RESEARCH ARTICLE

FORENSIC MEANING OF CORE TEMPERATURE - AN INDICATOR FOR ASSESSING THE SEVERITY OF HEATSTROKE IN AN ANIMAL MODEL

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ABSTRACT

Sudden deaths during efforts that are multifactorial and associated with exposure of the body to high temperatures beyond the power of thermoregulatory mechanisms are increasingly common. Autopsies are often performed, but the evidence is insufficient and non-specific. The research is aimed to determine the core temperature values of rats exposed to different water temperatures (37°C, 41°C, 44°C), before the start of the experiment (Tb), after immersion in water (Tu), after 20 minutes of exposure and at death (Ts) in rats for hyperthermia and heat stroke.

Forty rats were divided into five groups depending on the temperature and length of exposure to water: control group- CG37, G41-hyperthermia- group which exposure time was 20 minutes at 41°C, G41-heat stroke- group exposed until death at 41°C, G44- hyperthermia- group which exposure time was 20 minutes at 44°C, G44- heat stroke- group exposed until death at 44°C. A RET-4 probe was used to measure the core temperature of rats.

Significant changes in the body temperature of rats were observed during the lethal outcome, p<0.0005. After exposure to water temperature for a period of 20 minutes, depending on the group, it was observed that the body temperatures of rats differed significantly between G37 and G41, CG37 and G44, p<0.0005 and G41 and G44, p<0.0005. A significant difference was also observed in the *post mortem* temperature of groups G41 and G44, p=0.01, a significant difference between body temperatures in groups CG37, G41-hyperthermia, G41- heat stroke, G44-hyperthermia and G44-heat stroke (p<0.0005), and the significance of the differences in the CG37 group was p=0.044.

Exposure of albino rats to different water temperatures also led to the changes in the internal temperature; normothermia was established through thermoregulation in the control group, and in the other groups, hyperthermia and heat stress occurred.

Keywords: Heat stroke, rats, temperature, variation

INTRODUCTION

A large number of deaths both in Europe and in the United States of America have been attributed to exposure to high temperatures (Haines et al., 2006; Luterbacher et al., 2004). Central Europe experienced its hottest summer in 2003 since 1500, with average temperatures around 3.5°C above the upper air temperature limits. The number of deaths was between 22,000 and 45,000 in a period of two weeks, according to data published by the Red Cross Organization (IFRC, 2004). Common causes of death were dehydration and heat stroke in hospitalized patients in France (Ferron et al., 2006). A 7% increase in mortality recorded during the same period in Switzerland was associated with heat stroke (Grize et al., 2003). The core temperature of internal organs is regulated at approximately 36.6°C (Obermeyer et al., 2016), but due to exposure to high temperatures, it can vary significantly, from the lowest recorded temperature of 13.7°C experienced by human survivors (Gilbert et al., 2000) to a maximum of 41.5°C without any complications for the bodies (Racinais et al., 2019). Core body temperature in humans is the main regulated variable in thermoregulation (Kramer et al, 2022). Body core temperature is the result of local thermal balance depending on the place of measurement (Taylor et al., 2014). It has been proven that the influence of high air temperature triggered certain processes in the organism and that compensatory mechanisms failed. At autopsy, in cases of sudden death after exertion, atherosclerosis of the coronary vessels was found in more than 60%, left ventricular hypertrophy of unknown etiology in 7.8%, and valvular heart disease in 7.1% (IFRC, 2004; Ferron et al., 2006; Grize et al., 2003). Sudden death during exertion can depend on the predisposing heart disease. In comparison with the control group, a statistically significant association was observed between the subjects' heat exhaustion and changes in the heart tissue. Establishing absolute evidence in support of the diagnosis of death caused by heat stroke is a major problem in forensic pathology. Primarily, it is not routine to always measure the temperature of the deceased. In addition, macroscopic findings are not site-specific, especially if the survival time was short. The diagnosis of hyperthermia is based on investigative actions, circumstances, and reasonable exclusion of other causes of death.

The research aimed to determine the core temperature values of rats exposed to different water temperatures before the start of the experiment (Tb), after water immersion (Tu), after 20 minutes of exposure (Tu) and at death(Ts) rats for hyperthermia and heat stroke.

