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An Alternative Mechanism For Death by Crucifixion

by

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Dr. Bishop is a researcher in exercise physiology and ergonomics at the University of Alabama. He has studied suspension trauma with regards to fall protection applications. In the course of examining suspension trauma it occurred that this was a viable explanation for the mechanism for crucifixion. Dr. Church is a professor and researcher in exercise physiology at Arkansas State University. The manuscript was written solely by these two authors, and reflects only their own views, and not those of their institutions.

Introduction

For much of the western world, death by crucifixion holds a special significance. The most popular explanation for the mechanism of death by crucifixion^{1,2,3,4,5} was that mortality is attributable to respiratory failure. The mechanics of asphyxiation were attributed to positioning the thorax such that an upward push by the victim's legs was requisite for exhalation. An alternative explanation provided by several researchers is that death in crucifixions resulted from generalized traumatic shock.^{6,7,8,9,10} Others have suggested cardiac rupture as the primary mechanism of death.^{9,11,12,13,14,15}

We propose an alternative explanation that, in some crucifixions, orthostatic incompetence was a primary mechanism of death. The purpose of this paper is to elucidate the physiological evidence for and against these various explanations for death by crucifixion.

Methods

A thorough review of the literature yielded three major hypotheses regarding mechanisms for death by crucifixion cited above. Further review suggested the fourth possibility of death due to suspension trauma.

Death Due to Asphyxiation

Death by asphyxiation was originally proposed by LeBec¹ and supported by others.^{2,3,4} Barbet² cites reports from a World War II concentration camp wherein victims suspended from their hands fixed directly above their head did experience asphyxiation. Zugibe⁶ argues that this sort of hands-high suspension was not characteristic of crucifixion, rather crucifixion victims more typically would have their arms spread to approximately 70 degrees from a vertical axis. This angle was substantiated in Zugibe's experiments with living volunteers who also provided no physiological symptoms or reports of respiratory distress. However, these experiments were limited by the pain to 45 minutes or less, and longer duration could have induced respiratory insufficiency.

Death Due to Shock

A second common explanation for death due to crucifixion is profound hypovolaemic/traumatic shock secondary to blood loss, pain and general trauma precipitated by pre-crucifixion torture. In this explanation, the cumulative shock involved with beating the victim, driving spikes through the extremities, and denying food and water, would eventually lead to circulatory collapse leading to cerebral hypoxia. Depending on the extent of trauma, dehydration, and associated blood loss, death might take as long as several days.

Electrocutions, burning at the stake, hanging, firing squads, injections of toxins, are all examples of execution methods in which the execution method is primary to death. In each of these cases, death is normally both certain and reasonably rapid. If general shock were the primary mechanism of death, then crucifixion appears perfunctory and would chiefly serve as a means of immobilizing the victim or perhaps emphasizing any deterrent effect of the punishment.

It might be concluded that shock would likely contribute to death in crucifixion given the pain involved in having one's body mass suspended by being nailed or tied by the arms or hands to a cross. Edwards et al.⁹ postulated that it was a combination of hypovolaemic shock and fatigue-induced asphyxia that likely induced death in crucifixion.

Death Due to Cardiac Rupture

Another suggestion^{9,11,12,13,14,15} was that death in crucifixion was due to cardiac rupture. One mechanism is that the left ventricle could be damaged by a blow, or that general circulatory trauma could precipitate a

transmural infarct sufficient to rupture a ventricle.¹⁵ Some have suggested that ventricular rupture can occur rapidly¹⁵ though others insist it is usually considerably delayed.¹⁶

Modern observances of rupture of the heart suggest that it is typically secondary to coronary artery disease and occurs at least several hours after the infarction.¹⁶ Inducing cardiac rupture seems much more laborious and uncertain than alternatives. In cardiac rupture, the victim would be dead, or nearly so, before being crucified, rendering the actual crucifixion unnecessary.

A Novel Alternative Explanation

We hypothesize that a primary or contributory means of death in crucifixion would be orthostatic incompetence inducing hypovolaemic shock alone or adjuvant to pre-crucifixion injury. Our alternative explanation arises from observations of physiological responses to lower-body negative pressure, tilt tables, and harness suspension trauma.^{17,18,19,20}

Research studies utilizing lower-body negative pressure (LBNP), (a physiological analog of suspension trauma) have reported cases of vasovagal syncope, frank hypotension, and asystole.¹⁷

A less extreme example of suspension trauma is a tilt-table challenge, known to produce almost certain syncope, given enough time.¹⁸ In this paradigm, the participant is strapped supine to a table that is slowly raised from horizontal to within 20 to 40 degrees of vertical. If the person is maintained in upright posture, hypotension worsens and severe cerebral ischaemia can ultimately result in death. Baron-Esquivias et al.¹⁹ performed a large tilt-table study on patients with idiopathic syncope. They noted several cases of syncope and asystole during head-up tilt lasting from 3 to 90 seconds in their patient sample. They also note that asystole in tilting has been previously reported many times by other investigators. In LBNP, a period of asystole would likely be the primary mechanism of death, whereas in tilt table it could be asystole or unmitigated cerebral ischaemia.

