Table III. Contractile characteristics for skeletal muscle

Group	Peak twitch tension (g/g)	Twitch time (msec)	Rate relaxation (g/sec)	TPT (msec)
1. Normal	451 ± 51[a]	244 ± 28	23.6 ± 3.2	29.4 ± 2.9
2. Alcohol	393 ± 48	313 ± 22	19.6 ± 3.8	33.0 ± 1.6
3. Dehydrated-malnourished	357 ± 40	251 ± 31	18.8 ± 3.5	32.2 ± 2.7

[a]Mean ± SE. No significant difference occurred between any groups in any category.

troversial. BURCH et al.[6] have reported an experimental alcoholic cardiomyopathy in mice based on electron microscopic evidence, and MAINES and ALDINGER[7] showed a 70% decrease in ventricular systolic tension in rats on long term consumption of 25% ethanol. In both studies, as in our study, alcoholic animals consumed only a fraction of the fluid intake of normal controls. Clinically, most patients who carry a diagnosis of alcoholic cardiomyopathy have an accompanying decreased total fluid intake as an integral part of their alcoholism.

The decreased twitch tension in the cardiac muscles of both the alcoholic and chronically dehydrated groups must again raise the question of the role of malnutrition, specifically dehydration, and perhaps concomitant electrolyte abnormalities in alcoholic cardiomyopathy. As pointed out by BURCH and GILES[8], in published experimental animal studies, alcohol containing fluids served as the entire source of liquid intake. Control groups of these studies, however, were not designed to provide for a reduced fluid intake commensurate with that found in the experimental animals. Our studies did, however, attend to this matter, and our results force us to consider that dehydration per se may play an important role in the etiology of alcoholic cardiomyopathy.

It is of interest that while significant differences in peak twitch tension of cardiac muscle was demonstrated, no significant skeletal changes were induced. This fits well with the clinical observation that alcoholic cardiomyopathy is seldom accompanied by clinically detectable skeletal myopathy[4].

Summary. In vitro isometric contractile tension was measured in heart and skeletal muscle in 3 groups of mice: 1. a control group, 2. a group maintained for 27 weeks on 20% alcohol, and 3. a group whose fluid intake was restricted to the extent equaling that which occurred in the alcohol treated animals. Results showed a reduction in cardiac twitch tension in both the alcohol and fluid restricted group, as compared to normal controls. We therefore consider that dehydration per se may play an important role in the etiology of alcoholic cardiomyopathy.

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Hemodynamic and Ventilatory Effects of Skin-Cooling in Cattle

Cattle are often used to study cardiopulmonary responses to alveolar hypoxia[1–4]. Such studies are generally concerned with interactions among pulmonary and systemic hemodynamics, alveolar or arterial blood gases, and ventilatory minute volume. Although it is usually assumed that inspired oxygen tension is the only environmental variable of physiological importance, cattle use ventilation to help regulate body heat and the ambient temperature at which the animals are studied might have significant cardiopulmonary effects[5]. It is important, therefore, to determine if thermoregulatory ventilation at the usual laboratory temperature of 25 °C influences the inter-relationships among cardiopulmonary processes. Thus, we studied the effects of skin-cooling on pulmonary and systemic hemodynamics, arterial blood gases, and minute ventilation in cattle at an ambient temperature of 25 °C.

Materials and methods. Systemic and pulmonary blood pressures, cardiac output, and arterial blood gases were measured in 12, 3- to 4-month-old, unanesthetized, Hereford calves following catheterization of the thoracic aorta, pulmonary artery, and right atrium. The techniques used in this study have been described elsewhere[4]. Minute ventilation was measured with a muzzle mask and a dry gas meter. Skin and rectal temperatures were monitored with thermocouples. Measurements were made at an ambient temperature of 25 °C before and after the skin on the calf's back was cooled for 30 min with cold water and a stream of air.

