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Admiral Horatio Lord Nelson's Death at the Battle of Trafalgar: A Neurosurgeon's Forensic Medical Analysis

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Abstract

Admiral Horatio Lord Nelson, the greatest British naval hero, was fatally wounded during the Battle of Trafalgar. Conventional theory holds that he died mainly of an injury to a major blood vessel in the chest. However, a review of the empirical evidence-with the benefit of modern medical science-, suggests that the primary cause of death was a spinal neurogenic shock from transection of the mid thoracic spinal cord. These conditions do not necessarily exclude each other.

Keywords: Nelson's primary cause of death; Battle of Trafalgar; Musket ball shot trajectory; Hemothorax; Spinal neurogenic shock; Spinal cord transection at T6-7; Neurosurgical medical forensic analysis

Introduction

Lord Nelson's most famous naval engagement at Cape Trafalgar, saved Great Britain from threat of invasion by Napoleon Bonaparte, but it would be his last. He was killed while leading the attack on the combined French and Spanish fleet, shot by a French sniper during the battle. He died shortly after, becoming one of Britain's greatest war heroes.

The fatal wound was documented at the time by the surgeon of HMS Victory, Dr. William Beatty. In the "Authentic Narrative of the Death of Lord Nelson" published in 1807, Beatty suggested that a division of a large branch of the left pulmonary artery was the cause of Lord Nelson's early death [1]. While this has been much analyzed, discussed and interpreted ever since, it merits a 21st century review and commentary. The present neurosurgical analysis of the fatal wound leads to a conclusion somewhat different and more complete than the conventional historical explanation of the cause of death.

On Monday, October 21, 1805, shortly after one o'clock in the afternoon, in the midst of the naval battle, Hardy-the Captain of HMS Victory-realized that Nelson was no longer next to him. He turned to see Nelson kneeling on the deck, supporting himself with the left hand, his only one, before falling onto his side. He had been shot by a French marksman from the Redoutable, at a range of only 15 meters. Nelson felt "death enter with it", for when the horrified Hardy bent over his stricken friend and commander, he heard the terrible words, "Hardy, I believe they have done it at last... my backbone is shot through". Striking Nelson on the left shoulder at the epaulette, with a force that first threw him onto his knees, the French musket ball was described as smashing two ribs (the 2nd and the 3rd), tearing through his left lung and "severing a major artery on the way". Then, having fractured the spine at the level of the 6th-7th thoracic vertebrae (T6-7), and "wounding the spinal cord", it lodged 5 cm below his right shoulder blade in the muscles of his back. After falling, a sergeant-major of marines, Robert Adair and two seamen, carried Nelson down to the cockpit. He asked them to pause while he gave some advice to a midshipman on the handling of the tiller. Nelson then draped a handkerchief over his face to avoid causing alarm amongst the crew. He was taken to the surgeon, William Beatty, telling him, "Beatty, you can do nothing for me. I have but a short time to live. My back is shot through" [1].

He took over three hours to die, succumbing at 4:30 PM. For most of that time he was in shock and in agony. The entrance wound was in the left shoulder and the ball went down through the chest and through the spine. Beatty wrote that the "spinal injury by itself could have been mortal" but "from it alone he could have survived 2-3 days although in miserable condition." Beatty did not attempt to probe or explore the wound since the "back bone was shot through," and he did not want to cause any more pain. Eventually, he removed the ball aboard the Victory before arriving in England (December 1805) finding "a portion of the gold-lace and pad of the epaulette, together with a small piece of his Lordship's coat firmly attached to it." Nelson was fanned and given lemonade and watered wine after he complained of "feeling hot and thirsty." He asked several times for Hardy, who was on deck. He asked Beatty to remember him to Emma (his mistress), to his daughter, and his friends. Hardy came below decks to see Nelson just after half-past two and informed him that a number of enemy ships had surrendered. Nelson's words were carefully recorded during the three hours of his agony. He laid below in the cockpit in great pain but conscious until just before he died [1].

Discussion

Some of Nelson's recorded symptoms support the diagnosis of hemothorax: Nelson "found it difficult to breathe", felt "a gush of blood inside his chest", asked his valet to "turn him on to his right side" which "gave him scant relief", and he also said: "I feel something rising in my

left chest". But Mr. Beatty's first partial autopsy-done aboard the Victory the day after Nelson's death-and a second one (repeated later on the voyage home) showed that "The quantity of blood effused did not appear to be very great." Some of the other injuries, including that to the left lung-on the path of the musket ball-could not easily explain on their own merit his death so soon after the gunshot wound, considering that "the bleeding was just moderate." The backbone, the spine, was "transected by the ball." Also recorded was "loss of movement and sensation below the chest," implying spinal cord injury [1].

