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Introduction

The subject of heat stroke has received wide attention in the medical literature because of its dramatic clinical presentation and often fatal outcome. More commonly, physicians working in hot environments are called upon to evaluate casualties with heat cramps, syncope, or heat exhaustion. Classically, heat cramps have been felt to be a consequence of extracellular hyponatremia; heat syncope is the result of postural hypotension; and heat exhaustion has been defined as an inability to continue work in the heat due to salt or water depletion (1, 2). While conducting a field study of heat casualties at Fort Polk, LA, during the summer of 1970, we were impressed by the number of patients who were hyperventilating when first seen by a physician. In attempting to classify patients with heat injuries, it became apparent that many of the symptoms classically attributed to heat exhaustion, as well as heat syncope and heat cramps, could often be a manifestation of extreme hyperventilation in the heat. The present study was undertaken to obtain information about the acid-base picture in military recruits presenting to a heat ward with heat exhaustion, heat syncope or heat cramps.

Patients and Methods

All patients brought to the heat ward at Fort Polk, La. from July 12 through July 23, 1971, with heat stress illnesses were clinically evaluated by one of the investigators at the time of admission. All patients developed symptoms while training in the field. Upon recognition of acute heat illness, they were promptly moved to the shade, usually doused with water and evacuated by ambulance to the heat ward. The patients arrived at the heat ward from 10 to 30 minutes after the onset of symptoms. During the two week period of study, seventeen patients with heat exhaustion and one case of heat stroke were evaluated. Brachial arterial blood samples were drawn immediately upon admission in heparinized glass syringes after local anesthesia with 1% lidocaine. Venous blood for creatinine, electrolytes and magnesium was drawn, rectal temperature obtained, and a complete history obtained with attention directed to the events leading up to the patient's illness.

Arterial pH, pCO_2, and pO_2 determinations were performed on an Instrument Laboratories Model 113 pH Gas Analyzer. Samples were run within several minutes after arterial puncture and were kept on ice until analysis. Plasma lactate and pyruvate were determined on protein free filtrates of arterial blood using the Sigma reagent kit. Pyruvate was determined on the same day because pyruvate was not found to be stable with refrigeration. Optical density readings were taken on a Coleman Junior spectrophotometer at 340 mu and results calculated from standard curves and corrected for the weight of each sample. Serum electrolytes, magnesium (mg^{++}) and creatinine were subsequently determined on frozen serum by routine laboratory methods.

Results

The clinical data from the 17 patients with heat exhaustion are shown in Table 1 and demonstrates the difficulty in placing patients into distinct clinical groups. All patients were diagnosed as heat exhaustion by previous criteria cited in the literature (1, 2), but, in addition, 16 of 17 had abdominal or extremity muscle cramps as major symptoms, and 11 of 17 had experienced a syncopal episode. All patients were observed to have an increased respiratory rate on admission. Rectal temperature was elevated in 11 of 17 patients even though most of the patients had been doused with water.

All patients were in prior good health and related no history of a recent respiratory infection or gastroenteritis preceeding their admission. Twelve of 17 patients developed symptoms while speed marching and almost all casualties occurred in the afternoon. Examination of the WBGT index--a measurement which reflects humidity, wind velocity, and temperature, reveals this factor is consistently higher during the afternoon (1400 - 1600 hrs).

Serum electrolytes, mg++, and creatinine were in the normal range in the majority of the patients (Table 2). Hemoconcentration was observed in patients 4, 6, 11, and 15. Plasma lactate and pyruvate were also in the normal range in the heat exhaustion group.

The only patient with heat stroke seen during the course of this study had an increased respiratory rate of 30/min. As compared to the heat exhaustion patients, the heat stroke patient had a pH of 7.28, pCO_2 31.8 mm Hg, and an elevated arterial lactate of 4.22 mM/L. Serum electrolytes in this patient were: sodium: 137mEq/L, K^+:5.0mEq/L, Cl: 100mEq/L, and HCO_3^-: 3mEq/L.

Discussion

The results of the present study indicate that heat exhaustion, as seen in recruits in basic training, is associated with a marked respiratory alkalosis. Although Haldane first reported that men at rest hyperventilated in excessively hot environments (3), the prevalence of this finding in heat casualties had not been previously recognized. The first clinical report of hyperventilation and tetany in a tropical climate was in 1941, when Wingfield described a 27-year-old ship engineer who had attacks of tetany which occurred while in the intense heat of the Persian Gulf (4). These attacks would be produced by voluntary hyperventilation, and were similar to those produced experimentally by Landis (5) and others (6,7,8). More recent studies of the thermal ventilatory response of resting men have demonstrated that an increased tidal volume, rather than increased respiratory rate, accounted for the major protion of heat-induced hyperventilation (9).

