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沙漠干热环境创伤失血性休克猪模型的氧代谢特点*

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摘要 目的：探讨沙漠干热环境创伤失血性休克猪的氧代谢特点。**方法：**选择长白仔猪 40 头，随机分为四组：常温假手术组(NS)、常温创伤失血性休克组(NTHS)、干热假手术组(DS)、干热创伤失血性休克组(DTHS)，分别置于相应的环境暴露 3 小时后，进行麻醉，动静脉置管，NTHS 组和 DTHS 组分别自剖腹术后，行左下叶 1/4 肝脏切除及脾切除术后，再快速放血至平均动脉压(MAP)降至 45± 5mmHg；NS 组和 DS 组仅行腹中线剖腹术。持续检测计算动脉、混合静脉氧饱和度、氧含量及氧输送(DO_2)、氧耗(VO_2)、氧摄取率(O_2ER)和动脉血乳酸(Lac)。**结果：**整个病程中，各组动脉氧饱和度均无显著变化。DTHS 组混合静脉氧饱和度和氧含量均较相同时间点的其他各组低，DO_2、VO_2、O_2ER 均显著高于常温环境组($P<0.05$)。模型成功后，NTHS 组和 DTHS 组 DO_2 均经历 " 下降 - 代偿 - 稳定 " 的过程，但 DTHS 组短暂稳定后立即呈进行性快速下降至到动物死亡。在实验过程中，DTHS 组各时间点氧摄取率(O_2ER)均高于相同时间点的其他组，差异具有统计学意义($P<0.05$)。NTHS 组和 DTHS 组氧 O_2ER 均在休克后 0 h 出现明显变化，而动脉血乳酸(Lac)在休克后 1.5 h 才出现明显变化，但 DTHS 组动脉 Lac 增高较 NTHS 组升高更加明显($P<0.05$)，且进展迅速。**结论：**(1)沙漠干热环境创伤失血性休克较高的氧代谢，是机体代偿能力弱、病程变化快的重要原因之一；(2)VO_2、O_2ER 等直接氧代谢指标可作为早期评估监测机体氧代谢的敏感指标；(3)血 Lac 浓度可能是反映干热环境创伤失血性休克严重程度的重要指标。

关键词：沙漠；干热环境；创伤失血性休克；猪；氧代谢

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The Characteristics of Oxygen Metabolism in Swine Model with Traumatic Hemorrhagic Shock in Dry-heat Environment of Desert*

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ABSTRACT Objective: To establish investigate the characteristics of oxygen metabolism were compared and analyzed. **Methods:** 40 cases of Landrace piglets were randomly and equally divided into the normal temperature sham operation (NS) group, normal temperature traumatic hemorrhagic shock (NTHS) group, dry-heat environment sham operation (DS) group, dry-heat environment traumatic hemorrhagic shock (DTHS) group. After being exposured to each envionment for 3h respectively, the swine were anaesthetized and catheterized, and then laparotomy were performed , after laparotomy, NTHS group and DTHS group underwent left lower lobe 1/4 liver resection and splenectomy, further rapid bleeding to mean arterial pressure (MAP) was approached 45± 5 mmHg to establish the traumatic hemorrhagic shock model, NS group and DS group underwent laparotomy alone. The arterial, mixed venous oxygen saturation and oxygen content, oxygen delivery (DO_2), oxygen consumption (VO_2), oxygen uptake rate (O_2ER) and arterial blood lactate (Lac) were continuously measured. **Results:** There was no significant change in the arterial oxygen saturation during the whole course of the disease. The mixed venous oxygen saturation and oxygen content of the DTHS group were lower than the other three groups at the same time point. After being exposured for 3 h in dry-heat environment, DO_2 and VO_2 were significantly higher than those in the normal temperature environment group ($P<0.05$). After the traumatic hemorrhagic shock model were successfully established, the DO_2 of NTHS and DTHS groups underwent the process of "falling-compensatory-stabilization" in each group, but in the DTHS group, after shorter stable stage, the DO_2 was falling quikly to the death. O_2ER was significantly higher in the DTHS group than other three groups at the same time point ($P<0.05$). In theNTHS and DTHS group, the O_2ER had significant changed early in the just establishment of the shock model (0 h). How-