The fact is that the injuries were caused by a musket ball, that according to ballistics, is of known slow velocity and of a straight trajectory. It should be noticed that there is a significant anatomic distance between the branches of the pulmonary artery and the spine. Assuming that the left pulmonary artery or a large branch thereof was indeed injured, anatomical studies based on atlases and dissection on cadavers fail to support a simple straight-line course of the musket ball that could have both divided the artery and also damaged the spinal cord on its path forward [2].

Chest injury's life-threatening conditions (like Nelson's) include cardiac tamponade, tension pneumothorax, open pneumothorax, massive hemothorax, and flail chest, from multiple rib fractures. Management of massive hemothorax includes volume replacement and chest decompression. According to Beatty, difficulty in breathing was brief, despite of the fractures of the 2nd and 3rd ribs and the lung injury, and Nelson kept talking freely and frequently until the end, ruling out a fatal flail chest or a potentially fatal tension pneumothorax.

Nelson's pulse was "weak, small and irregular," compatible with shock. This condition can be produced by factors that attack the strength of the heart as a pump, decrease the volume of blood in the system (like in an external or in an internal hemorrhage), compress/displace the heart/lungs (like in a hemo or in a tension pneumothorax), or permit the blood vessels to increase in diameter (as in sepsis, anaphylaxis and in spinal neurogenic shock). The commonest type of shock is hypovolemic (or low-volume) shock. It happens when blood or plasma is lost in such quantities that the remaining volume cannot fill the circulatory system despite constriction of the blood vessels. It is characterized by a significant arterial hypotension, and compensatory tachycardia. Patients are pale, cold and clammy. The treatment of hypovolemic shock requires replacement of volume-i.e. blood transfusion-, stopping the bleeding (especially if arterial), and evacuating the hematoma if its volume is such that it compresses or displaces vital structures (heart, lungs). Paralyzed and bleeding internally, Nelson laid in cramped midshipmen's quarters in the after part of the ship, below the waterline, and in the dark (walls were painted dark red). The attendants gave him fluids as he watched Beatty work on the wounded, while the lower deck men cheered after each French ship was struck. Aware of his fatal injury, Nelson gave Hardy instructions for the care of his family and requested that his body not be thrown overboard. Hardy agreed and upon his death, and after the engagement, interred the corpse in a leaguer, the largest shipboard cask, which was filled with brandy. Thus embalmed, Horatio Lord Nelson returned to England and to a hero's burial.

A thorough neurosurgical study of the fatal wound suggests that Lord Nelson died primarily from and in spinal shock following a total spinal cord injury at T6-7. There was complete loss of all neurological activity below the level of the cord transection including motor, sensory, reflex and autonomic function. Lord Nelson's complaints were well recorded: "My backbone is shot through," (vertebral injury), "All power of motion below my breast is gone", and "I have no feeling below the breast," (spinal cord damage). He felt "thirsty" and "hot" pretty much all the time and asked for "Fan, fan" and "Drink, drink." Only at the end was he cold [1].

Nelson was paralyzed and in spinal neurogenic shock following the spinal cord injury/transection, presenting with flaccid paralysis of the lower extremities, areflexia and anesthesia below the level of injury, and with severe autonomic effects.

Spinal shock was first defined by Whytt [3]. Neurogenic spinal shock is a type of distributive shock in which there is a sudden loss of autonomic sympathetic tone, resulting in arterial hypotension, bradycardia and impaired autoregulation (leading to secondary injury) [4]. The mechanism for spinal shock involves the sudden interruption of conduction in the spinal cord as well as damage of neurons at the level of the injury. There is a loss of somatic and autonomic reflex activity below the level of the damaged spinal cord segment. Autonomic dysreflexia occurs when the spinal cord reflex arcs that are immediately above the injury, may also be severely disrupted from a loss of cranial regulation leading initially to extreme arterial hypertension, loss of bladder/bowel control, sweating, headaches, and other sympathetic effects. It is difficult to ascertain precisely whether this took place in the Nelson's case since obviously, no modern medical documentation of vital signs was available then [5].