Anyone can increase their tolerance of LBNP, tilt table, quiet standing, or suspension by intentionally contracting their leg muscles. In crucifixion, eventually syncope, fatigue, or injury reduces the muscle contraction to a level insufficient to maintain blood pressure and syncope ensues.

Suspension Trauma

In suspension trauma, the legs are immobile with a victim in an upright posture. Gravity pulls blood into the lower legs, which have a very large venous storage capacity. Enough blood eventually accumulates so

that return flow to the inferior vena cava is reduced such that cardiac output begins to fall. Heart rate increases to maintain sufficient cerebral blood flow, but if blood return to the right atrium is sufficiently reduced, tachycardia is ineffective, and the vasovagal reflex (or possible exhaustion of catecholamine stores or receptor tachyphylaxis) abruptly induces bradycardia. Typically this induces syncope resulting in a prone or supine posture relocating the legs, the heart and brain to the same level, minimizing the hindrance of gravity. Blood flow is restored to the right atrium and the victim recovers quickly. In a vertical suspension, the victim cannot fall to a horizontal posture so cerebral blood supply falls below the critical level. Once syncope ensues, the victim loses the contribution of the leg muscle pump and hypotension progresses until fatal cerebral ischemic hypoxia or myocardial infarction occurs. Seddon²⁰ provides a complete review of suspension trauma.

Suspension trauma doesn't occur often in healthy people because it requires that the legs remain completely relaxed, straight, and suspended below heart level. If the leg muscles are contracting, the muscle pump insures sufficient return to the inferior vena cava. If the upper-legs are horizontal, the vertical pumping distance is greatly reduced, so quiet sitting is not problematic.¹⁸

Recovery from Crucifixion

Death is still a potential sequela after a living victim is rescued from suspension. Post-rescue death apparently results from the heart's intolerance of the abrupt increase in blood flow after removal from suspension. Apparently, metabolic products secondary to ischaemia induce damage similar to that seen in recovery following periods of myocardial ischaemia in acute coronary syndromes treated with percutaneous revascularization or following aortic crossclamping and cardioplegic arrest in cardiac surgery. First aid procedures for suspension trauma are to slowly move the victim from a kneeling posture, to sitting, to supine.²⁰

There is circumstantial evidence to support suspension trauma as a mechanism in crucifixion. Suspension trauma would have induced death more certainly than general shock. Victims who incidentally periodically contracted their leg muscles could fight off syncope until fatigue and cramps finally overwhelmed their ability to resist. This would produce an extended torture fitting the descriptions of prolonged crucifixions.¹⁶ Likewise, breaking the victim's legs, as apparently was a common practice, inhibited the willingness to contract the leg muscles and hastened death whether secondary to shock, respiratory insufficiency, or suspension trauma. It is important to recognize that any torture that induced hypovolaemic or traumatic shock, or hastened fatigue would speed the

onset of suspension trauma. After syncope in a vertical posture, death is assured given there is no longer the ability to utilize the muscle pump.

Discussion

Our purpose was to propose a plausible novel alternative mechanism of death in crucifixion. There are empirical tests of our hypothesis. In his experiments simulating crucifixion, Zugibe found no evidence of hypotension or leg edema (F.T. Zugibe, personal communication, 2004), but he does state that his subjects engaged in considerable leg contractions in an effort to relieve shoulder discomfort which would preclude suspension hypotension by engaging the leg muscle pump. The Zugibe simulations did not exceed 45 minutes, and some were as short as 5 minutes. Had they gone on long enough for fatigue to ensue, we believe suspension trauma would have evidenced itself. Likewise, the relatively short duration of these simulations could not preclude asphyxiation, and his simulations were lacking traumatic shock.

Whereas it is possible that asphyxiation due to respiratory failure could have been a primary mechanism of death, it has never been verified empirically. Tenney⁸ pointed out that diaphragmatic action should provide sufficient ventilation at rest even in the presence of immobilization of the chest wall. Frans Wijffels, a Dutch physician, reviews a paper published in German in 1949 by radiologist H. Modder at the following:

<http://www.shroud.com/pdfs/n52part3.pdf>

According to Wijffels, Modder suspended medical student volunteers by the wrists and found that vital capacity fell from 5.2 to 1.5 L, with this final tidal volume sufficient to sustain life. Likewise, heart rates rose to 140 pm, and blood pressure fell from 120 to 70 mmHg, ECG and x-rays indicated coronary and cerebral hypoxia. These responses implicate suspension trauma as a key contributor to death in crucifixion. Although the total time of suspension was not clearly indicated, these were likely very short suspensions which would not necessarily fully simulate the longer suspensions in crucifixions.

