Stages of death

Life, Death



Stages of death – Paper Example

An accurate estimation of time since death (TSD) is often a crucial component of a death investigation. The success or failure of many investigations hinges directly upon precise assessment of the time between death and discovery. TSD is one of the hardest determinations to make because of the lack of reliable scientific means. In light of this, a large amount of forensic anthropological and pathological research has focused on developing and improving methods for estimating post-mortem interval (PMI).

Forensic pathologists have primarily focused their research on the initial interval between following death, concentrating on rates of autolysis. Forensic anthropologists have focused their research on the later postmortem interval, concentrating on rates of putrefaction. Both fields depend greatly on the advancements of the hard sciences, that is, analytical chemistry, biochemistry, and physics, for the development of technologies that can be applied to measuring rates of autolysis and putrefaction.

The study of time since death, while remaining a challenge, is an evolving area of research. The first step in measuring rates of autolysis and putrefaction is developing an understanding of the mechanisms that drive both processes. At death, organisms systematically begin the decay process by passing through a physiochemical and gross continuum of tissue breakdown from fresh to skeletal.

Even though systematic, the transition through several well-documented stages (created for scientific convenience) of decomposition is guided by the effects of several factors that include body physique, cause, mechanism, and manner of death, depositional context and the environmental conditions that may serve to temporarily arrest, retard of accelerate this process. Initially, autolysis, the irreversible cascading events of cell death, destroys cellular integrity and the cell-to-cell junctions that progressively result in widespread tissue necrosis.

The by-products of autolysis subsequently fuel putrefaction; the consumption of the body tissues through the progressive proliferation of bacteria. Given appropriate time and environmental conditions, these two internal processes are sufficient, even in the absence of insects and carnivores, to reduce a body to the skeleton. An understanding of decomposition is born from a fundamental knowledge of the normal biochemical function of living cells. Adenosine triphosphate (ATP) provides the energy for the biochemical and physiological pathways of the cell.

In aerobic organisms, ATP is produces by respiration, the oxygen-dependent extraction of energy from food (Berne and Levy, 1993). In anaerobic conditions, some aerobic organisms produce ATP through fermentation, converting pyruvate to lactate. A by-product of fermentation is the reduction of intracellular pH. The anaerobic pathway of ATP production is inefficient and the net gain of energy is insufficient to maintain cellular physiology (Gill-King, 1997; Tobin and Morel, 1997).

At death circulatory stasis and the consequent loss of aerobic ATP synthesis insults cellular integrity leading to microscopic (cellular) and eventual macroscopic (tissue) morphologic changes. The membrane transport system is destroyed by denaturing proteins in the cell membrane. With loss of the cross membrane transport system, molecules and ions essential for cell survival are unable to pass across the concentration gradient (Tobin and Morel, 1997).

Meanwhile, damaged membrane selectivity allows extra-cellular matrix to leak into the cell causing it to swell. Lysosomes, cellular organelles housing hydrolytic enzymes that function in intracellular digestion, rupture and releasing their contents. During aerobic cellular conditions, lysosomes fuse with the membrane of the phagosome and release the hydrolytic enzyme into the phagocytic vacuole, digesting the entity and releasing nutrients into the cytoplasm. The destructive enzymes remain locked within a membrane throughout the digestive process.

With membrane structural integrity compromised, liberated hydrolytic enzymes leak in the cytoplasm; activated by the lowered pH of the cytoplasm, they begin to consume the cell (Junqueira, Carneiro & Kelley, 1991). Finally, with continued disintegration of the cell membrane, cell to cell junctions dissolve, causing localized or focal death and eventual organ tissue necrosis. During this stage, decomposition becomes observable at the gross level as tissues become subjectively paler. In addition, breakdown of the cellular junction occurring between the layers of epidermis and dermis results in gross slippage of the epidermis (Spitz, 1993).

During life, normal metabolic pathways maintain the body at a core temperature of 98. 7°F. When these pathways diminish, the body begins gradually, then more rapidly, to cool to ambient temperatures. This is referred to as algor mortis. Livor mortis, or hypostasis, is the pooling of blood

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in the body. Blood pools in the capillary beds of regions of the body experiencing the greatest gravitational pull such as the feet of a hanging victim. Initially, livor is unfixed, meaning pressure will force the collected blood out of the capillaries, allowing the skin under pressure to blanch white.

With time, the capillary blood and surrounding fat coagulates, trapping the blood. At this point blood does not recede from the capillary under pressure causing livor fixation (Coe, 1993; DiMiao and DiMiao, 1996; Clark et al. 1997). Rigor mortis is the stiffening of muscles from the binding together of fibers within cells. Muscle cells consist of two fibers, myosin and actin, which bind and pull across each other during contraction. At rest, the binding site on the actin fiber for the myosin fiber head is bound by a troponintropomyosin complex (Berne and Levy, 1993; Junqueira et al. , 1991).

Calcium ions released from the membrane interact with the complex moving it from the binding site. These ions enable the myosin fiber head to bind to the actin fiber. At this point energy is released from ATP associated with the structures causing the myosin head to bend and the two fibers to pull across each other. The muscle cell relaxes after the calcium ion is pumped from the sacroplasm back into the cell membrane releasing the troponin-tropomyosin complex to obscure the myosin-binding site. At death, calcium ions are released from the disintegrating membrane allowing the two fibers to bind.

However, in the absence of ATP, the fibers do not slide across each other and calcium ions are not pumped out of the sarcoplasm. Hence, during rigor mortis the muscles do not contract, but stiffen via fiber binding. The decreased intracellular pH also causes the cytoplasm to congeal which contributes to rigor mortis. With time, the fiber breaks away from their anchoring site at the end of the cell gradually causing rigor mortis to dissipate. Following death are several other processes explaining the decomposition of the body.

To discuss briefly, putrefaction follows after the initial death. Putrefaction, or bloating, is the alteration of an organism through bacteria activity. The release of nutrients from autolyzed cells coupled with the decreased intercellular pH from loss of the buffer system creates a rich environment for endogenous bacterial proliferation (Spitz, 1993). The visible gross morphological changes with bacterial activity and the generation of odor are by-products of putrefaction. After the putrefaction comes the black putrefaction. In this stage, the bacteria create gases.

These gases then mixes with the blood making the skin colored green and after four or five days, becomes black. The smell gets worst because of the sulfur gases, putresine and cadaverine. After this stage comes butyric fermentation in which the corpse starts to dry out. The gases produced in the black putrefaction process lifts the skin from the muscle causing it to fall off. Finally, the body is desiccated and the rate of decay becomes slower. Perhaps a deeper study at the process of human decomposition should be undertaken as this is a crucial component of a medico-legal investigation.