The autonomic nervous system-or visceral efferent nervous system-has two divisions: sympathetic and parasympathetic. They are antagonistic. Pharmacological modulation is based on neurotransmitters and receptors, two neurons and two synapsis: preganglionic and postganglionic. The sympathetic neurons are located from T1 to L2 in the intermedio-lateral cell grey column of the spinal cord. When needed, i.e. after blood loss, an intact sympathetic nervous system triggers various compensatory mechanisms by releasing epinephrine and norepinephrine. These neurotransmitters initially cause an increased heart rate, faster breathing, and sweating. They cause vasoconstriction to shunt blood away from the extremities and towards the vital organs. This in turn leads to increased blood pressure and a pale, cool skin. In end-stage shock, the patient generally exhibits very low blood pressure, pale and cool skin, weak and fast pulse, and rapid and shallow breathing. In the case of neurogenic shock though, there is a sudden loss of sympathetic stimulation to the blood vessels, that causes them to relax (vasodilation) resulting in a sudden drop in blood pressure from decrease of peripheral vascular resistance. Severe hypotension and bradycardia in an acute spinal cord injury (complete lesion) happens because of an imbalance of the autonomic control with an intact parasympathetic influence-via the vagal nerve-and loss of sympathetic tone from disruption of the supraspinal control. Hence, the body loses its ability to activate the sympathetic nervous system and cannot trigger compensatory mechanisms. The parasympathetic tone remains. Neurogenic shock includes hypotension due to a sudden, massive vasodilation. Warm, flushed skin is from both vasodilation and from inability to vasoconstrict. Priapism, occasionally present, is also due to vasodilation. An acute transection of the spinal cord's descending pathways interrupts the supraspinal control of the sympathetic nervous system, with initial paralysis and disturbance of the sympathetic activity below the injury level. That, and as mentioned above, the lack of inhibition of the parasympathetic nervous system, has severe acute, subacute, and chronic effects. An acute and complete mid-thoracic (T6-7) spinal cord injury-like Nelson's-is characterized by motor and sensory deficit below injury level, cardiovascular, thermoregulatory

and broncho-pulmonary instability, urinary, gastrointestinal and sexual disruption. In summary, patients in spinal neurogenic shock present with cardiac bradyarrhythmias, hypotension, abnormal temperature control and disturbance of sweating, and vasodilation.

Spinal neurogenic shock causes great internal body disarray and turbulence. For example, transthoracic echocardiography in neurogenic shock shows impairment of the left ventricular cardiac function. There is also adrenal insufficiency. Decrease of systemic vascular resistance leads to relative hypovolemia because of the increase in venous capacity. Administration of isotonic fluids is frequently necessary, but arterial hypotension from spinal shock is frequently resistant to reanimation with fluids. Regarding bradycardia, atropine is required for a heart rate of less than 50/minute; a nodal rhythm with ventricular escape and dys-arrhythmias require higher doses of atropine or even pacing. Also, oxygen and inotropic agents such as dopamine are required. As to arterial hypotension, sympathy co-mimetics vasopressors, such as ephedrine are indicated after failure with a fluid resuscitation trial. Dopamine is often used, either alone or in combination with other inotropic agents, and with antidiuretic hormone (ADH). Phenylephrine may be used as a first line treatment or secondarily when there is no response to dopamine. Cardiac pre-load, contractility, after-load, heart rate, mean arterial blood pressure (through arterial catheterization), central venous pressure, pulmonary capillary wedge pressure (through Swan-Ganz catheterization), and other extensive hemodynamics monitoring, are of great help in the management of this condition [6,7].

Lord Nelson was in neurogenic shock. He was most likely hypotensive and bradycardic, and with red, warm, dry skin and feeling hot-at least initially-followed by hypothermia after vasodilation and heat loss. Because his lesion was at T6-7 (between C5 and T10), there must have also been respiratory intercostal muscles involvement associated with hypoxia, hypercapnia, and bronchial congestion. If injured today, Admiral Nelson would receive competent initial surgical care aboard a modern-day Navy ship followed by care at a tertiary neurosurgical hospital unit on land [8]. He would survive, but unfortunately, as a paraplegic, and as per Dr. Beatty's autopsy report: "The rest of the body showed no abnormalities. The heart and lungs were sound... (the body) resembled ...a youth...giving every evidence that His Lordship might have lived to a great age." He was only 47 years of age.

Conclusion

This forensic neurosurgical analysis of Lord Nelson's fatal wound at Trafalgar, reaches a different and more comprehensive conclusion regarding the primary cause of his demise, than the conventionally accepted one.

Blood loss from a torn pulmonary artery or from one of its branches and/or compression of vital structures by a hemothorax, was neither the only nor the main cause of his death, considering that the bleeding was just moderate and that modern ballistics excludes a major vessel in the trajectory of the musket ball.

Lord Nelson, hot at first and cold thereafter, paralyzed, with back pain and a weak arterial pulse, died mostly of and in spinal shock, following complete damage of the mid-thoracic spinal cord. The cord was transected at T6-7 by the passage of a ball shot by a French musketeer, resulting in loss/paralysis of all neurological activity below the level of the lesion, including motor, sensory, reflex and autonomic function. This prevented the body of the Admiral-despite its youth-from compensating for the chest bleed, contributing to end-organ cellular dysfunction/death from tissue hypoperfusion.

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