographs were then placed in position and control observation made over ten to fifteen minutes. Further control observations over eight