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PATHOGENESIS OF SUDDEN DEATH FOLLOWING WATER IMMERSION (IMMERSION SYNDROME)

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Translation of "Sympatho-adrenal Aktivität bei akutem Kaltestress. zum Mechanismus plötzlicher Todesfälle im Wasser", Zeitschrift für Rechtsmedezin, Vol. 83, 1979. pp 121-127.

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16. Abstract Predominantly vagal cardio-depressive reflexes discussed besides currently known mechanisms of sudden death after water immersion. Pronounced circulatory centralization in diving animals as well as following exposure in cold water indicates additional sympathetic activity. In cold water baths of 15°C authors' measurements indicate increase in plasma catecholamine levels by more than 300%. This may lead to cardiac arrhythmias by the following mechanism: Cold water essentially induces sinus bradycardia. Brady- and tachycardiarrhythmias may supervene as secondary complications. Sinusbradycardia may be enhanced by sympathetic hypertonus. Furthermore, ectopic dysrhythmias are liable to be induced by the strictly sympathetic innervation of the ventricle. Myocardial ischemia following a rise in peripheral blood pressure constitutes another arrhythmogenic factor. Some of these reactions are enhanced by alcohol intoxication.

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PATHOGENESIS OF SUDDEN DEATH FOLLOWING WATER IMMERSION (IMMERSION SYNDROME)

M. Bühring and H. F. Spies[1]

Primary submersion [37], water stroke [19], the "immersion syndrome" [24] or hydrocution [23] present a special type of death by drowning. In typical cases the swimmer, after being in the water for only a few moments, suddenly disappears from the view of the astonished observer and drowns. In some cases drowning is preceded by a brief struggle. /122*

This sort of death is relatively frequent [1, 17, 20, 21, 25, 33, 37]; for the assessor it is often hard to decide, whether the case is one that entitles to compensation in the sense of general accident insurance conditions [1, 10, 12, 20, 21, 25, 31, 33, 38, 43].

It happens repeatedly, that for many such subjects death intervenes when they are under the influence of alcohol, often only to a slight degree [13, 16, 18, 20, 24, 30, 32, 33, 36], so that the amount of intoxication is not an adequate explanation for an crdinary drowning through impairment of coordination.

It is usually assumed, that in such cases death involves a reflex event. In addition to anaphylactic cold reaction, immersion, Goltz and vestibular reflexes, the discussion centers especially around vagomediated cardiodepressive reactions. These are assumed to be released by sensitive structures in the laryngeal area following aspiration of water and of abnormally created anxiety with the possibility of psychogenic unconsciousness [38]; in the end anxiety

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*Numbers in the margin indicate pagination in the foreign text.

and bodily exertion are assumed to result in stress respiration, which in syncotropic types [8] induces abnormal decrease in BP or impaired rhythm.

In most clarification experiments there is a unilateral assessment only of the part played by vagus reaction processes. They arose as the result of the well known cold bradycardia and in part of impressive immersion bradycardia found in vertebrates that live in water and breathe with their lungs [3, 4, 11, 26] and in humans [28, 34].

However, with bradycardia there was also the development of a significant centralization of circulation. In this context air oriented animals show a minimal lifespan for attaining longer and longer submersion periods. In humans this centralization may be the reason for a surprisingly longer survival of children who have been submerged [2, 22, 40]. Recently this phenomenon occasioned a large number of revival experiments with people who had remained below water for as long as 38 (!) minutes [27].

In addition to the familiar transformations in the direction of vagotonia, these observations already indicate simultaneous sympathetic activation of autonomic regulation. Thus, at least for humans, water temperature appears to be significant: the observations referred to above were made exclusively in a cold water context. Nemiroff [27] gives 21°C as the critical temperature for an adult.

We wondered, whether sympathetic activity could be demonstrated under cold stress. This would produce new points of view for the genesis of primary submersion. We thought it would be a good idea to determine the plasma catecholamine level[2].

Test Procedure

The test subjects were 20 healthy males aged 22-37 ($\bar{x}$ = 27.3). Following an hour's preliminary rest on a lift platform they were

lowered with it into 15°C water, which a circulation system kept in constant movement. Following 10 min of this, the water temperature was uniformly raised 1.5°/min until it was one degree below actual rectal temperature (Fig. 1).

