The Rude Unhinging:
A Study of Shock

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Dedication

For Sten and Tove.

Abstract

In the early 19th century, the basic intellectual tenets of medicine underwent significant change. From a practice of nosology, disease classification, and a focus on the subjective and the symptom emerged an epistemology that looked for objective clinical signs that denoted the presence in the body of disease-defining pathologic lesions. Yet this physical, tangible identity of disease was challenged by the presence of functional diseases, which were readily identified but difficult to define, as they left no diagnostic mark upon their sufferers' bodies. Shock, a readily apparent, omnipresent phenomenon which could complicate injury, childbirth, certain disease states, and medical therapies, was one such condition. This study looks at how physicians and surgeons in the 19th century attempted to create an intellectual model for shock, so as to better define, recognize, and treat it. We will explore how technological change and social conditions affected this understanding, and how both traditional and novel theoretical models were invoked to explain it. We will also look at attempts to understand shock as examples of Kuhnian normal science, for even as technology changed in the early 20th century, and shock could be expressed and described in very modern-seeming ways, the underlying models and concepts that defined shock changed very little.

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Chapter 1 – A disease with an essence, but no seat

On 5 May 1842, a London police officer named Moss saw a suspicious young man lurking about a forested area on his beat in Highbury-Barn. Thinking he met the description of a man wanted for several recent robberies, Moss approached the suspect, who turned on him with a pistol and shot him in the arm. A second policeman and a local citizen, seeing the incident, began to chase the suspect, who produced another pistol and shot the second officer. The citizen, a man named Mott – either a baker or a broker, depending upon whose account you believe – was apparently made of stern stuff, and he continued the pursuit, driving the fleeing suspect into a narrow, dead-end lane. At this point, a third constable arrived, 42-year-old Timothy Daly, a married, 13-year veteran of the force. Putting his back to a hedge and brandishing his pistols, the suspect – a 22-year-old bricklayer named Thomas Cooper – threatened to shoot the first man in the gathering mob who touched him. Daly looked to Mott, declared, "I don't believe those pistols are loaded," and the duo rushed the suspect. Cooper, who had reloaded on the run, responded by firing both weapons simultaneously. Mott took a ball through the shoulder, and Daly fell instantly, "shot dead through the heart" according to press accounts. Cooper, described as a middle-sized man who "seemed like a person who had suffered much from want and weariness," was taken into custody and ultimately found guilty of murder. He was hanged for his crime at Newgate prison on 4 July.[1]

[1] Details on the story of Cooper and Daly from H. Daniell, "The phrenological society," The *Zoist* 1 (1843):46-48; Henry Hamilton Fyfe, *The Annals of Our Time*, 2nd ed. (London: MacMillan and Co., 1880), 107-108; T. Romeyn Beck, "Instant death from a wound of the stomach," *The American Journal of the Medical Sciences* 4 (1842):219; Hepworth Dixon, *The London Prisons: With an Account of the More Distinguished Persons Who Have Been Confined in Them, to Which is Added a Description of the Chief*

While this was certainly a case that captured the public interest – Daly was only the fourth constable of the Metropolitan Police to die in the line of duty since the force's inception in 1829,[2] Cooper's execution is mentioned in several reference books of dates, and a cast of Cooper's head was the centerpiece of discussion at the London Phrenological Society's Ladies' Night meeting on 21 November 1842[3] – the story of Daly passed through the medical literature without fanfare. For our purposes, though, it will bring up some important ideas.

Mr. Edward Drewry, the surgeon called to the scene of the shooting, had made a preliminary examination of Daly, arriving by his account three minutes after the event and finding the constable with "a slight muscular action of the jaw" but clearly dead, and without a pulse. Removing the body of the unfortunate Daly to the local tavern, Drewry found that "the ball entered and fractured the seventh and eighth ribs, and then passed transversely through the stomach, and out the other side below the floating rib, which is fractured." The ball did not pass through the chest, asserted Drewry, and so thus not through the heart.[4] The coroner directed Mr. Robert H. Semple, the parish surgeon, to

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Provincial Prisons (London: Jackson and Walford, 1850), 219; Joseph Timothy Haydn, *Dictionary of Dates and Universal Reference*, 3rd ed. (London: Edward Moxon, Dover St., 1845), 517; "The murder of the policeman at Highbury," *The Morning Chronicle* (London) 6 May 1842:6-7; "Trial of Thomas Cooper for the murder at Highbury," *The Era* (London) 19 June 1842:8.

[2] Per the historical timeline of the Friends of the Metropolitan Police, www.fomphc.org.uk/faq.php?cat_id=6&rowstart=0, accessed 31 March 2011.

[3] Daniel, "The phrenological society," 46.

[4] "The murder of the policeman at Highbury," 6.

perform a formal postmortem examination upon Daly's body, a report of which Semple submitted to the *Lancet* on 9 May.

On general examination, Semple noted that the body was well-formed and healthy-appearing. "A considerable quantity of blood had flowed from the wounds," he noted, "so as completely to saturate the flannel-waistcoat which he wore next to his skin." The bullet wound, the size of a six-pence, was below the left seventh rib; a second wound was evident on the right flank. The brain, chest, heart, and lungs were unremarkable, and there was no blood in the thorax. The stomach was of interest to Semple, as it was "distended with half-digested food," and had "an aperture with blackened edges, of about the size of a shilling, an inch below the junction of the oesophagus with the stomach on its posterior surface, and another corresponding aperture on the anterior surface." The liver and bowels were otherwise unremarkable, and he found no injury to the aorta or "important vessel." Semple's conclusion:

> In this case death occurred almost instantaneously after the discharge of the pistol; and must have been caused by the sudden shock given to the nervous system by the passage of the bullet through the distended stomach. No other cause of death can be assigned, for no other viscus was wounded, nor was any important vessel

ruptured."[5] Further, in his testimony before the coroner, he remarked "the sudden shock the digestion received was the cause of death, and not internal hemorrhage.[6]

Semple's case report likely made publication in the *Lancet* because of more general interest in a high-profile murder case; his findings, and diagnosis – death from shock – would have drawn no particular attention from his contemporaries. But, to the modern researcher, they raise a question. Why, despite the decedent's clothing being saturated with blood, and despite the abdomen being soiled with the contents of the perforated stomach, did Mr. Semple settle on "shock" as his final diagnosis? Why not hemorrhage, or peritonitis? What about this particular case produced this decision?

In the context of 19th-century medical literature, Semple's interpretation of the Daly murder is not unique. Other case reports draw the same conclusions, and raise the same questions. Take, for instance, an article penned by Richard Corwin, a resident physician at St. Luke's Hospital, Chicago. He describes the case of "Master E.S., aged fourteen," who attempted to hop a freight train by running alongside and hoisting himself into a car. He fell instead, and was dragged under the train and run over by six cars before being pulled free. His injuries sound devastating, commensurate with the mechanism: the right leg crushed, the "frontal, parietal and superior portion of the occipital bones were fractured in many directions" with multiple scalp lacerations, through which "a greater or less amount of brain matter had escaped." What brain remained in the skull was begrimed

[5] R.H. Semple, "Instant death from the shock of a bullet," *Lancet* 1841-1842 (2):250.
[6] "The murder of the policeman at Highbury," 6.

with cinders, bone shards, and dirt. The child was insensate and unresponsive, and breathing with ragged, shallow breaths. Corwin noted with interest if he elevated one of the depressed pieces of bone off the brain, that the patient's breathing slowed and became more regular. When he allowed it to sink back, labored and shallow breathing resumed. "This I repeatedly did," he reports, "and in each case the effects were the same," remarking that the heart's action never seemed to change despite this maneuver. The patient "resisted the shock" for eighty minutes before succumbing.[7]

Corwin's piece goes on to describe seven similar cases – crush injuries to the legs, arms and torso, degloving injuries of limbs, violent impacts to the head – and concludes these patients all died, not from local effects of injury, or physical disruption of the brain, or hemorrhage, but from shock.[8] In this litany of mutilation, what about shock makes it take precedence over everything else as the cause of death?

Further examples like this abound throughout the 19th- and early 20th-century literature: Walter B. Cannon, investigating battlefield injuries on the Western Front, reports a case of a German prisoner with low blood pressure and "a big hole in his chest" from some projectile and determines that the man suffers not from hemorrhage, or a sucking chest wound, or exposure, but from shock.[9] Numerous authors in the late 19th century relate the story of a King's College janitor who ran afoul of some rowdy undergraduates and died

[7] Richard W. Corwin, "Death by shock," *The Physician and Surgeon* 2 (1880):497-498.
[8] Corwin, "Death by shock," 498-499, 535-536.
[9] Saul Benison, A. Clifford Barger, Elin L. Wolfe, "Walter B. Cannon and the mystery of shock: A study of Anglo-American co-operation in World War I," *Medical History* 35 (1991):224-225.

from shock after they surprised him with a slap across the back of the neck with a wet towel.[10] The British Army medical department reported a case of a soldier in the Transvaal who stood too close to a pony and received a hoof to the chest; crying "I'm done for," he fell to the ground, dead, and the autopsy revealed nothing as to the cause. "The history and post mortem appearances of this case," wrote the surgeon, "apparently point to sudden death, the result of shock to the sympathetic system of nerves paralysing the cardiac plexus, and thus causing cessation of the heart's action."[11] What about these apparently divergent mechanisms of injury – a playful slap, sudden blunt force, high-energy penetrating trauma – causes them to produce death in the same way? How does shock unify them?

To answer these questions, we need to look more deeply into the identity of shock. More specifically, we need to examine what shock meant to the physicians and surgeons who described it. And, what we learn, is that shock – though so commonly discussed, and so seemingly universal and self-evident – changed identities frequently over time, and between authors. Shock, which seems so simple at the outset, was seen as bewildering, intellectually confusing, and poorly understood. The shock of 1842 is not the shock of 1880, or of 1918, and the differences between these iterations cannot be explained simply by assuming an ongoing, evolutionary revision and refinement of ideas about shock. Instead, shock is characterized by successions of ideas that, while holding certain things

[10] A good example is found in T. Lauder Brunton, "On the pathology and treatment of shock and syncope," *The Practitioner* 11 (1873):248.
[11] Arthur Harding, "Report of a case of sudden death the result of a kick by a pony at Pretoria, Transvaal, 14th June 1880," *Army Medical Department Report* (London) 21 (1881):325.

in common, represent deeply different systems of thought about physiology, the response to injury, and of the identity of disease itself.

Sampling narratives of shock in the medical literature reinforces the idea that shock has changed little over the centuries. Hermann Fischer, a surgeon in Breslau, in 1870 published a case report of a young man in shock after a carriage accident: "He lies as we see perfectly quiet, and pays no attention whatever to anything going on around him." His skin was pale, cold, and glistening with perspiration, his countenance apathetic and distant, the racing pulse barely perceptible. He breathed irregularly, sometimes sighing, sometimes breathing not at all; he could answer questions, but only with great effort. His urine had stopped.[12] Compare this with a modern description of shock: "Obtunded and pale, the patient now lies in an ICU bed before you. His condition is poor: his blood pressure is low, pulse rapid and thready, urine output nonexistent."[13] The language may differ, but the clinician's gaze alights upon similar details in each case. The parallels seem obvious.

But, on a closer investigation, the similarities turn out to be quite superficial. Although to the modern medical reader it is tempting to pick out the clinical features of Fischer's patient and concur with the diagnosis, a hundred years removed, of "shock," such an

[12] Brunton, "On the pathology and treatment of shock and syncope," 243-45; also in Benison, Barger and Wolfe, "Walter B. Cannon," 218-9.
[13] David C. Elliott, "An evaluation of the end points of resuscitation," *Journal of the American College of Surgeons* 187 (1998):536. Although the patient described here is hypothetical, it represents a sort of Platonic ideal of the presentation of shock that any surgeon would immediately recognize.

exercise (no matter how "correct" the diagnosis would actually be) would represent the imposition of a modern framework of thought onto a much different world. The underlying epistemological structures are so remarkably different from one another that the two conditions – though on the surface so tantalizingly similar – are in no way interchangeable.

One could argue that 19th-century surgeons simply looked at different parameters to define shock; that beneath their qualitative, bedside assessments and speculative theories lie the kernels which would form the modern understanding of shock. A review of the literature does not bear this out. The classic view of the past 200 years of clinical medicine is one of increasing reduction and precision, from Morgagni's organ-level seat of disease to Bichat's textures to Virchow's cells to the 20th century's molecular biology and epigenetics. But, while the modern definition of shock involves pathology at a subcellular level, a metabolic derangement at whose heart lies an imbalance of oxygen supply and demand, the practitioner of a century before found in shock a quandary: Shock could be clinically described, yet it failed to produce the necessary histopathological changes that would have allowed it to be assigned a proper diagnosis. Shock was enigmatic, mysterious, elusive, and frustrating – a common, easily diagnosed clinical condition that was capable of extinguishing life yet which couldn't be properly categorized. To define shock, to understand it in some rational way, to treat it, practitioners were forced to either rely on older notions of the body's function – often through vitalist-sounding concepts of life force, sympathy, and the animal economy – or

posit novel physiologic models, wherein the mind, the nerves, and the heart were enmeshed in a reciprocal relationship, where thoughts and emotions could alter the body's physiology as readily and as profoundly as a bullet, where powerful sensory impressions could permanently scar the brain and snuff out life itself. Even after the turn of the 20th century, the shock paradigm remained one of nervous dysregulation; a conception which remained fixed, independent of changing medical technology. As medical thought became more quantitative – as chemical analysis and blood pressure measurements replaced references to "high-colored urine" and "thready pulses" – and biochemically oriented, shock continued to command respect as a primary pathology of the nervous system leading to a general failing of the powers of life. At a conceptual level – at an intellectual level, at a history of ideas level – this shock has virtually nothing in common with the shock of modern medicine. Some critical shift in thinking had to occur to separate these two different phenomena.

The goal of this study is to try and understand the nature of this change; to investigate how the construction of "shock" changes from the early writings on the subject at the end of the 18th century through the period around World War I. Ultimately, the changing identity of shock represented changes in an intersecting network of social concepts, medical practices, and professional needs; blending, in the words of Jacalyn Duffin, "ideas about the illness and ideas about the people who are likely to suffer from it."[14] The changing meanings and definitions of shock reflect not only changes in physiologic and

[14] Jacalyn Duffin, *Lovers and Livers: Disease Concepts in History* (Toronto: University of Toronto Press, 2005), p. 8.

pathologic theories, but also offer clues into the intersection of social norms and professional values.

Why shock?

In *The Rise of Causal Concepts of Disease*, K. Codell Carter discusses how the persistence of language structures in medicine contributes to an artificial sense of continuity in medical thought. "What appears to be an essentially scientific vocabulary may cloak beliefs, concepts, and objectives totally alien from our own," he writes. "In the early nineteenth century, physicians spoke so much as we now speak that we see continuity where there was fracture and we overlook strands of their language that bind them inextricably to other systems of thought."[15]

Shock, as a disease that engendered so much confusion, concern, and debate among practitioners in the 19th and 20th centuries, represents a useful case study for investigating these other systems of medical thought. T.S. Kuhn, Gaston Bachelard, and Georges Canguilhem all argued that science produces objects and does not merely describe them, or in other words that pre-existing theoretical structures inform and shape the experience and interpretation of any scientific data. Michel Foucault referred to this as the "interpretive grid" – an internalized heuristic device, through which anything perceived by an observer is interpreted and organized according to the given observer's

[15] K. Codell Carter, *The Rise of Causal Concepts of Disease* (London: Ashgrove), 22.

preconceptions; in other words, the mental filter through which received data are fit into the observer's paradigm.[16] Facts without a paradigm, in Kuhn's terms, are "*mere* facts, unrelated and unrelatable" to any given situation, bereft of significance or applicability.[17] Meaning is not inherent to the array of signs and symptoms displayed in shock; rather, for these phenomena to be understood, to be shaped into what was called shock, meaning had to be imposed. And, by looking at these various meanings and interpretations of very similar clinical data – the clinician's approach to the shock patient – we can in turn try to understand something of the changes in the intellectual apparatus of medicine. We can, in Kuhn's terms, attempt to understand medical thought through its paradigmatic organization; or in Foucauldian terms, we can learn something about the "clinician's gaze."[18]

Shock as an entity represents the opportunity to investigate how medicine responds to a source of intellectual tension. To the 19th-century clinician, shock was at once both an easily diagnosed phenomenon that any medical man should instantly recognize, and a quandary, a confusing, vague collection of symptoms that seemed to belong simultaneously to many disorders. It was both ubiquitous and out of reach; something you knew when you saw it, but couldn't quite define. No small amount of 19th century

[16] Gary Gutting, *Michel Foucault's Archaeology of Scientific Reason* (Cambridge: Cambridge University Press, 1989), 136; Michel Foucault, *The Birth of the Clinic: An Archaeology of Medical Perception* translated by A.M. Sheridan Smith (New York: Vintage, 1994), 137; Thomas S. Kuhn, *The Structure of Scientific Revolutions* (Chicago: University of Chicago Press, 1970) 2nd edition, 35-39; Georges Canguilhem, *On the Normal and the Pathological* translated by Carolyn R. Fawcett and edited by Robert S. Cohen (Dordrecht, Holland; Boston: Reidel, 1978).
[17] Kuhn, *Structure of Scientific Revolutions*, 35.
[18] Foucault, *The Birth of the Clinic*, 88-91.

medical literature lamented the nonspecific use of the term "shock" and its application across wide spectra of problems with no real concern for establishing a "true" definition. The source of the problem with establishing this definition, in part, can be traced to the fact that in a time of materialist, solidist medicine shock remained elusive, producing no consistent post-mortem changes that could have been used to differentiate it from things like inflammation, delirium, brain injury, drunkenness, syncope, and the like. Shock had an essence, but no seat and in a sense, then, was something of a holdover – an 18th-century disease persisting within a 19th-century medical epistemology. This in turn challenged the materialist disease paradigm, and physicians struggled to explain it. Shock in the 19th century emerged as a functional, physiologic disease in an era of structural pathology, with medical consensus gradually establishing the definition of shock as a collection of stereotyped clinical findings associated with traumatic events and the absence of pathological findings upon dissection – almost making it a disease identified by its lack of identity.

From a broader perspective, shock is important in terms of how its presence affected medical practice. Fear of shock influenced many decisions on operation – how to operate, whether to use anesthesia, how radical an operation to attempt, when to operate after injury – and discussions of how to avoid and manage shock filled the surgical literature of the 19th century. Although most writers claim shock's entrance into the medical parlance occurred in the mid-18th century, there is a period of relative quiescence on the subject until the mid-19th century, when medical interest in shock suddenly took off; a

phenomenon both remarked upon in the medical literature and reflected in the steady increase over time in the number of publications related to the condition.[19] One rough – and admittedly nonscientific – quantitative means of corroborating this impression is via the holdings of the U.S. Surgeon-General's library. In the library's first catalogue series, covering the years 1880-1895 (but incorporating titles dating back to the 15th century), 211 articles and monographs are referenced to "shock." The second series, for 1896 through 1916, cites 525 works; the third series (1918-1932) at least 1,000.[20] The ascent of shock in the medical-surgical consciousness of the late 19th century was related to contemporary changes in the interrelation of society, science, and medical practice. A means of considering plausible explanations within this framework can be – somewhat arbitrarily – conceived of in quantitative and qualitative terms.

One argument is that perhaps the apparent spike in interest was an artifact, and that something purely quantitative was at work: More articles were published on shock simply through the growth of medical journalism and publication as the century progressed.[21] While this doubtless played a role, it doesn't account for the tenor of the articles themselves. Although medical authors tended to write about shock as though it has been with us since time immemorial, shock was widely treated by authors as a poorly

[19] Guy C. Kinnaman, "An experimental research into the temperature relationship existing in shock," *Annals of Surgery* 1903 38:846.
[20] National Library of Medicine, *Index Catalogue of the Library of the Surgeon-General, United States Army* series 1-3; accessed on-line 16 April 2008 at indexcat.nlm.nih.gov.
[21] See Thomas H. Broman, *The Transformation of German Academic Medicine* (Cambridge: Cambridge University Press, 1996), p. 85; Broman describes the creation of a public sphere for medical knowledge particularly through the medium of medical periodicals, publication of which blossomed at the end of the 1700s.

understood subject. A common rhetorical device was to open one's arguments by first decrying the apparent disinterest paid to shock by generations past: In the words of one author, shock was "a subject of the highest importance to the surgeon, and yet one that has been generally avoided by surgical writers."[22] Edwin Morris, a London surgeon, had similar sentiments in the introduction of his treatise on shock and railway accidents. "*Shock,* in a surgical sense, involves much that is interesting to the practical surgeon, and demands his whole consideration," he wrote. "In the whole range of surgical literature," however, "little notice has hitherto been taken of the subject, and then only incidentally."[23] Morris lamented the indiscriminate use to which the term "shock" was put by both physicians and the public, citing specifically the free use of phrases like "the shock of operation" and "sustaining a great shock." He concluded that shock, therefore, was poorly understood: "The above are fair examples of the manner in which *shock* is alluded to by surgical writers generally, in the present day: no explanation, no detail of what *shock* is, and the reader is left to draw his own conclusion, and form his own opinion relative to the condition of the system meant by the mystical term *shock.*"[24] There was a sense of deficiency, a gap in medical knowledge waiting to be addressed that makes this seem like less an issue of bystander effect – i.e., that more articles were written on shock because more articles were written overall – than the rise of a focused, increased interest in shock itself.

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[22] E.H. Woolsey, "Report on surgery," *Transactions of the Medical Society of the State of California* 1878-1879:64.
[23] Edwin Morris, *A Practical Treatise on Shock After Surgical Operations and Injuries; With Especial Reference to Shock Caused by Railway Accidents* (London: Robert Hardwicke, 1867), 1.
[24] Morris, *A Practical Treatise on Shock*, 6-7.

Given that the one generally agreed-upon tenet of shock was that it was caused by injury, another quantitative assessment could be that the demographic changes of the 19th century – industrialization and urbanization – allowed shock to become more of a medical issue simply based on greater exposure of a susceptible urban population to a hazardous environment. A larger number of injured patients would translate into a larger number of potential cases that could receive a diagnosis of shock. (Such a model would be analogous to the growth of medical interest in hospitalism in the 1850s and '60s.) Clearly, the city offered numerous opportunities for injury to those individuals involved in the dangerous work of industrial labor, and the urban streets exposed city dwellers to the risks of accident and violence. For example, New York surgeon Lewis Stimson, in a widely cited article on gunshot wounds of the abdomen, remarked that "the possession of parlor rifles and of pistols of small and medium caliber in all classes of the community has become very common," and that this free availability of firearms – combined with people's passions, anger, and poor judgment – was leading to an increasing frequency of gunshot injuries presenting in civilian surgical practice.[25] Along with urbanization and industrialization also came mechanized transportation, whose steamboats, streetcars, and trains were seen as prime sources of shock-producing injury.[26] The railway in particular, was well-represented in the shock literature, and shock was referred to as "one of the

[25] Lewis A. Stimson, "On gunshot wounds of the abdomen, with especial reference to wounds of the intestines," *The New York Medical Journal* 50 (1889):449.
[26] H. St. George Hopkins, "Shock—What is it?" *Pacific Medical and Surgical Journal* 24 (1881-2):206.

most dangerous and difficult injuries known to railway surgery."[27] Railroad injuries were sudden, violent, and emotionally traumatizing, hence the shock they produced was seen as particularly severe; it is rare to find a 19th-century review piece about shock that doesn't at least make a nod to the railroads.[28]

To assume a simple, direct correlation between patient numbers and professional interest seems tenuous, though. Far more important, and far better-documented, are two qualitative changes in medical thought and practice that are associated with the late 19th century. First, this period was marked by a rising interest in functional – that is, non-structural – causes of disease. States previously deemed to be behavioral traits or moral failings – drunkenness, thievery, laziness – acquired medical connotations – inebriety, kleptomania, chlorosis.[29] The idea that the physical capacity of the body to function could somehow be exhausted and a disease state made manifest in the absence of any pathological findings gained greater acceptance, as evidenced by the appearance (and popularity) of neurasthenia in the medical canon. A disorder which, according to George M. Beard, caused more suffering than death itself, and ran "in families more demonstrably than scrofula, or cancer, or consumption," neurasthenia produced a protean

[27] George Brannan, "Diagnosis and treatment of shock," *Fort Wayne Journal of the Medical Sciences* 8 (1888):258.
[28] Furneaux Jordan, "The Hastings Prize Essay, 1866, on Shock after Surgical Operations and Injuries," *British Medical Journal* 1 (1867):222; P.C. Little, "Railway and other accidents: Cases and observations," *British Medical Journal* 2 (1869):530.
[29] Bert Hansen, "American physicians' 'discovery' of homosexuals, 1880-1900: A new diagnosis in a changing society," in Charles E. Rosenberg, Janet Golden, editors, *Framing Disease: Studies in Cultural History* (New Brunswick, NJ: Rutgers University Press, 1992), 106; Barbara Sicherman, "The uses of a diagnosis: Doctors, Patients, and Neurasthenia," in Judith Walzer Leavitt and Ronald L Numbers, editors, *Sickness and Health in America* (2nd ed.) (Madison, WI: University of Wisconsin Press, 1985) 22-35.

array of symptoms yet left no physical evidence of its passing in the cells and tissues of its victims. Instead, a depletion of the nervous energy of the body caused the disease's effects. Well-developed clinical skills were deemed necessary to pick the subtle findings of neurasthenia out from the tangle of a suffering patient's complaints.[30] The parallels between neurasthenia and shock are striking – both were common diseases with potentially grave results (death from shock, insanity from neurasthenia), mediated by a failing of nervous energy, and whose true identities could be seen only in the living patient at the bedside, not in the corpse at autopsy.