MATERIAL AND METHODS

The study was processed as a prospective experimental, randomized study based on the albino Wistar rat model of hyperthermia. The research was conducted at the Faculty of Medicine, University of Sarajevo, by the Principles for the Care of Laboratory Animals (De Labra, 2021). After the approval of the Ethics Committee of the Faculty of Medicine of the University of Sarajevo (registration number 02-3-4-1253/20, Bosnia and Herzegovina), the total number of adult Wistar rats with body weight from 200 to 300 g were included in the experiment. Target temperature points in the water bath were determined, and one rat at a time was exposed to a total of 40 rats included in the experiment. Rats were divided into five groups depending on the temperature and length of exposure to water: control group exposed to a water temperature of 37°C (CG), groups exposed ta o water temperature of 41°C and 44°C for 20 minutes (G41-hyperthermia; G44-hyperthermia), groups exposed to a water temperature of 41°C and 44°C with exposure time until death (G41-heat stroke; G44-heat stroke). Heat stroke is defined as an increase in core temperature above 40.5 °C (Benzinger, 1969; Racinais et al., 2019).

The rats were anesthetized with ketamine in a dose of 1.2 mLtog tw + /- 10% (USP Rotexmedica-Germany) (Režić-Mužinić et al., 2018). The experimental protocol was made according to the previously described methodology of induction of hyperthermia and heat shock (Dervišević et al., 2022). A probe (RET-4 probe for mice and rats) was used to measure the core temperature

of rats, and the core temperature was read on a thermometer (Physitemp Instruments Clifton, Physitemp Thermalert Model TH-8, USA).

RESULTS

The basal temperature was the lowest in group G44 at 38.02±0.55°C, and the highest in G37 at 38.25±0.12°C. The immersion temperature ranged from 37.84°C in G41 to 38.67±0.90°C in G44. In group CG37, the lowest temperature of 37.75°C was observed after 20 minutes of exposure, and the highest in G44, 43.09±0.79°C. The death temperature was lower in G41°C 38.34±3.59°C compared to 41.51±2.73°C in G44 (Table 1).

Table 1 Mean values of measured body temperature of rats of experimental groups at time points

T (°C)	Group	x̄	±SD	95% IP DG	GG
T-b (°C)	CG37	38.25	0.12	38.13	38.37
	G41	37.80	0.48	37.53	38.07
	G44	38.02	0.55	37.72	38.32
T-u (°C)	CG37	38.17	0.23	37.95	38.39
	G41	37.84	0.50	37.56	38.12
	G44	38.67	0.90	38.19	39.15
T-20 (°C)	CG37	37.75	0.38	37.40	38.11
	G41	40.04	0.43	39.79	40.28
	G44	43.09	0.70	42.71	43.46
T-s(°C)	G41	38.34	3.59	36.35	40.33
	G44	41.51	2.73	40.05	42.97

± X̄- mean value; ±SD-standard deviation; IP-Confidence Interval; DG-Lower Bound; GG- upper limit; T-b- basal temperature of rats; T-u immersion temperature of rats; T-temperature in the 20th minute; T-s- temperature of death; p-probability CG37- control group of rats exposed to water temperature of 37˚C; G41-group exposed to water temperature of 41˚C; G44-group of rats exposed to a water temperature of 44˚C

By analyzing the mean values of body temperature measured at four-time time points, showed that the basal body temperature of rats of group CG37 was not significantly different from the basal temperature of rats in G41 and G44, p=0.125, so multiple comparisons between groups were not performed. The temperature after water immersion depending on the group was significantly different between groups G41 and G44, p=0.005. After exposure to water temperature for a period of 20 minutes, depending on the group, it was observed that the body temperatures of rats differed significantly between G37 and G41, CG37 and G44, p<0.0005 and G41 and G44, p<0.0005. A significant difference was also observed in *post mortem* temperature of groups G41 and G44, p=0.01 (Table 2).