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ever, the level of arterial blood lactate had significant changes until 1.5 h after the establishment of shock model. And the level of lactate was higher in the DTHS group than that of the NTHS group at the same time point. **Conclusion:** (1) The traumatic hemorrhagic shock in desert dry-heat environment could induce higher oxygen metabolism, which is one important reasons for the weak compensatory ability and rapid deterioration of the course of disease; (2) Direct oxygen metabolism (such as VO_2, O_2ER) can be use as a sensitive index for early assessment of oxygen metabolism monitoring; (3) Blood Lac concentration can be used as an important index to assess the serious degree of shock of traumatic hemorrhagic shock in the dry heat environment of desert.

Key words: Dry-heat environment; Trauma; Hemorrhagic shock; Swine; Oxygen metabolism

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前言

失血性休克是由于多种致伤原因导致的血管内血容量急剧减少,氧输送不足,重要组织器官灌注不足,细胞缺氧,最终导致多器官功能障碍甚至衰竭的临床常见危急重症[1]。本课题组前期的研究显示沙漠干热环境的火器伤及创伤失血性休克对机体损伤发生早、程度重、生存时间短等特点[2-4]。研究表明组织氧代谢障碍是导致创伤失血性休克患者死亡率高的重要原因[5],但目前国内外尚未见沙漠干热环境创伤失血性休克的氧代谢特点相关的文献报道。因此,本研究通过建立沙漠干热环境和常温环境创伤失血性休克猪模型对比研究沙漠干热环境创伤失血性休克过程中氧代谢特点,以期为临床病情评价及救治提供理论依据。

1 材料与方法

1.1 实验动物及主要材料

雄性长白仔猪:购自新疆天康畜牧生物技术股份有限公司;模拟环境在西北特殊环境人工实验舱(新疆军区总医院研制)内进行;麻醉呼吸机 EX-60:深圳迈瑞生物医疗电子股份有限公司;血气分析仪:GEM Premier 3000,USA;心电监护仪(T8):深圳迈瑞生物医疗电子股份有限公司;BL-420 生物测温仪:中国成都泰盟;血气针:北京海富达科技有限公司,实验经新疆军区总医院实验动物伦理委员会审查通过。

1.2 实验分组

本地雄性长白仔猪 40 头(25 kg-35 kg,7-9 周),随机分为常温假手术组(Normal temperature sham group,NS 组,n=10),常温创伤失血性休克组 (Normal temperature traumatic hemorrhagic shock group,NTHS 组,n=10),干热假手术组 (Dry-heat sham group,DS 组,n=10),干热创伤失血性休克组(Dry-heat traumatic hemorrhagic shock group,DTHS 组,n=10)。

1.3 环境设置及麻醉

在人工实验舱内设置环境条件,常温环境:温度 25℃± 1℃,湿度 35%± 5%;沙漠干热环境:温度 40.5℃± 0.5℃,湿度 10%± 2%。按以上分组将实验动物分别置于已提前达到相应环境的实验舱内分别暴露 3 小时。诱导麻醉使用氯胺酮 20 mg/kg、阿托品 0.05 mg/kg 肌肉注射;使用 1.5%-3%的七氟烷混入 50%的氧气中维持麻醉,维持 BIS 值在 40-60 之间。

1.4 置管及监护

连接心电监护仪,BL-420 体温监测,连续监测生命体征及体温。分离暴露右侧颈外静脉置入漂浮导管以抽取混合静脉血液样本;右侧股动脉插管监测动脉血压,左侧股动脉用于放血及血液样本的收集。