Second, the 1800s were a period during which the care of injured urban patients – often poor, single, and socially displaced individuals – increasingly fell to the hospital, an ancient institution which in the 19th century transitioned, as Morris Vogel put it, from a center for the care of the poor to a center for the care of the sick. The hospital also became a place of knowledge production, shaped by, yet also reciprocally molding, an emerging form of medicine affiliated with technology, education, and clinical and scientific research. The close relationship of universities and medical schools to teaching hospitals brought practical medicine into more close apposition with changes in scientific thought. The hospital acted as the point of interface between the injured patient and

[30] George M. Beard, *A Practical Treatise on Nervous Exhaustion (Neurasthenia); Its Symptoms, Nature, Sequence, Treatment*, 5th edition, edited and with additions by A.D. Rockwell (New York: E.B. Treat and Company, 1905; reprint New York: Kraus, 1971), 24-25.

physiologic theory, through the medium of medical and surgical practice; thus, the hospital became what Foucault would describe as a "surface of emergence" for shock.[31]

Hospital care made contact between the wounded patient and one of the beneficiaries of hospital medicine – the surgeon – more likely; and the importance of shock to the increasingly powerful surgical profession likely influenced its prominence in the literature. From the standpoint of professional authority, surgeons needed to control how shock was defined – they had to portray themselves as not only able to understand shock, but also as able to recognize, prevent, and treat it. The reason for this was practical: Surgeons, by the nature of their practice, were etiologic agents of shock. The introduction of volatile anesthetics and the developing consciousness regarding the need for antiseptic or aseptic precautions had brought surgical practice into the hospital, where the profession experienced a rapid, steady growth in its breadth, scope, and prominence. Not only were surgeons attempting (often to the horror of older surgeons) operations which would have been inconceivable a generation before, the sheer number of capital operations being performed rose dramatically.[32] Operations were simultaneously the base upon which surgery built its power and the source of its greatest danger, since surgical operations were considered prime causes of shock; the fear of shock thus became the axis around which revolved debates over the propriety of extending the reach of surgical

[31] See Morris J. Vogel, *The Invention of the Modern Hospital: Boston 1870-1930* (Chicago: University of Chicago Press, 1980), particularly 1-96; Paul Starr, *The Social Transformation of American Medicine* (United States: Basic Books, 1982), 145-179; Michel Foucault, *The Archaeology of Knowledge*, translated by A.M. Sheridan Smith (New York: Pantheon, 1972), 41.

[32] A brief review can be found in W.F. Bynum, "The rise of science in medicine, 1850-1913," in W.F. Bynum, Anne Hardy, Stephen Jacyna, Christopher Lawrence, E.M. Tansey, *The Western Medical Tradition 1800 to 2000* (Cambridge: Cambridge University Press, 2006), 155-160.

therapy. In the United States, at least, these debates were influenced in part by fear of malpractice suits – since surgeons there could be criminally prosecuted if patients died following an operation, shock took on considerable importance to the practitioner.[33]

One professional use of shock was to bolster calls for surgical restraint. More conservative surgeons warned of the mortal danger associated with surgical intervention, deeming many new operations "vicious, and unnecessary," and reminding their more adventurous peers that "shock follows all serious disturbances of the abdominal cavity," particularly handling of the bowel or incision of the peritoneum.[34] Pursuit of technical virtuosity for its own sake was seen as the mark of inexperience: "The young surgeon, by exercise in anatomy, acquires a workman's feeling of his subject, that every thing may be accomplished by dexterity; and he engages in operations which the powers of life cannot sustain."[35] Furthermore, the adoption of Listerism and the ready use of anesthesia were vilified for making surgeons too adventurous: Control of pain and infection allowed surgeons to operate on more and more inaccessible parts of the body, detractors argued, and gave them license to take longer in operating. One late 19th-century British surgeon, lamenting the days before Lister, opined in the *British Medical Journal* that the "exaggerated slowness of modern surgery" with its "prolonged cutting, pinching, and dissecting," and its "chilling of the vital heat" from a combination of anesthesia, exposure

[33] R.E. Bulander, "'A sharp knife and a clean pair of hands': Surgical debates on the role of laparotomy, 1880 to 1900," *Journal of the American College of Surgeons* 204 (2007):501-503.
[34] T.H. Manley, "The surgery of the abdomen with some of its responsibilities," *Journal of the American Medical Association* 15 (1890):701-704, 737-742; quoted in Bulander, "A sharp knife," 499.
[35] Charles Bell, "I. On the powers of life to sustain surgical operations, the effects of violence in wounds and in operations – and the causes of sudden death during surgical operations in some remarkable instances," *Practical Essays* 8 (Edinburgh: Maclachlan, Stewart, 1841), 2.

of the patient's skin, and "antiseptic irrigations, to vapour douches of similar agents, to applications of cloths wet in corrosive or carbolic solutions around the site of the operation" were all contributing to a greater risk of shock for the surgical patient than in the days when operations took minutes rather than hours.[36]

However, most surgical writers found that it was far preferable to define shock in such a way that it was preventable, treatable, or outside of their control altogether. For example, if the pain and suffering of operation were defined as etiologic factors in shock, then the surgeon who gave anesthesia could prevent, rather than induce, its development: James Paget remarked that "saving patients from the alarm and dread of pain" prevents what are "probably the chief causes of the fatal shock."[37] Some surgeons argued that the shock of operation actually encouraged healing: "It must not be supposed that shock is always hurtful, for at times and under certain circumstances it exerts a beneficial effect by inducing an increase of excitement throughout the system, which is called reaction."[38] Paradoxically, shock could also be constructed in such a way that it conferred protection against itself; with the minor shock of an injury rendering a patient refractory to further stimulus, and thus preventing onset of worsening shock from the subsequent operation.[39] Good patient care by a skillful surgeon was framed as the key to warding off shock:

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[36] Christopher Heath, "The clinical characteristics of tumours, and the shock caused by surgical operations," *The British Medical Journal* 1(1889):233-234.
[37] C.W. Mansell-Moullin, "Shock," in John Ashhurst (ed.), *The International Encyclopaedia of Surgery: A Systematic Treatise on the Theory and Practice of Surgery by Authors of Various Nations* (New York: William Wood and Company, 1881), 374; James Paget, "The address in surgery" (read at the 30th annual meeting of the British Medical Association), *British Medical Journal* 2 (1862):158.
[38] George McClellan, "Clinical lecture on shock and surgical fever," *Philadelphia Medical Times* 13 (1882-3):816.
[39] Jordan, "On shock," 222.

"Rough treatment and want of consideration for the feelings of the patient have a great deal to do with the causation of shock," wrote George McClellan in 1882, claiming that through a gentle, confident manner and careful, delicate operation the patient would "be less likely to suffer from shock."[40] If shock did occur, surgeons claimed a particular capacity for dealing with it: "The surgeon who can manage a patient when suffering from a surgical injury accompanied by shock, is indeed worthy of the name surgeon," announced one surgeon at a regional conference.[41] One of his contemporaries agreed, writing that shock "requires the immediate presence and best efforts of the most skillful surgeon in order to secure a reaction of the nervous system."[42] If the surgeons' attempts at prevention and treatment failed, surgeons took pains to clarify that deaths *after* operation were not the same as deaths *from* operation, and concluded not only that postoperative death from shock was rare, but that individual temperament played a considerable role in its development.[43] The physical and moral state of the patient was, after all, out of the surgeon's hands: "Men who have pursued convivial or intemperate habits for a long period, addicted to dissipation and late hours, have their nerves so weakened, and all the solids and fluids of the body so depraved and disorganized, that traumatism wins an easy victory."[44] Rather than accepting the mantle of being the causative agents of shock, forward-looking surgeons styled themselves the only ones competent at combating it;

[40] McClellan, "Clinical lecture on shock and surgical fever," 812.
[41] A. Rhu, "Shock," *Fort Wayne Journal of the Medical Sciences* 7 (1887):81.
[42] Brannan, "Diagnosis and treatment of shock," 258; also see Paget, "The address in surgery," 158.
[43] Paget, "The address in surgery," 157-158.
[44] E.T. Easley, "A study of shock," *Richmond and Louisville Medical Journal* 26 (1878):421.

instead of cowing surgeons into inactivity, shock could be used to gain them a measure of professional authority.

Shock in history

Despite its importance to physicians and surgeons, and to medical and surgical practice, shock has not received much study in non-medical historical literature. The two major works that address it – English's *Shock, Physiologic Surgery, and George Washington Crile* and Benison's "Walter Bradford Cannon and the mystery of shock" – focus less on examining the changing epistemological structures that come to define shock than on the pioneer spirit of individual researchers in contributing to the understanding of shock. There's a subtle difference in focus than what I aim for here: Shock, in these narratives, is something to be discovered rather than constructed, something unearthed bit-by-bit and put together rather than designed, a question waiting for someone to come along with the right answer.

Why has so little attention been paid to shock by non-clinical medical historians? I would posit the chief reason is that over the last four decades, medical history has trended toward the production of social, rather than intellectual, histories. To try to take apart shock at the level of its epistemological building blocks is to delve into a topic that at times becomes rather esoteric – filled with talk of pulses and pallor, reflexes and nerves, power and vitality – and theoretical. Shock as a disease lacks a compelling connection to

issues of medical disparity or injustice. Unlike breast cancer, shock is difficult to imbue with a narrative of the role of gender differences in disease management; unlike HIV/AIDS there is no hunt for a discrete etiologic agent buried under governmental pressures to vilify the disease's sufferers; unlike the isolation of insulin in diabetes there is no single, definable breakpoint that changes understanding of the disease.

Furthermore, shock carries with it a certain sense of immutability; an assumption that shock, like "cancer," or "diabetes," or any other condition denominated by a term passed to us from antiquity, is itself somehow fundamental and necessary to the human condition. The phrase "he was in shock" carries an almost self-evident meaning in a way that, say, "he developed acute interstitial nephritis" does not; that some condition of "shock" clearly exists and has always existed is a difficult idea to relinquish. As we've discussed above, the work of previous clinicians makes 19th-century descriptions of shock sufficiently present and familiar that a degree of continuity seems difficult to refute.

Essentially all other histories of shock in the literature are clinical in origin, written by and for a medical audience. They are positivist in tone, and in general consist of chronological lists that stress themes of discovery, accretion of fact, and production of finished "correct" versions of shock. These histories follow the same general pattern. Virtually all start their enquiry with Henri François LeDran, whose 1743 *Treatise or Reflections, Drawn from Practice on Gun-Shot Wounds* is broadly credited with

introducing the term "shock" to the medical literature: "The whole animal Machine participates more or less in the Shock and Agitation which is communicated to the Part, the Moment it is struck."[45] (As an interesting linguistic aside, "shock" was the English translation of LeDran's terms *secousse*, literally translated as "jarring," and *ébranlement*, literally "shaking";[46] however, by the end of the 19th century, French writers were transliterating the English "shock" into *choc*, as a synonym for what they in turn had taken to calling *commotion* or *stupeur*.[47]) Trauma was the key element here. Violence, either physical or psychical, was necessary, as it produced the jarring, the commotion, the force – the shock, if you will – that could derange the workings of the animal machine and snuff out the powers of life.

The mechanical, tactile physicality of LeDran's shock became far more intangible as the 19th century proceeded. The shock paradigm became one of nerves: In the words of Peter English, "Nearly every clinical article on shock in the nineteenth century proposed a faulty nervous system."[48] Rather than a simple brute force that overwhelmed the animal economy, shock acquired a connection to reflexes; it became a phenomenon of

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[45] Henry Francis LeDran, *Treatise or Reflections, Drawn From Practice on Gun-Shot Wounds*, translated anonymously "from the French original" (London: John Clarke under the Royal Exchange Cornhill, 1743), 1-2. While LeDran is certainly not the first author to discuss the notion of shock, his treatise is almost universally cited by authors in the 19th century, making it a useful common starting point.
[46] Robert M Hardaway, "Wound shock: A history of its study and treatment by military surgeons," *Military Medicine* (2004) 169:265; Peter C. English. *Shock, Physiological Surgery, and George Washington Crile: Medical Innovation in the Progressive Era*. (Westport, CT: Greenwood Press, 1980), 3.
[47] See, for example, M.L. de Santi, "Du choc traumatique et de la stupeur locale, principalement dans les plaies d'armes a feu," *Archives de Médicine et de Pharmacie Militaires* (1883) 2:225. de Santi also cites LeDran as performing the first study on shock.
[48] Peter C. English, *Shock, Physiological Surgery, and George Washington Crile: Medical Innovation in the Progressive* (Westport, CT: Greenwood, 1980), 10.

physiologic over-reaction. Building on an example published by Leopold Goltz – who described experiments wherein he struck frogs on the belly and found their hearts either slowed or stopped as a result – the human circulatory system was conceptualized as a fragile network which could collapse when its associated nerves were strained in some way.[49] Through the web of reflexes, injury produced a deep depression, either of some central nervous force itself, or of the parts of the brain and spinal cord that control the smooth muscle of the vascular system.

George Washington Crile stands out in most reviews as the next major participant in the shock story, around the turn of the 20th century. Crile is generally credited with re-defining shock in terms of quantifiable parameters, specifically low arterial blood pressure.[50] He posited a loss of arterial tension through the exhaustion of the vasomotor center – which controlled the degree of constriction or relaxation of major blood vessels – of the central nervous system, with subsequent pooling of blood in the veins, leading to insufficient filling of the heart and circulatory collapse. As an extension of his theories, Crile developed what he called "anoci-association" as a means of preventing surgical shock. The system used a cocktail of general anesthesia (usually nitrous oxide), scopolamine (to induce amnesia), morphine (to relieve pain and anxiety), and cocaine or other local anesthetic in the operative field to completely block all nervous impulses from the surgical site, consciously perceived or not, from reaching the brain, or from being

[49] Goltz is discussed frequently in the primary literature of the time, in secondary review literature see Fielding H. Garrison, *History of Medicine* (4th ed.) (Philadelphia: W.B. Saunders, 1929), 540.
[50] English, *Shock*, 88-90; Alan P Thal, E. B. Brown, Arlo S. Hermreck, Hugh H. Bell, *Shock: A Physiologic Basis for Treatment* (Chicago: Year Book Medical Publishers, 1971), 19.

processed there.[51] Crile reasoned that if the brain was fully unaware of the stimulus of injury there could be no reflex reaction to the injury, thus no fatigue of the vasomotor centers, no circulatory collapse, and no shock.

Other researchers adopted the hypotension model, though they quibbled over the underlying physiologic mechanisms. Crile's vasomotor exhaustion argument was met by counter-theories: the nervous system was not exhausted but rather thrown into imbalance between excitatory and inhibitory states; the vessels dilated not as a neurologic phenomenon but in response to some chemical change caused by injury, like the release of histamine or the development of acidosis; plasma leaked into tissues and led to emptying of the vascular tree in this manner.[52] Ultimately, the work of Walter Cannon emerged from the early 20th century as the leading shock model, and after this point the descriptions of shock become almost purely circulatory: Injury produced an alteration of the circulation, which caused low blood pressure and led to failure in cellular metabolism, accumulation of acids in the blood (now seen as a byproduct of shock, rather than its cause), and onset of shock, which could be treated with administration of intravenous solutions of sodium bicarbonate.[53] Arterial hypotension with resultant failure of the circulation to deliver oxygen to cells was seen as the key element of shock by World War II, and by the 1950s, definitions of shock had assumed something like their present form: Shock involved "peripheral circulatory failure," which could result from

[51] English, *Shock*, 165.
[52] English, *Shock*, 130-140; Thal, et. al., Shock, 19-20; Carl J. Wiggers, *Physiology of Shock* (New York: Commonwealth Fund, 1950), 8-13.
[53] Benison, Barger and Wolfe, "Walter B. Cannon," 220-234.

any number of diverse etiologic processes – loss of blood or plasma volume, loss of vascular tone, loss of cardiac power – which led to cellular anoxia and its resultant metabolic dysfunction as the final common pathway of injury.[54]

Continuity or discontinuity?

Foucault described the traditional historian's mission as one of constructing stable structures beneath the chaos and change on history's surface, of building connections between events, of organizing the past into an analytical, structural framework.[55] By this definition, the extant history of shock in the literature is a traditional history: We see a gradual progression of an idea through time, changing subtly but retaining its identity through what on closer investigation are periods of theoretical incompatibility.

A key feature in these histories of shock is the incorporation of modern definitions of shock into older writings, anachronistically drawing those conditions recognized as "shock" in the modern sense into the narrative. Evidence of this practice is seen in the inclusion in histories of shock of work done by O'Shaugnessy and Latta in 1830s on cholera, or by Fagge in the 1870s on diabetic coma. These men took care of patients *in extremis* from terrible dehydration – the cholerics through intractable diarrhea, the

[54] Robert M Hardaway, "Wound shock: A history of its study and treatment by military surgeons," *Military Medicine* (2004) 169:268; HA Davis, *Shock and Allied Forms of Failure of the Circulation* (New York: Grune and Stratton, 1949), 5-8; Alfred Blalock, "Reminiscence: Shock after thirty-four years," *Review of Surgery* (1964) 21:233-234.
[55] Foucault, *The Archaeology of Knowledge*, 5-7.

diabetics from uncontrolled urination. Latta cited malignant cholera as leading to a "hopeless state of collapse," in which "there is a very great deficiency both of the water and saline matter of the blood."[56] Fagge likewise related the polyuria of diabetes to the flux of cholera: "What suggested to my mind the advisability of injecting a saline solution into the blood in this case was the idea that the coma was due to the drain of water from the system, caused by the diabetes. I suppose that the hypothesis upon which I acted was essentially similar to that which formed the basis of the like treatment in the collapse of cholera."[57] All three men replenished their patients' desiccated bodies with infusions of saline solution, either given into the gastrointestinal tract (from above or below) or injected under the skin or into a vein; all reported success exceeding their "most sanguine anticipations," with moribund patients awakening, communicating, and behaving like their old selves again.[58] Yet, none ever mentioned shock. Modern writers gloss this over as a mistake, a conceptual misstep. Such dehydration now falls under the rubric of hypovolemic shock, a failing of the circulation as a result of insufficient blood to fill the vascular tree; a condition treated – just like Latta and O'Shaughnessy and Fagge did – with intravenous injection of saline fluids. In other words, to the modern eye these three *were* treating shock, they just didn't know it.

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[56] Thomas Latta, "Malignant Cholera: Documents communicated by the Central Board of Health, London, relative to the treatment of cholera by the copious injection of aqueous and saline fluid into the veins," *Lancet* (1831-1832) 2:274-275; see also an appended note from W.B. O'Shaughnessy, quoted on p. 281 of this article.

[57] C. Hilton Fagge, "A case of diabetic coma, treated with partial success by the injection of a saline solution into the blood," *Guy's Hosiptal Reports* (1874) 19:177.

[58] W.B. O'Shaughnessy, "Note from Dr. O'Shaughnessy: Proposal to combine Stimulants and Astringents with the Aqueous and Saline Injections," appended to Latta, "Malignant Cholera," *Lancet* (1831-1832) 2:281.

Regardless of the fact that Latta's patients suffered from modern hypovolemic shock, this approach exemplifies Kuhn's "invisibility" argument, wherein past scientific work is implicitly treated as having the same methods, worldview, and goals of the current ruling paradigm (hence older paradigms become invisible, by being subsumed and absorbed into the current version). For example, a 1940s textbook on shock reported in its prefatory remarks that, among the early commenters on "shock and shocklike states," were "O'Shaughnessy (1831) [who] described the presence of severe circulatory failure in patients suffering from cholera," and "Fagge (1874) [who] noted its presence in diabetic coma."[59] Another review volume from 1971 also remarks that Latta's 1831 essay "describing his treatment of the hypovolemic state in cholera is a classic in therapeutics."[60] A substitution of ideas has taken place here: The 20th-century diagnosis of shock (as circulatory failure, or as hypovolemia) has been made and retrospectively imposed upon the sources. Latta, O'Shaugnessy, and Fagge, who described themselves as treating *blood* which "has lost a large proportion of its water" and "a great proportion of its neutral saline ingredients," are repurposed by later writers, and considered to be treating *shock*.[61] To the 19th-century practitioner, these are not equivalent tasks; in the retrograde approach, they are. By modern definition, patients with profound fluid loss develop shock, and replacement of their intravascular volume is the treatment. These historical patients had lost fluid and their physicians gave intravascular volume; hence, Latta and Fagge diagnosed and treated shock. Yet, to a surgeon of Latta's time, shock

[59] Davis, *Shock and Allied Forms of Failure of the Circulation*, 1
[60] Thal, et al, *Shock*, 17-18.
[61] Quotation is from W.B. O'Shaughnessy, "Experiments on the blood in cholera," *Lancet* 1831-1832 (1):490.

was a completely different entity, relying instead upon an unexplained death, a history of trauma, an association with a set of clinical findings that were interpreted as showing depression of the nervous system.

The work of Crile receives similar treatment. What is perhaps most interesting is that Crile's theories are never interpreted as anything other than a stepping stone into a new era – English's book titles its introductory chapter "Surgical Shock Before Crile," implying the existence of a premodern-modern breakpoint at the work of this individual.[62] "This imaginative investigator and master surgeon conducted pioneering experiments in animals and in a prophetic way defined the importance of venous pressure and the responses to volume loading," wrote one clinical historian.[63] Yet, as we will discuss in detail later, Crile's paradigm of shock was one of nervous collapse; and though he described shock in the language of circulatory insufficiency – low blood pressure – he conceived of shock in terms of a neurologic failing – exhaustion or overstimulation of the nervous system leading to vascular paralysis. That this represents a completely different line of thought from his successors and apparent intellectual heirs is not addressed.

Shock has become, in available histories, what Foucault described as a "unity of discourse": A set of "statements different in form, and dispersed in time," which are posited to "form a group if they refer to one and the same object." Yet such a group is artificial, and trying to define unity around an object like shock "does not enable one to

[62] English, *Shock*, 3.
[63] Thal, et al, *Shock*, 20.

individualize a group of statements, and to establish between them a relation that is both constant and describable."[64] In other words, the shock of LeDran is not the shock of Gross, nor the shock of Crile. Instead of attempting to construct relationships between these disparate versions of shock and thus create a narrative history of progression, our goal here will be to evaluate the changing theories and concepts themselves, to see what they can tell us about the interrelations of society, medical theory, medical practice, and the production of scientific knowledge.

To accomplish this objective, I have relied primarily on medical literature of the late 19th and early 20th centuries. Reading these early descriptions of shock, however, leads the investigator into dodgy linguistic territory – in the late 18th and early 19th centuries, "shock" is often interchangeable with terms like "collapse" and "commotion," terms which themselves are subject to fluid usage and definition between different authors – and this creates a problem for a study of this sort. How does the historian identify shock in the sources, when the sources themselves show no agreement? LeDran, so often credited with giving us the term "shock," never really uses it after the second page of his treatise. He discusses the "sudden Depravation or total Suspension in many of Nature's Operations" that follow profound injury, and goes on to describe it in terms that sound familiar to the context of the later shock literature: the onset of "Numness and Weight" followed by "convulsive Motions, such as Hiccup, Vomiting, irregular Shiverings, or tonick Stiffness," a change in complexion to yellow, green, or leaden, the

[64] Foucault, *Archaeology of Knowledge*, 31-32.

superimposition of syncope. Yet LeDran notes that this state of disrupted animal economy is properly labeled "Commotion."[65] It was left to later commentators to lump his description in with "shock." Further difficulty emerges in later writings, when shock subtypes diverge into both physical and emotional variants: Does what comes to be called "shell-shock" relate, medically speaking, to what remains "traumatic shock"? What about other entities, like "spinal shock" or "railway shock"? At which point are we allowed to divorce the concept from the language?

This issue puts a certain degree of jeopardy into writing a history of shock. One courts the danger of simply constructing an alternate presentist version by applying the term "shock" independently of the sources' use of it; yet at the same time, ignoring everything but the actual term "shock" in the sources runs the risk of ignoring important lines of thought that are relevant to the study of shock. The compromise I hope to reach is to allow the sources to serve as the arbiters of what is and isn't shock; we will treat as valid parts of the shock literature those influences they cite, alternate definitions they propose, and predecessors they claim. For example, even though LeDran didn't write about shock per se, his successors took his description as ground substance for their own version of shock; they internalized it, referenced it, built upon it, and – perhaps most importantly – continued to work within the same paradigm. My hope is that proceeding in this manner will allow a useful analysis of 19th-century shock concepts, without trying to shoehorn

[65] LeDran, *Treatise or Reflections*, 2-5. I have preserved the original text's spelling and capitalization.

other states under the shock label or attempting to construct an artificial, unifying definition.

In the succeeding chapters, we will explore the theories, concepts, and intellectual currents of shock, beginning – as do many reviewers – with the work of Henri Le Dran in 1745. We will use three widely cited monographs on shock – a textbook chapter written by Samuel Gross, a prize-winning research paper by English surgeon John Furneaux Jordan, and an essay by Southern surgeon E.T. Easley – to serve as archetypes in discussing different approaches to the shock problem. We will end with a brief discussion of George Crile, Walter Bradford Cannon, and the changing worldview of shock research at the time of the Great War. Basing our analysis primarily on major review articles and textbook chapters, with contextual cues provided by other pieces from the medical literature, we will attempt to reconstruct what shock meant to successive generations of clinicians. We will assess, then, how changes in the concept of shock reflect changes in medical epistemology.

Chapter 2 – Common ground

While Ambrose Pare in the 16th century described "commotion," a "deathlike state" induced by trauma that resembled what came to be known as shock, many writers attribute the term "shock" to the writings of Henri François Le Dran.[66] The work of Le Dran includes themes that become the ground substance for much of the discussion of shock for a century and a half to follow. Shock is a clinically identifiable disorder that serves the purpose of explaining the effects of injury by describing how something localized, discrete, and apparently self-limiting can become something systemic, global, and terminal. It depends upon the assumption of a holistic view of the human organism; it demands as a prerequisite the idea of the body as a set of inter-related phenomena and responses.

