CARDIOVASCULAR CHANGES DURING VARIED THERMAL STRESS

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Summary: The effect of thermal stress on healthy mongrel dogs was studied at ambient temperatures of 40, 45 and 50°C. At 40°C there was a linear increase in cardiac output, heart rate and oxygen consumption and cardiovascular failure did not occur even after 6 hours of exposure. At 45°C the circulation was hyperdynamic upto 60 min but at 90 min there was a steep fall in oxygen consumption, cardiac output, stroke volume and mean arterial pressure whereas heart rate increased significantly. At 50°C the hyperdynamic changes reached peak values in 30 min; but by 60 min, there was a collapse of cardio-vascular function leading to rapid decline and ultimate death. It seems that the point at which there is a sudden fall in oxygen consumption, cardiac output, stroke volume and mean arterial pressure could be taken as an index of significant heat injury.

Key words: thermal stress, heat injury, cardiac output, heart rate, mean arterial pressure, oxygen consumption

INTRODUCTION

Physiological responses to higher environmental temperatures have been the subject of many reports in the past but controversy continues to exist regarding the role of cardiac output (CO) and oxygen consumption (O_2 consumption) in man's adaptation to acute heat stress (1,8). Burch and Hyman (1) Daily a d Harrison (3) and Koroxenidis *et al.*(8) reported an increase in CO on exposure to high environmental temperature whe eas Damato *et al.*(4) reported no increase in CO at temperatures of 100 to 115°F. Keeping this in view and the recent work of Bynum *et al.* (2) justifying the valid ty of an anaesthetized dog as a heat stroke model, the present work has been undertaken to study some of the basic cardiovascular parameters at different temperatures and time intervals.

MATERIALS AND METHODS

Thirty healthy mongrel dogs (12-18.5 *kg*) of either sex and having the same fur characteristics were randomly divided into group I, group II and group III of 10 animals each. The dogs were kept on *ad libitum* food and water schedule before use. Each dog was fasted overnight and then anaesthetized with an intravenous injection of Nembutal sodium (30 *mg/kg* body weight). Control studies were done in all the groups at room temperature of 35 $\pm$ 0.5 C and relative humidity (RH) of 20 $\pm$ 10%. Rectal temperature (T_{re}) was recorded with a thermometre and none of the dogs were given any fluid replacement during the experiment. Each dog served as its own control.

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Parameters studied

Cardiac output (CO) was determined by single injection indicator dilution technique as described by Hamilton *et al.*(6) using Evan's blue (T_{1824}) as the indicator substance. Oxygen consumption (O_2 consumption) was measured by closed circuit method using Palmer's spirometer; heart rate (HR) was determined by lead II of the electrocardiogram; mean arterial pressure (MAP) was recorded from the femoral artery by mercury manometer on a fluidrive kymograph. Stroke volume (SV) was calculated by dividing CO with HR.

After control studies, group I animals were exposed to an ambient temperature (T_{amb}) of 40±0.5°C and observations were made at 120, 240 and 360 min after exposure. All the animals in this group survived the duration of the experiment. Group II animals were exposed to 45°C T_{amb} and the parameters were recorded at 30, 60 and 90 min after which 8 dogs died. Group III animals were exposed to 50°C T_{amb} and the measurements were done at 30 and 60 min after which all the ten dogs died. None of the dogs were given any fluid replacements. Relative humidity was kept at 20±10% in all the groups.

