PAPERS AND ORIGINALS

Cardiac Responses to Thermal, Physical, and Emotional Stress

PETER TAGGART, PETER PARKINSON, MALCOLM CARRUTHERS

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Summary

We have studied the effect of a short period of exposure to the intense heat of a sauna bath on the electrocardiogram and plasma catecholamine, free fatty acid, and triglyceride concentrations in 17 subjects with apparently normal hearts and 18 persons with coronary heart disease. Similar observations were made on 11 of the 17 normal subjects and on 7 of the persons with coronary heart disease in response to exercise.

Exposure to heat was associated with an increase in plasma adrenaline with no change in noradrenaline, free fatty acid, or triglyceride concentrations. Exercise was associated with the expected increase in both plasma noradrenaline and adrenaline concentrations. A heart rate up to 180 beats/min was observed in response to both heat and exercise. Apart from the ST-T changes inherent to sinus tachycardia, ST-T segment abnormalities were frequent in response to heat in both the subjects with normal and abnormal hearts, but little change occurred in the ST-T configuration when the subjects were exercised to produce comparable heart rates. Ectopic beats, sometimes numerous and multifocal, were observed in some subjects of both groups in response to heat, but not to exercise. It seems likely that the net unbalanced adrenaline component of the increased plasma catecholamine concentrations (which is also seen in certain emotional stress situations) is predominantly responsible for ischaemic-like manifestations of the electrocardiogram in susceptible subjects. The observations provide further validation for previously reported studies that it is the increased plasma noradrenaline in response to emotional stress that is associated with the release of free fatty acids and ultimate hypertriglyceridaemia, of probable importance in the aetiology of atheroma.

Department of Cardiology, Middlesex Hospital, London W.1

PETER TAGGART, M.R.C.P., Lecturer in Medicine

PETER PARKINSON, M.R.C.P., Charles Wolfson Fellow in Clinical Cardiology

Institute of Ophthalmology, London W.C.1

MALCOLM CARRUTHERS, M.B., B.S., Senior Lecturer in Chemical Pathology

Introduction

At 8.45 a.m. on 24 April 1971 a 30-year-old jockey entered a sauna bath. At 11 a.m. he left the room coughing and in "distress," asked for a glass of water, and climbed on to the massage table. Within a few minutes he collapsed pulseless and unconscious. Despite the attention of a doctor who was at hand, including intubation and ventilation, he could not be resuscitated. He had been accustomed to using the sauna bath on a Saturday morning to reduce his weight. In the preceding months, because of increasing difficulty at the scales, he had supplemented this regimen with frusemide, taking up to 280 mg before entering the bath. At necropsy an old inferior (posterior) infarct was found, but although there was disproportionate intimal disease of the coronary arteries, no occlusion or recent infarction was apparent. The temperature of the bath was 125°F (51.6°C).

Incidents of this type have been described before, and it was recently reported at a medical meeting in Oslo (*The Times*, 13 April 1972) that 67 people died in Finland in 1970 while taking sauna baths. We therefore consider it timely to present certain observations on both normal subjects and persons with coronary heart disease using sauna baths at temperatures up to 230°F (110°C).

Striking electrocardiographic and biochemical changes have been observed in normal subjects and subjects with coronary heart disease exposed to certain emotional stress situations (Taggart *et al.*, 1969; Somerville *et al.*, 1971; Taggart and Carruthers, 1971). The electrocardiographic abnormalities have included tachycardia, often of an extreme degree, ST-T segment abnormalities, and multiple ventricular ectopic beats associated with increased plasma catecholamine, free fatty acid, and triglyceride concentrations.

Certain stress situations are associated with an increase in body temperature (Taggart and Carruthers, 1971), and it is therefore important to define the effects of heat on the values with which we are concerned. For this purpose a study was designed to investigate the effects of the intense heat of a sauna bath on a group of normal subjects and a group of persons with coronary heart disease. The finding of an increased plasma adrenaline concentration with no change in noradrenaline concentration provided a unique opportunity to study some of the effects of a situation associated with an increased circulating concentration of endogenous adrenaline. In order to enable a comparison to be made with the effects of noradrenaline the study was expanded to include observations on the same subjects before and after exercise, which might be expected to result in a

predominant increase of the latter hormone (Vendsalu, 1960). Comparative studies on the plasma concentrations of adrenaline and noradrenaline have so far been limited to infusion studies (Lepeschkin *et al.*, 1960; Carlson and Oro, 1965; Jurand and Oliver, 1970).