Le Dran was a military surgeon, active in the mid-18th century, who served as the chief consulting surgeon to the soldiers of the French army. He was also the director of the Royal Academy of Surgery in Paris, a fellow of the Royal Society of London, and chief surgeon of the Hôpital de la Charité.[67] In accord with his times, Le Dran espoused a mechanistic physiology, conceiving man as a machine of flesh animated by the breath of life; reminiscent of that described in Descartes' *Treatise of Man*. In Le Dran's framework, the human was envisioned as a holistic system, wherein injury or stimulus to one part of the body produced distant effects through primarily physical means. "It is

[66] Among them Davis, 1; Hardaway, "Wound shock," 265; Thal, et al, , *Shock*, 7; Carl J. Wiggers, *Physiology of Shock* (New York: Commonwealth Fund, 1950), 2.
[67] Henri-François Le Dran, *Consultations sur la pluspart des maladies qui sont du ressort de la chirurgie*. (Paris, p. Fr. Didot le jeune; 1765.)

well known," he wrote, "that all Parts of our Body, are connected by Means of a reticular Substance. This is a Kind of Net-Work that serves as a Canvas, if I may be allowed the Expression, in which all our Vessels are interwoven."[68] Tightness, or constriction, along this body-mesh had two major effects: crimping the blood vessels suspended in the mesh, as if by ligature, and impeding the "stream of animal spirits," free flow of which was necessary to maintain sensibility and motion. Obstruction of the animal spirits, in turn, led to symptoms such as "Numness and Weight," syncope, "convulsive Motions, such as Hiccup, Vomiting, irregular Shiverings, or tonick Stiffness," and change in complexion to yellow, green, or leaden. The severity of the symptoms, in general, paralleled the degree of obstruction.[69]

The chief etiology of this "spasmodick Convulsion" in the meshwork was injury, particularly the violent injury produced by gunshot wounds. "The Bullet, or whatever Body it is, thrown by Gun-powder, acquires such a rapid Force, that the whole animal Machine participates more or less in the Shock and Agitation which is communicated to the Part, the Moment it is struck."[70] This mention of shock, occurring on the first page of the treatise, is the first of two times Le Dran uses the term – in general, "commotion" and "agitation" supplant it – though he continued with a physiologic and clinical discussion that laid the groundwork for future authors' treatments of shock. Specifically, Le Dran

[68] Henry Francis LeDran, *Treatise or Reflections, Drawn From Practice on Gun-Shot Wounds*, anonymously translated "from the French original" (London: John Clarke under the Royal Exchange Cornhill, 1743), 4. I have preserved the source's punctuation, spelling, and capitalization scheme in quoted material.
[69] Ibid., 4-5.
[70] Ibid., 1-2.

addressed the idea of a localized injury becoming a global, systemic phenomenon; he discussed issues of proportionality between injury, symptoms and physiologic derangement; he described a clinical picture that matches well to future writers' descriptions of shock; and he discussed the role of patient temperament and habits as factors in susceptibility to shock.

The key element in Le Dran's physiology was interconnectedness: "Anatomy teaches us, that there is such a Connection and intimate Sympathy betwixt all the Parts of our Body, that they all mutually stand in Need of one another."[71] While allowing the organism to function as a unified whole, this mutual need had disadvantages, for it allowed the whole of the animal machine to be "thrown into universal Disorder, by a Blow received from Fire-Arms upon any one Part alone."[72] The "commotion" of injury, by causing spasms and tightening of the physiologic body-mesh, made the nerves "galled and irritated," communicating the effect of the injury "to the whole Machine." Fear and pain from the local injury further impeded the flow of "nervous Fluid," which in turn affected the heart, disturbing or stopping its action momentarily.[73] If the wound were allowed to suppurate and produce fevers, it could poison the body through its humors: "there being a continual and mutual Communication betwixt the wounded Part and all the rest of the Body, by this Means some of the extravasated Fluids may be conveyed into the Rout of the Circulation, and there disturb that intestine motion which the Author of Nature has imprinted on our

[71] Ibid., 61.
[72] Ibid., 62.
[73] Ibid., 3-5, 62.

Fluids, and on the Nature of which their good or bad Qualities depend."[74] The local area of injury – of tissue destruction, pain, contusion, and hemorrhage – thus imprinted itself upon the body's function as a whole; in other words, local derangements, through sympathy and the impedence of the pneuma, changed the very makeup of the whole body. Echoes of some of these concepts – that a physical change in one part of the body produces a whole-body chemical change, the notion that whole-body irritability could produce pathology if unbalanced, and that fear could influence this process – would be seen repeatedly in the works of later writers.

Le Dran treated hemorrhage in his discussion of injury, though he assigned it no direct causative role in the production of post-injury effects. Loss of blood could worsen the "universal Coldness" caused by contracture of the body-mesh, and the fluid loss associated with hemorrhage could predispose a wounded man to develop a systemic response to injury.[75] Loss of blood into an enclosed space – i.e., bleeding into tissues causing contusion or ecchymosis (bruising) – could increase local tension within a limb or a wound, causing fullness, suppurations, local circulatory failure, and gangrene in the affected part.[76] While he noted that stopping ongoing hemorrhage was necessary, Le Dran advocated bleeding patients suffering from undue tension, instructing surgeons to make incisions to make "a bleeding Wound of that which was a contused one."[77] The

[74] Ibid., 63.
[75] Ibid., 6, 62.
[76] Ibid., 63, 178.
[77] Ibid., 19.

therapeutic goal is clear – reduce tension, allow resumption of free flow within the body framework, and restore the normal state.

Le Dran described the physical changes in a patient suffering from a wound as reflections of tension's effects on the life-force, and alterations of the physical humors through extravasation. The pulse becomes "convulsive and palpitating," either through a scarcity of animal spirits or by their adulteration by blood and tissue fluids: "this Disorder arises in the Heart through a Deficiency of animal Spirits, which cease to flow with their usual Tides: And as it is the first Mover of all the Fluids, the whole Machine must go to Wreck, so soon as it deserts it's [sic] Office."[78] These cardiac alterations – it is noteworthy that the pulse is described in qualitative terms, particularly the energy and efficiency with which it discharges its duty – are complicated by coldness and numbness, the result of "Tension or Crispature, which is a kind of tonick Convulsion, [that] choaks up all the little Vessels." Furthermore, "Circulation is carried still on with Freedom in all the Trunks and larger Branches; but it is suspended in all the capillary Subdivisions of these, as well in the Trunk of the Body as in the Extremities, which is demonstrated by the freezing Coldness that is perceivable there." The patient's limbs and countenance thus become pale and cold, circulatory flow may lock up and cause vessels to burst as blood fails to return to the body's center; the constricted stomach, esophagus, and salivary glands fail to moisten the mouth and terrible thirst develops; restlessness and inquietude set in; and the patient may die: "The Shock with which the Patient sometimes finds

[78] Ibid., 182.

himself, as if it were, thunderstruck at the Time of the Blow, together with the Commotion or Agitation, may be followed by very fatal Consequences."[79]

The severity of these symptoms did not necessarily reflect the severity of a wound in a given patient. Le Dran, like surgeons after him, commented on the nature of gunshot wounds to produce symptoms that did not seem to be consistent with the extent of the initial injury: "All Sorts of Gun-Shot Wounds, whatever be their Degree of Largeness, may be followed with bad Symptoms, which shall not appear till some Days after receiving the Injury."[80] The cause of these symptoms – the aforementioned restlessness, thirst, coldness, feeble pulses – was not necessarily known, Le Dran wrote, beyond the assumption that alterations in the body's tension and circulation of animal spirits were to blame. This sense of a small wound provoking a large response is nearly universal in the shock literature; though the role of proportion in the degree of wounding to its effects is complex and often contradictory, even within individual writings.

Le Dran, for his part, looked to personal temperament and physics as a means of explaining the apparent discrepancy in magnitude between wound and effect. The degree of dysfunction, he argued, was influenced by "a stronger or weaker Habit, a greater or less Disposition in the Fluids toward Inflammation, or to the Nature of the wounded Part, besides many other Circumstances." (Fatigue and pain were two items Le Dran

[79] Ibid., 62, 176, 180-182. Not just fatal, but *very fatal*.
[80] Ibid., 62.

specifically counted among these "other Circumstances.")[81] Personal qualities – in particular *sang-froid* – could also influence a wound's development, as Le Dran noted, since the moment a man realized he was struck with a bullet, he was in danger of being overcome with fear and dread, verging on panic, that could "seize the whole animal Œconomy," and lead to a "sudden Depravation or Suspension in many of Nature's Operations."[82] As important in the process, however (and in accordance with Le Dran's very physical approach), were simple mechanics: "It is scarcely possible that a solid Body driven by Gun-Powder, should strike any Part, without communicating at the same Time an Agitation proportionable to it's [sic] Quantity of Matter, to it's [sic] Velocity and to the Resistance of that Part."[83] The size and speed of the projectile, as well as the nature of the part it struck, could be mathematically related to the disturbance it caused. By the 19th century this interest in injury kinetics would be downplayed in favor of softer theories, which placed more emphasis on the emotional state of the patient than on the physics of the injury. The dichotomy of fear and courage in relation to shock, for example, would receive much greater attention; and socially normative or admirable traits – temperance, courage, restraint, patriotism, heritage – would acquire an almost talismanic protective value against shock.

Le Dran wrote about shock only in the eyes of people who came after: His "shock" was a process of physical dissolution through excessive tension, a situation in which the

[81] Ibid., 2-3.
[82] Ibid., 2-3, 62.
[83] Ibid., 3.

physical effects of injury, through restriction of the flow of animal spirits, affected the nerves, the heart, and the humors and caused whole-body disruption. Le Dran didn't discuss shock as an entity; rather, "shock and alarm" is the inciting event in a process of derangement ("agitation" or "commotion"). This very finite, active sort of connotation persisted throughout the 19th century: Patients will be "shocked" rather than "in shock," patients will *receive* a shock rather than *lapse into* shock. Le Dran finds his way into the shock canon because many of the processes he described – global propagation of a local injury, the involvement of the nervous system, apparent collapse of the organism, a questionable relationship to hemorrhage and syncope, the inexplicability of death in some patients – came to represent a grouping of clinical phenomena that later writers placed under the heading of "shock." As disease definitions passed from a medicine of species to a medicine of pathology, shock would become important in explaining deaths not attributable to any specific pathologic event.

Themes in 19th century shock

The work of Le Dran is informative for a study of shock primarily because it opens the door to common themes that emerge in shock literature in the 19th century. Despite the fact that explanations and conceptualizations of shock are fragmented, discordant, and often contradictory, descriptions of the clinical phenomena attached to the term "shock" change little, whether you find them in Le Dran's 18th-century treatise or in a modern textbook. Independent of the investigator and the period of investigation, themes emerge

that remain fairly constant; as a prolegomenon to the dissenting ideas regarding the how and why of shock, we should first consider the what of it.

In reviewing a century's worth of material, certain major points of concordance emerge. First, most writers treat shock as a product of the human condition – it has a sense of universality and eternality to it, of omnipresence. It could be associated with nearly any forceful stimulus, both physical and mental. Personal characteristics – particularly as they related to emotional state, character, and personal habit – played a large role in the propagation and outcome of shock. Shock was an event rather than a state in the 19th-century literature, a discrete and definable impact upon the animal system, and the processes it initiated in the body were interpreted as pathological and harmful. These processes were expressed in terms of a generalized depression of function – though *which* function, specifically, changed with time – and they presented with characteristic clinical findings. These findings represent the systemic manifestation of a localized stimulus; shock amplifies and propagates a discrete impulse into one more global. Frequently, the proposed mechanism for such a systemic effect was built upon notions of aberrant tension or relaxation – irritability, in a Brunonian sense – frequently of nerves or blood vessels. This assumption of altered irritability was generally accompanied by an idea of systemic reserve, or exhaustibility; shock exceeded the capability of the body to recover.[84] Shock was classically associated with the absence of any unique physical

[84] See Janet Oppenheim, *"Shattered Nerves": Doctors, Patients, and Depression in Victorian England* (Oxford: Oxford University Press, 1991), 81-86, for a discussion of nervous exhaustibility. Particularly interesting in the Victorian context is the use of financial and economic metaphor to describe the function

lesion that could grant it pathologic identity. This, in turn, led to a sort of consensus opinion among writers that shock was mysterious, baffling, and difficult, if not impossible, to characterize effectively. In particular, shock was difficult to sort out from the effects of hemorrhage, with which shock was frequently accompanied. Finally, shock was associated with lethality – it was the pathway through which apparently minor injuries and impulses became fatal.

"As old as the everlasting hills"

In 1912, British surgeon H. Tyrell Gray and physician Leonard Parsons gave a lecture on shock to the Royal College of Surgeons that began with the statement: "The problem of shock is one of the oldest in medicine, and is still certainly the most formidable obstacle to modern surgical endeavour. It is almost as old as the 'everlasting hills'; certainly co-existent with the existence of man."[85] This statement is quite representative of 19th- and early 20th-century shock literature. In what remains a classic and widely used tactic of medical writing, authors of the period who undertook the project of writing up a treatment of shock began with a discourse of its history; invariably, they found they could create a long backstory, going back centuries. A nice example is found in a piece by C.C. Seabrook, a surgeon from Harrisburg, Pennsylvania, who gave a lecture on the pathology of shock to the assembled delegates of the Medical Society of the State of Pennsylvania

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of nervous power; in an era where capitalism and commerce drove the culture, the body's function was visualized as a microcosm of society.

[85] H. Tyrrell Gray and Leonard Parsons, "The Arris and Gale Lectures on the mechanism and treatment of shock," *British Medical Journal* 1912:938.

in 1881. Seabrook was a recent graduate of the University of Pennsylvania medical school – where he had received a share of a hundred-dollar prize for an essay written on his physiological experiments – and was active in his local medical society, serving as the treasurer.[86] In his prefatory remarks, Seabrook drew upon historical example to establish the context of shock as a long-standing human problem. William Clowes, he noted, in 1568 "gave a very excellent description of the symptoms of shock," to which Heister added in 1763, and about thirty years later the Scottish surgeon James Latta applied "the term *shock* … to this state of the system … and to-day this term, with the significance given it by him, is still in use."[87] This last statement is telling, in that Seabrook here drew a direct line between Latta and contemporary theory and practice; the unifying power of the term "shock" transcending the intervening century with its attendant changes in medical *savoir*. Shock is a part of the human condition, assumed to have always been there, unchanged and unchanging; shock is not a part of medical consciousness, changing as the frame upon which it is understood and constructed changes.

While shock had a long past with mankind, it also had a pronounced sense of presence. It was ever at hand, ready to strike: "Every distress, mental or physical," wrote Arkansas surgeon E.T. Easley, "is accompanied by corresponding shock."[88] Its physical causes were manifold: Shock could be produced by "a blow on the abdomen, sometimes even

[86] Anon., *Transactions of the Medical Society of the State of Pennsylvania* 13 (1881):858; Anon., *The Medical Record* 13(1878):239.
[87] C.C. Seabrook, "The pathology of shock," *Transactions of the Medical Society of the State of Pennsylvania at its Annual Session* 13, part 2 (1881):653. It is interesting to note that Seabrook credits Clowes with describing "the symptoms of shock" and not shock itself; this awaits Latta's turn-of-the-century denomination.
[88] Easley, "A study of shock," 518.

when the blow is by no means severe," "injuries to the skull and spine: gunshot, railway, steamboat and factory wounds, dislocations with fractures, violent sprains, burns and scalds, and the *severe capital* operations of amputations and removal of tumors," operations on the chest or abdomen, anesthesia, animal and insect bites, amputations, and manipulation of the urethra or bladder, among other things.[89]

The likelihood of a particular injury producing shock seemed to hinge upon two factors – its location, and its degree of violence, though this latter element was less constant in the literature. Injuries to structures in the midline of the body were seen as particularly shock-producing; the general sense was that they were associated with more direct connections (real or perceived) to the central nervous system. "The great nerve center, the solar plexus," lying in the region termed the epigastrium (just below the tip of the sternum) was one such connection, that allowed "a severe blow on the stomach [to kill] instantly."[90] Shock likewise often accompanied impacts upon the genitourinary tract (the testicles, in particular, are often cited), the head, the neck, or the spine. Fractures seemed to have peculiar shock-generating capabilities, as stimulus to the bone appeared to affect the organism disproportionately, perhaps, some argued, as a result of greater sensitivity of nerves that traveled in bone.[91]

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[89] Brunton, "On the pathology and treatment of shock and syncope," 247; Hopkins, "Shock—What is it?", 206; Jordan, "On shock," 76.
[90] Easley, "A study of shock," 520; Bell, "I. On the powers of life to sustain surgical operations," 3.
[91] Samuel D. Gross, *System of Surgery: Pathological, Diagnostic, Therapeutic, and Operative* (Philadelphia: Blanchard and Lea, 1859), 430; Brunton, "On the pathology and treatment of shock and syncope," 247-248.

Independent of the location of a particular wound, transfer of considerable force to a patient was an additional risk factor for shock. "The force which will break a bone," wrote William Savory, "may annihilate, without any visible mark, the functions of a nerve centre."[92] Gunshot wounds, railway crashes, crush injuries, burns, compound fractures, and other forceful mechanisms of injury had, in general, greater potential to produce shock.[93] This was, however, open to interpretation among practitioners. While shock "readily follows a blow on the abdomen," noted Samuel Gross, it could also occur "sometimes even when the blow is by no means severe."[94]

Furthermore, beyond the physical aspect, emotional and psychological factors were seen at play in shock. "Joy, grief and terror may produce every degree of mental or nervous shock," wrote Easley, noting that "sorrow, uncomplicated, sometimes kills outright."[95] A common feature in descriptions of shock is an emphasis upon personal traits as either prophylactic toward the development of shock, or salutary in terms of recovery from it. Personal courage and optimism, trust in the surgeon, personal habits, parentage, and race all entered into these discussions, from which frequently emerged an interesting sense of the mind and body relationship to the 19th-century practitioner. It almost seems as though this grew from a syllogistic need – if shock was mediated by the central nervous system through the nerves investing and controlling the tissues, then the formations and impulses

[92] William S. Savory, "Notes with causes, on some of the immediate and remote effects of injuries to the spinal cord." *St. Bartholomew's Hospital Reports* 5 (1869):46. Destruction of a "nerve centre" implied a sort of overload and burn-out of the portion of the brain and spinal cord associated with the injured part.
[93] Jordan, "On shock," 75-76.
[94] Gross, *System of Surgery,* 430.
[95] Easley, "A study of shock," 424.

of the central nervous system must necessarily be capable of influencing the physical body as well.

This sense of mental construction becoming physical reality is most frequently seen in discussions of fear, which in and of itself was sufficient to incite shock in the susceptible patient. "Is this state the result of fear and nervous susceptibility, or is it caused by a physical lesion?" wrote one surgeon, who concluded that "in many cases, doubtless, the effect is entirely mental."[96] The oft-repeated (in the literature) story of the King's College janitor is one example – caught up in a student prank, a night-janitor is held down, blindfolded, and told he is to be beheaded for some imagined crime; after tormenting the poor man for a time, to build the tension, the students slap him across the back of the neck with a wet towel, producing no injury but killing the man nonetheless, through the agency of fear alone.[97] "The *shock* produced by terror," wrote surgeon Edwin Morris, "is of a more violent nature than that caused by grief."[98] In the injured patient, fear of the dreadful implications of the wound – the operation, the pain, the protracted recuperation, the loss of self – could overwhelm even the otherwise strong and healthy. One railway case described a young man who was struck by a train and had a local crush injury to the leg; yet despite his strength and strong constitution, not to mention his sober character, he died in three hours. The reason, again, was emotional: "The terror induced by the

[96] Hopkins, "Shock—What is it?", 206.
[97] Brunton, "On the pathology and treatment of shock and syncope," 247; also Woolsey, "Report on surgery," 69.
[98] Morris, *A Practical Treatise,* 15.

contemplation of the consequences of the fall was sufficient in his case to produce a state of mental depression from which it was impossible to recover."[99]

Fear was an accompaniment of injury and accident, though it clearly played a role in the surgeon's elective practice as well. Case reports abound of surgical patients who manage to convince themselves that death was imminent or that they were fated to a poor outcome, only to be proven right in a sort of self-fulfilling prophecy. One such report filed in the *Richmond Medical and Surgical Journal* by S.V.D Hill – who had served as a Confederate Army surgeon in the Civil War[100] – described a case of "death from mental impression."[101] The patient was 65 years of age, presenting for the elective amputation of a gangrenous foot. The surgeon described finding the patient healthy enough to bear up to the amputation, so he sent for some whiskey to fortify the patient for the operation. "While waiting for the whisky," the surgeon noted, "I noticed his becoming very nervous, restless and apparently alarmed about the operation." The surgeon administered morphine to calm the patient. As the operation commenced, the assistant prepared the chloroform for administration, and the surgeon began to apply an Esmarch bandage to prepare the leg for making the incision, when "the patient suddenly had a slight rigor, his eyes became fixed and suffused, he gasped once or twice, and was dead."[102] The chloroform had not been given, the incision not been made, no blood had been lost, no pain had been

[99] C.B. Higgins, "Shock in railway injuries," *Fort Wayne Journal of the Medical Sciences* 3 (1883-4):20.
[100] Anon., *Journal of the Congress of the Confederate States of America 1861-1865 v. 3* (Washington: Government Printing Office, 1904), p. 201.
[101] S.V.D. Hill, "Letter to the editor," *Richmond Medical and Surgical Journal*, 27 (1879):185.
[102] Hill, 185.

inflicted. Why, then, did the patient die? "I believe [the patient could have survived the operation] but for the terrible fright and dread of the knife, which so *shocked* his nerve centers as to crush out life instantaneously. There was a fatal sedation of the brain and spinal cord, under the influence of the peculiar mental emotions harassing him at the time."[103]

While fear was deleterious, courage and optimism helped the patient hold the line: "He who is cheerful and hopeful in submitting to the knife," wrote Easley, "has vastly the advantage of him who is in an opposite or desponding frame of mind."[104] Personal stoicism conferred protection, though even the overly emotional patient could, under their external drama, cling to a thread of hope that "may be regarded as the psychical equivalent of physical endurance."[105] The patient was not alone in the responsibility for keeping the tone upbeat. The surgeon's behavior was as strong an influence as the patient's on the outcome. George McClellan, writing in the 1880s, warned that "rough treatment and want of consideration for the feelings of the patient have a great deal to do with the causation of shock." He further admonished surgeons they needed to carry out their duties with gentleness, confidence, and delicacy: "If you can gain the confidence of the patient and make him feel that you will not injure him unnecessarily, he will submit himself to your care more readily and be less likely to suffer from shock."[106]

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[103] Hill, 185-186. Italics in original.
[104] Easley, "A study of shock," 513.
[105] Woolsey, "Report on surgery," 67.
[106] McClellan, "Clinical lecture on shock and surgical fever," 816.

Any alteration of consciousness – or at least of the ability to perceive – could potentially mitigate the influence of shock. This makes sense when considered in the light of a shock model in which emotion has the power to amplify physical sensations or experiences. An abrogation of consciousness – a stripping of the mind's analytical ability and ability to process fear, anxiety, or depression – would limit shock to simply the gross physical effect of the *secoussement* on the nerves themselves.

Intemperance, or at least the habitual use of alcohol, was seen as depletive; it deprived the patient of the ability to rally in the face of shock's onslaught to the nervous system. Intoxication, as an acute phenomenon, on the other hand was potentially beneficial, removing as it did the fear and apprehension associated with injury or operation. An essay in *The Medical Record* from 1880 describes a hypothetical patient with osteomyelitis of the hip at the surgeon's office for a debridement, a painful, bloody procedure of scraping, curetting, and cutting infected bone back to healthy tissue. Instead of being anxious and fearful of the coming pain, the patient is happy, with a flushed face, "eyes suffused, her skin warm and natural, her pulse full and slow, her breathing quiet and undisturbed." Why?

> She is partially intoxicated, or 'half seas over,' using a common but expressive phrase. We have purposely brought her into this state in order to prepare her for the operation. During the last five hours she has been supplied with whiskey at regular intervals, until she has taken six ounces. … She is insensible to danger,

> and in the most hopeful state of mind and body, in good condition for the operation.[107]

Alcohol intoxication, wrote the author, was safer than quinine or opium, reduced the requirement for ether, fortified the circulation, and – perhaps most importantly – cooled the emotions, making the patient "quite indifferent, or even bold and daring."[108] Inducing this state in recovering patients could be important as well, as Easley remarked that if a patient was too anxious to allow proper healing, "the moral element of the case must be eliminated at any cost" and the patient "made oblivious to the situation" with opium.[109]

In much the same way, madness, though certainly not a socially desirable quality, was described by some authors as protective against the nervous sequelae of injury. "An insane spinster, aged 38, eluded her attendants and threw herself in front of a moving train, receiving injuries which necessitated amputation of both her legs," wrote railway surgeon CB Higgins in 1883. "In her case, there was no hemorrhage, and no shock," he continued, and furthermore, she returned to her "right mind" upon awakening from anesthesia.[110] (Notably, the cure of this woman's madness through the mechanism of physical injury speaks to the sense of interrelationship and enmeshing of the physical and the emotional in the body's physiology.) Mitigation of the emotional consequences of

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[107] Stephen Smith, "On the value of partial intoxication in the prevention of shock during operations," *The Medical Record* 18 (1880):701.
[108] Ibid., 701.
[109] Easley, "A study of shock," 537.
[110] Higgins, "Shock in railway injuries," 20.

injury, or perhaps elimination of the ability to consider repercussions, prevented the brain and nervous system from burning energy and reserve that could potentially be put toward recovery.

A global depression

Shock could accompany any injury, operation, or overly powerful mental impression, and for the most part in the 19th-century literature, "shock" appears as a verb. A person is "shocked," or they receive a shock; they do not lapse into a state of shock. "The shock through the medium of the brain is such as to suspend the faculties of sense and volition, and to act directly upon the heart as a powerful sedative," noted Morris in his monograph. "In fact," he argued, "it is a *shock* or violence to the whole nervous system, producing great prostration of nervous power" that leads to the classic clinical presentation of shock, which almost universally manifested as a global depression of function, particularly of the consciousness.[111] (There are exceptions – notably during the mid- to late 19th century – where an agitated variant of shock was possible; but for the most part depression was the rule. [112]) A late 19th-century text described shock thus:

[111] Morris, *A Surgical Treatise*, 21, 24.