The hyperventilatory response observed in patients undergoing heat stress, or associated with the development of symptomatic heat exhaustion, may be related to concomitant anxiety which is also a cause of the hyperventilation syndrome. It appears, however, that in the present study the patients with heat exhaustion and hyperventilation tetany and/or peripheral numbness or paresthesias were unable to perceive a subjective feeling of anxiety prior to the onset of symptoms. It is also very likely that the syncopal episode experienced by heat exhaustion patients in the study were not those classically described as "heat syncope" (10-14) thought to be related to venous pooling. It may very well be that many cases of "heat syncope" may be hyperventilation-induced syncope. Further studies are warranted to examine this hypothesis.

Only two of the 17 heat exhaustion patients (4, 11) appeared to be severely dehydrated as assessed by hypernatremia and serum creatinine elevation. Interestingly, these two patients had the lowest arterial pH values of the entire group. In evaluating heat casualties more carefully, one may find a group of patients who are not salt or water depleted, but have a marked respiratory alkalosis.

Hyperventilation is accompanied by hyperlactatemia in some disease states in man (15-19). Sustained mechanical hyperventilation in dogs is associated with an increase in blood lactic and pyruvic acid (20-22). The lactatemia of hyperventilation in dogs does not appear to be the result of tissue hypoxia, lactic acid release from muscle, or decreased liver utilization of lactate, but rather from a stimulation of red blood cell glycolysis by hyperventilation (23).

Huckabee considered the lactatemia of hyperventilation to be a benign process (24), since he found no oxygen debt and the lactate/pyruvate ratio (L/P) unaltered in dogs which were hyperventilated for one hour (22). In contrast to Huckabee's findings, changes in the L/P ratio and excess lactate as a result of hyperventilation do occur in longer term studies in dogs (19). Thus, it is theoretically possible that hyperventilation in the heat might lead to excess lactate formation in man. In the only two cases of heat stroke in the literature in which blood lactate was measured, both patients were acidotic with arterial pH of 6.9 and 7.17 with marked elevation of arterial lactate to 11.2 and 6.4 mM/L, respectively (25). These investigators observed mitochondrial damage in liver biopsies obtained within minutes of death. They felt that hyperpyrexia may result in irreversible changes in mitochondrial structure and function, with an associated effect on aerobic metabolism resulting in excess lactate production and acidosis. Althought the mechanism of the lactic acidosis in these patients is apparently due to irreversible tissue damage, it may be that hyperventilation alone might eventually contribute to the acidosis of heat injury by raising the lactate out of proportion to the pyruvate. Although a number of patients were found to have lactate/pyruvate ratios greater than 10, this study did not indicate that heat-induced hyperventilation results in a rise in lactate that was clinically significant.

Although the role of hyperventilation in heat stress syndromes is speculative, several points should be stressed in relation to the present data. The symptoms of heat exhaustion are nonspecific and many heat casualties seen in basic training could have symptoms secondary to hyperventilation. This finding may be important since previously the etiology of heat exhaustion has been felt to be due to either salt or water depletion, or both, which did not appear to be a major factor in the military recruits seen

with heat exhaustion in the present study. The majority of these patients had an adequate fluid intake by history on the day of admission and their electrolyte data does not indicate significant dehydration.

Secondly, heat cramps, which occur commonly in heat exhaustion, may often be the result of hyperventilation-induced muscle spasm and does not necessarily reflect intracellular overhydration or chloride loss. Although the patient with the most severe cramps had hyponatremia, 15 of the 16 remaining patients with cramps had normal to elevated serum sodium and chloride, arguing against the more classic etiology of heat cramps in the patients in this study.

Thirdly, many of the patients who faint while exercising in the heat may do so on the basis of hyperventilation. The clinical and experimental literature demonstrate that the syncope is due to pooling of blood in the extremities and splanchnic vascular bed resulting in orthostatic symptoms (10-14). Hyperventilation with its resulting decrease in cerebral blood flow (26-28) could account for a significant number of military recruits who are diagnosed as "heat syncope".