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Effects of skin-cooling on hemodynamic, ventilatory, and arterial blood gas variables in cattle

Variables	Skin-cooling[a]	
	Before	After
Ts (°C)	38.6 ± 0.1	32.9 ± 0.5[b]
Tr (°C)	39.8 ± 0.0	39.7 ± 0.1
HR (min^{-1})	96 ± 3	92 ± 2[b]
Q (ml/min/kg)	133 ± 7	129 ± 4[b]
SV (ml/kg)	1.39 ± 0.06	1.34 ± 0.05
P_{pa} (mm Hg)	30 ± 2	32 ± 2
TPVR (mm Hg/ml/min/kg)	0.24 ± 0.02	0.26 ± 0.02[b]
Pao (mm Hg)	104 ± 3	112 ± 4[b]
TSVR (mm Hg/ml/min/kg)	0.80 ± 0.06	0.92 ± 0.05[b]
f (min)	85 ± 7	40 ± 4[b]
V_E (ml/min/kg, BTPS)	400 ± 30	220 ± 10[b]
V_T (ml/kg, BTPS)	5.0 ± 0.3	6.5 ± 0.8
PaO_2 (mm Hg)	68 ± 1	66 ± 1
$PaCO_2$ (mm Hg)	36 ± 1	37 ± 1
pHa	7.46 ± 0.01	7.46 ± 0.01

Ts, skin temperature; Tr, rectal temperature; TPVR, total pulmonary vascular resistance; TSVR, total systemic vascular resistance. [a] Mean values ± SEM are shown for 12 animals (except $n = 10$ for Pao and TSVR). [b] Before and after values differ significantly, $p < 0.05$ (determined by a paired t-test).

Results and Discussion. The cardiopulmonary effects of skin-cooling are shown in the Table. In the cooled area, skin temperature was lowered by nearly 6 °C. The decrease in skin temperature probably evoked a peripheral vasoconstriction that elevated systemic arterial pressure despite a reduction in cardiac output. The elevated systemic pressure might have caused the fall in heart rate via increased baroreceptor activity. The increase in total pulmonary resistance could have been related to pulmonary vasoconstriction or to an increased left atrial pressure. If this pulmonary effect of skin-cooling is exaggerated in a cold environment, it might account for the higher incidence of hypoxic pulmonary hypertension and brisket disease in cattle during cold weather at high altitudes[1].

After the skin was cooled, respiratory rate and minute volume were reduced by about 50%. Arterial blood gases were not altered. These results suggest that although cattle hyperventilate at an ambient temperature of 25 °C, the increased ventilation represents panting and alveolar gas exchange is not affected. Environmental temperature must apparently approach 40 °C before thermoregulatory ventilation influences alveolar ventilation and arterial blood gases[5]. Thus, it can be inferred that in cattle at moderate laboratory temperatures arterial blood gases reflect alveolar ventilation as determined by non-thermoregulatory mechanisms. If variations in environmental temperature from 15 to 25 °C affected alveolar ventilation, then much blood gas data collected from normoxic and hypoxic cattle might be subject to reinterpretation[4,6–9]. The thermoregulatory-induced disassociation between minute and alveolar ventilation might account for the lack of correlation between changes in minute ventilation and arterial blood gases observed in cattle exposed to simulated high altitude[7] and to carotid body excision[10]. It is obvious that attention must be paid to ambient temperature when minute ventilation is used as a variable in studies of ventilatory control in cattle. Minute ventilation would probably be more closely related to alveolar ventilation if cattle were studied at an ambient temperature somewhat lower than 25 °C.

Summary. Cooling the skin of cattle at an ambient temperature of 25 °C decreased cardiac output and increased systemic and pulmonary vascular resistances. Minute ventilation was reduced by about 50%. There was no change in alveolar ventilation as measured by arterial blood gases. These results indicate that thermoregulatory ventilation has significant cardiopulmonary effects in cattle at normal laboratory temperatures.

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Seasonal Variations of Cardiac Output in Rats

Data on the cardiac output of rats given in the literature range between 142 ml/kg/min[1] and 457 ml/kg/min[2]. This great variability was attributed to different techniques, or to differences in the age, strain and sex of the animals[3]. In our laboratory determination of cardiac output were performed in rats of the some strain and sex and nearly the same age over 4 years; inspite of the constant experimental conditions, values varied greatly. Trying to find an explanation for this phenomenon, we have detected seasonal variations in the cardiac output of rats.

Materials and methods. We used male Wistar rats (conventional animals; dealer P. Bäumler, Wolfratshausen) weighing 320 to 400 g which were housed at 23 °C and fed with Altromin pellets and tap water ad libitum. The

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