[112] The importance of balance between activating and depressing forces comes into play here. Shock as a depressive phenomenon is an asthenic state, with loss of ability to initiate or coordinate the functions of life. Some writers felt that an over-excited imbalance was, by definition, also possible. This state was referred to as traumatic delirium. It sometimes represented a failure of the patient to recover from shock, and sometimes represented an iatrogenic complication of managing shock. For examples, see Easley, "A study of shock," especially pp. 526-528; Gross, *System of Surgery*, pp. 436-440; Mansell-Moullin, "Shock," 363. We will address this more fully in chapter 3.

> Everything denotes diminished energy of circulation: the pallor and coldness of the skin, the weakness and small volume of the pulse, the difficulty of respiration, the languor and general depression, all point to some failure among those forces that maintain the circulating fluid at the necessary tension; to some difficulty affecting the motive impulse of the heart, or the peripheral resistance of the capillaries, or the state of tone of the smaller vessels, arteries as well as veins; for these are the forces mainly concerned in keeping up the arterial pressure on which the circulation depends.[113]

This depression was manifest by a classical clinical appearance: a blunting of the sensorium, a slowing of the mental faculties, a loss of overall strength and power. Le Dran's 1745 description of the presentation of shock fits remarkably well with C.W. Mansell-Moulin's entry on shock in the 1881 *International Encyclopedia of Surgery*: "Few conditions are more characteristic than that of a patient suffering from the graver effects of shock; none resemble death itself more closely," with the patient falling quiet, still, and unaware of surroundings; he was conscious, yet with dulled thoughts, speech, movements, and perceptions; he had a weary expression, with sunken eyes and dilated pupils; there was blue discoloration of the nailbeds, coldness with sweating, an irregular and rapid pulse, irregular respirations, vomiting, and urinary retention.[114] This again compares remarkably well with a description from World War I: "The low-tension pulse, usually rapid, sometimes not, the shallow breathing, the pallor, sometimes going on into a

[113] Mansell-Moullin, "Shock," 357-358.
[114] Ibid., 362.

light cyanosis, the lack of apparent suffering, the carelessness as to one's surroundings, the preservation of a clear though lethargic mind up to the very last – these things are all familiar to us, and these were the symptoms which, in our experience, were constantly present."[115] Though the language in the examples differs, all three descriptions are fairly concordant despite nearly 200 years elapsing between them. In all, the patient is enervated and oppressed, weak- and ill-appearing, poorly reactive to his surroundings. There is a sense of loss – or suspension – of control, with involuntary movements, vomiting, and alterations in breathing. There is pallor, a loss of luster and vigor, and a countenance of exhaustion, weariness, and disinterest. The pulse has lost its strength, the respirations are feeble, and the patient seems perched at the edge of dissolution.

These global manifestations are another hallmark of shock. Shock had the power to transform a localized stimulus into a global, whole-body response. Notions of the animal economy and sympathy appear throughout the shock discourse, and starting with Le Dran, we see from the earliest writings this idea of shock representing a local trauma propagating. Samuel Gross, for example, described shock as "a depression of the vital powers," which may follow injury "however trivial," and produced a "perturbation of body and mind, extremely variable both as to intensity and duration."[116] Similarly, Silas Weir Mitchell and colleagues, in compiling cases from the Civil War, made note of the phenomenon of reflex paralysis, a condition related to shock which produced "paralysis

[115] Edward W. Archibald and W.S. McLean, "Observations upon shock, with particular reference to the condition as seen in war surgery," *Annals of Surgery* 66 (1917):282.
[116] Gross, *System of Surgery*, 430.

of a remote part or parts [which] has been occasioned by a gunshot wound of some prominent nerve, or of some part of the body which is richly supplied with nerve branches of secondary size and importance."[117] The nervous system provided the physical explanation for this apparent sympathy, as seen in Morris' argument that "when an injury of any kind is inflicted upon any part of the body every part of the system is affected by it, through the medium of the nervous system, more especially the more important organs of the body, such as the heart, stomach, and spinal marrow."[118]

The globally propagating force of shock produced a fundamental imbalance of the powers of life. The force of shock disturbed the function of the vital systems, in the words of one review article creating a situation of "the harmony of action of the great organs of the body becoming disorganized or disarranged."[119] These whole-body manifestations of shock were generally interpreted as the expression of a sort of depletion or exhaustion; that shock in some way destroyed the body's inability to sustain itself in the face of injury. Commonly, this was expressed in mechanical terms: A strong impulse in an organ or tissue travels along the nerves that invest it, destroying the center of the spinal cord or brain at the nerve's origin in a burst of overstimulation. Mitchell and colleagues remarked, "it appears to us possible that a very severe injury of a part may be competent so to exhaust the irritability of the nerve centres as to give rise to loss of

[117] S. Weir Mitchell, George R. Morehouse, W.W. Keen, "Circular No. 6 of the Surgeon General's Office," reprinted in George A. Otis, D.L. Huntington, and Joseph K. Barnes *The Medical and Surgical History of the Civil War Volume XII* (Wilmington, N.C.: Broadfoot, 1991); originally printed as *The Medical and Surgical History of the War of the Rebellion*; *Part III, Volume II: Surgical History* 2nd issue (Washington D.C: Government Printing Office, 1883), 729.
[118] Morris, *Practical Treatise*, 20.
[119] Hopkins, "Shock—What is it?", 205.

function, which might prove more or less permanent."[120] This loss of function could manifest as reflex paralysis, or it could also manifest as shock, a global depression beginning in the nerves and mediated by impaired function of the circulatory system, culminating in a pathway of paralysis and loss of function in the capillaries, alterations in blood flow, anemia of the tissues, and the hallmark depressive symptoms. The notion of altered tension – seen throughout the 19th century literature – is essentially unchanged from LeDran's Cartesian model, only expressed in newer terminology.

While Mitchell and colleagues relied on analogy for evidence of the idea that sensory impulses could destroy physical tissue – "reflecting then upon the close correlation of the electrical and neural force, it does not seem improbable that a violent excitement of a nerve trunk should be able to exhaust completely the power of its connected nerve centre"[121] – other writers argued that clinically observable evidence for this argument was seen in cases where what should have been an uncomplicated recovery was stifled by shock's sapping of the powers of life. Healing from pyaemia, for example, required a certain "reactive power," which "might be destroyed by an intense physical and mental shock," wrote English surgeon H. Colley March in 1868. He described the case of one Mr. Harrinson, an otherwise healthy man who sustained a compound fracture of the thumb. The patient had apparently, some months before, suffered a fracture of the leg, which had healed without complication, though "it seemed he went in dread of some other mishap, for he had just obtained a policy in an Accidental Death Insurance

[120] Mitchell, Morehouse, and Keen, "Circular No 6," 737-738.
[121] Mitchell, Morehouse, and Keen, "Circular No 6," 738.

Society." In this nervous and anxious state – which predisposes one, after all, to shock – Harrinson fell, incurring the fracture and receiving "a terrible shock, both mental and physical." Evidence of the shock persisted after injury, argued March, as during reduction and splinting of the fracture, the patient was "pale, faint, and sick, though no expression of pain escaped him." The next day, he remained shocked, as he evidenced a "peculiar, mottled, congested appearance of his face. His expression was dull, and his eyes were red." He developed vomiting and diarrhea, drowsiness, hallucinations, incontinence, and an irregular pulse, while the wound remained clean. The next day, with the thumb showing some blackness at the tip, Harrinson died; the postmortem confirmed an infection of the thumb. March concluded "it would seem that shock and putrid infection, meet hounds of death, hunted this unfortunate man, as it were, in couples. Shock held him down, while pyaemia fastened in her poisoned fangs; and so, while 'presently through all his veins ran/A cold and drowsy humour, which did seize/Each vital spirit,' shock, like a good dog, still held on, till the great killer cried "Dead"!"[122]

Shock's ability to deplete could be sufficient to supersede the conscious mind's attempt to ward it off. Morris cited a case of a young man having a leg amputation for scrofula. The patient was stoic and optimistic – exactly the sort of patient who should bear up to the procedure without complication – yet "genuine *shock* following operation" set in. The patient, he writes, was mentally prepared for "any amount of torture that might be supposed to be inflicted by so severe an operation; but the brain was unequal to the task,

[122] H. Colley March, "Transactions of Branches: Reading Branch, Report of the Reading Pathological Society: Anomalous Case," *British Medical Journal* 2(1863):633-634.

it was tried beyond its power of endurance, and a sudden collapse of the whole nervous system took place." The physical power of the system to maintain itself was depleted, courage and personal traits notwithstanding, and the patient remained confused and intermittently conscious until a regimen of stimulants, morphine, and warmth re-energized him and he returned to normal by morning.[123]

A disease with no seat

Shock was easily recognized clinically, and while it could make seemingly trivial wounds lethal, complicate otherwise skillfully executed surgical operations, and manifest unpredictably from patient to patient, it could not be fixed pathologically. Shock, in a 19th-century medicine of textures, tissues, and pathology, was a disease without a seat. This, more than anything, made the shock problem simultaneously important yet seemingly impossible to solve and also gave shock a veneer of mystery and menace. As one review text on the subject put it in 1867: "It is these sudden collapses of vitality, attributable to no apparent pathological changes in the animal structure, which involve the whole subject of *shock* in so much mystery."[124] Interestingly, despite all of the changes in the approach to and definition of shock that occurred between the 19th and 20th centuries, this particular sense of uncertainty never really leaves the shock literature: Walter B. Cannon in 1918 conceded "that investigation of shock has been difficult and

[123] Morris, *A Practical Treatise*, 35-36.
[124] Ibid., 23.

baffling is indicated by the number and variety of theories put forth to account for it"[125]; Owen Wangensteen in the 1970s still referred to it as the "baffling shock syndrome."[126]

Part of the issue underpinning the confusion is that nobody seemed to be able to agree on what shock actually was. E.H. Woolsey, who ultimately became the president of the physiology section of the American Medical Association, quoted the *Medical and Surgical History of the War of the Rebellion* in an article on shock, noting that it was "a subject 'which has perplexed pathologists for centuries.'"[127] That pathologists are called out as being unable to fix the identity of shock speaks to a quandary in 19th-century investigation of shock: Is the identity of shock to be sought in the tissues and the postmortem body, remaining there buried and out of reach? "We know that the nerve-centers are affected in shock," Woolsey wrote, "we know that emotional impressions and impressions of injury are sent into the body and reflected back as the symptoms of shock, but just what takes place at the nerve-depots or centers, which receive these impressions, we do not know, and do not at present possess any means of knowing."[128] Or is shock, as writers like Furneaux Jordan asserted, a product of the living body? "As no one doubts," argued Jordan, "pathology is merely disturbed or diseased physiology."[129] The need to articulate a language of physiology as a living variant of pathology, to give the sort of weight and credence to data gleaned from the living body that had been previously

[125] W.B. Cannon, John Fraser, A.N. Hooper, "Some alterations in distribution and character of blood in shock and hemorrhage," *Journal of the American Medical Association* 70 (1918):526;
[126] Owen H. Wangensteen, "Foreward," in Thal, et al, *Shock,* 8.
[127] Anon., *Journal of the American Medical Association* (1895) 24:498; Woolsey, 64.
[128] Woolsey, "Report on surgery," 64-65.
[129] Jordan, "On shock," 74.

reserved for the anatomic specimen, is found through the shock literature through the latter part of the 19th century. That nobody can agree on issues such as what should be measured, how to measure it, and what importance to ascribe to data collected contributes to, rather than alleviates, this sense of confusion.

One particular situation that vexed writers was differentiating shock as a pure pathologic/physiologic entity from the very similar clinical appearance produced by hemorrhage. Interestingly, hemorrhage is dealt with in fairly disinterested terms by surgical writers; they did not seem to give it the same weight as shock in terms of physiologic disturbance, and there is almost a sense that blood is an inexhaustible resource. Blood was physical, visible, and seemed to have less of a mysterious, deeper connotation than did the function and strength of the psyche. "My impression," argued James Paget in 1862, "is that the tendency of the present day is to attribute too much to the loss of blood, and too little to the impression on the nervous system, which being, through anesthetics, not consciously perceived, is apt to be forgotten."[130] Blood had connection to vital power, and while loss of it rendered the patient susceptible to infection and "other subsequent calamities, and certainly makes him less fit to bear them if they do befall him," it lacked the underlying menace of shock's "violent impression on the nervous centres, which, whether consciously perceived or not, is reflected from these centres, not upon the heart alone but upon all the organs of organic life."[131]

[130] James Paget, "The address in surgery" (read at the 30th annual meeting of the British Medical Association), British Medical Journal 2 (1862):157.
[131] Ibid., 157.

Theodor Billroth argued that the blood had a fixed volume – "you will often have occasion to see in the clinic how freely the blood spurts at the commencement of an operation and how much less it will be toward the end. … Thus decrease of the total volume of blood may cause spontaneous arrest of hemorrhage" – but that it was shock, rather than hemorrhage, that led to the weakening of the heart's action in the injured patient. The recovery from shock, he contended, would produce improved strength of the heart's contractions and potentially lead to bleeding from blood vessels previously not identified during an operation.[132] A similar avenue of thought held that shock represented a congested state of the circulation – "one finds in most cases a manifest dilation of the venous system, more marked in the large trunks and abdominal cavity, in particular, that are engorged with blood" – which would argue against the co-existence of hemorrhage and shock.[133] Enervation, not emptying, of the heart was at the root of shock.

The absence of significant hemorrhage in the postmortem setting was also seen as corroborating the idea that shock and hemorrhage were unrelated. "In every case where death resulted from pure and uncomplicated shock, without hemorrhage," argued a piece on the medico-legal implications of trauma, "the right side of the heart has been uniformly distended with fluid and clotted blood." Meanwhile, the postmortem finding of

[132] Theodor Billroth, *General Surgical Pathology and Therapeutics in Fifty-One Lectures*, with additions by Alexander von Winiwarter, translated by Charles E. Hackley (New York: D. Appleton and Company, 1883); 22, 167.
[133] D. Carmelo Aranda y Martinez, "Del choque traumatico," *La Cronica Médica* 5(1881):232.

a heart "empty and contracted" was strong "negative evidence against the theory of shock."[134] Shock, in other words, killed without bleeding the victim dry.

However, others freely listed shock as a corollary of hemorrhage. In an unusually confident statement on the topic in the early shock literature, the Philadelphia Academy of Surgery noted at a 1901 meeting that "Shock in such cases [gunshot wounds] is almost invariably associated with hemorrhage, and its severity is almost directly proportional to the amount of blood lost." Yet, this opinion was modulated by the belief that shock was a primarily neurologic phenomenon, as the authors noted, a few lines later, that shock without hemorrhage was possible, and implied "very extensive injury" to the viscera or nerve trunks.[135]

One means of reconciling these viewpoints was to approach shock as part of a constellation of clinical situations which followed some loss of vital fluid or force, with corresponding result on the appropriate system. In shock, this would be the nervous system, in the case of hemorrhage the circulatory. "Syncope is a condition dependent upon cerebral deprivation of blood, and may result from simple hemorrhage," wrote E.H. Woolsey in a paper published in 1878. "It is true that the *symptoms* of shock simulate those of syncope and collapse, but it does not necessarily follow, that there is no

[134] Harold N. Moyer, "Medico legal relations of shock," *Medico-Legal Journal* 7 (1889-90):498-499.
[135] De Forest Willard (chair), "Transactions of the Philadelphia Academy of Surgery, Stated Meeting October 7,1901: Gunshot wounds of the abdomen," *Annals of Surgery* 25 (1902):113-114.

difference between these several conditions."[136] Shock in this view terminates with multiple other etiologies along a sort of final common pathway; a neurological route to physiologic dissolution that could also be traveled via bleeding, or fluid loss, or toxemia.

From an intellectual standpoint, shock was challenging, difficult, and interesting; described by one writer as one of the "fertile streams of study and observation ... one of the most terrible accidents that complicates the serious traumatisms which, disgracefully, are so common in our times."[137] And, while the academically oriented could study and research and dispute cause and effect and pathology or the lack thereof, medical and surgical practitioners still had to deal with shock as an everyday, often lethal, part of their practices. Over the course of the next several chapters, we will explore how they attempted to do just this – organize shock into something they could understand, rationally approach, and ideally manage successfully.

[136] Woolsey, "Report on surgery," 71-72. Emphasis in original.
[137] Aranda y Martinez, "Del choque traumatico," 167-168.

Chapter 3 – Shock as a clinical/qualitative phenomenon

As has been well-described, notions of disease identity changed markedly between the 18th and 19th centuries.[138] Nosologic classifications, the idea of "ideal" diseases superimposed upon variable patients, iatromechanics and humoral medicine were abandoned, through an epistemological revolution, in favor of disease classification based upon the lesion. The clinical-pathologic approach to medicine sought to explain the physiologic processes that could produce, as their end results, the tubercles of phthisis or the hobnailed liver of cirrhosis. In this setting, shock emerged as something of a pathologic enigma – authors assumed its existence, yet they could not locate its associated pathologic changes. And while it was a disease with an essence but no seat, it was an ever-present danger to the medical practitioner of the 1800s. The lack of a clear and agreed-upon definition of the disease, and of a consensus regarding its identity and its rules, did not prevent shock from affecting real-world practice of medicine and surgery. How then were the practitioners to establish a systematic, rational approach to shock such that they could practice effectively despite its presence?

In the next several chapters, we will investigate three different approaches to creating an understanding of shock, from three different practitioners using three different ideas as to shock's nature. Building from the same set of clinically observed phenomena – the depressive symptoms, the whole-body response, the relationship of shock to trauma – we

[138] For examples, see Russell Maulitz, *Morbid Appearances: The Anatomy of Pathology in the Early Nineteenth Century* (Cambridge: Cambridge University Press, 1987); Foucault, *The Birth of the Clinic*.

will arrive at three different conclusions using three different epistemological models; in which, hopefully, we can discern something of how medical knowledge is constructed.

To accomplish this goal, we will use three primary authors to function as archetypes, to illustrate three separate perceptual grids that tried to assemble the clinically observed components of shock into an intelligible picture. Such an approach has inherent limitations – the selection of authors naturally limits the scope of our discussion, and one could argue that the selection of one author over another is arbitrary – but, I would argue, considering that a key feature of the shock literature is its widely dispersed variety of theories, opinions, and concepts, establishing some limits will at least allow us to impose some structure and order upon our analysis. The study of 19th-century shock is not one of progress and linear accretion of ideas, as we have discussed, and picking out recurring themes is a useful way to establish some sort of analytical order in these multiple, parallel streams of thought.

We will assess the work of Samuel Gross, John Furneaux Jordan, and Edward Tandy Easley as our archetypes. All of these men were surgeons, and worked and practiced in the mid-19th century, with Gross on the earlier end (1850s) and Easley toward the latter (1870s). They held in common most of the items we discussed in the previous chapter; they posited variations on a theme of nervous system dysfunction as the physical mechanisms underlying the clinical presentation of shock. Gross was perhaps the most empiricist of the trio, basing his analysis on anatomic knowledge and inferring nervous

function using analogies to the blood. He hypothesized the existence of a nervous fluid, which functioned for the brain and nervous system much in the way blood does for the circulatory, and approached shock as a clinically recognized, qualitatively understood phenomenon reflecting a depletion and depression of nervous system function. Jordan, while keeping the notion of finite nervous power, was more mechanistic and sought to characterize shock in quantifiable, mechanical ways in the physiology laboratory. Easley can be described as rationalistic, almost to the point of being philosophical and holistic as he ascribed primacy to the emotions and mind/body interrelationship in the causality of shock. These three approaches represent three philosophically distinct attempts to make sense of a phenomenon which broke from existing rules.

The rude unhinging

A characteristic mid-19th century assessment of shock is found in Samuel Gross' *System of Surgery*. Gross was a university-educated surgeon, holder of the chair in surgery at the University of Louisville and later professor of surgery at the Jefferson Medical College. He was a prolific author and researcher, with an interest in dissection, pathologic anatomy, and physiologic experiment.[139] *System of Surgery* is a multi-volume reference work, widely translated and reprinted in multiple editions, and it provides a good central reference point for further discussion. In it, one finds shock listed in chapter 10 of volume 1, "Effects of Injuries Upon the Nervous System." The nervous system is the key player

[139] Samuel W. Gross and A. Haller Gross, "Memoir of Samuel D. Gross," in *The Autobiography of Samuel D. Gross*, Samuel W. Gross and A. Haller Gross, eds (Philadelphia: WB Saunders, 1893), xiii-xxxi.

in Gross' framework; it is the means by which injury is reflected more globally onto the organism and the system through which the patient will either recover or succumb.

Shock, in Gross' definition, was primarily a neurologic phenomenon with particular clinical hallmarks: it followed injury; its severity may or may not reflect the severity of injury; it was generally a depressive state with collapse of the nervous system, though it may paradoxically present as agitation; and its severity and character reflected the disposition, habits, and idiosyncrasies of the patient. Fundamental to Gross' model of shock were the two complementary ideas that shock represented a depletion or exhaustion of some patient-specific reserve of energy, and that some imbalance in the patient's system contributed to shock's onset or propagation.

"Shock," Gross began, "may be defined to be a depression of the vital powers, induced suddenly by external injury, and essentially dependent upon a loss of innervation." Shock could be sudden, like lightning, and if severe enough could cause instantaneous death. Gross illustrated his definition by setting up a parallel, and a distinction, between shock and syncope, or fainting: "It bears, in effect, the same relation to the nervous system that syncope does to the vascular; in one case, the result is caused by a diminution of nervous fluid, in the other by a diminution of blood; in both, the consequence is more or less prostration, with perturbation of body and mind, extremely variable both as to intensity and duration."[140] The appearance of "nervous fluid" here is interesting; it seems to take on

[140] Gross, *System of Surgery,* 430.

a role not unlike that of the "animal spirits" of LeDran. It makes sense, contextually: Nerves were known to be required for life and for consciousness, yet their mechanism was elusive. Galvani had, by this point, demonstrated the effect of electricity upon the sartorius muscles of frogs, so there was a sense that nerve function related in some way to electrical conduction. Electricity, in turn, was posited to function through a fluid medium. Gross thus inferred that nerves would use this same fluid mechanism to carry their own impulses. That no anatomist had seen nervous fluid was no obstacle; it was assumed to be beyond the limits of perception to be detected: "We can never acquire any intimate knowledge of an agent so subtle as the nervous fluid; like the electric, or galvanic, which it is supposed to resemble, we can know it only by its effects."[141] The very subtlety of this fluid made the nervous hypothesis of shock more attractive: While the surgeon could appreciate ongoing loss of blood – a "vital fluid, so necessary has its integrity always been regarded to the well-being of the system and the maintenance of healthy action" – in the wounded patient, he could not quantify or even detect loss of nervous fluid. And, because men could die from exhaustion of this nervous fluid without a drop of blood being spilled, "of the two fluids, the nervous is, in every respect, the more important, because the more essential to life."[142]

While the nerve-fluid held primacy, Gross – unlike many later writers – allowed for hemorrhage to have a role in shock. In this model, shock was essentially the loss of a vital fluid from the body, either "purely nervous, or partly nervous and partly

[141] Ibid., 431.
[142] Ibid., 430-431.

hemorrhagic." Loss of either fluid was capable of depressing the total-body vital power, though loss of nervous fluid did it faster, and (presumably) at much smaller volumes of loss.[143] The physiologic reaction to this loss of fluid was highly individualized in any given patient, as Gross noted that even heroic men, like soldiers who would face enemy fire, could be laid low by a "trifling" injury or even faint at the sight of blood; while there were others "whom hardly any accident, however severe, can shock." His explanation for this dichotomy was almost humoralist: "In the former case the individual is all nerve, all sensibility; in the latter, all blood, all muscle." The nervous, "sensible" – or sensitive – temperament felt the loss of nerve-fluid more readily than a patient whose physiology was built around the more primal vital power of blood and muscle. Personal temperament thus influenced the response to injury or operation, an issue of particular concern to a surgeon. "The fat and corpulent are prone to be affected," by shock, Gross warned, and he encouraged surgeons to carefully assess a patient's qualities when considering them for operation. A patient who fainted at the sight of blood or when pricked with a needle or catheterized with a bougie, was a fragile soul indeed. "Touch them," Gross admonished, "and they are sure to die."[144]

The causes of shock were manifold. Trauma played a role: gunshot, railway, steamboat, factory injuries; falls; sprains or dislocations; burns and scalds; severe operations with or without hemorrhage; blows or crush injuries to the nervous centers of the skull, spine, and solar plexus. Internal disease processes that were regarded as particularly liable to

[143] Ibid., p. 431.
[144] Ibid., 431-432.

produce shock included bowel perforation, obstetrical labor, apoplectic seizure, gallstones, and kidney stones. Further, "violent mental emotion of almost any kind" – particularly terror and fear – could lead to shock.[145] In line with his placement of greater value on the nervous fluid, Gross felt that if physical and mental causes of shock co-existed in a patient, the mental would predominate. He discussed a hypothetical case of a soldier who is gravely wounded, yet he "is sanguine of recovery, and cheerfully and manfully bears up" and does well until the surgeon tells him he is likely to die. Then, "the system is overwhelmed with perturbation and excitement; the vital powers are depressed to the utmost; and death takes place" sooner than would be expected.[146] Note here the importance placed on the soldier's attitude: he is "sanguine," "cheerful," and "manful," all favorable qualities, and his death follows descent into "perturbation" and "excitement." There is an interesting connection placed here between socially normative values and normal physiologic function, as loss of the soldier's calm and manly reserve is all it takes to sap his remaining strength and push him into the void.[147]

The first common factor in this diverse array of etiologies here was the application of some violence, either mental or physical, to the patient. The underlying assumption was that the force – of injury, of pain, of fear, of hopelessness – dissipated the nervous energy of the patient, sapping his physical system of the power to sustain his life. For this to occur, we need to consider a second common feature, which was that all of these

[145] Ibid., 430-432.
[146] Ibid., 433.
[147] We will see further discussion of this aspect of shock in chapter 5.

mechanisms relied upon the interconnectedness of the nervous system. The bowels and other intra-abdominal structures, for example, from anatomical dissections were known to be connected directly to the brainstem through the ramifications of the vagus nerves; profound extremity injuries were communicated to the nervous system through the large peripheral nerve trunks. The forceful, excessive, sudden stimuli associated with shock-producing trauma produced sufficient impact across this meshwork of nerves to upset the body's internal balance of forces – generally by exhausting some internal reserve of power – and this imbalance was at the heart of how shock manifested itself.