What would constitute a substantive test of our hypothesis? As Zugibe has reported,⁶ pain precludes lengthy crucifixion simulations in the laboratory. The only ethical experiments would be those involving suspension in a body harness or 70 degree head-up tilt. Those experiments have already been done, as we have reported herein. It seems unethical to repeat these experiments solely to verify that syncope and asystole can be induced in crucifixion simulations. In fact, definitive tests of crucifixion require pre-crucifixion scourging or some means of inducing pre-

crucifixion shock. There have already been laboratory tests verifying our proposed mechanism. Short of an actual crucifixion no better studies can be done.

Scripture and Suspension Trauma

Doubtlessly there were different causes of death among the thousands of crucifixion victims. Suspension trauma would have unavoidably been present in crucifixion regardless of the primary cause of death. There are several aspects of the Gospel accounts that give some clues to the mechanism of death. Probably the best known aspect of Christ's crucifixion were the pronouncements He made from the cross (Matt 27:46; Mk 5:34; Luke 23:43,46; John 29:26-30). Both shock and suspension trauma could permit the unimpeded pronouncements from the cross reported in the Gospel accounts, whereas respiratory failure would normally prohibit loud vocalization. Of course, an appeal to the miraculous can overcome any physiological explanation.

A key philosophical aspect of the crucifixion is the sovereignty of Christ. In Luke's and John's accounts of the crucifixion, the language seems clear that Christ died voluntarily, as opposed to the executioners "taking it from him" (see John 10:7-8). In the case of Christ, who certainly knew human physiology, he could "give up the ghost" by simply relaxing his legs. Had He continued to contract his leg muscles, the crucifixion could have gone on for several hours, which was often the case. Eventually He would have succumbed involuntarily.

Fainting could logically lead to the practice of testing the pain response of unconscious victims by stabbing them with a spear. This is particularly important in suspension trauma, since this method would result in an occasional error whereby a victim was prematurely removed from the cross, and upon being laid down spontaneously recovered from the attempted execution. Routine spontaneous recovery seems a bit less likely for the other two scenarios, asphyxia and shock. Finally, general shock or suspension trauma would result in the thirst reported in the account of the crucifixion of Jesus (John 19:26).

Whereas there can be no definitive mechanism for death by crucifixion ascertained from Scripture, circumstantial support for suspension trauma is present. These historical accounts seem to more strongly support suspension trauma than asphyxiation. It appears, as in many cases, that a conclusion can never be certain, but merely the product of considering the evidence and weighing the probabilities.

Summary

Whereas no one can say definitively the exact mechanism of death in any particular crucifixion, there are several possible contributors. Of the potential etiologies, it is likely that orthostatic incompetence independently, or combined with asphyxiation and hypovolaemic/traumatic shock, was a primary mechanism of death in most cases.

Even with a complete pathology report, it would be challenging to determine the exact aetiology of death in the face of several potential contributors. Because of this, it is impossible perhaps to give a definitive answer to the aetiology of crucifixion, but it certainly seems that orthostatic intolerance independently, or combined with asphyxiation and hypovolaemic/traumatic shock, would qualify for consideration.

References

1. A.A. LeBec, *Catholic Med. Guard.* **3**, 126 (1925).
2. P. Barbet, *A Doctor at Calvary*, (P.J. Kennedy & Sons, New York, 1953).
3. N.D. DePasquale, G.E. Burch, *American Heart Journal*, **66**, 434 (1963).□
4. R. Lumpkin, *J Med Assoc Ala.* **47**, 8 (1978).□
5. C.T. Davis, *Ariz Med.* **22**, 183 (1965).□
6. F.T. Zugibe, *Sindon, N.S.*, Quad. #8, 109 (1995).
7. W.B. Primrose, *Hibbert J.* **47**, 382 (1949).
8. S.M. Tenney, *American Heart Journal*, **68**, 286 (1964).□
9. W.D. Edwards, W.J. Gabel, F.E. Hosmer, *JAMA*, **255**, 1455 (1986).□
10. F. T. Zugibe, *Can. Soc. For. Sci. J.* **17**, 1 (1983).
11. W.A. Stroud, *Treatise on the Physical Cause of Death of Christ* (Hamilton and Adams, London, 1874), pp 36-156.
12. H.C. Cooper, *NY Med J.* **38**, 150 (1883).
13. S. Bergsma, *Calvin Forum*, **14**, 163 (1948).□

14. J. Whitaker, *Catholic Med. Guard.* **13**, 83 (1935).
15. D.A. Ball, *J Miss St Med Association.* **30**, 77 (1989).□
16. J.E. Holoubek, A.B. Holoubek, *Journal of Meedicine.* 26, 1 (1995).
17. F. Hilton, J. Giordano, S. Fortney, *Aviation, Space, and Environmental Medicine.* **60**, 61 (1989).
18. P. Madsen, *Aviation Space and Environmental Medicine.* **69**, 781 (1998).
19. G. Baron-Esquivias *et al.*, *European Heart Journal.* **23**, 483 (2002).
20. P. Seddon, "Harness Suspension: review and evaluation of existing information" (Health and Safety Executive. Research Report 451/2002).