Original Article

AStudy of Cardiac Function During Heat Stress using Systolic Time Intervals

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Eighteen healthy male volunteers were exposed to a hot environment (52 deg. dry bulb, 33 deg. wet bulb) for 50 min. Contractile function of the heart was studied using the systolic time intervals. The heat exposure was accompanied by a significant decrease in the pre ejection period/left ventricular time ratio (PER/LVET), the pre-ejection period index (PEPI), and an increase in the left ventricular ejection time index (LVETI). The QS2I (the electro-mechanical time index) showed a reduction. The results suggest an increase in myocardial contractile function in the initial 40 min or so of the heat exposure, but towards the end of the exposure, the relative reduction in the PEP/LVET ratio tends to reduce. This may mean a relative decrease in the heart contractility. This finding may be of relevance in the generation of heat induced syncope, and in the cardiovascular status of a patient of heat stroke.

Key words: heat stress, myocardial contractily, STI.

Exposure to moderately severe heat stress is met with in military and industrial situations, and is likely to affect body physiology adversely. The problem is particularly severe during high speed low level flying in the summer when aircrew are often exposed to high cockpit temperatures for 40-50 min (in fighter operations), resulting in whole body heating[1,2]. It was suggested by Rowell[3] that inotropism of the heart increases with an increase in body temperature, and this effect helps to maintain the stroke output and the systolic blood pressure (SBP) eventhough there is a substantial peripheral vasodilatation which would tend to reduce these two variables. Frey and Kenney[4] used systolic time intervals (STI) to examine myocardial contractility of normal subjects during heat exposure to conclude that this variable increased only after about 15 min of exposure. They did not report on the PEP/LVET ratio, and the electromechanical index (QS2I) which are reliable indicators of myocardial contractile activity[5,6,7,8], and based their conclusions only on the decrease in the pre-ejection period (PEP) index. Also, during moderately severe heat exposure (as met with in low level flying in the summer) the body temperature increases to about 30°C[1,2,9], while the core temperature in the reported study increased only to about 37°C. The method used by these authors to increase the body temperature of their subjects was different to the whole body heating in a hot cockpit used by us.

The findings of the Frey and Kenney study therefore may not be applicable to the heat stress situation met with by aircrew during low flying. In view of this, we have measured the myocardial contractility of our subjects exposed to moderately severe heat stress using STI. The study has relevance to the physiological tolerance of military aircrew exposed to heat stress during high speed low-level flying.

Material and Methods

Eighteen healthy male volunteers (mean SD of age 32.0 ± 6.3 yr; height 170.9 ± 4.6 cm; weight 64.0 ± 5.7 kg.) were recruited for the study which was ethically approved. The subjects were clinically examined to ascertyain their fitness to undergo the experiments. Their prior consent was obtained.

Precordial electrodes, the carotid pulse pickup transducer (British Physical Lab., TF 111-S), and the BPL phonocardiograph (TK-111-S) were applied to record a CM5 ECG lead, the carotid pulse tracing and the phonocardiograph record. The output of the two transducers was amplified in a BPL 1083 A input box, and recorded simultaneously with the ECG on a Grass 5d polygraph at 100 mm/sec. The electromechanical systole (QS2), the left ventricular ejection time (LVET) and the pre-ejection period (PEP) were derived by using standard method[8].

The oral temperature was measured using an Ellab (Copenhagen) thermocouple probe and galvanometer. The skin temperature was recorded using a Naina temperature recording system with the skin sensors placed on the chest,

the right arm, right thigh and the right leg, and the mean skin temperature was derived from this data using Ramanathan's formula[10].

After the resting STI and body temperatures were recorded at room temperature (25C), the subjects underwent a 50 min exposure to an average dry bulb and wet bulb temperature of 52C and 33C respectively (Oxford Index 35.9C) in the IAM hot cockpit in which desired temperature/humidity conditions can be maintained satisfactorily[2]. During the heat exposure the physiological variables were measured at 10 min intervals. The fluid and electrolyte intake of the subjects was not regulated. The subjects were free to abandon the experiment at any time. All the subjects completed their exposures without difficulty. Skin temperature could be obtained satisfactorily in only 10 of the subjects.

The STI were measured from at least 5-7 cardiac cycles. The PEP/LVET ratios were derived from uncorrected (for heart rate) values[5]. The others were corrected for heart rate using regression equations formulated from the data of various STI recorded over a heart rate range of 43-140 beats/min in 43 healthy male subjects[11], and were then reported as indices[5].

The students paired t test ($p < 0.05$) was used to statistically compare the values of the pre-exposure STI and PEP/LVET ratio to their values measured during the heat exposure at 10 min intervals. It was not considered it necessary to assess the heart rate and body temperature responses in this manner.

Results

The heart rate and the oral temperature increased almost linearly with heart exposure duration while the skin temperature increase also followed the usual pattern (Fig.1)[1].

The STI response is given in table I. The PEP/LVET ratio had reduced significantly by the 10th min of exposure, reaching its lowest value by 40th min, but thereafter increased (relatively) slightly. The PEPI reduced as did the QS_2I. The LVETI however increased slightly with increasing

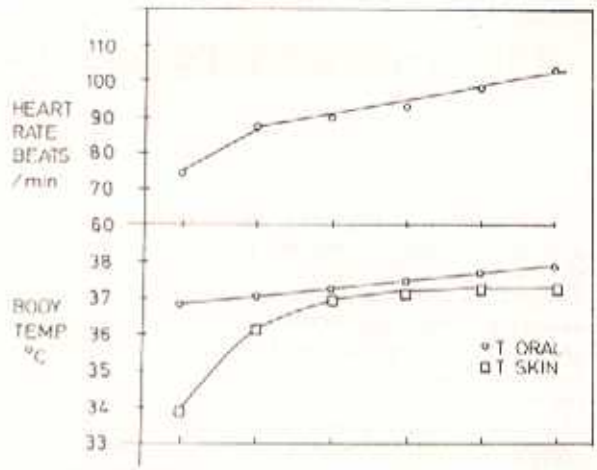

Fig 1 Mean values of oral temperature (Toral), mean skin temperature (Tskin) and heart rate (beats/min) for subjects exposed to heat stress (Oxford Index of 35.9°C) for 50 min in a hot cockpit.

duration of heat exposure. The mean percentage change in the PEP/LVET ratio is given in fig. 2.