Shock as a clinical disease

Gross hewed to a model of shock that did not differ markedly from that of his contemporaries. In it, shock is defined and understood as what I will term a clinical-qualitative phenomenon. By "clinical," I refer to those things that can be observed, addressed, and understood at the bedside, through the practitioner's physical examination of the patient. In a medical epistemology increasingly interested in precision and pathologic diagnosis, shock is notable in that shock-related discourse retains a very qualitative nature: The language of shock is descriptive and clinical, the disease has antecedent factors, standard examination findings, and a reasonably predictable course. It retains almost a sense of 18th-century medicine of species, existing in known variants that can be categorized and described. Absent are any attempts to describe shock in more quantitative or concrete terms. In explaining shock through the agency of the nervous

system, we encounter themes of depletion and exhaustion – be it nerve-fluid as in Gross' account, or blood, heat, irritation, or anything that makes up the more subtle and less-definable "life force." Shock in these writings also reflects a situation in which a body has become out of balance – there is an abundance or paucity of excitation in the patient's physiology. Finally, Gross makes no arguments that suggest the mysterious, lethal particulars of shock could be elucidated through study or research. Particularly as compared with scientific and medical writings of the 20th century, there is a distinct lack of positivism in discussions of shock by Gross and his contemporaries.

Benjamin Bell, writing in the late 18th century, discussed sudden collapse without evidence of injury – what later writers would call shock – as a situation in which the powers of life were in some way deranged though the agency of a nervous system dysfunction, "but which does not leave such marks of its existence behind it as to render it capable of having its real nature ascertained by dissection."[148] This definition fits the tone of much of the 19th-century work on shock. Bell's comment reminds us that disease is manifest and understood by the marks it leaves behind in the body's tissues, but shock refuses to be known in this way. In the absence of a definable "real nature," shock becomes something known by its appearance. Shock is thus defined in a natural historian's sort of way: To be present, shock needed an inciting event, it needed to produce an appropriate set of clinical findings in the victim, and it needed to follow a

[148] Benjamin Bell, "Section IV – Of Concussion or Commotion of the Brain," *A System of Surgery* (Boston: Isaiah Thomas and Ebenezer T. Andrews, 1791), 180.

particular course as it evolved to its terminus. Shock was thus a clinical disease, diagnosed by elucidating the altered physiology of the living patient through information that was chiefly available to the sensory faculties of the examining physician.

As we discussed in the previous chapter, the key factor in considering whether shock was present was the history of trauma. "Trauma" was somewhat loosely defined, though in general for it to be sufficient to produce shock the insult needed to be of adequate intensity and severity that it could transcend its own local effects and produce the full-body sequelae that defined shock. Gross' list of potential etiologies as enumerated above is instructive – often shock-producing injuries were ones in which the corporeal integrity of the patient was put asunder, in which significant force was transmitted, or in which the nervous system was directly affected: railway crashes, crush injuries, amputations, burns, head and spinal injuries, gunshot wounds. Powerful mental stimuli, often associated with the fear of death or pain, were sufficient to produce the shock cascade because the conscious mind was itself attached to the anatomic/neurologic circuitry presumed to lie at the core of shock. Stimuli could be perceived from the outside, or stimuli could be created from within – the end result on the organs in the system was the same. Trauma served as an inciting event by providing an unbalanced force to the nervous system. The nervous system, in turn, acted as the means by which this force – be it physical or mental – was transmitted across the other bodily systems.

There are a couple of approaches to the problem of how this occurred. They are not mutually exclusive, and authors in the 19th century seemed to have little trouble fluidly moving from one to another as needed. Shock could operate through an agency of nervous exhaustion – in other words, the impact of the stimulus could provide such a shock to the nervous system that it was depleted of its motive force and became unable to carry out its functions, resulting in a global loss of physiologic function. A variant of this idea is that shock functioned like a reflex – a stimulus was applied and a response was generated across an anatomically defined nerve pathway. The resultant shock was then the result of burnout of the nerve center at the crossroads of the reflex arc leading directly to loss of its function, or indirectly to its exhaustion by over-activation of the center's known normal physiologic function.

The basis of these ideas seems to arise from relationships in the body posited from gross anatomy. Even though shock eluded microscopic diagnosis, anatomy still provided a framework for theories of shock's mechanism. In the human, the abdominal viscera, spinal cord, brain, and heart can be seen as linked to one another through the anatomic wanderings of the vagus nerve. The vagus is also known as the tenth cranial nerve, and it arises from the hindbrain, near the spinal cord, forming a thick, easily identifiable nerve fiber that travels in close proximity to numerous important structures – the carotid arteries and jugular veins in the neck, the esophagus in the chest – before visibly ramifying into tiny, innumerable branches across the stomach and bowel. A second large, readily identified nerve – the phrenic – arises in the mid-neck from large nerve roots on

the spinal cord, traveling downwards into the chest, across the surface of the pericardium – the tissue sac that surrounds the heart – and spreading out finally across the surface of the diaphragm. From an anatomic standpoint, these two nerves – both of which are paired structures – form a grossly identifiable means by which the autonomic portion of the brain and a high portion of the spinal cord could be connected with the heart and the viscera. Furthermore, the spinal cord served as a conduit connecting these visceral nerves with the sensory nerves of the periphery. With this underlying communication framework, any part of the body could influence any other – a sort of anatomically definable version of the older, more ephemeral idea of sympathy.

This framework allowed clinicians to make anatomically based arguments on why shock seemed capable of killing its victims so quickly, and why it was capable of producing effects remote from the site of injury. Charles Bell, discussing shock in 1841, remarked that "when a blow on the stomach kills, it is by an influence reflected back on the great nerve of the stomach to the centre of all these vital motions." Bell noted that this neurologic effect was even more profound, and had more rapid action, than the physical destruction of the traumatized organ itself: "And so I can believe that a blow on the heart may have a similar consequence, though a bayonet through the heart has no such instantaneous effect."[149] Shock went to the source of life, essentially hitting an off switch, abruptly unbalancing the vital forces that kept the patient from dissolution.

[149] Charles Bell, "I. On the powers of life to sustain surgical operations," 21-22.

This source of life was generally considered to be the medulla oblongata. The medulla is the portion of the brain which controls or influences many of the basic, autonomic functions of life. This whole-body governing power, combined with direct connections to not only the higher centers of the brain but also the spinal cord and bodily periphery, made the medulla a logical choice as a point of origin for shock. Baltimore physician Frank Bressler noted that the seat of shock must be in the "medulla oblongata, and is dependent upon some molecular change, since shock involves the cardiac, respiratory, vasomotor, secretory centres, etc., in every case, and in proportion to the depression of these centres do we have the intensity of shock."[150] While the "molecular change" could not be readily demonstrated, the anatomic and functional relationship of the hindbrain to the physiologic system as a whole was appealing.

A newer way to express this relationship was in the language of reflexes, which functioned as a somewhat more specific version of the same anatomically derived model. The concept of the reflex was relatively new in Gross' time, with initial publications on the phenomenon in the 1820s and 1830s.[151] Actions of nerves in association with the spinal cord were demonstrated to control physiologic functions when stimulated; researchers posited the existence of functional nerve-organ reflex arcs, in which particular peripheral nerves evoked organ-specific responses through the spinal cord. The vagus nerve was believed to influence the function of the heart primarily as a result of

[150] Frank C. Bressler, "Atropine as a remedy for shock," *Therapeutic Gazette* 13 (1889):240.
[151] Karl E. Rothschuh, *History of Physiology* (Huntington, NY: Robert E Kreiger Publishing, 1973, translated and edited by Guenter B Risse), 184-186.

experiments performed by Leopold Goltz in the 1860s. Goltz showed that, in a living frog, tapping on the abdomen would produce a vagus-mediated slowing of the heart rate.[152] This correlated, on a theoretical level, to a similar reflexive response in the human. "Now, when a painful impression is made on a sensory nerve, it is not infrequently carried up to the medulla oblongata," noted a review on shock from 1870, "where it is transferred to the vagus nerve, which, as you know, has the power of stopping the heart, and by setting this nerve into action arrests the cardiac pulsations."[153]

Thus explained, some understanding of the mechanics of shock seemed in hand – theoretically, at least, with an anatomic rationalization substituting for what could not be fixed pathologically. How then, did this nerve/reflex event become the clinical entity recognized as shock? As we have discussed, shock as it began to materialize in the mid-19th century was envisioned as the product of some derangement of the nervous system, either initiating therein or mediated and propagated through its pathways. Hence, the initial signs and symptoms of shock were recognized, organized, and understood through the lens of neurological disruption.

Recognizing shock in the 19th century

Shock, to the surgeon of the 1800s, was a clinical phenomenon; it was recognized at the bedside, through gleaning a history and examining the patient. The signs and symptoms

[152] See, for example, Seabrook, "The pathology of shock," 660.
[153] Brunton, "On the pathology and treatment of shock and syncope," 253.

of shock, according to E.T. Easley, were not subtle, and the diagnosis not difficult: "The diagnosis of shock requires little remark; it is not likely to be mistaken, and is generally, if not always, instantly manifested on the cause producing it."[154] Easley's confidence seems a bit surprising, given the fact that no real consensus existed on how to actually *define* shock, but none of his contemporaries contradicted him; the true difficulty in shock, apparently, was less in the recognition of it than in the explanation of it.

One of the more comprehensive lists of the characteristic physical findings of shock comes from A. Rhu's 1887 address to the surgeons of the Chicago and Atlantic Railway:

> Prostration with Excitement – Expression of the face blanched.
>
> Features – Pinched, drawn small and shriviled. [sic]
>
> Eyes – Lusterless, pupils dilated and react slowly.
>
> Skin – Cold, clamy [sic] and pale.
>
> Resp. – Slow, shallow, irregular and abnormally deep
>
> Pulse – Small, feeble and varriable. [sic]
>
> Arteries – Small and tension low.
>
> Chilly – Sensations of the extremities and along the spine.
>
> Retention – Of urine as a rule.
>
> Psychical – Depression great.[155]

[154] Easley, "A study of shock," 516.
[155] Rhu, "Shock," 82.

The symptoms of shock traversed a broad range of severity, according to Gross. Mild shock manifest as pallor, weakness, dark vision, and confusion that passed in a short time without complication. More severe shock, however, was far more arresting: "The patient, unable to support himself, falls to the ground, often as he does so inflicting serious violence upon his person." Unconsciousness supervened, and on examination the surgeon found that the patient's "countenance is deadly pale, the respiration is slow and feeble; the pulse is small, fluttering, and, at times, altogether imperceptible; the extremities become rapidly cold; and the surface is soon bathed with an abundant clammy sweat. Gasping and sighing, with convulsive tremors, are often present." Vomiting and incontinence were not uncommon; death frequently followed.[156]

Changes in neurological function and behavior dominated descriptions of shock and in general they gave the impression of someone stunned and speechless, apathetically gazing about, slowly blinking, unable (or unwilling) to interact: "He lies as we see perfectly quiet, and pays no attention whatever to anything going on around him."[157] The classic presentation of shock was manifest by this hazy, indeterminate state of consciousness – not quite comatose, yet not quite fully alert. Furneaux Jordan claimed the shock sufferer was "as pale, motionless, as indifferent to the outward world, as if the injury had already terminated in death," though the actual level of depressed awareness depended upon the patient's own constitution and the sort of injury they had sustained –

[156] Gross, *System of Surgery,* p. 433.
[157] Hermann Fischer, quoted in T. Lauder Brunton, "On the pathology and treatment of shock and syncope," 243.

"every level of intensity" was possible.[158] Some patients were conversant and alert, others barely rousable.[159] Speech and perceptions tended to be dull and slow, the eyes lost their sparkle and became sunken and dilated, the patient lay still and rarely moved.[160]

Emergence from shock was heralded by a stage termed the "reaction," defined as "a gradual, or more or less rapid resumption of the functions of the different organs, which awake, so to speak, from their slumber, and spring back again into life and happiness." Again, this was something recognized through observation: The color returned, the pulse strengthened, the breathing normalized, and often, the patient vomited.[161] The physician needed to be wary, however, as a false reaction could occur. If the vital powers lacked "sufficient stamina to maintain the action of the brain and heart," the patient would again collapse. Even more dangerous, and ominous, was the onset of what Gross termed "insidious shock." In this setting, the symptoms of shock were "of a masked character," and struck as the physician and patient both felt the danger had passed. The patient's condition had improved, but subtle disturbances were there, if you knew where to look: "the pulse too soft and frequent," the complexion too sallow, the patient "too composed and tranquil for the amount of injury he has sustained." In fact:

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[158] Jordan, "On shock," 136.
[159] See Richard W. Corwin, "Death by shock," *The Physician and Surgeon* 2 (1880):497-499, 535-536. Corwin presents eight cases of death from shock, all with railway crashes as the mechanism of injury; half of the patients are conscious at the time of presentation, half unresponsive.
[160] Mansell-Moullin, "Shock," 362.
[161] Gross, *System of Surgery*, 433.

> A more careful examination soon serves to show that deep mischief is lurking in the system; that the machinery of life has been rudely unhinged, and the whole system profoundly shocked; in a word, that the nervous fluid has been exhausted, and that there is not enough power in the constitution to reproduce and maintain it.

Despite the surgeon's ministrations, the patient would quietly slip away. Again drawing explicit connection between the emotional inner life and the physiologic function, Gross described how to best recognize insidious shock: "The countenance, in this form of shock, has often a peculiar melancholy expression, as if foreshadowing the fatal event; a sad smile plays upon the lip, and illumines the lower part of the face, while the upper part, on the contrary, wears a gloomy aspect." Death occurred, "from mere exhaustion," a few days later.[162]

Gross posited a third variant of shock, aside from the more acute versus subacute model as described above. If depressive shock was an outcome of injury that reflected depletion of the vital powers and a failing of physiologic functions, almost like a battery wearing down, the flip side of the coin was traumatic delirium, a state of agitation, paranoia, religious frenzy, and "anger at inanimate objects."[163] This was also referred to as erethismic shock or prostration with excitement. It could manifest as the initial form of shock, or could supervene onto an apparently uneventful recovery from injury.

[162] Ibid., 433-434.
[163] Ibid., 436-437.

Alternatively, it could co-exist with regular shock, creating a mixed over-/understimulated clinical picture that was difficult for the surgeon to read. And, perhaps even more so than in the case of pure depressive shock, the patient's personal characteristics influenced the development and outcome of the delirium.

Gross enumerated the symptoms of traumatic delirium as including "[a] confused, wandering, or flighty state of mind, with excessive vigilance; incoherency of speech and manner; absence of fever; an open, moist state of the skin; and little or no excitement of the pulse. The eyes have generally a wild expression." The bowels were costive, the urine dark-colored and scant, the extremities cold.[164] Other authors made the condition seem more dramatic: "The countenances of patients suffering from it are distorted, and express a nameless anxiety and excruciating agony. They toss wildly about, groan and scream, and complain of a fearful oppression and want of breath, oppressive presentiments of death, and a feeling of total annihilation." This state was accompanied by flushed skin, fast breathing, a faint pulse, and, often, exhaustion with secondary onset of torpid shock.[165]

While particular injuries seemed to produce delirium more than others – notably burns and scalds, railway crashes, compound fractures, and deep lacerations – temperament, character, and habit strongly influenced whether a patient would develop delirium. Gross

[164] Ibid., 437-438.
[165] Brunton, "On the pathology and treatment of shock and syncope," 245-6; Mansell-Moullin, "Shock," 363-64.

stated that "the affection is generally of a purely nervous nature, arising from the effects of the commotion inflicted upon a delicate and highly susceptible constitution."[166] Alcohol use factored heavily into its onset, with "the inordinate use of alcoholic spirits, suddenly interrupted by the occurrence of a severe injury" as a prime cause of traumatic delirium, which could be distinguished from the delirium tremens of alcohol withdrawal by the absence of tremor. Heavy use of coffee, tea, and tobacco also predisposed a person to delirium; all of these substances were categorized (along with alcohol) as stimulants, presumably the overstimulated patient would be more likely to become agitated than depressed after trauma. Children, the obese, and "nervous, hysterical females" were also particularly susceptible.[167] As with depressive shock, the socially normative values of self-control – as manifest by moderation of food, alcohol, and other vices, as well as emotional restraint – and the male sex were proof against delirium.

The three shock subtypes held in common that they represented an alteration of central nervous function following trauma. Whether the patient immediately falls limp and obtunded or rapidly expends their energy on frenzy and delirium, shock is a situation in which the brain and nervous centers fail to discharge their duties of control and coordination over the animal system. This dissolution of the body's unifying principle, in turn, led to the other defining characteristics of shock: The "suspension of mental influence" brought about by the "intensity of the concussion" or the reflex-mediated

[166] Gross, *System of Surgery*, 437-438. "Commotion" here refers to the physiologic disruptions associated with injury.
[167] Ibid., 437-440.

suppression of nervous function created a global, rather than local, physiological disturbance.[168] Influences of the deranged central nervous system were seen as manifest in signs observed in the cardiovascular, respiratory, gastrointestinal, and genitourinary systems.

Cardiac power was widely thought to be diminished, if not suspended, in shock. The pulse in shock is almost invariably described as weakened, described by adjectives like "small," "slow," "diminished in intensity," "feeble."[169] The cause of this failing was a loss of stimulation from the nervous centers responsible for the heart: "The heart losing its normal stimulant, becomes weaker in its action, until it ultimately ceases to beat altogether."[170] Weakness was the key feature of the pulse in shock, far more so than rate, because weakness was an outward manifestation of the posited nervous system exhaustion. An increase in the heart's rate was seen by some authors as a sign that shock had passed – the heart, in this line of thought, was only able to increase its rate when it regained sufficient sensibility to compensate for its weak beats.[171]

Respiratory function was likewise compromised, though there was substantially more inter-author variability in describing these changes than with the pulse. Respirations could be fast and shallow or slow and abnormally deep, "oppressed and sighing,"

[168] Little, "Railway and other accidents: Cases and observations," 530; also Jordan, "On shock," 257.
[169] Multiple sources, including Aranda y Martinez, "Del choque traumatico," 267; Brunton, "On the pathology and treatment of shock and syncope," 249-250; Hopkins, "Shock—What is it?", 206.
[170] Edwin Morris, *Practical Treatise,* 24.
[171] Jordan, "On shock," 258.

gasping, labored, or irregular.[172] The key finding was the departure from comfortable, normal breathing – disarray, disorder, ineffectiveness were the characteristics of shock-oppressed respiration. As with the circulation, this perturbation was presumed to be centrally mediated: "There is but one spot, which, being injured, shall have this effect – the *medulla oblongata*, giving origin to the nerves called Respiratory."[173]

Coldness featured into most descriptions of shock – sometimes of the entire body, sometimes just of the extremities. Along with the coldness traveled pallor and cyanosis – a bluish tinge to the lips and fingernails.[174] The coldness could be explained in several ways: A failure of heat production as a result of disruption of the heat-producing centers of the brain, a failure of heat production at the level of the tissues from dysfunction of the "thermic" or heat-promoting nerves, a loss of warm blood being pumped to the extremities as a result of the cardiac weakness, or some combination thereof.[175] The organism was presumed to suffer from an overall depressed vitality – failure of the sufferer's vital powers led to a failing of heat production.[176]

Impaired gastrointestinal and urinary functions were seen as further evidence of the suspension of nervous influence. "The peristalsis of the intestines is suspended from

[172] Examples taken from Brunton, "On the pathology and treatment of shock and syncope," 246, 249; Easley, "A study of shock," 517; Corwin, "Death by shock," 497-499, 535-536.
[173] Bell, "On the powers of life to sustain surgical operations," p. 21.
[174] Morris, *Practical Treatise*, 41, Brunton, "On the pathology and treatment of shock and syncope," 251.
[175] Aranda y Martinez, "Del choque traumatico," 233, 295; Brunton, "On the pathology and treatment of shock and syncope," 251.
[176] Jordan, "On shock," 281. Jordan here discusses the use of brandy to treat shock; brandy is seen as capable of generating "internal heat" which could be transformed into a vital force.

simple shock," wrote one investigator.[177] This aperistalsis – also known as ileus – was viewed as a beneficial side effect of shock: If the shock had arisen from bowel injury, a lack of digestion and intestinal motility allowed to the bowel to rest and heal.[178] Further, the overall systemic failure was deemed to extend to the kidneys – in shock "the kidney suffers: the secretion of urine is suppressed" – and to the liver.[179]

Differential diagnosis

Shock, of course, was not the only depressive disorder to affect the 19th-century patient. While clinical descriptions and definitions were generally able to convey the sense of shock to the reader, distinguishing between shock and other states of depression could be difficult, and there was little consensus between writers on how best to accomplish this. Shock, syncope, hemorrhage, and intoxication could all manifest with a similar constellation of clinical signs. "Profound alcoholism may be mistaken for shock," warned one author, adding opium narcosis, hemiplegia, and sunstroke to his list of differential diagnoses.[180] The general attenuation and failure of the patient's physiology was also associated with conditions such as septic collapse, fatty embolism, inflammation, injuries to the head and/or spine (often termed "concussion," or "compression" if bleeding into

[177] Frederick Churchill, "Cases illustrative of shock and visceral lesions," *St. Thomas's Hospital Reports* 1 (1870):497.
[178] Bulander, "A sharp knife and a clean pair of hands," 499.
[179] Bell, "On the powers of life to sustain surgical operations," 7; Easley, "A study of shock," 529.
[180] Brannan, "Diagnosis and treatment of shock," 257.

the skull occurred), and traumatic fever.[181] The two conditions considered most difficult to distinguish from shock, however, were hemorrhage and syncope; making the distinction was as much a function of asserting the identities of the respective diseases as it was of eliciting clinical findings.

The relationship between hemorrhage and shock was a subject of considerable and long-lasting controversy, and no real consensus on the subject emerged in the literature until shock was more radically re-defined in the 20th century. Generally speaking, in a neurological depression paradigm, hemorrhage was not seen as an etiologic agent of shock. They were different disease processes, defined by different mechanisms. They produced remarkably similar clinical pictures, though they arose from different sources; furthermore, distinguishing between them was vital, because the treatment of the two conditions was different.[182] Yet, some compromise existed in this domain. Some authors accepted that hemorrhage could contribute to shock by depleting the system of strength and rendering patients susceptible to shock-generating nervous influences.[183] Patients already ill, dissipated, or weak could succumb to hemorrhage-related shock in this manner. As Easley noted, "often a man, especially if his hold on life be a feeble one,

[181] Mansell-Moullin, "Shock," 360, 364; Bell, "On the powers of life to sustain surgical operations," 8; Jordan, "On shock," 223; McClellan, "Clinical lecture," 817; "Concussion of the brain," *Boston Medical and Surgical Journal* (1899) 140:46; Gross, *System of Surgery*, 437.
[182] Higgins, "Shock in railway injuries," 19.
[183] Gross, *System of Surgery*, 431.

perishes from the shock and exhaustion of a rapid loss of blood when the wound from which the hemorrhage proceeds is simple and unimportant."[184]

A second means of reconciling the similarities between hemorrhage and shock was to allow blood loss to cause shock if it could be fit to the neurological model. Blood lost internally or unexpectedly, blood lost with great rapidity, or blood loss that alarmed the patient were seen as particularly capable of killing from shock.[185] The sudden change in the physiologic system – or the mental anguish instigated by watching one's own blood spill – is what set off the shock cascade. The actual event of losing blood was what was important, far more so than the amount lost.

Sorting out shock from hemorrhage in a practical sense was a matter of exclusion: If bleeding had occurred, shock generally was not diagnosed. One case report from *Lancet* described a pregnant patient who had fallen and begun to bleed copiously. Upon the surgeon's arrival she was "collapsed to a marked extent, lying on a sofa in her bedroom, the pulse barely perceptible, the skin cold and clammy, extreme pallor of the face, an anxious expression of countenance, sighing respiration, and slight jactitation." All of the clinical findings that typified shock were present, but with the added feature of hemorrhage. No diagnosis of shock was made; the only mention of shock comes in regards to the author's plan of treatment: "viz., to arrest hemorrhage, to correct the malpresentation with the least possible shock, and, by cautiously employing stimulants

[184] Easley, "A study of shock," 434.
[185] Jordan, "On shock," 135; Easley, "A study of shock," 433-435.

and restoratives freely, to allow the patient to rally and deliver herself spontaneously."[186] Shock wasn't caused by the loss of blood, but it could be caused by the stimulation resulting from improper manipulation of the uterus and fetus – in other words, it could be *superimposed* upon the patient already suffering the ill effects of hemorrhage; it wasn't *produced* by the hemorrhage. Shock in this setting is conceived as a purely visceral, neurologic phenomenon.