1.5 模型建立

由于猪具有一个约为人类三倍大小的脾脏,储存较多血液,创伤失血性休克时可形成自体输血,因此两种环境的创伤失血性休克猪都将行全脾切除术[6]。沿腹中线开腹、膀胱造瘘。NTHS 组和 DTHS 组行左下叶 1/4 肝脏及全脾切除术,并输入三倍脾重的乳酸林格氏液[7,8]。休克模型采用固定血压的中度创伤失血性休克模型 [9],自髂外动脉快速放血致平均动脉压(MAP)45± 5 mmHg,稳定 20 min 后记录休克 0 时,稳定期间可通过再放血或者静脉输入乳酸林格氏液,稳定目标血压。NS 组和 DS 组按照上述方法完成动脉等监测后,行剖腹术及膀胱造瘘术后结束手术操作,不进行部分肝脏、脾脏切除和放血。创伤失血性休克组持续观察监测至动物死亡,NS 组和 DS 组在相应环境创伤 THS 组最长生存时间上延长约 30 min 后安乐处死。

1.6 标本收集、检测及氧代谢计算方法

分别于暴露后、休克成功后(计为休克 0 时)及休克后每 30 分钟自左侧股动脉抽取动脉血;血流动力学平均动脉压(MAP)、心输出量(CO)等从监护设施连接后即开始连续观察记录,NTHS 组和 DTHS 组直至动物自然死亡,NS 组至暴露后 11h,DS 组至暴露后 4 小时。动脉血液抽取后置于 4℃保温盒内 10 分钟内送至我院 ICU 血气分析仪 (GEM Premier 3000,USA)检测动静脉血气、乳酸。实验虽行全程对比研究,但本文为更为直观清晰的表达沙漠干热环境创伤失血性休克特点,制图时,仅选取与 DTHS 组对应时间点。

氧代谢指标及未成年猪的体表面积计算公式如下:DO_2=CO (L/min)·CaO_2 (mlO_2/100ml/blood)·10/ [body-weight] (kg);VO_2= (CaO_2-CvO_2)·CO (L/min)·10/ [body-weight] (kg);O_2ER=DO_2/VO_2= (CaO_2- CvO_2)/ CaO_2;Ca/O_2 (ml/dl)= Hb (g/dl)·1.34(ml/g)+ 0.003(ml/dl·mmHg-1)·PO_2(mmHg)[10];[body surface area](m^2)=0.073·[body-weight] 2/3[7]。

1.7 统计学处理

采用 SPSS 23.0 软件进行统计学处理,符合正态分布的计量资料以均数± 标准差($\bar{x}$± s)表示。采用 Sigmaplot12.5 软件作图。相同时间点两两比较,方差齐时采用单因素方差分析,并以 LSD 进行多重比较,方差不齐时采用 Kruskal Wallis 检验。P<0.05 为差异具有统计学意义。

2 结果

实验过程中,没有手术或麻醉等意外死亡动物。本研究沙漠干热环境创伤失血性休克组一般在 3.2-3.5 小时死亡,常温

环境创伤失血性休克组一般在 10.5-11.5 小时死亡，按实验方案，实验舱于实验动物进仓前达到相应环境设置要求，实验过程中环境稳定，无突然幅波动改变等现象。

2.1 基础数据

四组暴露后体重(BWAE)较暴露前体重(BWBE)均有所减少，但暴露前后差异无显著差异(P>0.05)；各组暴露前体温，切除肝脏与体重之比(RL/WT)、呼气末二氧化碳(CPE-TO2)及 BIS 值等基础值无显著差异(P>0.05)。

表 1 各组基础数据比较($\bar{x}\pm s$)

Table 1 Comparison of the basic date between different groups

	Groups			
	Normal temperature		Dry-heat temperature	
	THS group	NS group	THS group	NS group
Amount(n)	10	10	10	10
BWBE(kg)	29.61± 2.62	29.96± 2.34	29.67± 2.71	31.9± 2.5
BWAE(kg)	29.1± 2.69	29.38± 2.37	29.08± 2.55	31.2± 2.4
Temperature(℃)	39.03± 0.24	39.12± 0.29	38.99± 0.31	39.08± 0.2
$C_{PE\text{-}T}O_2$(mmHg)	39.7± 3.27	39.5± 3.06	40.4± 3.13	40.6± 3.06
RL/WT(g/kg)	49.53± 8.64	N/A	48.85± 6.99	N/A
BIS	48.9± 5.23	51.2± 4.69	39.7± 5.79	50.2± 4.37

Note: BWBE, body weight before exposure; BWAE, body weight after environmental exposure; CPE-TO2, end tidal carbon dioxide; RL/Wt, resection of liver / body weight; N/A, no hepatectomy.