In the majority of stress situations that we have studied the rise in plasma catecholamine concentrations has been mainly due to its noradrenaline content. Particularly in view of the known potency of adrenaline in the production of arrhythmias, it is of interest to attempt to separate the effects of these two hormones on the observed electrocardiographic changes in response to stress. It is also of interest to separate their effects on plasma free fatty acid concentrations in view of increasing evidence on the importance of circulating lipids and the incidence of arrhythmias (Kurien *et al.*, 1971). In addition, this study is intended to validate further the relation between plasma concentrations of noradrenaline, free fatty acid, and triglycerides in response to emotional stress (Taggart and Carruthers, 1971), which we have recently presented in support of a hypothesis on the aetiology of atheroma (Carruthers, 1969).

Material and Methods

Subjects with Normal Hearts.—Seventeen normal male volunteers aged 21–54 (mean 33) years were monitored during a 10-minute sauna bath. Eleven of these were monitored again during intense physical exercise on a rowing ergometer. All subjects in this group had a normal resting 12-lead electrocardiogram and none had a history to suggest any cardiac abnormality.

Subjects with Abnormal Hearts.—Eighteen male volunteers aged 43–62 (mean 52) years with a previous history of myocardial infarction were studied during their accustomed programme of carefully controlled rehabilitative exercise, which included the option of a brief sauna bath. Electrocardiograms were recorded on 24 occasions from the 18 subjects during the sauna bath and on 10 occasions from seven of them during exercise. Blood samples before and after the sauna bath were taken from 9 subjects, including 5 of the 18 subjects and an additional 4 persons.

Radio electrocardiograms.—The C and M (series 1) radio-electrocardiograph (Medical and Industrial Equipment Ltd.) was used. The lead connecting the two press-stud electrodes (Devices) to the radio transmitter was extended to allow the transmitter to be positioned outside the door of the sauna bath. The transmitted signals and simultaneous commentary of events were recorded on the two tracks of a UHER 4000 Report/L tape recorder. Technical specifications of the equipment, together with the principle of recording and playback procedure, are described in detail elsewhere (Taggart *et al.*, 1969).

Plasma Samples.—Details of collection and analysis of plasma samples for catecholamines have also been described (Carruthers *et al.*, 1970). Free fatty acids were measured on the Auto Analyzer II by the semi-automated fluorimetric technique developed in this laboratory (Carruthers, 1972). Triglycerides were measured by the standard Technicon semi-automated method on isopropanol extracts of plasma, and cholesterol by the direct automated Technicon method using the Lieberman-Burchard reaction.

Procedure

NORMAL SUBJECTS

Sauna.—The subjects were studied after a light breakfast. Control radioelectrocardiograms were taken with the exploring electrode in the V4 and V6 positions. Both leads were recorded in the standing, sitting, and lying positions in order to detect any postural variation. Initial records were obtained of body weight, blood pressure (standing, sitting, and lying), and mouth

temperature. Venous blood samples were taken, centrifuged immediately, and processed as described by Carruthers *et al.* (1970), a small quantity of whole blood being retained for analysis of haemoglobin, packed cell volume, and mean corpuscular volume (performed in duplicate). The subject then entered a sauna bath (Nordic Sauna Ltd.), where he remained sitting down for 10 minutes. The dry bulb temperature of the bath varied between 90 and 110°C. The exploring electrode was positioned in the V6 position. After the sauna bath tracings were taken with the exploring electrode first in the V6 and then in the V4 position to enable observations to be made on the T-wave configuration in a right precordial lead in addition to the usual left ventricular recordings. Blood samples were taken and processed as before, together with repeat observations of mouth temperature, blood pressure (standing, sitting, and lying), and body weight. In four of the subjects a third blood sample was taken one hour later and treated in the same manner as before. **Exercise.**—After an interval of at least two days a similar procedure was adopted for the study of exercise. Each subject exercised for seven minutes on a rowing ergometer, this having the advantage over the bicycle ergometer or many other forms of exercise in providing whole body exercise.