Hemorrhage could also be seen as incidental to shock, as we saw in the story of Timothy Daly. Loss of blood sufficient to soak the policeman's clothes raised no clinical suspicion; the nervous impulse from the perforation of the stomach, rather than a significant spillage of blood, was conceived as the proximate cause of death. The shock of visceral injury – Easley reported that "penetrating wounds of the abdominal parietes, in which the spleen, intestines, liver or kidneys, are implicated" were notorious for producing sudden death – and of the gunshot wound itself apparently outweighed the loss of blood.[187] A similar example was published by Mansell-Moullin, who described a case of sudden death from shock after an operation to drain a hepatic cyst with a sharp trocar. The penetration of the abdominal wall and "handling of the viscera" – and not the

[186] C. Egerton Jennings, "The intra-venous injection of fluid for severe hæmorrhage," *Lancet* 1882 (2):436-7. Malpresentation refers to the fetus being out of the normal head-down position, or to one of the fetal limbs protruding from the cervix; this would be corrected by external version, a maneuver involving manually pushing the fetus back into position so labor could continue.
[187] Easley, "A study of shock," 509.

incidentally noted laceration of the portal vein, which would have caused torrential, almost audible, bleeding – were held responsible for causing the fatal shock.[188]

Absence of hemorrhage, appropriately, thus lent weight to a diagnosis of shock after injury. Surgeon J.S. Geigley published a case report in 1883 about his trip to see a farmer kicked by a horse. The patient appeared sick: He was "cadaveric looking," languid, and diaphoretic; he kept his eyes half-closed and rolled back; his respirations were feeble, interrupted by the occasional moan; the radial pulse was not palpable. Geigley reported performing a thorough physical examination to rule out signs of hemorrhage; finding none, he "therefore concluded that [the] patient was simply suffering from shock."[189] Another case report, by William Cox, discussed a pregnant woman who, after a prolonged labor complicated by "rigidity of the os uteri," nearly died in childbirth:

> She suddenly fell into a most alarming state of collapse—syncope I can hardly call it,– tossing about wildly, muttering and laughing, the breath being hurried, panting, and precipitate, and soon settling into a condition which I feared for a time was death. The pulse was quite gone, the countenance perfectly ensanguine, the respiration imperceptible.

[188] Mansell-Moullin, "Shock," 261.
[189] J.S. Geigley, "Notes from private practice: Art. I – Case of shock," *The Peoria Medical Monthly,* 4 (1883-4):224-225.

His first thought was post-partum hemorrhage, but a quick exam ruled this out: She had lost little blood in the delivery, and the uterus had contracted well. In the absence of hemorrhage, he could conclude that the patient suffered from shock brought on by "excessive nervous prostration."[190]

Presence of specific neurological findings were also cited as a means of sorting true shock from hemorrhage. Failure of the personality, reason, and intellect were early signs of shock, according to Jordan; motor signs such as loss of deglutition indicated deepening shock; and the appearance of autonomic failure portended poorly: "Especially when the fifth and glosso-pharyngeal nerves fail to excite any response in the nerve-centres, the gravest fears may be entertained that respiration itself will momentarily cease."[191]

Related to hemorrhage and shock was syncope. Syncope implied a loss of consciousness as a result of some disturbance of the circulatory power and the consequent cerebral anemia – the brain failed to receive its necessary allotment of blood.[192] In the modern sense, syncope is essentially synonymous with fainting; to the 19th-century practitioner it held far closer association with dissolution and death. From a causative standpoint, syncope still had neurological connotations – "An influence of the mind, as in fright and sudden joy, which suspends the heart's full action, and diminishes the force of circulation

[190] William J. Cox, "Case of excessive nervous shock following delivery," *Lancet* 1853(1):556-557. The os uteri is synonymous with the uterine cervix.
[191] Jordan, "On shock," 137. The fifth cranial nerve controls the muscles related to chewing and carries sensory input from the face; the glossopharyngeal nerve is important in tongue control and swallowing.
[192] Aranda y Martinez, "Del choque traumatico," 233; Easley, "A study of shock," 522.

in the brain."[193] But mechanically, the manifestations of syncope were tied to circulatory function rather than neurological function, an issue of either arterial dilation or suspension of cardiac contractions.

As was the case with hemorrhage, syncope's exact relationship to shock was unclear and varied between authors. Gross, for example, drew a very clear distinction between the conditions: "[Shock] bears, in effect, the same relation to the nervous system that syncope does to the vascular; in one case, the result is caused by a diminution of nervous fluid, in the other by a diminution of blood; in both, the consequence is more or less prostration, with perturbation of body and mind, extremely variable both as to intensity and duration."[194] Others saw it as existing upon a continuum, with shock and syncope as different grades of the same depression syndrome: "So closely indeed, are syncope and shock connected that they were considered by the celebrated surgeon, Travers, to differ in degree rather than in kind."[195] However the mechanism was conceived, syncope was recognized and differentiated from shock chiefly by its brevity – patients both collapsed and recovered quickly in syncope, whereas in shock the transitions were less steep, and the systemic state of depression lasted longer.[196] The reason for this was simple – a weak circulation led to the fainting, but when the patient hit the floor, gravity assisted the

[193] Bell, "On the powers of life to sustain surgical operations," 2-3.
[194] Gross, *System of Surgery*, 430.
[195] Brunton, "On the pathology and treatment of shock and syncope," 241; Aranda y Martinez, "Del choque traumatico," 296.
[196] Aranda y Martinez, "Del choque traumatico," 269.

physiology: “The very fact of the head being lowered induces more blood to pass to it, and the normal condition is at once in many cases restored.”[197]

Understanding shock as a clinical problem

To summarize, shock was recognized in the 19th century by a constellation of clinical findings assumed to represent the depression of the nervous system: “Almost from the receipt of the injury the patient labors under a condition denominated shock,” surgeon George Brannan told his audience, the assembled surgeons of the Wabash Railway, in 1888, “some of the symptoms of which are, skin cold, exsanguinated and covered with cold clammy sweat, pulse is weak and tremulous, eyes are wholly or partially closed, pupils react very slowly, if at all to the stimulous (sic) of light, and respiration is slow and labored.”[198]

The findings that are highlighted are those that reflect some disarray, depression, or dissolution of the system – the patient’s appearance, skin color, temperature, and responsiveness are all called forward, described in terms of coldness, slowness, irregularity, shrinking. It is noteworthy that all of the abnormal findings are couched in qualitative language – the pulse, as a surrogate of arterial tension, is “weak,” but not expressed in terms of a manometric pressure; as a representation of the systemic strength, the pulse is described as “tremulous,” but not put into terms of a rate measured in beats

[197] Brunton, “On the pathology and treatment of shock and syncope,” 253.
[198] Brannan, “Diagnosis and treatment of shock,” 257.

per minute; the skin is described as "cold" rather than as having a specific temperature. Further, shock was distinguished from states that produced a related symptom picture by clinical, interpretive criteria – rapidity of response to therapy, presence of certain types of injury, absence of hemorrhage.

Descriptions of the physical appearance of the patient and the expected examination findings help establish a means to translate the observable phenomena of shock into the medical grammar of the underlying pathologic changes. However, one of the interesting things to note is that the qualitative elements that go into a diagnosis of shock are, in general, things which could easily have been quantified. There was no technological mandate that kept shock defined in a qualitative sense; there was no obstacle to it being characterized otherwise. The description of shock, its definition and diagnosis through purely clinical-qualitative criteria, is a conscious, deliberate epistemological choice on the part of practitioners.

Why would such a choice be made? I suspect there are a few things at play. First, the culture of 19th-century clinical medicine had a somewhat adversarial, or at least skeptical, relationship with laboratory and physiologic science. More quantifiable information could offer explanations for particular clinically observed phenomena, but in the eyes of many in the profession – particularly those educated in the earlier part of the century – clinical experience and observation held more weight in terms of caring for patients and

embarking on treatment.[199] There would be little need for a quantitative explanation of shock to a clinician whose frame of reference privileged the empirical.

Another consideration is that clinicians embraced the concept that the underlying physical derangements in shock were possibly unknowable, at least outside of the ability to recognize their presence indirectly through clinical findings. As noted above, Gross argued that physicians could "never acquire any intimate knowledge of an agent so subtle as the nervous fluid" in the study of shock, but could only "know it only by its effects." California surgeon EH Woolsey noted in his report to the state medical society that "we know that emotional impressions and impressions of injury are sent into the body and reflected back as symptoms of shock, but just what takes place at the nerve-depots or centers, which receive these impressions, we do not know, and do not at present possess any means of knowing." [200] This lack of positivist tone is interesting for the medical literature, but it also speaks to the fact that the mission of writers like Gross and Woolsey was thus not one of elucidation of mechanism, nor of generation of new theories, but rather one of refinement in recognition. Like the work of a natural historian, writings on clinical-qualitative shock sought to better demonstrate how to identify that particular beast when encountered in its habitat, not to discern any deeper appreciation of its meaning or function. It was a practical interest, not a revolutionary one; shock was a problem that needed to be diagnosed so it could be dealt with. The underlying mechanics

[199] For a discussion of the role of science in 19th century medical therapeutics, see John Harley Warner, "Ideals of science and their discontents in late nineteenth-century American medicine," *Isis* 82:3 (1991):454-478.
[200] Woolsey, "Report on surgery," 64-65.

were beyond the reach of physicians to influence and thus not as important. If shock was an issue of lethal alterations in the central nervous system, the quantification of pulse rate or temperature would not be particularly useful. The sense of depression of function produced in shock is more of the issue – clinicians were looking for outward evidence of nervous dysfunction, which does not require a numerical, quantified precision. We are not constructing shock as crossing a threshold of some measurable physiologic normal, as would be done by 20th-century writers; we are instead looking for shock to manifest signs of the underlying whole-organism dissolution and loss of vital force. Enervation, pallor, weakness of physiologic action – these observable, though unmeasured, phenomena are sufficient.

In this sense, clinical-qualitative shock fits the Kuhnian normal science model. There was not much interest among writers to better explain or understand the physiology of shock; there was a great interest in describing, recognizing, and discussing shock. An alternative approach, and one which would be favored as medicine's research and scientific focus became more physiologic and quantitative, was to express shock in more concrete, definable, measurable parameters. Interestingly, though, a shift toward a quantitative evaluation of shock would produce no revolutionary change in its underlying nervous dysfunction paradigm.

Chapter 4 – Shock and quantitative study

As we discussed in the previous chapter, that shock would be described in a clinical-qualitative way reflected more upon the epistemological priorities of 19th-century clinical medicine than it did upon available technology or physiologic theory. The ability to perform certain quantitative physiologic measurements had existed as early as the late 18th century. Taking a pulse rate required no technology more elaborate than a pocket-watch; the ability to easily and noninvasively measure vascular dynamics was introduced with Marey's sphygmograph in 1860; Wunderlich's study on clinical thermometry was published in 1868.[201] All of these technologies either pre-existed or developed contemporaneously with the rising prominence of shock in the medical literature. New technology did not generate a shift in the way shock was defined; rather, a re-alignment of medical priorities gave privilege to a different form of knowledge, and a demanded a different standard of evidence for its proofs. As Foucault described for the transition of disease theory from a medicine of species to a medicine of lesions, this sort of shift is a matter of "a recasting at the level of epistemic knowledge [*savoir*] itself, and not at the level of accumulated, refined, deepened, adjusted knowledge [*conaissances*]. … It is not a matter of the same game, somewhat improved, but of a quite different game."[202]

[201] Robert G. Frank Jr., "The telltale heart: Physiological instruments, graphic methods, and clinical hopes, 1854-1914," in *The Investigative Enterprise: Experimental Physiology in Nineteenth-Century Medicine*, ed. William Coleman and Frederic L. Holmes (Berkeley: University of California Press, 1988), 215-218; Stanley Joel Reiser, *Medicine and the Reign of Technology* (Cambridge: Cambridge University Press, 1978), 101-102, 117-118.

[202] Michel Foucault, quoted in Gutting, *Michel Foucault's Archaeology of Scientific Reason*, 136.

The latter part of the 19th century saw a shift in the *savoir* of medicine, from a bedside art defined by the semiotic mission of discerning the hidden lesions of disease through physical examination to a more quantitative, measured science; the practice of finding deflections of the functioning, living organism from some normal point. In the same way that clinical examination superseded rationalized systemic medicine for its purported objectivity, graphical, visible, and numerical representations of physiologic processes came to supplant the clinician's gaze as "truly" objective findings. The broadening use of the thermometer, sphygmograph, sphygmomanometer, electrocardiogram, and radiograph all point to a medical episteme in which hard data held greater prominence and worth. The individual, sensory nature of the clinical examination – in which each physician experienced the qualities of the patient's pulse, skin temperature and respiratory depth through the medium of their own perceptions – came to be seen as inadequate. Since each person's perceptions were different and each communicated them differently, there was no standard from which to proceed. On the other hand, information such as temperatures, rates, and curves obtained by machine could be communicated to other physicians, studied at length, and compared with other readings.[203]

Scientifically minded authors in the late 19th century sought ways to study shock through the use of physiological measurements – thermometers, sphygmographs, manometers, chemical assays. Correspondingly, shock began to take on a different identity: From the state of generalized depression shock emerged, by the end of the Great War, as a disease

[203] Reiser, *Medicine and the Reign of Technology*, 91-121; Frank, "The telltale heart," 212-213.

defined almost purely by physiological metrics and biochemical assays. The rude unhinging of the machinery of life, marked by a melancholy countenance, an overpowering coldness, a quiet flickering of the vital spirits ending in a poetic death instead became a state of measured variables: hypothermia, hypotension, tachycardia, acidemia. Interestingly, though, while the defining characteristics of shock changed – or, at least, were articulated differently – the identity and the underlying mechanics of shock remained mostly the same.

Furneaux Jordan and quantitative shock

John Furneaux Jordan was surgeon to Queen's Hospital and professor of surgery at Queen's College, Birmingham. Active in the mid- to late 19th century, Jordan was a prominent author and investigator. His essay, "On Shock After Surgical Operations and Injuries" was awarded the Hastings Prize of 1867 and published serially in the *British Medical Journal* over the course of that year; further, it seemed to enjoy a certain degree of popularity, being re-issued and reprinted later in the 19th century as a companion to his textbook *Surgical Enquiries*. Testimonial advertisements in the *BMJ* described his work as "able," of "utmost practical value," and as showing evidence of "earnest thought, much originality and thorough personal investigation." Admittedly, these are hardly unbiased statements – they are trying to sell a book, after all – but they also suggest to a degree the esteem in which Jordan's work was held. If nothing else, it is instructive to look at how the advertisers try to sell Jordan's work – the work is described as powerful

because it reflected his own experiences and was based on empirical observation of clinical cases: "It is a striking contrast to the encyclopedic and elaborate style that so largely prevails, but is a simple record of many interesting cases which have come under Mr. Jordan's care, or of careful investigations he has made. … It is essentially a personal book; it is Mr. Jordan, and himself alone, *his* experience, *his* views, *his* hopes, *his* fears; and as such it is a very valuable book."[204]

Jordan's investigation of shock is, in its theoretical grounding, quite similar to Gross' work. Shock exists as a neurologic phenomenon, a complication of injury or violence that physically affects patients through a medium not fully understood. Jordan, however, gives less of a role to the mind and nerves as vessels of some sublime *pneuma* and instead operates in a framework where the mind, through the medium of thought, reflex, and emotion, can create the body's reality – a theoretical approach which was taken further in the works of surgeons like E.T. Easley, and also in the decidedly nonsurgical realm of the functional diseases like neurasthenia. What sets Jordan's essay apart is that it is research-driven, reinforcing the initial theoretical discussion of shock with case-based observations and introducing ideas of quantification and reproducible measurement into the shock literature. What is interesting to note is that these change nothing with respect to the grounding theories of shock; but they do suggest the rumblings of new epistemological demands on medicine.

[204] "Surgical Enquiries including the Hastings Essay on Shock, The Treatment of Inflammations, and numerous clinical lectures by Furneaux Jordan," *British Medical Journal Advertiser No. 1063*, 14 May 1881. (not paginated)

"Shock," opens Jordan's essay, "may be defined in general terms as a peculiar condition of the animal system, characterised by depression of all its functions, the result of a powerful impression applied to the nervous centres, or to a portion, more or less considerable, of the peripheral nervous expansion."[205] Shock affected the body through the suppression of vital functions – chiefly the heart and circulation, which was broadly understood as the final common pathway of shock's effects – but was initiated and propagated through the nerves. Shock had confounded surgeons because of the absence of a truly pathological lesion associated with it; but Jordan argued that the answers to shock would instead be found through application of experimental physiology: "Pathology," he wrote, "is merely disturbed or diseased physiology."[206] Specifically, greater understanding of the physiology of the nervous system would give researchers greater understanding of shock.

Jordan's physiologic understanding gave primacy to the nervous system: "The nervous system is the loftiest in its functions, and the most complicated and elaborate in its construction."[207] It was the central organ system of the body, that which took the sensory, the volitional, the intellectual, the imagined and made them physical: "The cerebrospinal system is the instrument by means of which the will, the intellect, the sensibilities, the sensations, and all the varied modes of action, are brought into operation, and their

[205] Jordan, "On Shock," 73.
[206] Ibid., 74.
[207] Ibid., 73.

effects made manifest."[208] He allowed for two fundamental divisions within the nervous system: The "most exalted" higher seats of feelings, ideas, and the will; and the physical effector system for these higher seats, in turn divided into sensori-motor (volitional) and excito-motor (reflex) nerves. The higher functions of the brain were deranged first in shock, which caused no lasting problems; however, when the "manifestations of psychic power" via the sensori- and excito-motor nerves became impaired, the patient could die: "When sensori-motor action is arrested, life itself is in danger; and when excito-motor action in some of its manifestations (the suspension of all excito-motor action, it is needless to say, is inconsistent with life) is impaired, a fatal termination is extremely probable."[209]

Jordan posited "nerve force" as the driver behind nerve function. "Nerve-force is that which controls, influences, or modifies all other [vital forces]," he wrote. Nerve force was a physical, material thing, manifest in what Jordan termed nerve-substance, which seemed to correspond to the cytoplasm of the neuron and originated from "the forces residing in the materials of the external world."[210] Nerve-force was kept in balance with the other forces of the animal economy in a tightly controlled conservation of energy – "A given amount of nerve- or of any other force is interchangeable with (in shock we may perhaps say replaced by) some other force but the quantities of the forces are always

[208] Ibid., 73.
[209] Ibid., 73-74.
[210] Ibid., 74.

equivalent"[211] – and could be converted to and from nerve force in an almost chemical-reaction kind of way with these other forces. Hence, Jordan argued, nerves were myelinated to insulate them from losing nerve-force too readily. This fits contextually with late-19th century medical thinking on the function of the nervous system – that the nerve force, or *vis nervosa*, was a finite, exhaustible, ethereal physiologic parameter whose proper flow and conservation were crucial to the normal function of the organism.[212]

With this as background, Jordan addressed the causation of shock, which he argued could always be explained by one of four etiologies: "1. Those which act on the corporeal organization. 2. Those which act on the psychical organization. 3. Those which act on both the corporeal and the psychical in equal or unequal degrees. 4. The fourth head comprises cold, which, although fully admitted by physiologists, has curiously escaped the attention of surgeons."[213] Cold specifically was known to "equally and primarily" depress "all the manifestations of vitality,"[214] hence by Jordan's own definition cold was necessarily a form of shock. The third type of injury, "where more or less of mental influence is added to a severe bodily injury," was understood as the dominant type of shock seen in surgical practice. Interestingly, and it's unclear how the distinction would be made practically, Jordan argued that shock incited by profound emotional stress or physical injury alone would not be worsened by the addition of the other factor – the

[211] Ibid., 74.
[212] Oppenheim, *Shattered Nerves*, 79-80.
[213] Jordan, "On shock," 75.
[214] Ibid., 75.

body was "already in a condition in which its susceptibility to further depression is diminished."[215] Once the ebb and flow of *vis nervosa* was disrupted, to Jordan and his contemporaries, the system became refractory to further perturbation; shock, paradoxically, could prevent further shock.

In any case, there was a broad range of potential causes of these third-category states: railway injuries ("by far the most appalling [injury] which the mind can conceive"), burns, soft tissue injuries, compound fractures (especially of the thigh), amputations (also especially of the thigh), battlefield injuries ("where, in spite of bravery, there is also dread and anxiety"), "severe operations of any kind where there is much apprehension before chloroform is given, or where it is imperfectly or not at all administered," injuries and operations of the large joints, injuries to the thorax and thoracic organs, "injuries which implicate the abdominal organs" including "wounds which merely penetrate" the abdominal wall, irritant poisons, poisoned wounds, and the bites of poisonous animals, crush injuries of the eye or the testicle, spine injuries, rupture of the eyeball, rupture of the bladder or urethra, and hemorrhage. Further, "injuries of the head are among the most important causes of shock."[216] Certain threads of commonality can be picked out among the items of this list: proximity of the injury to the trunk, association with pain and fear for survival, crushing or penetrating mechanisms, and potential involvement of nervous reflex pathways, which will be discussed further below.

[215] Ibid., 75.
[216] Ibid., 75-76.

Purely psychical causes of shock were delineated as "the more powerful emotions – joy, grief, anger, and fear," and could produce death in susceptible patients. Purely physical causes of shock were seen to be "extremely rare," but shock could be suddenly induced if a "large or habitually distended bladder is completely emptied," in cases of "internal and unsuspected hemorrhage, as in rupture of an internal aneurism, or the rupture which occurs in extrauterine foetation," or "in injuries, or loss of blood."[217] Like most authors in the 19th century, Jordan didn't seem to settle on fully differentiating shock and hemorrhage. Much like Gross, he allowed hemorrhage to produce shock but didn't envision hemorrhage as a type of shock, in and of itself.

Although he conceived of the circulatory system as the pathway of shock – the effector system, essentially, where the manifestations of suspended nervous influence became clinically apparent and through whose agency shock produced death – Jordan viewed the nervous system as the key area of dysfunction in shock. Much like other authors – most notably E.T. Easley – Jordan allowed for mental formations to create physical, physiological reality in injured patients. Strong emotion was enough to change physical chemistry: "Changes take place in the blood in the shock which follows intense emotion. In such cases, the secretions are generally altered, and often have a different odour. … An eminent physiologist pertinently asks if the blood may not be suddenly so changed as to be no longer a nutritive fluid, but a deadly poison."[218] Strong emotion – generally always carrying a negative connotation of fear, anxiety, dread, cowardice – makes the blood so

[217] Ibid., 76. "Extrauterine foetation" in this case refers to ectopic pregnancy.
[218] Ibid., 223.

noxious that it develops a "disposition to the formation of coagula in the heart or blood-vessels," which in turn are embolized through the body to worsen the patient's outcome.[219]

Shock killed patients, in Jordan's terms, through "syncope and asthenia."[220] Patients exposed to great violence or force died suddenly from shock through "extreme spasmodic contraction of the heart, which leaves it empty and which is not followed by relaxation." The clenched heart could not refill, the blood could not flow, and the patient collapsed. Alternatively, "very much more frequently, the cause of fatal syncope is the sudden cessation of the contractility of the muscular fibre of the heart. Death occurs in this manner, in most cases of injury, from the several varieties of injury or from loss of blood."[221] Jordan's physiologic model required muscle to have both blood supply and nerve supply to contract; the cessation of nervous impulses from the shocked brain, combined with loss of stimulating blood, led the heart into a spiral of diminishing function that culminated in death. Jordan reported that muscle fibers taken from a man dead of shock could not be stimulated to contract in the laboratory – this, he argued, "illustrate[ed] most forcibly the potent effects of violent impressions on the nervous apparatus."[222] One gets the impression, upon reading such a description, of a single large discharge of energy through the nervous circuitry, exhausting all of the vital power of the tissues and depleting the body of the reserves it needed to keep going.

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[219] Ibid., 223.
[220] Ibid., 259.
[221] Ibid., 259.
[222] Ibid., 260.

Shock could be diagnosed clinically, recognized by its depressive signs and symptoms. The patient was "as pale, motionless, as indifferent to the outward world, as if the injury had already terminated in death." The countenance had the appearance of "bloodlessness," "the conjunctiva is lustreless, or even 'glazed,'" and the "features are smaller, especially the nose," with the face taking on a shrunken, withdrawn look. Sweating and cyanosis were evident. The pulse reflected circulatory depression, becoming softer and irregular in its rhythm and rate. Feeble, sighing respirations were characteristic, as was loss of sphincter control and failure of swallowing.[223] Much like Gross' description, a state of delirium and agitation was also possible: "In cases where the shock is due to great but not sudden loss of blood, the stillness of the muscular system will be replaced by the tossing restlessness, and possibly delirium, so characteristic of unchecked haemorrhage, either internal or on the surface of the body."[224] Complaints of coldness, stiff muscles, and nausea were more characteristic of mild shock; whereas objective findings reflecting failure of autonomic and central nervous functions – "especially when the fifth and glosso-pharyngeal nerves fail to excite any response in the nerve-centres, the gravest fears may be entertained that respiration itself will momentarily cease" – heralded a deepening, perhaps terminal course.[225]

[223] Ibid., 136.
[224] Ibid., 136.
[225] Ibid., 136. The fifth cranial nerve (the trigeminal) controls, among other things, the muscles required for chewing; and the ninth cranial nerve (the glossopharyngeal) controls muscles of the pharynx important in swallowing.

Yet, to Jordan, clinical diagnosis was imperfect and imprecise. It saw only the outer manifestations of shock, not the underlying processes that made it happen. The pathologic findings of shock, Jordan had to concede, were minimal; often the effects of the injury couldn't be distinguished from any concrete change in tissues. But, he argued, just because no change was evident didn't mean it wasn't there: "It by no means follows, with our imperfect means of observation and research, that we are justified in concluding that no change has taken place."[226] Electrical currents, he argued, often worked in similarly mysterious ways – able to coagulate a snail within its shell, for example, or burst the blood vessels within an egg, yet in neither case leaving a mark on the outside. Neurologic discharge, he concluded, likely functioned in a similar manner, wreaking havoc internally that we simply were unable to identify through the usual pathologic channels. Jordan argued that the changes associated with shock would be found in the living patient, rather than the anatomic specimen. He argued that shock could be best-understood through the lens of "dynamical pathology" – essentially applied physiology, or what modern scientists would term pathophysiology – rather than static, structural pathology.[227] Jordan set out to analyze shock's clinical features in dynamic pathology terms; spelling out a series of physiologic changes in the heart and circulatory systems that he explains through the agency of altered nervous stimulus:

- The unconsciousness that may supervene in severe injury is a direct result of nerve-force exhaustion.