2.2 动脉、混合静脉血氧饱和度和含量

本研究中，通气氧浓度为 50%，病程中 4 组动脉氧饱和度(SaO_2)基本处于 100%，各组间无显著差异(P>0.05)。而 DS 组混合静脉氧饱和度(SvO_2)始终显著低于 NS 组(P<0.05)，但两组均保持各自的相对稳定状态；DTHS 组和 NTHS 组 SvO_2 随休克进展呈下降趋势，但 DTHS 组下降更早更快，整个病程两组均呈显著差异(P<0.05)。实验期间，NS 组动脉氧含量(CaO_2)基本保持与暴露后处于同一水平；DS 组与 NS 组未呈现明显差异(P>0.05)，但由于干热环境血液浓缩，DS 组却呈逐渐上升趋势；0 h 后 NTHS 组始终低于 NS 组，除 2 h 时外，其余均呈现显著差异(P<0.05)，DTHS 组 0 h 后迅速代偿，虽与 NTHS 组未呈显著差异，但相同时间点始终高于 NTHS 组。观察期间，DS 组混合静脉氧含量(CvO_2)低于 NS 组，DTHS 组和 NTHS 组自 0h 后呈进行性下降趋势，但 DTHS 组下降更早更快。

表 2 各组动脉、静脉血直接氧代谢指标的比较($\bar{x}\pm s$)

Table 2 Comparison of the blood oxygen metabolism index of artery and vein between different groups

	AE	0 h	1 h	2 h	3 h	8 h	10 h
				Arterial oxygen saturation(SaO_2)			
N S	0.99± 0.0	1	1	1	1	1	1
NTHS	1± 0.01	1	1	1	1	1	1
D S	0.99± 0.01	0.99± 0.01	1	1	1		
DTHS	0.991± 0.01	1	1	0.99± 0.01	0.99± 0.01		
				Mixed venous oxygen saturation(SvO_2)			
N S	0.81± 0.04	0.8± 0.04	0.8± 0.04	0.79± 0.05	0.78± 0.04	0.68± 0.27	0.78± 0.03
NTHS	0.81± 0.03	0.64± 0.03[a]	0.64± 0.1[a]	0.57± 0.08[a]	0.54± 0.11[a]	0.37± 0.18[a]	0.19± 0.15[a]
D S	0.7± 0.03[d]	0.71± 0.05[cd]	0.71± 0.06[cd]	0.69± 0.04[cd]	0.69± 0.04[cd]		
DTHS	0.71± 0.03[d]	0.55± 0.02[b]	0.61± 0.04	0.47± 0.07[b]	0.21± 0.11[b]		
				Arterial oxygen content(CaO_2)			
N S	12.94± 0.61	12.75± 0.35	13.10± 0.55	12.94± 0.55	12.44± 0.37	12.29± 0.48	12.19± 0.48
NTHS	12.94± 0.51	10.92± 0.65[a]	11.47± 0.82[a]	11.47± 0.81	11.38± 0.51[a]	10.83± 1.30[a]	10.99± 1.65[a]
D S	12.26± 1.02	12.53± 0.91[c]	12.89± 1.01	13.29± 1.03	13.83± 0.93		
DTHS	12.82± 0.71	10.67± 0.60	12.06± 0.77	12.44± 0.96	12.19± 1.16		
				Mixed venous oxygen content(CvO_2)			
N S	10.44± 0.71	10.16± 0.71	10.54± 0.87	10.2± 0.82	9.72± 0.5	9.43± 0.35	9.45± 0.61
NTHS	10.5± 0.63	6.98± 0.59[a]	7.34± 1.46[a]	6.55± 1.22[a]	6.13± 1.57[a]	4.01± 2.24[a]	2.57± 2.63[a]
D S	8.6± 0.99[d]	8.91± 1.18[c]	9.18± 1.45[c]	9.23± 1.2[c]	9.6± 1.19[c]		
DTHS	9.05± 0.88	5.83± 0.48[b]	7.35± 0.69	5.79± 0.97	2.82± 1.13[b]		