SUBJECTS WITH CORONARY HEART DISEASE

The radioelectrocardiogram (V6 position) was monitored during a 10-minute period of carefully graded rehabilitative exercises in a gymnasium. Recordings were then taken from the same subjects during a five-minute period in the sauna bath, this being the maximum time that they were allowed to remain in the bath. Blood samples were taken from nine persons, both before and after the sauna bath, and analysed as described above. In these subjects the sauna bath followed within a few minutes of the end of their exercise period, unlike the normal subjects, where the observations on heat and exercise were made on separate days.

Results

The heart rates and ST-T segment abnormalities in both normal subjects and those with coronary heart disease are presented in Table I.* The strenuous exercise of rowing characteristically produced an immediate increase in heart rate to near the maximum values attained, after which a slow further increase occurred. A rapid initial increase occurred in the subjects with coronary heart disease in response to more gentle exercise, though to a lower level, after which a pre-determined pulse rate was maintained according to their prearranged programme of rehabilitative exercises.

Heart rates in both groups of subjects showed a progressive increase during the sauna bath, being roughly linearly related to the time in the bath. In the normal subjects, who remained in the sauna for 10 minutes, maximum rates in excess of 170/min were frequent and comparable to the rates observed in the same subjects during strenuous physical exercise. There was much variation in the tachycardia observed in different subjects during the sauna bath, some attaining extremely rapid heart rates, and although this was the finding in the majority a few showed only a modest increase.

Many normal subjects in response to heat rapidly developed ST-T changes resembling those of ischaemia, which were not apparent when the same subjects exercised to produce a comparable tachycardia (Figs. 1 and 2). The electrocardiograms of four subjects with coronary heart disease are similarly presented in Fig. 3, selected to include examples of four different ST-T changes—namely, isolated T inversion, flattening of the T wave, S-T segment depression, and “J”-type depression with

*More detailed information on these subjects can be obtained from the authors.

TABLE I—Heart Rates (Mean and Range) and ST-T Segment Abnormalities in Normal Subjects and Subjects with Coronary Heart Disease in response to Intense Moist Heat and Exercise (V6 Position)

Heart Rate	Normal		Coronary Heart Disease	
	Heat (n = 17)	Exercise (n = 11)	Heat (n = 24)	Exercise (n = 10)
Start	78 (53-96)	78 (56-92)	99 (85-115)	88 (75-109)
No. with ST-T abnormalities			13	2
After 5 min	123 (77-166)	163 (145-187)	125 (100-165)	114 (88-142)
No. with ST-T abnormalities	12	3	20*	4*
After 7 min	131 (79-164)	168 (148-192)	—	—
No. with ST-T abnormalities	12	3		
Maximum	145 (106-181)	168 (148-192)	126 (101-165)	121 (97-143)
No. with ST-T abnormalities	12	3		

*Increased ST-T abnormalities. In the normal subjects exercise followed heat by a number of days. The coronary subjects exercised first and proceeded to the sauna within a few minutes.

FIG. 1—Normal subject; radioelectrocardiogram, V6 position. Progressive ST-T segment changes at one-minute intervals during sauna bath, and exercise tracing at comparable heart rate showing normal ST-T.

FIG. 2—Normal subject; radioelectrocardiogram, V6 position. Progressive T-wave changes at one-minute intervals during sauna bath, and exercise tracings recorded at one-minute intervals during a seven-minute period of intense physical exercise showing normal T waves at a comparable heart rate.

TABLE II—Plasma Catecholamine Values before and after Intense Moist Heat (10 min) and Exercise (7 min) in Normal Subjects (Mean ± S.D.)

	Heat (n = 17)			Exercise (n = 11)		
	Total Catecholamines (μg/l.)	Noradrenaline (μg/l.)	Adrenaline (μg/l.)	Total Catecholamines (μg/l.)	Noradrenaline (μg/l.)	Adrenaline (μg/l.)
Before	0.72 ± 0.13	0.68 ± 0.12	0.03 ± 0.04	0.71 ± 0.16	0.69 ± 0.16	0.02 ± 0.01
After	0.90 ± 0.22	0.61 ± 0.14	0.29 ± 0.17	1.76 ± 0.53	1.44 ± 0.39	0.33 ± 0.19
P	<0.005	<0.1	<0.001	<0.001	<0.001	<0.001

FIG. 3—Coronary heart disease; radioelectrocardiogram, V6 position. Records from four subjects before and during sauna and exercise illustrating T inversion, T-wave flattening, S-T depression, and "J"-point depression during sauna but not exercise. Second control tracing included as sauna immediately followed period of exercise.

loss of T-wave amplitude, occurring during the sauna bath but not during exercise.