[226] Ibid., 260.
[227] Ibid., 257.

- The muscular weakness of shock is a product of both nervous and circulatory failure. Muscles require nerve-force to act, but also require adequate blood; shock renders them empty of both, through direct impairment of the nerve-force as well as failure of cardiac action secondary to lack of the nerve-force.
- Urinary retention occurs because there is insufficient nerve-force to empty the bladder; fecal incontinence occurs because there is no nerve-force to prevent the evacuation of the bowel.
- Diaphoresis (diffuse sweating) is the result of loss of nervous tone to sweat glands, which let go their sweat.
- The heart's actions are "modified" through alterations in the nerve-force to produce the circulatory and respiratory phenomena of shock: pallor, coldness, irregular and shallow breathing, and cyanosis.[228]

Jordan clearly believed that nerve-force and physiologic functions – in particular cardiac function – were linked. The task he thus set out for himself was to find a way of demonstrating this in a "dynamical" fashion; or, in other words, measuring one of these physiological parameters in a quantifiable way.

[228] Ibid., 258-259.

Thermometry and pulse wave interpretation

Jordan viewed coldness as a unique form or manifestation of shock. Coldness, conveniently, could be easily and quickly measured through thermometry. Using axillary temperatures, as had been described by Wunderlich, Jordan treated body temperature as a surrogate marker for the strength of the heart and the circulation in general. If the heart's action was strong, the blood would be circulating well and the body temperature would be normal; likewise, coldness and pallor would accompany asthenia of the heart caused by bloodlessness and failing nerve power. Listing as evidence a series of cases, accompanied by graphical representations of temperature curves, Jordan argued that shock was heralded by a slight drop in body temperature, and the recovery from shock manifested by a slight rise in same. The temperature deviations he measured were quite small – as fine as a fifth of a degree Fahrenheit – and he noted them to be most prominent whenever a bone was being divided. Jordan found this interesting, as bone is not particularly richly innervated compared with the soft tissues one must divide to reach it. He presumed, though, that given the smaller population of nerves available in bone, each nerve there must carry proportionately more physiologic energy than each one of the legion of nerves in soft tissue: "The most probable explanation of this singular phenomenon is, the greater laceration of the nerves, smaller and fewer as they are, which instantaneously gives rise to shock, as indicated by depressed cardiac action and lowered temperature."[229] Failure of the temperature to exceed 97 degrees Fahrenheit correlated,

[229] Ibid., 164.

according to Jordan, with a poor prognosis and failure to recover from the shock; while temperatures between 101 and 103 degrees, if reached not later than the morning of the second day, were consistent with a post-shock recovery. After the second day, fevers in excess of 102 suggested a process other than shock and reaction, and sustained temperature in the 103 degree to 106 degree range portended a fatal outcome.[230]

Several things are worthy of mention here. First, while he initially dealt with the diagnosis of shock using the same sort of clinical-qualitative, soft-measurement, bedside-exam sensibility common among other writers of the time, Jordan's goal was to develop a quantitative, precise way of making the diagnosis. Measurement of body temperature is numerical, can be charted over time and viewed with respect to its trend, and can be communicated in writing as a reproducible and broadly understood value, as opposed to the more qualitative approach of discussing lustreless eyes, lassitude, and pallor. Jordan's attention to temperature measurement is quite precise, with changes as small as 0.2 degrees noted as significant. Second, evidence of the researcher's heuristic frame of reference is visible through Jordan's statements regarding the temperature drop in shock – the fall in temperature is explicitly assumed to be the result of an attenuation of nerve function, which in turn dulls the heart, leading to diminution of the body's vital heat. Change in temperature was a proxy for cardiac power, cardiac power was likewise a proxy for nervous power, and nervous power was the key variable in shock. Jordan was not seeking to redefine the mechanism of shock, or elucidate some secret of it. Rather, he

[230] Ibid., 164, 167.

was trying to find a clearer way to express the clinical features he expected to find. Third, Jordan used this quantitative data to in turn establish a set of clinical guidelines and prognostic indicators – shock below a certain temperature, reaction above. In fact, Jordan found temperature to be the best available clinical marker of shock and recovery that he could identify: "The thermometer furnishes the must [sic] reliable information (more than the state of the pulse and respiration) of the real nature of shock and reaction, and its data afford the safest grounds for prognosis."[231] This reliance on the quantitative to make such bold declarations is not found among Jordan's contemporaries.

Jordan's thermometry cases are also instructive in terms of looking at his shock model. Interestingly, temperature is the only measured variable; the remainder of the clinical factors at play in a given case are of the familiar qualitative sort. As an example, Jordan listed a case of a woman undergoing mid-thigh-level amputation of the leg. Her temperature fell intra-operatively to 96.6 degrees and rose afterward only to 97.8 at a maximum; she became progressively enfeebled and died on the tenth postoperative day. While Jordan took his thermometric readings as a sign of poor response to injury – "the resulting shock was not severe; but reaction was late, and never marked" – he attributed her failure to tolerate the operation to constitutional and personality factors. "Her nervous system was weak, excitable, and unhopeful," he wrote. "Indeed, shock from psychical causes seems to have been present before the operation; and this probably was the cause

[231] Ibid., 167.

of the less marked change after the operation."[232] Another patient, who survived, was noted to have poor reaction and a prolonged recovery "owing, apparently, to the highly emotional temperature of the patient—an Irishwoman. More than once she said she was going to die."[233] Jordan's investigation of shock was less about elucidating a mechanism – as 20th-century researchers would attempt – than about creating a diagnostic and prognostic device. He did not question the origin or the propagation of shock, nor did he doubt the role of personal factors in the host response to injury. Rather, he looked for measurable data points within this framework, ones which were predictable, repeatable, and which fit the observed clinical phenomena. The mechanism was sound; in his interpretation, only the manifestations needed better definition.

Jordan also investigated shock through another quantifiable means, using the sphygmograph. Credited to Etienne-Jules Marey, the sphygmograph was similar to the modern sphygmomanometer in that it used a flexible diaphragm to measure pulsations over an artery, which were then transduced by a flexible arm onto a graphical medium.[234] While the sphygmomanometer provides numerical data – an indirectly measured blood pressure – the sphygmograph created a graphical depiction of the pulse wave, which

[232] Ibid., p. 165.
[233] Ibid., p. 167.
[234] Discussions of Marey's sphygmograph can be found in Karl E Rothschuh, *History of Physiology*, translated by Guenter B Risse (Huntington, NY: Robert E Krieger Publishing Company, 1973), 274; and in more detail in Reiser, *Medicine and the Reign of Technology*, 101-104. The pulse waves produced by the sphygmograph were graphical, permanent, concrete representations of otherwise fleeting physiologic data. Proponents of the device's use saw this as bringing a new objectivism and precision into medicine; physicians who were less convinced of the device's value argued that there was too much inter-observer variation in the acquisition and interpretation of this data, thereby limiting its value. This controversy suggests that the heuristic interpretive grid for such data did not yet exist as a consensus in the profession.

could then be analyzed with respect to its amplitude, frequency, slope, and the like. This tracing, in Jordan's estimation, provided useful data in assessing the secondary effect of shock upon the cardiovascular system: "There can be no doubt that while in shock, as a rule with very few exceptions, the impression of a severe injury is made first on the nervous system, most of the more marked phenomena are due to the secondary effects of the injury on the heart, and consequently on the circulation generally. Indeed, the degree of enfeeblement of the action of the heart will serve as a fair index of the reduction of vitality generally."[235] As with temperature, the sphygmograph provided reproducible, distributable, descriptive data. Although the meaning and significance of the various measurements applicable to analyzing a pulse tracing were open to interpretation, the information contained in the tracing itself was objective and quantifiable.

This objective character was something Jordan found extremely important. Clinical examination was useful, but the interpretation of the results struck Jordan as too variable from physician to physician. The sensation of a patient's pulse was not a piece of data that could be transferred in a pure form from person to person – it was available as primary, raw information to the examiner only, and as a second-order data point, pre-interpreted and necessarily with loss of fidelity, to anyone else. This, to Jordan, was fraught with imprecision: "The impression made on the finger by the pulse is so much less certain and decisive, that opinions formed on this basis possess only a relative

[235] Jordan, "On shock," 192.

value."[236] Six different clinicians, he argued, will give six different interpretations of any pulse; this in turn creates ambiguity and inability to effectively diagnose a disease. Graphical measurement, on the other hand, was more reliable: "The importance, then, of obtaining the sphygmographic characters of the pulse in every deviation from health is too obvious to require comment. The trace which I have given of the pulse in shock is peculiar, and differs from any other condition of the pulse."[237] The way to confirm a diagnosis was to identify the characteristic, measurable changes it effected upon living physiology. The graphical representation brooked no dissent, to Jordan, and erased the interpersonal variability of subjective pulse interpretation. This assessment of the graphical tracing – shock's peculiar pulse – is reminiscent of the Laënnec notion of finding disease in the lesion, and represents an attempt to apply the intellectual model of pathologic anatomy to a disease without a lesion.[238] It is important in that it is an appeal to pathologic *physiology*, an attempt to elucidate specifics of disease within a living system without the tissue-level changes. It looks to objectively assess function over structure.

[236] Ibid., 193.
[237] Ibid., 193.
[238] Laënnec's approach to clinical diagnosis was based upon the epistemological foundation that disease existed within the tissues as a discrete lesion. Mediate auscultation (i.e., using a stethoscope) was one example of a means by which direct, discernible evidence of a lesion could be identified in a living body, as opposed to in the cadaver at autopsy. This marks a significant shift in the approach to diagnosis, by privileging objective data identified by the physician over the subjective data as presented by the patient; this change in the physician-patient interaction would develop more fully over the course of the 19th century. For further discussion, see Reiser, *Medicine and the Reign of Technology*, 25-44; Foucault, *The Birth of the Clinic*, 160-161.

Jordan's explanation of the sphygmograph's utility in shock reflected his conceptual model. The nervous insult produced depressed cardiac activity, which in turn reduced blood flow through the body. Simultaneously, the "coldness" produced in shock led to capillary constriction that further diminished flow: "In shock, the impaired circulation in the capillaries is no doubt due mainly to the weakened propulsive power of the heart. That it is so altogether, is doubtful. The coldness which is present in shock probably leads to contraction of the smaller vessels and capillaries."[239] This arrangement is interesting in that it is an exact opposite of the model in favor fifty years later – and today, for that matter. Jordan posited shock was the inciting event that led to coldness, which led to capillary constriction, which led to circulatory impediment, which was the final common pathway toward patient demise. A modern read of the same patient – injured, cold, pale, pulseless – would interpret the injury leading to circulatory impairment (in particular from loss of blood), because of which the capillaries would constrict (to preserve blood pressure) and the pulses would diminish, leading to pallor and coldness that suggested the presence of a shock state. In this sense shock moved, between Jordan and 20th century authors, from an event to a state; from something which could incite a process to the end path of multiple processes.

As was the case with temperature, Jordan found shock-specific changes in pulse amplitude with his sphygmographic study: smaller amplitude of the pulse waves, more

[239] Jordan, "On shock," 192.

regular rhythm, and flatter descents.[240] Furthermore, sphygmographic tracings confirmed Jordan's temperature findings in the case of dividing bone, noting that the pulse amplitude dropped while the saw was in action, diagnostic, in his reading, of shock. Furthermore, the pulse amplitude recovered before the patient made a clinical return to normal, suggesting that the sphygmograph had greater sensitivity than clinical examination.[241]

The quantitative remaking of shock

Jordan's two quantitative assessments of shock fit nicely into Kuhn's model of the function of normal science: Facts obtained through observation are fit into and reinforce the existing paradigm.[242] The depression of the pulse in shock was not explained by Jordan in terms of a falling blood pressure, but rather as a cardiac failure; the cardiac failure in turn was not the primary pathology of shock but secondary to the failing of the nervous system. Jordan's use of technology produced no new knowledge – or, perhaps more specifically, produced no change in the way knowledge was applied to the problem of shock – but it did foreshadow coming changes in medical practice. The use of the sphygmograph, in particular, convinced Jordan of the value of physiological measurement as a part of medical practice: "It is not necessary for me to dwell on the advantages to be derived from the use of the sphygmograph in medicine and surgery. …

[240] Jordan, "On shock," 192.
[241] Ibid., 193.
[242] Kuhn, *Structure of Scientific Revolutions*, 35-42.

The impression made on the finger by the pulse is so much less certain and decisive, that opinions formed on this basis possess only a relative value." Unlike clinical examination, the sphygmograph was "clear, accurate, and beyond dispute," and Jordan envisioned a time when it would be used to diagnose all manner of diseases.[243]

Interest in the sphygmograph, at least with regard to shock, didn't really take off after Jordan. One prominent clinical lecture on shock in 1882 mentioned sphygmography only in passing, as a reference to Jordan's good luck with its use.[244] Interestingly, though, George M. Beard's treatise on neurasthenia brought up the use of sphygmography as a means of assigning a quantitative value to a functional disorder, remarking that the "sphygmograph is an aid in determining the amount of exhaustion; and by comparisons of tracings taken at intervals, the progress of the patient towards recovery can be estimated."[245] Beard faced a challenge with neurasthenia similar to that which his surgical contemporaries had with shock: as a functional disease, neurasthenia was something of a medical second-class citizen to the more broadly accepted and narrowly defined structural diseases.[246] By seeking to define the physiological changes of neurasthenia through the use of a quantitative medium, Beard was engaging turn-of-the-century medical epistemology on its own terms – even as he pleaded for a special status for neurasthenia as a functional disorder, he aimed to prove its existence through an accepted means of physiological measurement.

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[243] Jordan, "On shock," 193.
[244] McClellan, "Clinical lecture on shock and surgical fever," 813.
[245] Beard, *A Practical Treatise on Nervous Exhaustion (Neurasthenia)*, 87.
[246] Beard, *A Practical Treatise on Nervous Exhaustion*, 23-33.

While shock's existence was never in doubt, its nature remained elusive. Researchers after Jordan continued the practice of investigating shock through the use of clinical measurements. The gradual trend across the latter half of the 19th century was incorporation of more and more elements of physiologic measurement into the narrative – case reports more often included measured temperatures and heart rates, for example, as the 1900s approached.[247] The chief avenues of investigation, roughly chronologically, can be thought of as temperature measurement, cardiovascular measurements (heart rate and blood pressure), and assessments of blood chemistry. These categories are somewhat arbitrary, and by no means sharply distinct or representative of a progression from primitive to modern. What they do show, however, is the influence of physiologic measurements on disease identity; through them, ultimately, shock's nature becomes something expressible as a quantity rather than as a quality.

Clinical thermometry and shock

W.W. Wagstaffe, demonstrator of anatomy at St. Thomas's Hospital, London, published an investigation on the use of temperature in shock in 1870. Shock, to Wagstaffe, was "merely the expression of an effect upon the system." Given that other systemic effects – "exposure to cold, sustained mental exertion, alcohol, or a large meal when first taken" – could depress the temperature, he proposed that temperature could play a role in shock.

[247] An example is found in Geigley, "Notes from private practice: Art. I – Case of shock," 224.

He investigated thermometer readings on patients admitted to the hospital after various causes of shock, enumerated as "injury to sensitive nerves, as exemplified by burns, scalds, and skin and bone injuries," injuries to the brain and spinal cord, visceral injuries, poisonings, and hemorrhage. Though he was unable to confirm Jordan's data on temperature drop during the sawing of a bone, he determined that in fatal cases of shock the temperature fell to a greater degree than in survivable shock. Temperature thus had prognostic value, with risk of death proportional to the magnitude of the fall, but could not be used as a criterion to either define or diagnose shock.[248]

The use of thermometry as a prognostic aid in shock seemed to find some purchase in general practice. Geigley's case report of the horse-kicked farmer mentions a temperature drop to 94° F, a "fearfully low temperature" associated with "complete collapse" of the patient, marking it an "apparently hopeless case."[249] Another case report from Paris described the use of temperature as a guide to improving patient condition: a patient whose legs had been crushed by a train seemed to be recovering from his prostration based upon a rising temperature.[250] Other authors took a falling temperature as a sign of the magnitude of shock itself. Rhu argued that "all surgeons are agreed that it is in no sense justifiable to operate on a person the subject of severe and profound shock, and with a Temp. below 97 degrees F," the idea being that vitality was so depressed at that

[248] W.W. Wagstaffe, "On the temperature of shock in surgical cases," *St Thomas's Hospital Reports* 1 (1870, new series):465-483.
[249] Geigley, "Notes from private practice: Art. I – Case of shock," 224-225.
[250] M. Duret, "Plaie par écrasement des membres inférieurs; mort rapide; lésions des vaisseaux," *Bulletins de la Société Anatomique de Paris* 10(1875):317-318.

point an operation would be nonsurvivable.[251] Physiologically speaking, the temperature and the heart were seen as connected – as the body cooled, the heart lost power; thus awareness of falling temperatures and steps to correct hypothermia became central components of treating shock.[252]

Research performed at the beginning of the 20th century asserted that hypothermia could, in and of itself, cause shock. Guy C. Kinnaman, a surgical fellow at Rush Medical College, performed a series of experiments on dogs to investigate the role of temperature in shock. Kinnaman wrote that helping in his father's surgical practice put him onto the idea; specifically, he remembered that if injured patients were allowed "either from negligence or non-discovery, to lie upon the ground for any length of time, shock in every instance was much more severe than if the patient were immediately put upon a board or cot and well covered with blankets." Kinnaman's interest in warmth is couched in biochemical terms – normal temperature is necessary, in his view, to maintain the chemical processes of normal cellular physiology – and hypothermia takes on an etiologic, propagative role in shock itself rather than acting as a surrogate for cardiac power. He thus wanted to investigate the relationship of temperature to shock, to examine whether temperature data from shock patients could thus have prognostic value. His experiments were all predicated upon the belief that exposure of the intestines to air, as would occur in a surgical laparotomy, produces shock. Handling of the bowels was posited to worsen the problem. For his study, he anesthetized a series of dogs, surgically

[251] Rhu, "Shock," 8.
[252] Mansell-Moullin, "Shock," 371.

opened their abdomens, and produced shock either by spreading their intestines out while exposed to air, by submersing the dog and exposed bowel in warm water, or by "vigorously manipulat[ing]" the exposed viscera until shock developed before plunging the dog into a warm tub. All of the dogs in the air-only group died of shock, whereas he lost no dogs from the warm water groups to shock. He interpreted this result as indicating that shock was associated with a fall in body temperature, and that there was some proportionality between temperature and shock severity: "A fall of 5.7° C., with or without manipulation, leads either to death from shock or to a condition of fatal shock." Further, since actively rewarming the dogs led to an abatement of shock symptoms, the hypothermia must have had some role in either causing or perpetuating shock. Hence, he surmised, "temperature commands first place by its power of production, by its power of limitation, and by its power of amelioration of the composite condition, – shock."[253]

Kinnaman's study is interesting in that despite its very quantitative mode of expression and investigation, it retains an underlying paradigm of vital power. Heat, instead of being a measure of some nebulous life force, is the driving force behind the chemical reactions that characterize life; depression of this heat leads to failures of these reactions and ultimately extinguishment of life itself. His inspiration for the study – memories of cold patients falling into deep shock – gives a clue to this worldview: There is no necessary, obvious reason to select coldness as the attribute that made his father's trauma patients fare poorly, any more so than selecting "dampness" or "delay of care" or "left on the

[253] Kinnaman, "An experimental research," 843-877.

ground because they were too badly hurt to be moved safely" as the cause. Further, the experimental data could have been interpreted differently – exposing the dogs' abdominal cavities and intestines to room-temperature air could have been taken as the cause of falling temperature, rather than shock. The presumption of a relationship between temperature and shock here was a conscious choice, further reinforced by seemingly favorable data.

Kinnaman's essay marks the last major investigation of temperature and shock, at least insofar as any postulated cause-effect relationship was concerned. Once a ubiquitous description of the shock patient, "cold" began to take a backseat role in shock to the vascular system. While some authors continued to cite cold as a cause of shock – as in a 1911 article in *BMJ* that warned that the use of cold intravenous solutions may cause "no small degree of shock rather than any benefit" – most instead saw it as a contributing or complicating factor to pathology initiated by another source.[254] "I have no doubt that a low temperature is one of the most powerful aggravating causes of shock," wrote British surgeon John Malcolm in 1905, though he felt it led to over-constriction of the already clamped-down blood vessels, rather than sapping the body of energy in and of itself.[255] To Walter Cannon a decade later, cold affected the rheostatic and chemical properties of blood: The falling temperature in shock – now produced by exposure to the cool environment, rather than by shock itself – led to increased blood viscosity, impeding flow

[254] Quoted from Harold K. Waller, Gerald Walker, "The management of epidemic summer diarrhoea and vomiting, including the use of saline injections," *The British Medical Journal* September 16, 1911:594-595.
[255] John D. Malcolm, "A lecture on the condition of the blood vessels during shock," *Lancet* 1905 (2):578.

in the capillaries and leading to sludging of red cells, loss of plasma, and interference with the chemical reactions related to oxygen exchange.[256]

From feeble and fluttering to tachycardic and hypotensive – Circulatory measurements and shock

The heart and blood vessels were presumed to participate in shock from the earliest works on the subject, even if their exact role was unclear. Investigation of the circulation, as we have discussed, was initially performed through assessment of the pulse. In general, this was performed as a qualitative description of the speed and strength of the pulse that corresponded, in the mind of the examiner, to the vigor of the heart. There was no real consensus as to the relative value of a numerically determined pulse rate versus a qualitative sense of the pulse's rate in the mid-19th century. Indeed, debate on this subject had been going on for centuries before this point: Was the rate what was truly important, as opposed to the interval between beats, or the regularity of beats, or some other aspect of the physical sensation experienced by the examining finger? Clearly, the decision wasn't predicated on the availability of technology for pulse-counting – reports of using water clocks to measure the rate quantitatively date back to the fourth century B.C.E.[257]. Instead, the measurement of circulatory function in a quantitative sense depended upon physicians deciding they needed to do such a thing. As we have discussed, attempts to

[256] W.B. Cannon, "A consideration of the nature of wound shock," *Journal of the American Medical Association* 70 (1918):615.
[257] A review of pulse-related thought can be found in Reiser, *Medicine and the Reign of Technology*, 95-98.

quantify the heart's power began as early as the 1860s with the use of the sphygmograph, which ultimately changed little in terms of the conceptualization of shock. Riva-Rocci's sphygmomanometer, on the other hand, changed everything: The ability to easily, noninvasively, and portably measure blood pressure seemed to pique the interest of researchers, and within a decade of its introduction in 1896, shock was being described in hard terms of low arterial blood pressure rather than the more qualified language of circulatory weakness.[258]

In a neurological model of shock, heart rate was interpreted as an indicator of the power of the nervous system; given that the model for shock was one of global depression and slowing of function, the logical outcome for the heart rate was that it must be slowed in response to injury. The reason cited by Jordan for this phenomenon was predicated on the structural-anatomic fact that the heart is innervated by nerves which trace both to the nervous ganglia near the spinal cord (the sympathetic, or spinal nerves) and to the brainstem (the vagus, or pneumogastric nerves). The nerves worked in opposition to control the rate of the heart: "The physiological action of the pneumogastric seems to be, most singularly, to retard or inhibit cardiac action, and thus to oppose the influence of the ganglionic and spinal nerve-stimuli." Shock was presumed to be communicated to the heart through inhibition of the spinal nerves or stimulation of the pneumogastrics, leading to slowing of the heart rate and enfeeblement of the pulse. A relative rise in the heart rate indicated the shock had passed, and recovery was underway. As the depressive effect of a

[258] Ibid., 105.

shocked nervous system lifted, Jordan explained, "the pulse gradually succeeds in the attempt to compensate for its weakness by increase of action." The degree of this rise in rate was proportionate to the depth of the shock and severity of the injury.[259]

Overall, the significance of a numerical heart rate to 19th-century surgeons is unclear. Although Jordan assigned clinical value to the heart rate in relative terms, he valued its quantification less than he did that of the temperature. Morris's 1867 monograph on shock treated cardiac function in purely relative terms of strength and power; Easley likewise didn't mention the heart rate at all, concentrating instead on qualification of the heart's impaired vitality.[260] Rhu mentioned that "as a rule the Temp. and Pulse are our best guides to determine the severity and danger of shock," though it is unclear whether he was primarily concerned about the pulse's speed so much as he was its quality.[261] When heart rate was described in numerical terms, it often came in the context of using changes in pulse rate to follow patient progress. For example, Geigley's report described the patient's pulse rate as diminishing from 160 beats per minute during deep shock to 105 beats per minute by post-injury day 2, by which time the patient had mostly recovered – notably opposite to the Jordan notion that shock was identified by a slower rate.[262] Another report, from 1883, claimed superiority of digitalis to standard treatments of shock using normalization of the heart rate as evidence: The pulse, described as "feeble, just perceptible at the wrist, 165" in a patient whose "condition [was] evidently

[259] Jordan, "On shock," 258-259.
[260] Morris, *Practical Treatise*, 16-18, for example; Easley, "A study of shock," 519-523.
[261] Rhu, "Shock," 82.
[262] Geigley, "Notes from private practice," 224-225.

one of threatening dissolution," failed to respond to brandy, morphine, and ammonia, but fell to an easily palpable 108 after a hefty dose of digitalis.[263] Again, though, the change in quality of the pulse – feeble and just perceptible becoming easily palpable – holds as much importance as the rate.