Note: AE, after exposure; NS group, Normal temperature sham group; NTHS group, Normal temperature traumatic hemorrhagic shock group; DS group, Dry-heat sham group; DTHS group, Dry-heat traumatic hemorrhagic shock group. [a]P<0.05 NTHS compared with NS group; [b]P<0.05 DTHS compare with NTHS group; [c]P<0.05 DS compare with DTHS group. dP<0.05 DS group compare with NS group.

2.3 氧输送和氧耗

沙漠干热环境暴露后，氧供(DO_2)、氧耗(VO_2)均显著高于常温组(P<0.05)。随血液的丢失，DO_2 均快速下降后逐渐代偿至某一稳定水平，但 DTHS 组短暂稳定后呈快速进行性下降至动物死亡，其再次下降之前高于 NTHS 组。DTHS 组 VO_2 始终高于 DTHS 组，低于 DS 组。组间相同时间点 DO_2、VO_2 的差异见图1。

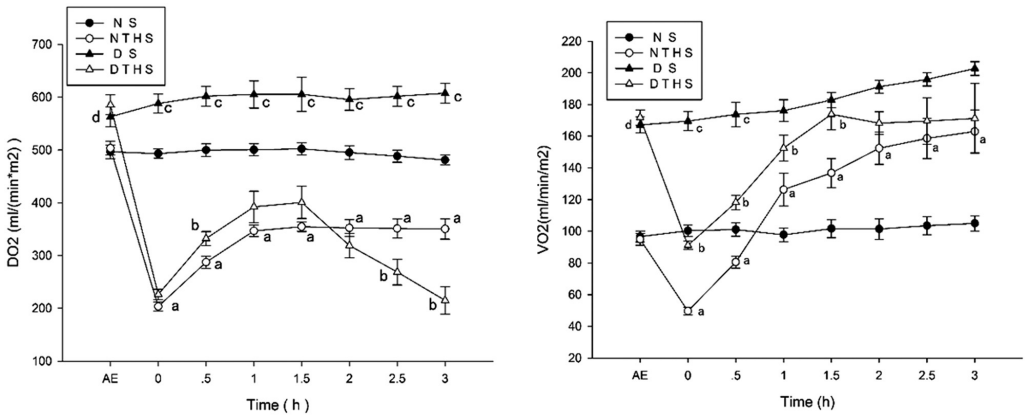

图 1 氧输送及氧耗变化趋势图

Fig.1 Trends of oxygen delivery and oxygen consumption

Note: DO_2, oxygen delivery; VO_2, oxygen consumption; AE, after exposure; NS group, Normal temperature sham group; NTHS group, Normal temperature traumatic hemorrhagic shock group; DS group, Dry-heat sham group; DTHS group, Dry-heat traumatic hemorrhagic shock group. ^{a}P<0.05 NTHS compared with NS group; ^{b}P<0.05 DTHS compare with NTHS group; ^{c}P<0.05 DS compare with DTHS group. ^{d}P<0.05 DS group compare with NS group.

2.4 氧摄取率和动脉血乳酸

环境暴露后，干热环境氧摄取率(VO_2/DO_2)显著高于常温环境(P<0.05)，DTHS 组氧摄取率在 0 h 时突然升高，整个病程呈进行性上升，且始终高于其他 3 组。动脉血乳酸暴露前后无显著差异，休克后，两休克组血乳酸均呈上升趋势，但 DTHS 组至 1.5 h 后即呈快速进行性升高，较 NTHS 组升高早且快。组间相同时间点 VO_2/DO_2、Lac 的差异见图 2。