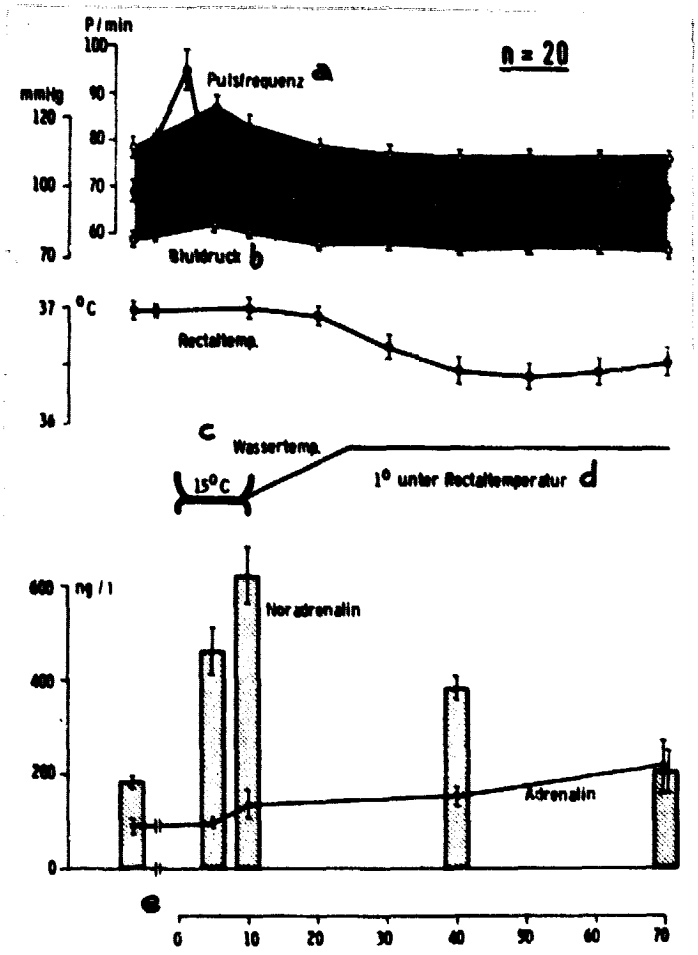

Fig. 1. Circulation values, body temperature and plasma catecholamine level during 10 min immersion in 15°C water. Preliminary rest and subsequent warming of water to within 1°C of actual rectal temperature. Range of mean error shown by brackets.

Key: a. pulse rate
b. blood pressure
c. water temperature
d. under rectal temperature
e. prerest in minutes

crease in the pulse rate.

At the start of prerest insertion of a venous catheter, blood sampling shortly before start of immersion and at minutes 5, 10, 40, 70 of immersion. Adrenaline and norepinephrine measured by Passon and Peuler method [29]. Continuous recording of pulse rate and rectal temperature, auscultatory BP measurement.

Results (see Fig. 1)

After 10 min immersion the noradrenaline levels are 340% of the initial values. Only with central cooling of the organism do the adrenalin levels rise considerably (up to 250% in immersion minute 70).

During the cold water phase the rectal temperature drops considerably and the elevated catecholamine levels are not expressed by the moderate and short term in-