Table 2 The difference in body temperature values of rats of experimental groups at three-time time points

T (°C)		Group	Group	p	95% IP	
					DG	GG
T-u (°C)		G44	G41	**0.005**	-1.42	-0.22
T-20 (°C) T-s (°C)	CG37		G41	**<0.0005**	-2.91	-1.65
			G44	**<0.0005**	-5.95	-4.71
	G44		G41	**<0.0005**	2.56	3.54
	G41		G44	**0.01**	-5.86	-0.46

T (°C) - Temperature in degrees Celsius; p-probability; IP-Confidence Interval; DG-Lower Bound; GG- upper limit; T-u immersion temperature of rats; T-temperature in the 20th minute; T-s- temperature of death; p-probability; CG37 (-control group of rats exposed to water temperature of 37˚C; G41-group exposed to water temperature of 41˚C; G44-group of rats exposed to a water temperature of 44˚C)

Repeated measurements of body temperature at three time points (T-b, T-u, T-20) by group showed a significant difference between body temperatures in all three groups. In the control group (CG37), a significant difference was observed in three measurements (p=0.044), and in groups G41 and G44, a significant difference in the body temperature of the rats was observed in three measurements (p<0.0005) with a drop in the temperature of CG37 group in 20 minutes and an increase in temperature in groups G41 and G44 (Table 3).

Table 3 Multicomparison of body temperature of rats of groups at three-time time points

a)

T (°C)	Group	Group	p	95% IP	
				DG	GG
T-u (°C)	G41-AM	G41-PM	**0.04**	-2.07	-0.03
	G41-PM	G44-AM	**0.03**	-2.03	-0.6
T-20 (°C)	CG37	G41-AM	**<0.0005**	-3.00	-1.25
		G41-PM	**<0.0005**	-3.2	-1.57
		G44-AM	**<0.0005**	-6.08	-4.39
		G44-PM	**<0.0005**	-6.27	-4.58
	G41-AM	G44-AM	**<0.0005**	-3.95	-2.26
		G44-PM	**<0.0005**	-4.14	-2.45
	G41-PM	G44-AM	**<0.0005**	-3.64	-2.00
		G44-PM	**<0.0005**	-3.82	-2.19

b)

T (°C)	Group	Group	p	95% IP	
				DG	GG
		G41-PM	**<0.0005**	-9.19	-4.29
	CG37	G44-AM	**<0.0005**	6.99	-409
		G44-PM	**<0.0005**	-12.01	-7.11
	G41-AM	G41-PM	**<0.0005**	-8.56	-3.66
T-s		G44-AM	**0.001**	-6.36	-1.46
		G44-PM	**<0.0005**	-11.38	-6.48
	G41-PM	G44-PM	**0.013**	-5.19	-4.58
	G44-AM	G44-PM	**<0.0005**	-7.39	-2.65
	G44-PM	G44-AM	**<0.0005**	2.65	7.39

T (°C) - Temperature in degrees Celsius; p-probability; IP-Confidence Interval; DG-Lower Bound; GG- upper limit; T-b-basal temperature; T-u- rat immersion temperature; T-s- temperature of death; CG37-control group of rats exposed to a water temperature of 37˚C; G41-AM (G41-hyperthermia)- *ante mortem* group exposed to water temperature 41˚C (length of exposure 20 minutes); G41-PM (G41-heat stroke) – *post mortem* group exposed to water temperature 41˚C (length of the exposure until death); G44-AM (G44-hyperthermia)-*ante mortem* group of rats exposed to a water temperature of 44˚C (length of exposure 20 minutes); G44-PM (G41-heat stroke) –*post mortem* group of rats exposed to a water temperature of 44˚C (length of the exposure until death)

DISCUSSION AND CONCLUSION

Large temperature fluctuations and climate changes affect the global temperature, with the period between 2011 and 2020 being the warmest in the last 140 years (Asheville, 2021; Global Climate Report-Annual, 2020; Périard et al., 2021; WMO, 2020). Since 2015, the warmest years are 2016, 2019, and 2020. The mean global temperature was 1.2°C above the level in 2020, WMO 2020 and between 2030 and 2052, it is predicted to rise by another 1.5 °C (Masson-Delmotte et al., 2018). These temperature changes in the environment directly affect the internal temperature of the organism because the basic temperatures increase and the loss of water in the body also increases the reserve consumption of the compensatory mechanisms of the organism (Masson-Delmotte et al., 2018).