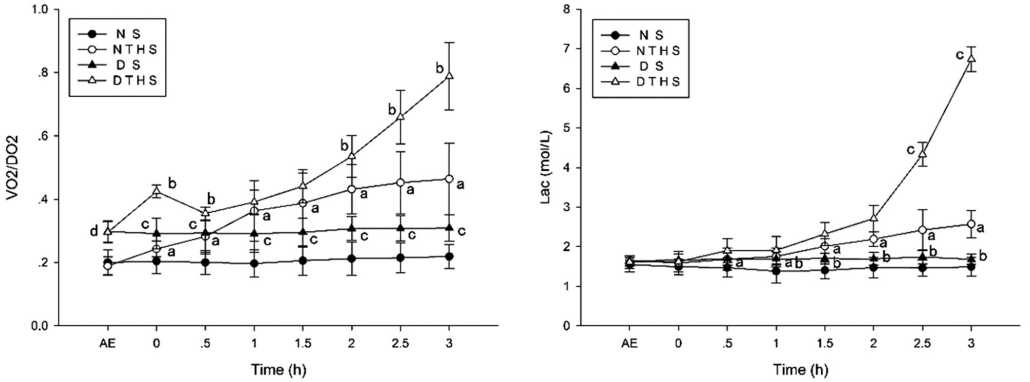

图 2 各组氧摄取率及血乳酸变化趋势图

Fig.2 Trends of oxygen uptake rate and blood lactic acid in different groups

Note: DO_2, oxygen delivery; VO_2, oxygen consumption; AE, after exposure; NS group, Normal temperature sham group; NTHS group, Normal temperature traumatic hemorrhagic shock group; DS group, Dry-heat sham group; DTHS group, Dry-heat traumatic hemorrhagic shock group. ^{a}P<0.05 NTHS compared with NS group; ^{b}P<0.05 DTHS compare with NTHS group; ^{c}P<0.05 DS compare with DTHS group. ^{d}P<0.05 DS group compare with NS group.

3 讨论

生理状况下，机体正常的生理活动有赖于组织氧代谢的供需动态平衡，即氧输送量≥ 氧耗量[5]。当机体由于某种原因或疾病导致氧供减少和(或)耗氧量增加，都将打破机体氧供需的动态平衡，导致组织细胞发生缺血缺氧。根据缺氧的时间及严重程度，可导致组织、器官损伤，直至衰竭致机体死亡[11]。创伤失血性休克的实质是组织灌注不足引起缺血、缺氧，导致组织器官损伤，甚至多器官功能衰竭[12]，但目前国内外尚未见沙漠干热环境创伤失血性休克的氧代谢特点报道。因此，本研究探讨了此环境的氧代谢特点。

氧输送是决定氧供给量的关键因素，主要依赖动脉血流量及动脉氧含量(CaO_2)[5]。热应激导致心率、心输出量显著增加[13]，从而显著增加动脉血流量。本研究结果显示暴露后的 NS 组和 DS 组 CaO_2 未见显著差异，因此干热环境假手术组(DS)和干热环境创伤失血性休克组(DTHS)环境暴露后氧供高于常温环境假手术组(NS)和常温环境创伤失血性休克组(NTHS)。休克模型建立后，全身总血流量迅速减少，导致 NTHS 组和 DTHS 组 DO_2 快速下降，继而导致组织缺氧、灌注不足。组织灌注不足、缺氧的第一个代偿反应是增加心输出量和增加氧摄取率 [5]，因

此在 DO_2 下降后即出现代偿性升高;热应激与失血性休克两种应激因素的协同作用，使 DTHS 组 DO_2 代偿及维持期间均高于 NTHS 组，但在 1.5-2 h 间开始 DTHS 组 DO_2 低于 NTHS 组,并呈进行性下降趋势。由此可见,干热环境创伤失血性休克在代偿期维持较高的氧输送,短暂代偿后的进行性下降是导致机体有限的氧供快速耗尽的原因,也是沙漠干热环境创伤失血性休克代偿期短、病程进展快的重要原因之一。