These findings do not allow a definite conclusion concerning the role of hyperventilation in the heat stress syndromes. The profound respiratory alkalosis in this group of patients, coupled with the development of symptoms characteristic of hyperventilation prior to a syncopal episode and then prompt recovery on removal from the heat without further therapy, suggest that many of the manifestations of heat stress syndromes as seen in military recruits are the result of an exaggerated hyperventilatory response to thermal extremes.

Table 1

CLINICAL DATA IN HEAT EXHAUSTION PATIENTS

PATIENT NO.	AGE	RACE*	ACTIVITY AT TIME OF SYMPTOMS	SYMPTOMS		RECTAL TEMPERATURE ON ADMISSION†	RESPIRATORY RATE ON ADMISSION
				SYNCOPE	CRAMPS		
1	19	C	Marching	Yes	Abdominal (Abd.)	99.6	24
2	20	C	Running Mile	Yes	Legs/Abdominal	98.4	30
3	20	C	Rifle Range	No	No	99.4	24
4	21	C	Rif. Rge./Aft. Mrchg	Yes	Hands	100.4	22
5	21	N	Marching	No	Severe/Abd./Legs	102.4	35
6	22	C	Marching	No	Legs	100.0	22
7	20	N	Rifle Range	No	Mild	100.0	22
8	20	C	Marching	Yes	Abdominal/Legs	100.8	30
9	20	C	Marching	No	Abdominal	101.4	24
10	18	C	Marching	Yes	Chest	100.8	18
11	19	C	Marching	Yes	Tetany	101.5	30
12	18	C	Marching	Yes	Severe	100.6	30
13	20	C	Marching	No	Mild	98.6	26
14	19	C	Marching	Yes	Abdominal/Legs	100.7	30
15	23	C	Rifle Range	Yes	Abdominal/Legs	101.0	32
16	18	N	Marching	Yes	Chest/Legs	101.2	28
17	17	C	Marching	Yes	Abdominal	100.6	22

* C: Caucasian
  N: Negro

† In degrees Farenheit

Table 2

SERUM ELECTROLYTES*, MAGNESIUM*, AND CREATININE† CONCENTRATIONS IN HEAT EXHAUSTION PATIENTS

PATIENT	Na^+	K^+	Cl^-	HCO_3^-	CREATININE	MG^{++}
1	142	4.5	100	20	1.15	1.73
2	145	4.2	102	7	1.50	1.92
3	143	4.7	102	22	1.05	2.10
4	162	4.3	116	17	1.84	1.30
5	141	4.3	102	10	1.40	1.64
6	152	4.3	--	--	1.30	2.12
7	140	4.1	100	18	.95	1.73
8	145	3.8	102	22	1.20	1.86
9	--	--	--	--	--	--
10	140	4.0	100	16	1.30	1.61
11	160	4.9	106	--	1.50	2.00
12	130	4.5	100	--	1.15	1.50
13	141	4.0	104	17	1.30	1.78
14	145	4.8	102	19	1.05	1.84
15	148	3.7	104	20	1.65	1.59
16	148	4.3	105	22	1.15	1.84
17	146	5.1	102	11	1.05	2.00

* mEq/L

† mg per cent

Table 3

ARTERIAL BLOOD GASES, pH, LACTATE AND PYRUVATE CONCENTRATIONS IN HEAT EXHAUSTION PATIENTS

SUBJECT NUMBER	LACTATE mM/L	PYRUVATE mM/L	pH	pCO_2 (mm Hg)	pO_2 (mm Hg)
1	--	--	--	--	--
2	--	--	--	--	--
3	--	--	--	--	--
4	0.75	.055	7.47	34.0	74.2
5	1.48	.081	7.50	32.4	69.8
6	--	.089	7.70	14.8	--
7	1.36	.061	--	--	--
8	0.82	.089	7.52	28.8	89.9
9	0.62	--	7.69	19.8	105.6
10	0.62	.079	7.56	29.4	88.2
11	1.44	--	7.44	34.2	77.9
12	--	.100	7.71	17.2	96.2
13	0.91	.085	7.77	15.2	110.8
14	0.49	.069	7.76	16.3	87.8
15	1.05	--	7.66	19.7	90.6
16	1.25	.073	7.78	14.7	93.3
17	0.67	.074	7.53	28.4	66.6
MEAN	0.955	.078	7.62	23.5	87.5
SD	0.35	.013	0.12	7.8	13.5
SEM	0.10	.004	0.03	2.2	3.9

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