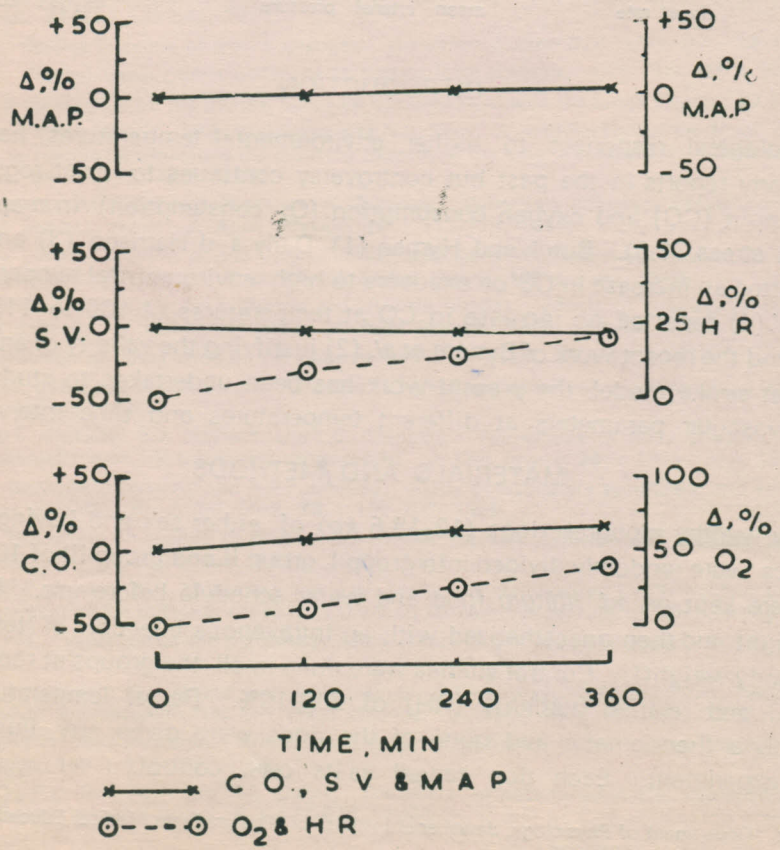

Fig.1 : Changes in cardiac output (CO), oxygen consumption (O_2), stroke volume (SV), heart rate (HR) and mean arterial pressure (MAP) in group I dogs exposed to 40°C ambient temperatrue.

RESULTS

Effects of exposure at 40 C

The results of group I animals exposed to 40°C T_{amb} are illustrated in Fig.1. It is quite evident that O_2 consumption, CO and HR increased progressively and had a linear relationship with the duration of exposure. However, SV did not show significant change but MAP was significantly elevated at 360 min duration. All the dogs survived the exposure of 360 min after which the experiment was terminated. Mean T_{re} at the termination of the experiment was 39.0±0.7 C.

Effects of exposure at 45 C

The effects of exposing group II dogs to 45°C T_{amb} are shown in Fig.2. O_2 consumption, CO, HR and SV increased more steeply as compared to group I dogs and these values were significantly higher after 30 and 60 min exposure. MAP also was significant'y

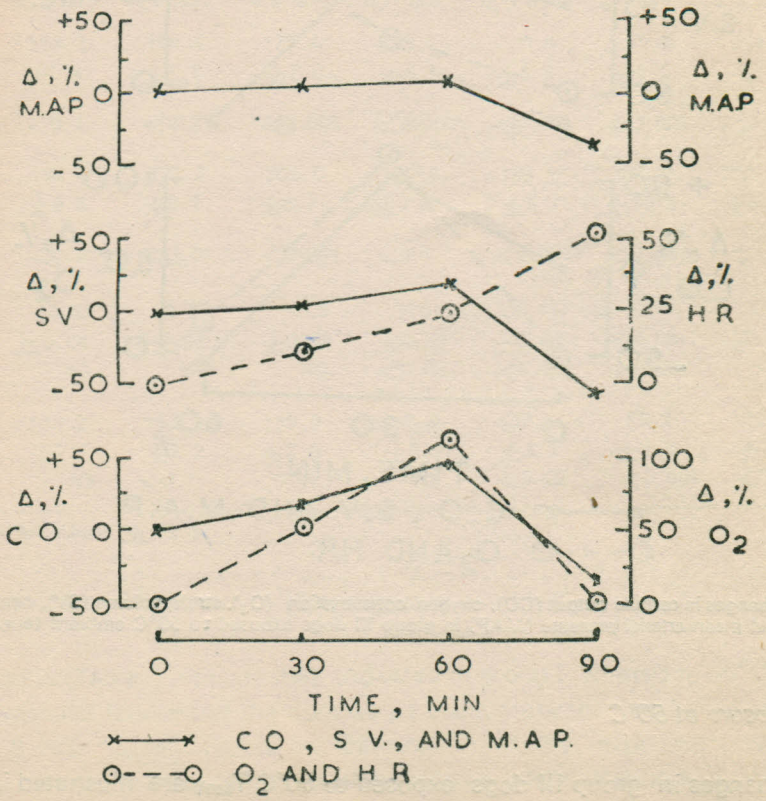

Fig.2 : Changes in cardiac output (CO), oxygen consumption (O_2), stroke volume (SV), heart rate (HR) and mean arterial pressure (MAP) in group II dogs exposed to 45°C ambient temperature.

elevated after 60 min exposure. But at 90 min, CO, SV and MAP were significantly lowe than the control values whereas O_2 consumption had decreased towards the control valu and the HR continued to rise significantly. Terminally there was bradycardia followed b asystole or ventricular fibrillation in eight out of the ten dogs which died at a mean T_{re} 44±0.5°C.