Ventricular ectopic beats were prominent and at times recurrent in four of the 17 normal subjects during the sauna, whereas none were observed in the 11 subjects studied during exercise (Figs. 4 and 5). Six of the subjects with coronary heart disease developed ectopic beats during the sauna bath, ventricular in five and supraventricular in one. In one subject occasional ventricular ectopic beats were observed during exercise but were frequent during the sauna.

Tall, pointed, symmetrical T waves were noted in a right precordial lead immediately after the sauna bath in six of the normal subjects (Fig. 6).

Plasma catecholamine concentrations in the subjects with normal hearts before and after sauna and exercise are presented in Table II, and for the nine subjects with coronary heart disease from whom blood samples were taken before and after the sauna in Table III. In both groups a significant and often spectacular increase in plasma adrenaline concentration was apparent after the sauna, whereas the noradrenaline concentration remained unchanged. The expected increase in both plasma adrenaline and particularly noradrenaline concentration was observed in the normal subjects in response to exercise.

Plasma concentrations of free fatty acids, triglycerides, and cholesterol before and after the 10-minute sauna bath and the

FIG. 4—Normal subject; radioelectrocardiogram, V6 position. Ventricular ectopic beats occurring during sauna (150/min).

FIG. 5—Normal subjects and subjects with coronary heart disease; radioelectrocardiogram, V6 position. Selection of subjects illustrating variety of ectopic beats. Note multifocal nature in subject No. 1 (second trace).

FIG. 6—Normal subject; radioelectrocardiogram, V4 position. Tall, pointed T waves after sauna (110/min).

TABLE III—Plasma Catecholamine Concentrations before and after Intense Moist Heat (5 min) in 9 Subjects with Coronary Heart Disease (Mean $\pm$ S.D.)

	Total Catecholamines (μ g/l.)	Noradrenaline (μ g/l.)	Adrenaline (μ g/l.)
Before ..	0.85 $\pm$ 0.20	0.77 $\pm$ 0.17	0.08 $\pm$ 0.06
After ..	1.61 $\pm$ 0.79	0.91 $\pm$ 0.36	0.70 $\pm$ 0.48
P ..	<0.02	<0.4	<0.01

TABLE IV—Levels of Plasma Free Fatty Acids, Triglycerides, and Cholesterol before and after Intense Moist Heat (10 min) and Exercise (7 min) in Normal Subjects (Mean $\pm$ S.D.)

	Heat (n = 17)			Exercise (n = 11)		
	Free Fatty Acids (μ Eq/l.)	Triglycerides (μ mol/l.)	Cholesterol (mg/100 ml)	Free Fatty Acids (μ Eq/l.)	Triglycerides (μ mol/l.)	Cholesterol (mg/100 ml)
Before ..	909 $\pm$ 498	1,253 $\pm$ 579	229 $\pm$ 48	823 $\pm$ 314	1,461 $\pm$ 1,020	233 $\pm$ 45
After ..	1,096 $\pm$ 632	1,246 $\pm$ 609	227 $\pm$ 39	955 $\pm$ 409	1,648 $\pm$ 1,059	234 $\pm$ 45
P ..	<0.2	<0.9	<0.7	<0.2	<0.005	0.97

seven minutes' exercise in the normal subjects are presented in Table IV. No significant difference was seen in the free fatty acid, triglyceride, or cholesterol levels in response to either heat or exercise under the conditions of the present studies. The similar findings for the nine subjects with coronary heart disease in response to heat are presented in Table V. A third sample was taken one hour after the sauna from four normal subjects. The plasma adrenaline concentration had invariably

TABLE V—Levels of Plasma Free Fatty Acids, Triglycerides, and Cholesterol before and after Intense Moist Heat (10 min) in 9 Subjects with Coronary Heart Disease (Mean $\pm$ S.D.)

	Free Fatty Acids (μ Eq/l.)	Triglycerides (μ mol/l.)	Cholesterol (mg/100 ml)
Before ..	717 $\pm$ 298	188 $\pm$ 123	250 $\pm$ 41
After ..	759 $\pm$ 262	186 $\pm$ 121	249 $\pm$ 42
P ..	<0.3	<0.7	0.3

returned to the pre-sauna resting value, and the plasma concentration of noradrenaline, free fatty acids, triglycerides, and cholesterol still remained unchanged from control values.