氧耗(VO_2)反映机体的总代谢情况,代表实时组织吸收的氧量。VO_2 和 DO_2 有着类似的病理过程[14]。与 DO_2 相似,干热环境暴露后,DS 组 VO_2 显著高于 NS 组,但 VO_2 增加幅度较 DO_2 增加幅度大,约 2 倍左右,提示干热环境暴露后,机体的耗氧量大幅提高。Haouzi 等指出失血性休克后机体氧耗量高于休克前机体氧耗量[15,16],而本研究结果显示 DTHS 组 VO_2 代偿虽然始终高于 NTHS 组,但未能超过其休克前的氧耗量,提示 DTHS 组始终保持较高的氧耗量，但其代偿能力可能较 NTHS 组低。因此,我们推测沙漠干热环境创伤失血性休克始终保持较高的组织耗氧量和较低的代偿能力。

氧摄取率(VO_2/DO_2,O_2ER)是反映组织内呼吸的指标[17]。机体代谢增强可通过提高氧摄取率来满足机体对氧的需求,增加组织呼吸,减少混合静脉氧含量(CvO_2)。本研究结果显示干热环境暴露后,DS 组 CvO_2 显著低于 NS 组，而 O_2ER 显著高于 NS 组，提示干热暴露后机体组织氧摄取增加。与 NTHS 组相比,DTHS 组 CvO_2 较早较快的呈进行性降低,而 O_2ER 较早较快的呈进行性升高,提示在干热环境和创伤失血性休克的双重打击下,机体内呼吸增强增快。正常机体都有一定的氧储存能力,保证机体在遭受不利因素刺激时,使氧供给尽可能的满足突如其来的耗氧量增加[18]。热暴露期间,机体可能已经耗尽体内生理氧储存,突然的创伤失血性休克加剧机体氧耗量,因此这可能是 DTHS 组在 0 h 氧摄取率突然升高的主要原因。

正常机体循环乳酸(Lac)浓度保持产生和消耗的动态平衡,由于应激或缺血会导致乳酸产生增加,或由于乳酸作为底物的能量代谢障碍,也将导致乳酸浓度增加,乳酸浓度也被认为是机体氧债大小重要参考依据[19,20],动脉乳酸浓度通常被认为是评估机体休克严重程度的敏感指标[5,20,21]。本研究结果显示动脉乳酸浓度在 DTHS 组自 0.5 h 开始较 NTHS 组显著增高，但二者均在 1.5 h 即呈明显升高趋势，且后续呈快速增长趋势,而 NTHS 组虽有升高,但较 DTHS 升高幅度和速度较小且慢。因此,我们推测沙漠干热环境创伤失血性休克的氧负担及酸中毒发生时间早,变化速度快,机体损伤程度重。

Kopterides 等发现由于组织产生乳酸与循环中乳酸出现有一定的时间差,血乳酸比组织乳酸出现的时间较晚、浓度相对较低[21-23]。本研究结果显示 DO_2、VO_2、O_2ER 在环境暴露后即呈现出显著差异,提示机体在干热环境暴露后机体已呈现氧的高代谢,而环境暴露前后动脉血乳酸无显著变化,乳酸盐的明显变化出现在休克后 1.5 h, DO_2、VO_2 等直接氧代谢指标变化与血乳酸变化呈现不一致变化的可能机制:(1)此过程增大的氧需求量由机体的本身氧储存补给[13],尚无缺氧发生,无乳酸生成;(2)机体虽有氧供不能满足氧需,已有无氧代谢至乳酸增多,但此阶段主要集中于组织内,尚未循环入血,因此在环境暴露与休克初期并无血乳酸增高的现象。因此,我们推测乳酸盐变化不是反映沙漠干热环境创伤失血性休克的早期敏感指标,而是反映其严重程度的有用指标。

综上所述,本研究结果表明:(1)沙漠干热环境创伤失血性休克加剧机体较高氧代谢，致使机体较快耗尽有限氧含量,是导致代偿能力降低、病程变化快的重要原因之一;(2)干热环境 VO_2、O_2ER 等直接氧代谢指标于暴露后即与常温环境呈现显著差异,因此其可作为沙漠干热环境创伤失血性休克早期评估监测机体氧代谢的敏感指标;(3)与直接氧代谢指标相比,血乳酸升高变化出现较晚,因此血 Lac 可作为沙漠干热环境创伤失血性休克评估监测机体休克严重程度的有用指标。

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