32 showed mean values of 24.53 ± 1.84 mm, and it was larger in the hearts of animals under the age of 3 years (Table 1).

Table 1: Mean values of the position of the ventricular septum and its relation with the aortic valve (in mm) (n=32)

| Measures | Mean value | Sex | | |
|-----------|------------|-------|--------|--|
| | | Male | Female | |
| A | 13.81 | 13.56 | 14.06 | |
| В | 14.72 | 14.13 | 15.31 | |
| C | 17.66 | 17.50 | 17.81 | |
| D | 23.28 | 22.69 | 23.88 | |
| Ratio C/D | 24.53 | 25.30 | 23.75 | |

The mean aortic diameter (D) of the bovine heart was 23.28 ± 2.85 mm and there were no significant sexual and age differences in the present study. The aortic diameter was smaller in hearts of animals under the age of 3 years, and it had showed a progressive increment with age but statistically it was insignificant (p>0.05). Jatene et al.4 also reported the similar observations in human heart and found that the mean diameter of the aorta was 21.8 ± 3.6 mm, and there were no significant sexual or racial differences, but the diameter increased progressively with the increase in age. They also added that the aortic heart valve annulus did not show a perfect circumference with some variations in the measurements of the annulus, in the cusps and in the relation with the ventricular septum. In the present study, it was also revealed that the ratio C/D was higher in hearts of animals <3 years of age and progressively decreased with</p> the increase in age. This may suggest that perhaps left ventricular outflow tract (LVOT) shows a trend to proportionally decrease its area in relation to the aortic diameter with increasing age.

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Regulation of serum total protein and cholesterol level in cold-exposed rat

Several factors involved in the regulation of serum protein and cholesterol levels of humans as well as rodents. Cold exposure is the major sympathetic and environmental stimuli regulating metabolic functions¹. After prolonged exposure of rats to cold, a sustained increase in systolic blood pressure occurs, giving rise to a cold-induced model of hypertension². Activation of the sympathetic nervous system is thought to contribute to the development of hypertension in cold-exposed rats³. Sympathetic input to the kidney increases the release of renin, which in turn contributes to the production of Angiotensin II. The sympathetic output from the hypothalamus in response to cold exposure has been shown to regulate glycogen metabolism, degradation of glycogen to glucose and appeared in the higher glucose concentration in Moreover, cold-induced the serum. thermoregulation is associated with an increase in lipid metabolism^{4, 5}. Triglyceride degradation after sympathetic stimulation is predominantly observed. The resulting higher free fatty acids in the serum interact with serum binding protein like bovine serum albumin. It is speculated that cold exposure stimulates the accumulation of serum protein as well as cholesterol because of the constriction of blood vessels by higher blood pressure. The prolonged exposure may result vasodilatation regulating homeostasis of these constituents. Therefore, the present study has been undertaken whether cold exposure is involved in the regulation of serum constituents like protein and cholesterol.

Male rats weighing 200 to 260 g were used. They were housed in the cages at ambient temperature and given free access to laboratory foods and water. In the day of experiment, cold exposure (4-8°C) were given to the different groups of rats in the cold chamber for 30 min, 1 hour, 2 hour and 4 hour with full aeration and with free access of water. After cold exposure treatment, the rats were immediately anesthetized with diethyl ether and

were quickly decapitated. Blood was drawn from the jugular vein and was centrifuged at 3,000 rpm for 10 min. The serum in the supernatant was kept at -20°C.

Amount of serum total protein was estimated by the Folin-Lowry method⁶ and serum cholesterol was estimated according to Sockett's method⁷. All data were analyzed using analysis of variance (paired t-test) by Stat View software.

Table 1 shows serum protein for control and coldexposed rats. For control rats kept in ambient temperature, protein content was 9.3 ± 0.32 g/dL. After 30 min exposure of cold, the protein content was 9.5 ± 0.2 g/dL indicating only 2.1 % increase in concentration. After 1 hour of cold exposure, protein content was 12.1 ± 2.1 g/dL in the serum showing 30.1% significant increase (p<0.05). After 2 hour and 4 hour of cold exposure, total protein contents were 8.0 ± 0.9 g/dL and 5.5 ± 0.15 g/dL demonstrating 13.9% and 40.8% decreased significantly (p<0.05) in concentration respectively compared to the control rats. Serum protein was increased up to 1 hour and tend to reduce from 2 hour to 4 hour of cold exposure.

Table 1: Effects of cold exposure on serum protein content and cholesterol level of rats

| | Control rat | Cold-exposed rat | | | | |
|---------------------------------|-----------------|------------------|-----------------|---------------|----------------|--|
| | (n=3) | 30 min (n=3) | 1 hour (n=3) | 2 hour (n=3) | 4 hour (n=3) | |
| Serum protein content (g/dL) | 9.3 ± 0.32 | $9.5 \pm~0.2$ | 12.1 ± 2.1 | 8.0 ± 0.9 | 5.5 ± 0.15 | |
| Serum cholesterol level (mg/dL) | 100.0 ± 1.8 | $125. \pm 2.5$ | 155.5 ± 3.0 | 115. ± 3.3 | 85.6 ± 2.9 | |

Serum total protein maintains colloidal osmotic pressure of blood and aids in regulation of the disturbance of fluid between blood and tissue. Serum protein also maintains viscosity and blood pressure. It acts as buffer and antibodies. In the present study, serum total protein was significantly lower in 2 hour and 4 hour cold-exposed rats compared to the control (p<0.05). The amount of total serum protein in cold-exposed rats increased at the initial stage (1 hour) and decreased later and is supported by others⁸. Cold-exposure is known to induce concomitant increases in the levels of adrenomedullary tyrosine hydroxylase, RNA, protein and enzyme activities. Adrenomedullary bioprotein is increased rapidly after the onset of cold exposure. Our findings also suggest that cold stress induces increases in the levels of the tyrosine hydroxylase cofactor and may represent a key event in the sympathoadrenal system response to cold stress. The reduced serum total protein in cold exposed rats may be due to decrease secretion of insulin, because insulin has a global effect on protein metabolism increasing the rate of protein synthesis and decreasing the rate of protein degradation. Sympathetic input inhibits insulin secretion through α2-adrenergic receptor. High adrenergic stimulation generated by exposure of cold for homeothermic animals reduces insulin secretion.

Cholesterol, a derived lipid, is an important constituent of the blood. It exists in free and esterified form in the blood. In the present study, the mean values of serum cholesterol in cold-exposed rats of 30 min and 1 hour was significantly

higher (p<0.05) and declined at 4 hour compared with control. The significant reduction of serum cholesterol after 4 hour of cold exposure are caused by the decline in the sympathetic response to cold and an enhancement of the vagal response after repeated exposures to severe cold. Increased cardiovascular disease mortality has been related to thrombosis due to hemoconcentration in the cold. It is speculated that the increased sympathetic activity after short-term exposure producing higher arterial pressure causes accumulation of serum cholesterol. Hemoconcentration in the cold can in part explain the increase concentration in clotting factors and serum lipids.

Collectively, cold exposure has the dynamic effect on serum protein and cholesterol to maintain the homeostasis of the biological system. Short-term exposure activates the sympathetic nervous system and prolonged exposure to cold, however, may reduce the activity of sympathetic nerve rather tends to maintain homeostasis of the serum constituents.

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