



E43 Death From Hypothermia During a Training Course Under “Extreme Conditions”: Two Related Cases

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After attending this presentation, attendees will understand that in typical and clinically confirmed cases of death from sub-acute exhaustion hypothermia in subjects with an optimal natural defense system, none of the signs observed in the autopsy (flaccidness and coloring of the skin, cerebral and pulmonary edema, pink markings on the lungs) are specific. Although some recent publications have addressed the utility of postmortem biochemical markers when establishing a diagnosis with no anamnesis, no knowledge or analysis of the circumstances of death, and without an *in situ* examination of the body, it appears difficult, if not impossible, to confirm that death was caused by hypothermia.

This presentation will impact the forensic science community by the relevance of these typical cases. Comparing typical hypothermia cases of dead and living subjects demonstrates that, despite the advances in research on biochemical markers, these will remain non-specific.

Death from hypothermia following exhaustion or from various complicated pathologies is no longer a frequent cause of death among combat troops. During a training course under “extreme conditions” in the French Alps, two young African officers died. Confronted with these two clinically confirmed cases of hypothermia, the unknown anatomopathological and biological specificities associated with death from hypothermia were highlighted. In these typical and clinically confirmed cases of death from sub-acute exhaustion hypothermia, none of the signs revealed by the autopsy were specific.¹⁻³

During the autopsy, the following were noted in both subjects: congestive and edematous brain tissue, pulmonary edema with large pink lesions on the anterior border of the lungs associated with a small number of darker lenticular lesions, congestive abdominal organs, and a full bladder. Based on the microscopic examination, the following was observed: non-specific, anoxic cerebral lesions; alveolar edema with intra-alveolar hemorrhagic alterations; non-specific gastritis lesions; and blood in the peripancreatic fat with no other architectural modifications of the gland that could have resulted from resuscitation maneuvers.³

Overall, the necroscopic signs were scarce and completely lacking in specificity.

Finally, a substantial increase in thyroid hormones with a concomitantly and paradoxically clear increase in the Thyroid-Stimulating Hormone (TSH) content was observed, which is indicative of an immediate and total involvement of the hypothalamo-pituitary and thyroid axis at the outset of the adverse weather conditions, to support the “first-line” pituitary hormones.⁴⁻⁸

At the biological level, the total urinary catecholamines remained at normal levels (1.2 and 1.5 $\mu\text{mol}/24\text{ h}$ (0.8-2.1)), whereas their derivatives had significantly lower values than normal (0.7 and 1 $\mu\text{mol}/24\text{ h}$ (2.1-4.2 $\mu\text{mol}/24\text{ h}$)). Major hypoglycemia (1.2 and 1.3 mmol/l (4.2-6.6 mmol/l)), a highly significant increase in transaminases (ALAT at 738 and 1012 UI/l (8-65 UI/l)), and an increase in serum creatinine (160 and 135 $\mu\text{mol/l}$ (62-106 $\mu\text{mol/l}$)) were also found.

In the literature, it is observed that urinary catecholamines, free fatty acids of the blood, blood corticosteroids (especially cortisol), and free urinary cortisol can increase following death from hypothermia, independent of blood ethanol concentrations. Although this biological data may confirm the diagnosis of death from hypothermia, it is important to emphasize that the quality of conservation of the biological specimen and the time between sampling and analysis will have a significant influence on the stability of urinary catecholamines. Thus, normal levels of urinary adrenalin in such cases of suspected death from hypothermia do not allow such a diagnosis to be excluded.⁹⁻¹³ Similarly, an increase in the concentration of free fatty acids and corticosteroids in the blood cannot be used as the sole criterion for the diagnosis of fatal hypothermia; however, an increase or decrease in corticosteroid concentrations in the blood and urine can also be a symptom of pre-existing diseases, leading to the conclusion that glucocorticoids, just as other biochemical parameters, can be considered potential markers of fatal hypothermia when all of the other postmortem results are taken into account.⁹⁻¹³

In conclusion, although some recent publications have addressed the utility of postmortem biochemical markers when establishing a diagnosis, with no anamnesis, with no knowledge or analysis of the circumstances of death, and without an *in situ* examination of the body, it appears difficult, if not impossible, to confirm that death was caused by hypothermia.



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