Heat stroke leads to oxidative cellular stress and impaired thermo-compensatory mechanisms, and can range from heat exhaustion to death. An organism engages compensatory mechanisms of heat- release through sweating, tachycardia, and ultimately the activation of pro-inflammatory and inflammatory cytokines. Heat stroke is defined as a state of elevated body temperature of 40.05°C and above this value, which is a consequence of overemphasized compensatory mechanisms and exhausted body reserves with interindividual variations (Nzvere et al., 2020; Suzuki and Hori, 2014).

Heat stroke, a condition occuring in the range of 10 to 50%, leads to death through cardiac dysfunction with global hypokinesia (Rajan et al., 2017). Given that the number of deaths caused by heat stroke is increasing, it is necessary to understand the behavioral response of the organism to increased environmental temperature, which is directly related to global warmings (Barney and Kuhrt, 2016). In the world, the number of deaths that occur in closed spaces with an increase in air temperature inside the room, for example during bathing, in saunas, Turkish baths, and among athletes due to strenuous training, especially in the summer months, is increasing. Two significant causes of sudden death in athletes are death due to arrhythmia and heat stroke. Death caused by arrhythmia is mate in the center of medical attention. while heat stroke is less considered

(Yankelson et al., 2014). Autopsy rates remain low and vary between countries, and protocols for performing autopsies in cases of suspected sudden death also differ (Illing et al., 2019).

Determining heat stroke as the cause of death is very difficult in forensic medicine due to the non-specificity of biochemical analyses. In our rat model of thermal climate, a thermoregulatory response was included by exposing rats to different water temperatures (37, 41, and 44°C). An esophageal probe was used to measure core temperature. The proven physiological body temperature of rats is 37.5-37.7°C (Bathini et al., 2020), and the research aim was to determine the core values of rats exposed to different water temperatures (37, 41, and 44°C) before the start of the experiment (Tb), after immersion (Tu), after 20 minutes of exposure (Tu) and at the moment of death (Ts) in rats with hyperthermia and heat stroke. Basal temperature was the lowest in group G44 and the highest in the control group, which is in correlation with inter-individual variations. The death temperature was lower in G41 compared to G44, which considers the physiological state and the absence of pathological processes in the rats before the experiment started. Given that the analysis of basal temperatures in the grower was not significant, this points to the conclusion about the organism's physiological state and normothermia in rats before the start of the experiment. A significant difference appears during water immersion, after 20 minutes, and at the moment of death, which points to the control mechanisms of the thermoregulation center. Heat stroke was detected in rats exposed to the highest temperature of 44 degrees.

Our results are in accordance with the literature data that indicate that the minimum time required for the onset of heat stroke with the compensatory mechanisms of the organism involved is 20 minutes from the moment of the target temperature (Suzuki and Hori, 2014). Basal temperature is the result of the organism's physiological thermoregulation itself, while increased ambient temperature and the length of exposure of the body affect the increase in internal temperature and engage the compensatory mechanisms of the organism with the exchange of heat between the body and the environment. The temperature of rats at the time of death was 44.02±0.38°C in group G44-heat stroke, and 41.20±0.76°C in G41. The rats exposed to water temperatures of 41°C and 44°C had longer survival in group G41 of 175.50 minutes, compared to rats in group G44 of 4.14 minutes (p=0.001). Therefore, the period of exposure to high temperature caused more rats of group G44 not reaching the temperature threshold for heat stroke. In a study by Quinn et al. (2014), a predetermined temperature is used as the temperature standard of heat stroke, which was also done in our research, and is considered an inter-individual variation (Danzl, 2002; Hori, 2014; Sweeney, 2002; Yoder, 2004). In a study of healthy individuals, the internal temperature reached values of 40°C after 10 minutes of exposure. In addition, deterioration of brain and heart function was observed after immersion in water at a temperature of 44°C, but no brain and heart damage was observed after immersion in water heated to 40°C. The degree of damage depends on the temperature of the environment and the length of exposure of the body to that environment (Hori, 2014). Heat stress induces systemic inflammation through increased cytokine and white blood cell concentrations, which contributes to hyperthermia and reduced metabolic activities, as metabolic reservoirs are depleted to deploy homeostatic mechanisms. Excessive inflammation in heat stress induces the release of damage-associated molecular ligands from apoptotic cells in the final stage of pyroptosis (Geng et al., 2015).