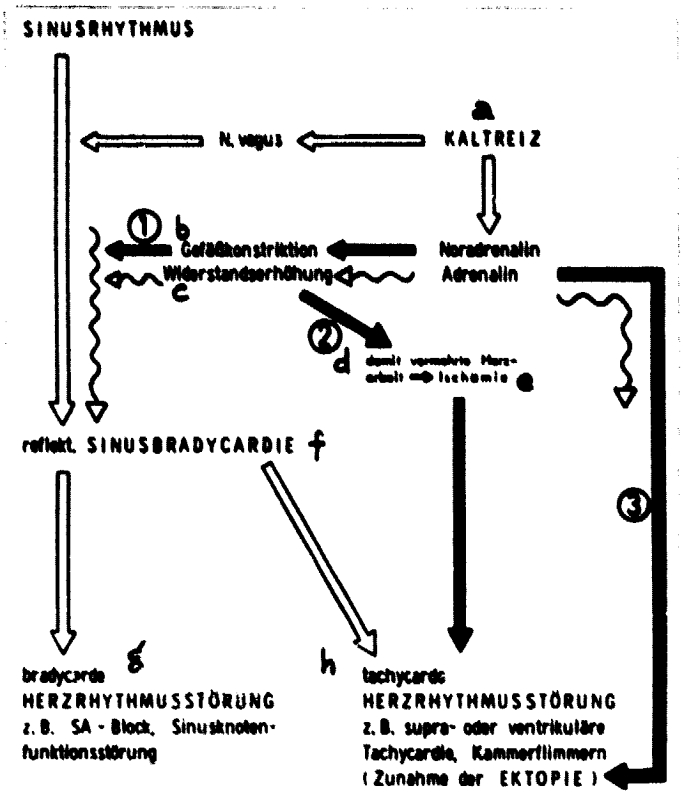

Fig. 2. Vagal (white arrow) and sympathetic (black arrow) reactions in cold stress may induce bradycardiac and tachycardiac rhythm disturbances in several ways. Some reaction processes are intensified by alcohol (wavy arrow). Details in the text.

Key: a. cold stimulation
b. vascular constriction
c. heightened resistance
d. attendant increased cardiac effort leading to
e. ischemia
f. reflex sinus bradycardia
g. bradycardiac disturbance of heart rhythm, e.g. SA block, disturbances of sinus node functioning
h. tachycardiac disturbance of heart rhythm, e.g. supra or ventricular tachycardia, ventricular fibrillation (increase of ectopy)

Discussion

Using balanced catecholamine measurements from the urine, Wennmalm [41] made a plausible point for the first time in regard to humans of a sympathetic activation in cold water. Now that increased adrenaline and norepinephrine in the plasma have been demonstrated, one must rethink the genesis of cases of sudden death in water. They involve pathological reflex processes affecting the heart with fatal rhythm impairment under certain conditions (Fig. 2).

A first danger is already to be found in reflex sinus bradycardia itself, which yields numerous possibilities for bradycardiac and tachycardiac rhythm disturbances. How the bradycardia comes to be cannot yet be stated with certainty. Is it released directly by cutaneous thermoreception and vagal reflex, or /124 is it to be regarded only as the result of a cardio-

depressor reflex following primary hypertonia? Simultaneous sympathetic stimulation takes hold at different places:

1. The heightened peripheral resistance occasioned by norepinephrine results, via baroreceptors, in reinforced bradycardia. Latent disturbances in the functioning of the sinuatrial circuit and in the buildup of stimulation in the sinus nodes may appear with additional vigor.

2. Increased resistance in the arterial system induces cardiac overload in the form of increased afterload. In cases of hemodyna- /125
mically effective coronary heart disease there is danger of tachycardiac, usually ventricular, rhythm impairment.

3. A third possible complication is associated with the special manner in which the heart reacts to autonomic stimuli. Within the His-Purkinje system only the sinus node and the AV node show a sensitive reaction to stimulation by the vagus as well as the beta-sympathetic system. The ventricular myocardium has exclusively sympathetic innervation and with stimulation there is a lowering of the fibrillation threshold. When at the same time there is retarded stimulation following the ordinary pathway from the cranial, there is particularly great danger of a tachycardiac ventricular rhythm disturbance.

The initial situation when alcohol is in the picture is that of an enlarged periphery, even if intoxication is moderate. The norepinephrine encounters a wider open vessel, instant resistance increase in some cases is greater and along with it a reflex type sinusbradycardia. Another possibility would be increased flow of catecholamine to produce the required vasoconstriction. Here however there is the danger of ectopy in the form of the abovementioned selective sympathetic innervation in the ventricular area.

We consider this concept significant for the following reason: /126
numerous descriptions have been given of rhythm disturbances in cases of sudden cold effect [5, 26, 28, 34, 35, 44]. Cases of sudden

death were noted by Lartique [23] only for temperatures below 18°C. In other authors there is little data about water temperature. It is likewise true that the statistics cited above for the survival of drowned children and adults count only for cold water.

In regard to cold sensitivity it is to be noted that cold reception is possible only in the range of 18-34°C [14]; colder temperatures take the pain receptor path. It may be that the colder temperatures are treated by the CNS in a completely different way.

In immersion bradycardia the specific cold sensitivity of the face is important [5, 9, 12]. We ourselves have shown the fact of increased sensitivity of the upper body when compared with the lower [6]. Thus, where there is question of certifying cases of sudden death in water, we consider it imperative that the water temperature and the topography of the cold effect be considered.

Footnotes

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2. We followed a suggestion from the Pharmacology Center in the Clinic of the Johann-Wolfgang-Goethe University. We are grateful to Prof. D. Palm, M. D. and Mrs. E. Appel, Ph.D. for sharing their findings with us.

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