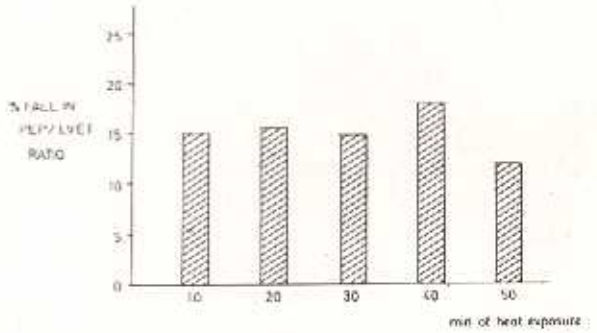

Fig 2 The mean percentage fall in the PEP/LVET ratio from its control value in subjects exposed to a heat stress of 35.9°C Oxford Index for 50 min. The values were significantly lower ($p < 0.01$) upto 40 min of the heat exposure, while the 50th min value was significant at the 5% level.

Discussion

There was a steady increase in heart rate and body temperature with heat exposure (Fig.1). These effects are well documented[2,3,9]. The initial rapid increase in the skin temperature has been associated with a feeling of discomfort and performance decrement[12], while tolerance time is dependent upon high core temperature[3,2].

In this study, an increase in myocardial contractilty was well established by the 10th min

Table-I Mean and SE values for the PEP/LVET ratio, the pre-ejection period index (PEPI), left ventricular ejection time index (LVETI) and the total electromechanical time index (QS2I) before the heat exposure (pre-exposure) and at 10 min intervals during the heat exposure.

		Pre exposure	10 min	20 min	30 min	40 min	50 min
PEP/ LVET	M	0.396	0.339	0.335	0.337	0.325	0.348
	SE	0.020	0.010 ***	0.012 ***	0.013 ***	0.010 ***	0.010 **
PEPI	M	106.0	91.0	89.0	87.0	84.0	87.0
	SE	3.0	2.8 ***	3.0 ***	2.8 ***	2.8 ***	2.8 ***
LVETI	M	361.0	370.0	370.0	369.0	372.0	372.0
	SE	3.3	2.8 **	2.4 **	2.6 *	2.6 **	2.4 **
QS2I	M	466.0	460.0	459.0	457.0	457.0	459.0
	SE	3.8	5.2	3.5	4.0 *	3.8 *	4.5

The * indicate the statistical difference of each value compared with its pre-exposure value using a Students 't' test. * = p 0.05; ** = p 0.01; *** = p 0.001. the indices are in milliseconds (ms).

of heat exposure (Table-I) because PEP/LVET ratio and the PEPI which are reliable indicators of myocardial contractility[5,6,8] decreased significantly. This effect is likely to be dependent more upon the initial rapid increase in the mean skin temperature rather than the oral temperature change because the contractilty increase appears to have reached its peak with the mean skin temperature peak, while a negligible change in the variable under discussion occured even though the oral temperature continued to rise linearly with increasing duration of the stress. Allan and Gibson[12] reported severe discomfort in subjects whose skin temperature increased rapidly during heat exposure. Such a stressful situation is likely to excite the sympatho-adrenal axis and cause an enhancement of myocardial contractility.

The LVETI in our subjects increased with heat exposure. An increase or a decrease in this variable may occur with an increase in myocardial contractility. The former effect is brought about by an increase in the pre-load and a sustained contraction of the myocardium while the latter is expected with an increase in the velocity of fibre contraction[5]. A combination of all these factors will decide the final outcome. The peripheral vasodilatation which occurs with whole body heating excludes the increase in the pre-load as a factor which prolonged the LVETI. Our findings are supported by the decrease in the QS_2I. This variable is relatively unchanged over a wide range of heart rates, but is known to decrease only with increase in the inotropism of the heart [5]. Frey and Kenney[4] did not measure this variable, nor did they report their skin temperature data, but chose to base their conclusions on their PEPI and LVETI data, (both the variables showing a decrease) making it difficult to fully interpret their findings. They also concluded that in the early phase of the heart stress, a vagal withdrawal occurs.

It is also possible that the variation in the findings of this study and that by Frey and Kenney occured because of a difference in the methodology used by them (dipping legs in hot water at about 42-45°C while our subjects underwent whole body heating as it occurs in the flight situation, and therefore may be considered more realistic.

Ethnic differences between the subjects used in the two studies may explain the variation in the findings of these studies. Indians are known to have a good tolerance to severe heat stress[2].

An interesting observation in our series of experiments was the relative decline of the

enhanced contractility at 50 min of exposure (Table-I; Fig.2). At this juncture, it is difficult to interpret this finding which may be an experimental variability, but could also mean that the myocardial contractility starts to decline when the body temperature reaches about 38C. More studies will have to be done to confirm this observation. If the latter hypothesis is true, it may well be that the finding could have a relevance on the causation of heat syncope in susceptible individuals, and may also help to monitor myocardial function of patients with heat stroke.

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