The modern epoch of climate change also caused research on the topic of thermoregulation of the organs with adaptation to high temperatures. Thermoregulation of heat is described by the central integrator model of peripheral and central heat inputs that activate thermoeffector reactions when the core temperature moves above physiological. However, further research should consider more complex models that include several integrators and other afferent signals taking into account the stages of the heat flow mechanism and the inter,individual variations of the response that

cannot be excluded.

Exposure of albino rats to different water temperatures also led to a change in the internal core temperature. In the control group, normothermia was established through thermoregulation, and in the other groups, hyperthermia and heat stress occurred.

Taking into account the hypotheses of our research, we confirmed the working hypothesis. In addition, there are certain limitations to the study. It is necessary to work on the development and expansion of existing protocols for the diagnosis of heat stroke, given that the climate changes are becoming more frequent, compensatory mechanisms are unpredictable, and that heat stroke is an increasingly common cause of death at autopsy. It would be necessary to validate the results on human material and create registers of causes of death.

CONFLICT OF INTEREST

None declared.

CONTRIBUTION

Concept – ED, MD, AB; Desing – LD, AS, EM; Supervision – ED, MD, AB; Materials – ED, ZA, AB; Data Collection and/or Processing – AS, LD; Analysis and/or Interpretation – ED, LD, ZA; Literature Search – AB, ZA, ED, MD; Writting Manuscript – ED; Critic Review – EM, ZA, AS.

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FORENZIČKO ZNAČENJE UNUTRAŠNJE TEMPERATURE – INDIKATOR PROCJENE TEŽINE TOPLOTNOG UDARA NA ŽIVOTINJSKOM MODELU

SAŽETAK

Iznenadne smrti tokom napora, koje su multifaktorijalne i povezane s izlaganjem tijela visokim temperaturama, a prevazilaze snagu termoregulatornih mehanizama, su sve učestalije. Obdukcije se često izvode, ali su dokazi nedostatni i nespecifični. Cilj istraživanja je određivanje vrijednosti unutrašnje temperature pacova izloženih različitim temperaturama vode (37°C, 41°C, 44°C), prije početka eksperimenta (Tb), nakon potapanja u vodu (Tu), nakon 20 minuta izlaganja i u trenutku ugibanja (Ts) kod hipertermije i toplotnog udara.

Četrdeset stahora je podijeljeno u pet grupa prema temperaturi i dužini izlaganja vodi: kontrolna grupa-CG37, G41-hipertermija - grupa čije je izlaganje iznosilo 20 minuta na 41°C, G41 - toplotni udar – grupa izložena do ugibanja na 41°C, G44 - hipertermija- grupa čija je dužina izlaganja iznosila 20 minuta na 44°C, G44 - toplotni udar - grupa izložena do ugibanja na 44°C. Za mjerenje osnovne temperature pacova je korištena RET-4 proba.

Kod smrtnih ishoda su zabilježene signifikantne promjene u tjelesnoj temperaturi stahora, $p < 0.0005$. Nakon izlaganja temperaturama vode u trajanju od 20 minuta, u ovisnosti od grupe, uočeno je da su se tjelesne temperture stahora signifikantno razlikovale između G37 i G41, CG37 i G44, $p < 0.0005$ i G41 i G44, $p < 0.0005$. Također je uočena signifikantnost razlika u *post mortem* temperaturama između grupa G41 i G44, $p = 0.01$, kao i signifikantnost razlika u tjelesnim temperaturama između grupa CG37, G41 - hipertermija, G41 - toplotni udar, G44 - hipertermija i G44 - toplotni udar ($p < 0.0005$), dok je signifikantnost razlika u CG37 grupi iznosila $p = 0.044$.

Izlaganje albino stahora različitim temperaturama vode je, također, dovelo do promjene unutarnje temperature; normotermija je u kontrolnoj grupi uspostavljena termoregulacijom, dok je u ostalim grupama došlo do pojave hipertermije i toplotnog stresa.

Ključne riječi: Stahori, toplotni udar, temperatura, varijacija