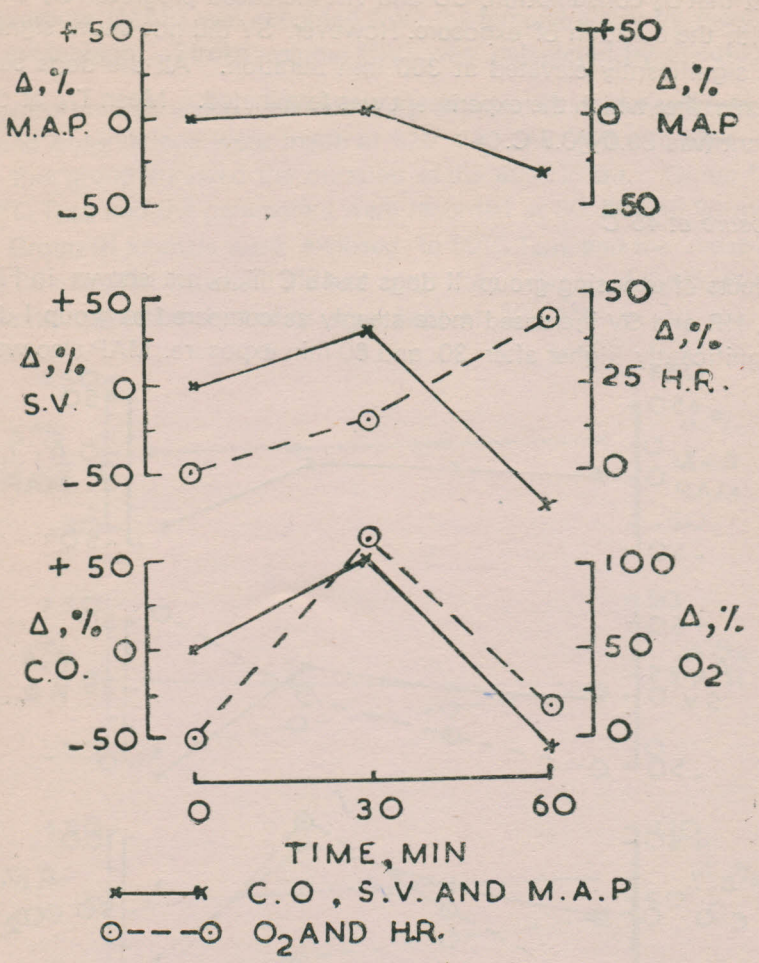

Fig.3 : Changes in cardiac output (CO), oxygen consumption (O_2), stroke volume (SV), heart rate (HR) and mean arterial pressure (MAP) in group III dogs exposed to 50°C ambient temperature.

Effects of exposure at 50°C

The changes in group III dogs exposed to 50°C T_{amb} are illustrated in Fig.3. CO, HR, SV, MAP and O_2 consumption increased rapidly and were significantly higher than the

control values after 30 min exposure but after 60 min there was a sudden and significant decrease in all the values except that of HR and O_2 consumption which were significantly higher than the control. Death of the animals was preceded by bradycardia followed by asystole or ventricular fibrillation as in group II animals. All the ten dogs died at a mean T_{re} of 44.8±0.3°C.

TABLE I : Effect of varied thermal stress on anaesthetized dog.