Cuff blood pressures recorded in the lying position in the 17 normal subjects before the sauna were (mean $\pm$ S.D.) 130 $\pm$ 18 mm Hg systolic and 80 $\pm$ 9 diastolic, and after the sauna 130 $\pm$ 22 systolic and 51 $\pm$ 29 diastolic. Recordings obtained from the same subjects in the sitting position were 126 $\pm$ 13 systolic and 79 $\pm$ 10 diastolic before the sauna, and 113 $\pm$ 20 systolic and 47 $\pm$ 29 diastolic after the sauna. Again the difference in response of certain individuals should be emphasized, some persons showing a profound degree of hypotension immediately after the sauna. The values obtained in the 11 subjects studied during extreme physical exercise were 129 $\pm$ 18 systolic and 78 $\pm$ 13 diastolic before and 166 $\pm$ 20 systolic and 72 $\pm$ 15 diastolic after exercise. Mouth temperatures increased from 98.2° $\pm$ 0.17° to 100.6° $\pm$ 0.59° in the 17 normal subjects after sauna, and to 98.7° $\pm$ 0.65° in the 11 normal subjects after exercise. All subjects lost weight in the sauna bath, in the case of the normal subjects from an overall mean of 158.5 $\pm$ 21.3 lb to 157.2 $\pm$ 20.8 lb (P < 0.001).

Haemoglobin measured in the normal subjects in duplicate before and after both sauna and exercise may be taken as a rough indication of changes occurring in plasma volume. The values obtained before and after sauna were 15.1 $\pm$ 0.73 and 15.5 $\pm$ 0.8 g/100 ml respectively (P < 0.01), and before and after exercise 15.0 $\pm$ 1.1 and 16.0 $\pm$ 1.1 g/100 ml (P < 0.001). No significant changes occurred in serum potassium, sodium, and chloride, plasma bicarbonate, or blood urea either after sauna or after exercise.

Discussion

The degree of tachycardia in the sauna bath and the short time required for very fast heart rates to be attained was impressive. The tachycardia in these subjects was more pronounced than that reported by others (Eisalo, 1956), although the temperature of the bath was higher in our studies. The linear response of heart rate to the time spent in the sauna seems to facilitate the management of persons in whom the combination of fast heart

rates and hypotension are undesirable by curtailing the length of time spent in the sauna.

A high proportion of the normal subjects and those with coronary heart disease developed "ischaemic-like" ST-T patterns during exposure to heat. Minor alterations of ST-T configuration have been reported by others in studies at lower temperatures, including lowering of T-wave amplitude in limb leads (Eisalo, 1956) and S-T segment depression in left precordial leads (Sherif *et al.*, 1970). Such changes have been variously attributed to the tachycardia (Rasanen, 1951), haemodynamic changes (Sherif *et al.*, 1970), or "sympathetic tone" (Ott, 1948). The ST-T segment changes in the present subjects were more profound than those described by others (Eisalo, 1956) although, as stated above, the conditions in our study were probably more extreme.

The absence of such ST-T changes when the same subjects were exercised to produce a comparable heart rate seems to exclude tachycardia as the sole cause. A postural variation in the ST-T segment configuration has been shown to occur in some subjects (Lachman *et al.*, 1965). This effect can confidently be excluded from the present study for the following reasons. Recordings were taken of the lead position used in all subjects in the standing, sitting, and lying positions both before and after the sauna and exercise and no such postural effect was observed. Furthermore, the subjects did not alter their position during the test periods, and yet the ST-T changes were progressive. As expected, both systolic and diastolic blood pressures were lower during the sauna (Eisalo, 1956), whereas the expected increase in systolic pressure with variable but small changes in diastolic pressure occurred during exercise (Hanson *et al.*, 1966). The resulting decrease in coronary artery diastolic filling pressure during the sauna, together with other haemodynamic changes, particularly increase in left ventricular work (Sherif *et al.*, 1970) and reduced diastolic filling time as a consequence of the tachycardia, may result in myocardial anoxia despite an increased stroke volume and cardiac output (Sherif *et al.*, 1970).

The tachycardia and ST-T segment abnormalities in these subjects in response to heat bear a strong resemblance to those seen after infusion of adrenaline (Mitchell and Shapiro, 1954; Lepeschkin *et al.*, 1960). Lepeschkin *et al.* (1960) noted such changes in response to adrenaline but not to noradrenaline infusion, and cited their own experimental data of alterations in the shape of phases 2 and 3 of the action potential in response to adrenaline infusion as the mechanism for these changes.

Ventricular ectopic beats (Figs. 4 and 5) are a known consequence of intravenous adrenaline infusion (Lepeschkin *et al.*, 1960) and of adrenaline secreting pheochromocytomata (Page *et al.*, 1969). The number of persons who developed ectopic beats was comparatively small compared with those who developed ST-T segment abnormalities. Nevertheless, when they did occur they tended to be spectacular in their frequency, and were at times multifocal. We have previously documented the occurrence of ventricular ectopic beats in subjects with normal hearts speaking before an audience (Somerville *et al.*, 1971) and in persons with coronary heart disease (Taggart *et al.*, 1969) while driving in traffic. Both situations were associated with increased plasma catecholamine concentrations, and it is tempting to postulate a cause-and-effect relation.

The fact that there was no apparent correlation between the incidence of ectopic beats and the plasma adrenaline concentration in the present study does not exclude a causative relation, since the plasma adrenaline concentration was not measured at the height of ectopic beat formation. There was again no apparent correlation with their incidence and the measured levels of plasma adrenaline concentration or other variables such as the degree of hypotension or body temperature. In particular, there was no apparent correlation with the incidence of ectopic beats and the development of ST-T segment abnormalities. This suggests that there may be a fundamental difference in the response of the myocardium in people with normal hearts in addition to those with coronary heart disease,

such that some are specially prone to arrhythmias, perhaps from an early age, while others may be more liable to develop localized areas of ischaemia.

The tall, pointed T waves appearing in the right precordial leads in some of the present subjects after exposure to heat were single waves, not combination waves (superimposition of a giant early U wave on the T wave). Although this was suggested by Lepeschkin *et al.* (1960) as the explanation for their occurrence during infusion of adrenaline, separate, distinct U waves can be seen in our subjects (Fig. 6). However, although it is not the purpose of this article to enter into the vexed and itself emotional subject of the electrophysiological aspects of the U wave, we feel that there is equal reason to accept that the algebraic sum of the subendocardial minus subepicardial late after-potentials would produce such a picture and be perfectly in keeping with an adrenaline effect. The serum potassium in these subjects showed no significant change in the sauna bath. Tall, pointed T waves did not occur in the sauna subjects after exercise.

While these T-wave changes may be a reflection of subendocardial ischaemia (Sodi Polares *et al.*, 1971) as a consequence of hypotension and tachycardia, it is also possible that they are influenced by the *net* unbalanced effect of high circulating adrenaline concentration. Similar appearances have been reported in some subjects due to anxiety (Mitchell and Shapiro, 1954). It is unlikely that this was an important factor in our subjects, as none was aware of subjective feelings of anxiety, nor had the subjects with normal hearts an important degree of tachycardia on entering the sauna bath. Nevertheless, it is probable that both heat and anxiety may produce such tall, pointed T waves through the intermediary of adrenaline secretion. The ST-T segment abnormalities and ectopic activity seen during heat exposure therefore raise the possibility that a net unbalanced increase of plasma adrenaline may be of prime importance in the aetiology of certain arrhythmias, and particularly of the immediate ischaemic-like manifestations in subjects with abnormal and even normal hearts.

As no rise in plasma free fatty acid concentration was seen on exposure to heat it seems to validate our contention that the noradrenaline response to emotion in our previously reported data on racing drivers (Taggart and Carruthers, 1971) was responsible for the observed rise in plasma free fatty acid concentrations, rather than the associated smaller increase of plasma adrenaline concentration or the increased body heat due to the thick fireproof clothing.

It is of interest to compare the effect of heat, exercise, and emotion with respect to plasma noradrenaline, adrenaline, and free fatty acid concentrations. In Fig. 7 the alterations in the plasma concentrations of these substances are represented diagrammatically for the normal subjects in the present study before and after heat exposure and exercise, together with previously documented values obtained in a group of racing drivers (Taggart and Carruthers, 1971). Comparison of the effects of heat and emotion supports the contention that the rise of plasma free fatty acid is predominantly a result of noradrenaline secretion rather than adrenaline (Weake, 1966). The much smaller increase in free fatty acids after exercise is likely to be a consequence of their utilization as a metabolic fuel

FIG. 7—Changes in plasma adrenaline (A), noradrenaline (NA), and free fatty acid (FFA) concentrations during heat and exercise in the present study, and emotion (right-hand group) taken from a previous study.