		Group I			*Grpup II*			*Group III*	
	Control	*Time after exposure at 40°C*			*Time after exposure at 45°C*			*Time after exposure at 50°C*	
		120 min	*240 min*	*360 min*	*30 min*	*60 min*	*90 min*	*30 min*	*60 min*
O_2 consumption (*ml/kg/min*)	6.16 ±0.05	6.96 ±0.15	7.76 ±0.15	8.68 ±0.12	9.34 ±0.22	13.26 ±0.66	6.36 ±0.31	12.75 ±0.11	7.02 ±0.26
P value		<0.001	<0.001	<0.001	<0.001	<0.001	NS	<0.001	<0.005
CO (*ml/kg/min*)	151.0 ±2.1	159.2 ±5.4	166.4 ±6.1	173.4 ±7.0	183.0 ±5.3	230.6 ±3.9	99.5 ±6.9	224.3 ±3.1	67.4 ±1.2
P value		<0.1	<0.2	<0.005	<0.001	<0.001	<0.001	<0.001	<0.001
HR (beats/min)	168.0 ±4.3	184.4 ±8.5	191.2 ±6.7	202.0 ±5.7	180.8 ±7.3	198.1 ±5.7	243.8 ±13.5	203.0 ±9.8	251.0 ±13.1
P value		<0.1	<0.01	<0.001	<0.1	<0.001	<0.001	<0.005	<0.001
SV (*ml/kg*)	0.91 ±0.03	0.87 ±0.04	0.87 ±0.03	0.86 ±0.03	1.02 ±0.04	1.17 ±0.03	0.41 ±0.03	1.12 ±0.05	0.27 ±0.01
P value		NS	NS	NS	<0.05	<0.001	<0.001	<0.001	<0.001
MAP (mm Hg)	128.5 ±1.4	132.2 ±2.2	133.2 ±2.2	135.0 ±2.0	132.8 ±2.9	137.4 ±3.4	82.2 ±2.3	136.0 ±3.0	89.6 ±7.9
P value		NS	<0.1	<0.01	NS	<0.02	<0.001	<0.05	<0.001

Values given are means ± SEM

DISCUSSION

Our results (Table I) indicate that exposure of group I animals to 40°C T_{amb} did not lead to cardiovascular failure and the dogs could easily fight the heat stress and survived the exposure for 6 hrs. Mean T_{re} at the termination of the experiment was 39°±0.7°C. Compensatory mechanisms for termal regulation come into operation and we find that there is progressive and linear increase in HR, CO and O_2 consumption (Fig.1) throughout the

duration of the exposure. These findings though contrary to those of Damato *et al.*(4) are in agreement with the work of Burch and Hyman (1), Daily and Harrison (3), Grollman (5) and Koroxenidis *et al.*(8) and are consistent with the view that in the initial stages of heat stress the circulatory demand is met by increased CO as a result of peripheral vasodilatation and increased venous return (12).

Thus the increase in HR boosts the cardiac pump as the pre-ventricular sumps remain adequately filled up. But exposure to greater ambient temperatures of 45°C and 50°C as in group II and group III respectively, lead to cardiovascular failure. Group II animals showed rapid compensatory cardiovascular changes like group I animals leading to hyperdynamic circulation at 30 and 60 min duration similar to that described by O'Donnell and Clowes (11) in patients suffering from heat stroke. But at 90 min there was a sudden fall of CO, SV, MAP and O_2 consumption whereas HR increased significantly above the control values. Eight out of the ten dogs in this group died at a mean T_{re} of 44 ± 0.5 C. In group III animals, hyperdynamic circulation was seen at 30 min (Fig.3) but at 60 min there was a steep fall in CO, SV, MAP and O_2 consumption whereas HR was significantly higher than the control and all the ten dogs of this group died at a mean T_{re} of 44.8 ± 0.3°C.

These findings are similar to those reported recently by Bynum *et al.*(2). Analysis of this data demonstrates a close relationship between the attainment of peak HR, collapse of MAP, and a large reduction in CO associated with decreased SV. This could be attributed to decreasing venous return as a result of massive veno-dilatation. Wright *et al.*(13) while working on rats have reported veno-dilatation and venous pooling at colonic temperatures of 42-43°C. They also found a decrease in responsiveness to nor-epinephrine with temperature elevation which suggests that the deterioration of vasoconstrictor response may also contribute to the development of cardiovascular failure. Apart from these factors decrease in myocardial contractility at T_{amb} of 40°C or higher (10) may also contribute to a sudden reduction in CO. Our results (Table I and Fig.2 and 3) further show that the failure of cardiovascular function in group II and group III animals is associated with abrupt decrease in O_2 consumption and therefore the possibility of hypoxic damage to myocardium cannot be ruled out. In addition, heat causes a direct damage to the myocardium as reported by Knochel *et al.* (7) and Malamud *et al.*(9) in victims of heat stroke. Our cardiovascular data also supports the view point of Bynum *et al.*(2) that the point at which CO abruptly falls to very low levels could be utilized to define the occurrence of significant heat injury.

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