(Johnson *et al.*, 1969). This is a further demonstration of the biochemical consequences of imbalance between levels of emotional and physical activity. It provides some additional encouragement for those investigating the place of "exercise therapy" in the prevention and treatment of cardiovascular disease.

In addition, this study indicates that sudden exposure to intense heat, as in sauna baths, Turkish baths, and presumably the "coffee baths" becoming fashionable in Japan, may contain an element of risk in those prone to coronary insufficiency. This group includes a number of middle-aged men with tiredness, lethargy, and "fibrositis" in the chest and shoulder who will seek out the most gruelling of these artificial environments and stay in longest to "sweat it out of their system." Apart from descriptions such as "my heart was thumping" and "I thought my heart was going to stop" appearing in recent articles on the subject (*Observer*, 1971), sudden syncopal attacks just on standing to leave the sauna are not uncommon.

If any recommendations can be made on the basis of the limited number of subjects in this study perhaps a cautious approach for the middle-aged and elderly sauna novice might be indicated. The cooler, lower levels away from the heater should be tried in the high temperature saunas being promoted in Britain with a time limit of five minutes. However, at the lower temperature of 60-70°C more common in Finnish saunas, Eisalo's (1956) work has shown that a more leisurely approach can be used without apparently undue strain on the cardiovascular system. The recumbent posture customary in these Finnish saunas also helps adequate coronary artery perfusion, rather than sitting or standing in the smaller units found in Britain. For many healthy people the sense of relaxation and well-being induced by a sauna will continue to outweigh the potential dangers of the circulatory gymnastics involved. On the basis of

these experiments, however, it appears to be neither health-giving nor heroic to prolong a sauna bath past the point where a person feels he has had enough.

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Continuous Recording of Direct Arterial Pressure and Electrocardiogram in Unrestricted Man

W. A. LITTLER, A. J. HONOUR, P. SLEIGHT, F. D. STOTT

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Summary

A system has been devised which records direct arterial pressure, electrocardiogram, and other variables in man with no restriction of activity. The advantage of this system is that the record is on magnetic tape, and it is possible to replay the records at various speeds to analyse each beat or to produce an entire 24-hour record on a few feet of paper. A marker allows the timing of relevant events. Initial experience with this procedure indicates that it is safe and reliable.

Introduction

Automatic sphygmomanometers operating at intervals can approach the ideal of continuous recording of arterial pressure, but the apparatus is bulky and the information gained, though

valuable, is incomplete (Richardson *et al.*, 1964). A small, completely portable apparatus was therefore developed which permitted continuous direct recording of both systolic and diastolic pressures over periods of up to 24 hours, the movements of the subject being entirely unrestricted with no observer present except to service the apparatus every 12 hours (Bevan *et al.*, 1969). The pressures measured with this original apparatus were recorded on 1 in (2.5 cm) bromide paper moving at 6 in (15 cm) an hour, and its small size made it necessary to use an episcopes to enlarge the trace between five and six times, as the arterial pressures had to be read directly from the record, which was viewed through a calibration grid. This made the analysis of the record tedious and time-consuming and, because of the slow paper speed, made resolution of individual beats impossible, and much information, particularly of short-lived events, could not be resolved.

Further development of this apparatus has been made which overcomes the deficiencies of the photographic record. By using a miniature analogue multichannel tape recorder electrical signals representing systolic and diastolic pressures, electrocardiogram, event marker, and other values are impressed on magnetic tape. A playback deck enables the signals to be displayed simultaneously on an oscilloscope or to be converted to a permanent record by using a Consolidated Electrodynamics (5-127) (UV) recorder.

In conjunction with the Atomic Energy Research Establishment, Harwell, ancillary electronic equipment (Harwell 6000

Radcliffe Infirmary, Oxford

W. A. LITTLER, M.D., M.R.C.P., Lecturer, Cardiac Department
 A. J. HONOUR, M.A., D.PHIL., First Assistant, Department of the Regius Professor of Medicine
 P. SLEIGHT, M.D., F.R.C.P., Consultant, Cardiac Department

M.R.C. Clinical Research Centre, Northwick Park, Middlesex
 F. D. STOTT, M.A., D.PHIL., Member of Scientific Staff