By the early 20th century the prominence of the heart itself began to fade in discussions of shock, replaced instead by interest in the arteries and veins. Correspondingly, blood pressure began to assume the dominant role in investigations of the circulatory system. The availability of a means to noninvasively and easily measure blood pressure likely contributed to this shift in focus, but a changing medical epistemology – one colored by the Progressive era, which valued scientific precision – is what gave it meaning. "It by no means behooves us to disparage the value of an educated touch as a means of estimating vascular qualities," wrote Harvey Cushing in 1903, "but the tactile and muscular sense, no matter how well trained, must give way to some method more precise."[264] Interpretation of the pulse was the quintessential clinician's skill, something abstract, internal, sensory, and open to inter-observer variability. Blood pressure, as measured by the sphygmograph, was accurate, reproducible, and objective: "In place of the loose and indefinite terms applied to degrees of tension one learns to interpret them with some measure of numerical accuracy, and recognizes a 'weak' or 'compressible' pulse as one with a tension perhaps of 80, a 'hard' or a 'bounding' pulse as one, for instance, of

[263] F. Cauthorn, "Digitalis in shock," *The College and Clinical Record* 4 (1883): 195-196.
[264] Harvey Cushing, "On routine determinations of arterial tension in operating room and clinic," *Boston Medical and Surgical Journal* 148 (1903):250.

260."[265] Cushing, who attended medical school at Harvard and undertook his surgical training at Johns Hopkins, would have spent time pursuing physiological laboratory research during his medical education.[266] His valorization of quantifiable, precisely measured blood pressure is reflective of a contemporaneous movement in surgery to more closely ally physiological principles with clinical practice. Monitoring of the patient's intra- and post-operative vital signs, hemoglobin levels, urine output, and other physiological markers became accepted standards of surgical care in the early 20th century.[267] "The mechanics of surgery," wrote American surgeon George W. Crile in 1905, "long ago reached [their] zenith." Instead of drawing upon the static sciences of anatomy, pathology, and bacteriology, he continued, modern surgery was dynamic, drawing its knowledge from an understanding of the function of the living system: "Surgical practice rests largely upon altered physiologic actions, or upon surgical physiology."[268]

Crile, for his part, is generally remembered as the individual who inextricably linked blood pressure and shock. Declaring in 1903 that "control of the blood pressure is synonymous with the control of life itself," Crile performed a series of experiments in which laboratory animals were subjected to shock-producing injuries. In his published results that year, Crile noted that the most consistent finding in shock was a reduction of

[265] Cushing, "On routine determinations of arterial tension," 250.
[266] Particulars of Cushing's life from Elizabeth H. Thomson, *Harvey Cushing: Surgeon, Author, Artist* (New York: Henry Schuman, 1950), pp. 39-93.
[267] For a brief review of the physiological surgery movement, see English, *Shock,* 34-37; see also Joel D. Howell, *Technology in the Hospital: Transforming Patient Care in the Early Twentieth Century* (Baltimore: Johns Hopkins, 1996), pp. 93-94.
[268] George W. Crile, "Surgical physiology," *Bulletin of the Johns Hopkins Hospital* 16 (1905):269.

blood pressure.[269] Though his methods were criticized broadly in the physiology community, apparently Crile's results were compelling: Even his detractors conceded the point to him, focusing their critiques instead on Crile's proposed mechanism. British surgeon John Malcolm, for example, noted that, based on Crile's work, "the most popular theory at present, however would appear to be that the essential factor in the production of the condition of shock is a fall of blood pressure." But this is the last point of accord before Malcolm takes Crile to task on a point-by-point basis, using the American's own data to support a completely different interpretation of shock physiology.[270] Similarly, later writers didn't always feel that arterial hypotension was an absolute requirement for shock, though they tended to grant it a central role when it did occur.[271] Other investigators used arterial hypotension as a marker for disease severity – suspicion of shock was raised by clinical findings, and then its severity categorized by blood pressure.[272] The key point, however, relates less to the lack of consensus on mechanism than it does to the shift in standards of proof: The older language of arterial tension, cardiac weakness, and vascular failure had dissipated by 1905, and after this point discussion of shock in the literature is essentially a discussion of the blood pressure.

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[269] George W. Crile, "A research into the means of controlling the blood pressure," *Boston Medical and Surgical Journal* 148 (1903):247-250; English, *Shock, Physiological Surgery, and George Washington Crile*, 88-89.
[270] Malcolm, "A lecture on the condition of the blood vessels during shock," 573-579.
[271] See Henry H. Janeway and Ephraim M. Ewing, "The nature of shock," *Annals of Surgery* 59 (1914):158-159; Archibald and McLean, "Observations upon shock," 285.
[272] Cannon, Fraser, Hooper, "Some alterations," 527.

Chemical assessment of shock

By the early 20th century, chemical analysis was certainly not a new technique in the investigation of disease. Reports in the *Lancet* from London's 1832 cholera epidemic, for example, show researchers attempting to discern some useful bits of data via laboratory assays. W.B. O'Shaughnessy, in particular, found that something of a balance existed between the choleric flux and the patient's peculiar serum abnormalities: The blood "has lost a large proportion of its water" and "neutral saline ingredients," he reported, and furthermore, "all of the salts deficient in the blood, especially the carbonate of soda, are present in large quantities in the peculiar white dejected matters."[273] The blood, according to O'Shaughnessy's contemporary Thomas Latta, was in a "thick, black, cold state," after these losses; this pathologic situation led to the "most distressing symptoms" and the death of the patient.[274] O'Shaughnessy described his analytical approach as "chemical pathology," allowing for the disease to be identified by a chemical signature that could be seen in the living patient. That cholera made some mark that could be detected chemically before it was detectable pathologically suggested that a finer level of investigative focus was available to the researcher than the gross clinical findings and postmortem changes of cholera.[275]

[273] O'Shaughnessy, "Experiments on the blood in cholera," 490.
[274] Latta, "Malignant Cholera," 274-275.
[275] W.B. O'Shaughnessy, "Chemical pathology of cholera," *Lancet* 1831-1832(2):225-232.

This sort of thinking foreshadows post-1900 investigations into shock. Attempts to elucidate physiologic mechanisms of shock increasingly incorporated biochemical assays after the turn of the century, and laboratory abnormalities were likewise invoked as contributing to more effective diagnosis and treatment of the condition. Measured findings in the blood of patients suffering from shock included lowered pH (reflective of loss of buffering capacity of the blood, or accumulation of acids), increased blood viscosity, changes in the size of red blood cells, and a gradient in the hematocrit between central vessels and capillary beds that suggested marginalization of red blood cells to the periphery; acidosis was by far the most-studied and most-discussed parameter.[276] We will discuss this in further detail later.

Quantity, quality, and shock

The transition of shock from a clinical-qualitative syndrome into a quantitative phenomenon roughly parallels the change in medical epistemology over the 19th century. As the Paris model that revolutionized clinical medicine in the early part of the century gave way to a more German-influenced system, the medical profession's sense of self shifted: instead of identifying as clinical in nature, it began to view itself as scientific. Interestingly, in both cases members of the medical *avant-garde* saw themselves as making medicine more objective – first by privileging clinical signs over patient

[276] Cannon, "A consideration of the nature of wound shock," 611-617; Cannon, Fraser, and Hooper, "Some alterations," 526-531. The hematocrit is the proportion of solid components to liquid components in the blood; expressed as a percentage and determined by centrifuging a sample of whole blood to force the cells to settle out of the serum.

complaints, later by granting more value to numerical physiologic measurements over these same clinical findings. [277]

This central change in the concept of how medicine was practiced in turn changed the identities of diseases themselves, and shock was not the only disease entity to shift from a clinical-qualitative phenomenon to something quantified by physiological measurement during the Progressive era. The diagnosis of anemia, for example, moved from a clinical decision made on the basis of pallor to a confirmation by laboratory assay demonstrating deficiencies of hemoglobin concentration and red blood cell count; likewise a positive Widal reaction (an immunologic assay) became the definitive diagnostic sign of typhoid fever.[278] The identity of diabetes changed from a disease of ceaseless urination – "a melting down of the flesh and limbs into urine," as described in the first century C.E. – to, as Michael Bliss noted, "a condition in which glycosuria exists."[279] The acquisition of an associated quantifiable abnormality could function to bolster arguments for legitimacy of functional diseases. George Beard, for example, claimed increased urinary oxalate, urate, and crystals as well as albuminuria as diagnostic of neurasthenia; his colleague

[277] For discussions of the rise of scientific medicine, see, for example, John Harley Warner, *Against the Spirit of System: The French Impulse in Nineteenth-Century American Medicine* (Baltimore: Johns Hopkins, 2003) 304-315; Paul Starr, *The Social Transformation of American Medicine* (United States: Basic Books, 1982), 134-141; William G. Rothstein, *American Physicians in the 19th Century: From Sects to Science* (Baltimore: Johns Hopkins, 1985), 323-326.
[278] Howell, *Technology in the Hospital*, 191-193, 201-205.
[279] Michael Bliss, *The Discovery of Insulin* (Chicago: University of Chicago Press, 1982), 20-21. Glycosuria is the presence of glucose in the urine.

A.D. Rockwell added rapid fluctuations in the blood pressure and an abnormal sphygmograph trace as further evidence.[280]

Medical interest in shock arose during a time when disease was constructed and understood through a model of bedside observation and pathological correlation. The earlier writings on shock reflect this model – shock was a disease of depression, expressed in a language of signs reflective of this central disruption: feebleness, shrinking, coldness, collapse, dissolution. Though the qualitative description of physical signs was not new to the 19th century, the underlying grammar of the signs was – Laënnec's pectoriloquy and egophony were not merely adventitious lung sounds, they were physically perceptible representations of an underlying pathological process.[281] Shock refused to submit to such an assessment, however; it offered a multitude of clinical signs in the utter absence of pathologic findings. It thus remained through much of the century a purely descriptive entity – stereotypical, identifiable, predictable but not easily classifiable.

The shift in the medical consciousness to favor measurement over examination in the latter half of the century brought new means of describing and recording clinical phenomena that rendered information previously accessible only as sensory input into new forms. Through the medium of the sphygmograph, for example, the pulse moved from something experienced only through touch into something visible and quantifiable.

[280] Beard, *A Practical Treatise on Nervous Exhaustion*, 85-87, 102-103, 174.
[281] Foucault, *Birth of the Clinic*, 160.

Further, it became universal and standardized: No longer did each individual clinician experience a patient's pulse only through his own senses, instead the graphical representation of the same data was available to all.[282] The interpretation of physiologic parameters gained a degree of transparency, if nothing else: Rather than discussing a pulse in a manner not unlike discussing the characteristics of a glass of wine – hard and bounding, a trifle fast, a bit fluttery, a note of threadiness … or perhaps of narrowness? – the transcribed curve of the pulse was a representation that could be discussed in terms – a slope of *x* degrees, a maximal height of *y*, a dicrotic notch of depth *d*, a descent of *z* – that lent themselves to less inter-observer variation (or, even if the interpretation was open to argument, the measurements themselves were there for everyone to see). Appropriately, as this standard of evidence changed, the identity of shock changed. As medicine invested more meaning into those things that could be quantified and mathematically analyzed, shock became something that was expressed as a phenomenon of aberrant or deranged function; it could be put into seemingly more objective quantifiable terms. Although it still defied being categorized as a disease of structure, identified through application of pathological anatomy, it could now be diagnosed through its identity as a disease of function, diagnosed, described, and defined through physiological measurement.

[282] Reiser, *Medicine and the Reign of Technology*, 95-104, reviews this conversion.

Chapter 5 – Shock as a moral phenomenon

In the 19th century, physicians conceived of the clinical manifestations of shock as the result of interactions between the central nervous system and the cardiovascular system. Stimulus was applied to the periphery, a signal crossed through a relay point in the brain, a peripheral effect followed. The brain, however, was not only a passive recipient and processing center for outside information; it was a source of stimuli on its own. Therefore, the intelligence, the emotions, the thoughts and ideas of a patient – the mind, in other words, or the "psychical apparatus" – were thought to be capable not only of influencing the course of shock, but of producing shock independent of any physical injury the patient incurred. In a time when the medical profession increasingly tried to characterize shock in terms of measurable data, a concurrent and contemporaneous theoretical framework looked at shock more in terms of the emotions and the inner life. The nervous system, in this construction, becomes less of a simple relay network for shock and more of a producer of physiologic reality in its own right. In addition to centers of power burning out and simple reflex inhibitions being suspended, the very chemistry, tissue composition, and biologic function of the organism were fundamentally altered, solely through the agency of thought or emotion.

In this sense, shock could be seen in terms of a moral phenomenon. Looking at shock through this lens provides a different set of insights than when shock is considered purely as a medical condition. We get a sense of the ideas physicians held about human life and thought, about how mind and body were related, about human frailty and resilience.

Shock becomes able in this context to serve social as well as medical functions. In the setting of moral/emotional shock, socially normative values could be ascribed protective roles, surgeons could call upon different standards of evidence for causation and diagnosis, and surgical practice could be influenced. It is in this guise that shock closely resembles neurasthenia, another late 19th-century disease of function that suffered from a difficult lack of pathologic findings despite its ubiquity in the clinical setting.

"Sorrow, uncomplicated, sometimes kills outright"

Mimicking the standard template, E.T. Easley's posthumously published treatise on shock begins with a lament as to the poorly understood nature of the condition. "No definition of shock," he wrote, "has apparently been given which is sufficiently precise and yet so comprehensive as to embrace all its phenomena." Shock, to Easley, was imperfectly understood, and surgeons seemed little inclined to address the problem.[283] As evidence, he cited the plethora of shock definitions in the literature – "fatal sinking," "constitutional irritation," "general depression of vital powers," and, perhaps most illustrative of Easley's own thinking, "the harmony of action of the great organs of the body becoming deranged."[284]

Easley was a surgeon who practiced in Little Rock, Arkansas. Born in 1842 and educated by private tutors, Easley volunteered as an infantryman in the Civil War. Captured at

[283] Easley, "A study of shock," 414-415.
[284] Easley, 414.

Cold Mountain, he spent the remainder of the war in a Union prison camp. Following the war, Easley attended the University of Louisville medical school, from which he graduated in 1870. In his early career, Easley became a prolific writer, publishing on amputations, orthopedics, gynecologic operations, sanitation and public health, and pharmacology. He was rapidly rising to professional prominence as well, serving two terms as the secretary of the American Medical Association section on Surgery and Anatomy, before his untimely death from yellow fever in the Memphis epidemic of 1878.[285] He became something of a romantic character in the Southern medical literature after he died; that he contracted the disease while working as a volunteer physician in a relief effort for the embattled city only served to add to this aura.[286]

It is somehow fitting, then, that the Easley memorialized as a tragic hero would conceive of shock in a far more Romantic sense than many of his contemporaries. His monograph is notable for its allusions to literature and poetry and the prominent role it allows for personal characteristics, temperament, and character to enter into the understanding of shock physiology. Basing his physiologic principles on the contemporary, vitalist-inspired view that the human organism carries a finite amount of life energy, he took a more rationalist than empiricist approach to shock and appealed to common sense as much as to clinical observation to make his points. Easley sounds archaic, or at least anachronistic, in his late 19th-century context, as his contemporaries focus on clinical

[285] Julius F. Miner, ed., *The Buffalo Medical and Surgical Journal*, 1877 (8):391; William Biddle Atkinson, ed., *The Physicians and Surgeons of the United States* (Philadelphia; Charles Robson, 1878), 347.
[286] J.M Keating, *History of the Yellow Fever Epidemic of 1878* (Memphis, TN: Howard Association of Memphis, Tennessee, 1879), 389.

parameters, laboratory values, and numerical physiologic metrics. Instead of these systems, Easley argued, shock – a necessary part of the human condition, existing through our own frailties and circumstances – was best understood through metaphor.

Shock, to Easley, was a mind and body phenomenon, and while the two were inextricable, the mental and nervous aspects were key: Shock was a "condition of suddenly depressed vital or nervous action, produced by varied causes, of greater or less severity and duration, and not invariably associated with violence to the physical system." This somewhat prevaricating description contains in it a very important clause, namely that physical injury – while potentially devastating – was not necessary for the production of shock: "It is safe to say that every case of shock, however transitory or important, is attended with more or less mental depression or excitement, and that it may exist to a dangerous or fatal extent when corporeal structure is not appreciably involved."[287] Furthermore, the severity of shock was not necessarily proportional to the power of the inciting stimulus – indeed, a hallmark of shock was its propensity to occur to a fatal extent in seemingly trifling injuries or emotional strains.

Easley argued four possible etiologies for shock: "(1) causes predisposing or remote, (2) those which are exciting and direct, (3) such as effect chiefly the brain and its nervous centres … (4) those which operate by some lesion of structure of the physical system."[288]

[287] Easley, 415.
[288] Ibid., 416.

Functionally, this collection reduces to a susceptible host receiving a sufficient stimulus, either mental or physical.

The remote causes of shock, which established susceptibility, had a certain amount of predictability. Easley believed age to be a strong predictor of shock – "as life moves on beyond its meridian, the mortality list from disease and accident swells in a corresponding ratio" – though he did not feel the same about sex. "Women, apparently very fragile," he wrote, "frequently display an astonishing degree of nerve and recuperation."[289] Racial characteristics were far more important to Easley's conception of shock; this racial model was closely intertwined with an almost Hippocratic subtext that stressed the interplay of climate and parentage. Treading a well-worn path, Easley espoused the virtues of "bracing and invigorating air" over living in a "warm, miasm-infected region," noting that "the inhabitants of torrid climes, although vindictive and treacherous, are notoriously timid and destitute of moral or physical courage," while the "North American savage," denizen of a temperate climate, displays a "stolid heroism."[290] Easley directed a fair amount of attention toward the health of the newly liberated slaves of the American South: "the "Negro," he wrote, is "more profoundly depressed by shock than any race of people on the face of the earth."[291] Perhaps this is not surprising, considering Easley's upbringing in the deep South and his coming of age during the Civil War. In contradistinction to his admiration of the rugged individualism and free spirit of

[289] Ibid., 417, 423.
[290] Ibid., p. 418.
[291] Ibid., 418-419.

the American Indian (and their associated salutary effects), Easley somewhat bitterly remarks that emancipation has made African-Americans weaker, inhibiting their ability to heal wounds, mount a rally against injury, or recover from shock.[292] From a practical standpoint, he argued, this meant that black patients should be treated with more aggressive therapies much earlier in the course of their disease.

Personal characteristics extended to the ability to tolerate surgical operations. Easley noted that the chance of fatal shock supervening on operation rested chiefly upon patient-dependent factors – their habitus, their habits, their general health, their disease state, and, chiefly, their state of mind. "He who is cheerful and hopeful in submitting to the knife," Easley wrote, "has vastly the advantage of him who is in an opposite or desponding frame of mind." Just like for the soldier, whose chances of surviving a battlefield wound depended upon his patriotism and steadfastness, the civilian surgical patient required that socially normative element of courage to bear up to his burdens.[293] As evidence, Easley stated the apparently well-known fact that lunatics had few surgical complications, because "the moral element in this case is eliminated" – 'moral' taken here in the sense of courage, perseverance, and fear. Essentially, people who can't worry about the outcome of their operation do not sink themselves into shock through fear. Children, on the other hand, had just the opposite situation – inconsolable, easily chilled, intolerant of

[292] Inid., 419.
[293] Ibid., 513.

hemorrhage, and unable to stand up to pain – and were thus defined by Easley as particularly susceptible to shock.[294]

While a person's heritage and temperament were beyond willful control, habits and proclivities were not, and in Easley's model these also played a role in shock. In a setting where thoughts had the capacity to manifest as physical symptoms and physiologic dysfunction, the ability to bear up and control the emotions in the face of adversity – disease, injury, or the threat thereof – had obvious power. Consistently, the personal virtues protective against shock were the virtues considered ideal for a man in the author's social circles. For Easley, these included patriotism (which allowed a wounded soldier to survive injury better than a civilian), self-control in the setting of both temperance and diet (as the sober and thin were stronger than the intoxicated or obese), and stoicism (as the anxious and irritable "suffer terribly from shock"). Conversely, deviation from socially normative behaviors – essentially exhibiting a lack of discipline and restraint – led to danger: "Men who have pursued convivial or intemperate habits for a long period, addicted to dissipation and late hours, have their nerves so weakened, and all the solids and fluids of the body so depraved and disorganized, that traumatism wins an easy victory."[295]

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[294] Ibid., 514.

[295] Ibid., 420-421. For further discussion of what constituted "manly" ideals to a 19th-century Southerner, refer to Stephen W. Berry II, *All That Makes A Man: Love and Ambition in the Civil War South* (Oxford: Oxford University Press, 2003), especially pp. 17-43. A similar discussion of British ideals is found in Adam Nicolson, *Seize the Fire: Heroism, Duty, and the Battle of Trafalgar* (New York: Harper Collins, 2005).

While a person's response to a shock-inducing stimulus could be predicted to some degree by their associated demographic factors, idiosyncrasy often played a frustrating confounding role. Easley noted that "of two men of apparently similar health and habits the one will linger long and doubtfully under a depression which the other will overcome with ease."[296] In practice, this allowed for the physician to explain deaths from shock that otherwise failed to fit the model – even Gross noted that courageous men could be laid low by a trifling wound.[297] The key feature that made an individual more susceptible to shock was a depleted physiologic state, which functionally translated into a lack of sufficient nervous energy to bear up under the shock. Nervous energy, of course, was the domain of the mind.

Chief among the direct mental causes of shock was the emotional state. That "joy, grief and terror may produce every degree of mental or nervous shock, is a fact both rational and well attested," Easley argued, commenting that "sorrow, uncomplicated, sometimes kills outright."[298] Citing Shakespeare, he noted that King Lear's death at his final, tragic moment of recognition and clarity is an example of emotionally induced shock. In fact, he argued, the simple fact that so many writers explore the themes of collapse and death from emotional causes in literature is ample evidence that such a physiologic phenomenon actually exists. This is quite a departure in terms of standard of proof and epistemological approach from the thermometric and plethysmographic experiments of

[296] Easley, 417. "Depression" here refers to shock, in the sense of "sinking" or "depletion."
[297] Gross, *System of Surgery*, 431.
[298] Easley, 424

Furneaux Jordan or the subsequent clinical-laboratory models of Crile and Cannon. Fear, ultimately, was the enemy; not only was it not a socially normative emotion, it wore patients out, prostrating them, using up the energy and nerve force that could be applied to recovery: "When a man has passed *through any imminent deadly peril* or has been in mortal dread of his life, although no physical harm be done, more or less collapse or prostration of all his energies ensues."[299] Fear of death could ruin an otherwise uncomplicated convalescence; fear "impair[ed] the integrity of glandular products and reparative material"; fear could produce a sort of "contagious shock," especially in the hospital situation, where the wounded man watched other wounded men suffer and die.[300] Fainting at something frightening was a "fine example of the effects of shock," a sort of *petit-mort,* showing the powerful effect of emotion, just in a transient, less severe way.[301] Pain itself could apparently produce shock, as Easley cited neuralgia as a cause. Importantly, nonphysical causes produced death "with no textural alterations" whatsoever, running contrary to the very defining concept of disease in the post-Laënnec, post-Bichat worldview of pathologic medicine.[302] Shock could thus be real but undefinable, existing in the clinical sphere but not the anatomic. Perhaps recognizing this quandary, Easley argued that a pathologic change was possible, but medicine simply didn't understand what it was.[303] But even in the absence of discernible diagnostic changes, Easley argued that shock was real, and if "we exclude as causes all wounds but

[299] Easley, 426.
[300] Ibid., 422-425.
[301] Ibid., 426.
[302] Ibid., 426.
[303] Ibid., p. 521.

such as show a visible mortal lesion, we shall have a large number of deaths undoubtedly due to violence unexplained."[304] Common sense and experience taught the surgeon that shock could kill without leaving a mark.

Easley was unique among his contemporaries for the strong importance he placed upon the emotional ramifications of shock – both in relation to etiology and to pathology. Humans had emotions, emotions were the product of the mind, and the mind was the ultimate arbiter and shaper of human reality: "In our view of the subject, every distress, mental or physical, is accompanied by corresponding shock; for as we can not understand a body without sentient nerves, so it is impossible to conceive a nature so callous as to be indifferent to those calamities and griefs which affect the moral feelings."[305] Human beings respond to noxious stimuli, including thoughts, because they are *supposed* to; nature dictates it to be so. The mind had such power over the body that if an emotion were sufficiently forceful, actual physical injury could occur: "It may be regarded as proved that the exciting passions, when violent or accompanied with agony or conflict of mind, are competent to induce bloody sweat, and when still intenser, even rupture of the heart." This rupture of the heart "consequent on tremendous mental agony, of which many authentic cases are recorded, is, of course, followed by mortal shock." Proof, argued Easley, was found in the biblical gospels, which state that when the body of the crucified Jesus was pierced with a spear, a stream of water and blood issued forth. Easley seized upon this as documentation that crucifixion led to death from cardiac rupture –

[304] Ibid., p. 430.
[305] Ibid., 518.

because of the pain, the fear, and the suffering of the event – and subsequent pericardial tamponade, a filling of the confined space around the heart with fluid or blood, which prevented the heart from filling and beating effectively. Carrying this line reasoning to its conclusion, Easley credited the Roman soldier in the story with performing an inadvertent pericardiocentesis, draining from around the heart the separated serum (which would appear watery) and clotted blood.[306] He found further proof, interestingly, expressed in an almost Jungian collective-consciousness sort of way: Because humans use terms like "I must catch my breath," and "my heart leaps for joy" or "my heart is oppressed," and because these terms, used in literature and poetry, are universally understood and experienced, they must represent actual insight by man into the workings of his own body.[307] These physical sensations of emotion, intangibles put to some concrete approximation in language and agreed upon by common usage, represented to Easley no mere metaphors, but rather true physiologic phenomena.

This particular element of Easley's paper is by far the most fascinating. It assigns the emotions a powerful role as shapers of reality, it suggests that control over one's emotions is important in health, and, most interestingly, it uses a unique evidentiary standard that seems out of place in the late 19th-century literature. In an era where quantification was becoming more important, where experimental physiology was rising to a level of parity with pathology in terms of investigation of disease, where medicine clearly was moving toward a much more rigidly scientific epistemology, that Easley

[306] Ibid., 525.
[307] Ibid.,. 518-519.

could so readily trot out appeals from anecdote seems jarring. (Indeed, that he pulled fiction, literature, and myth into his proofs interchangeably with clinical data, setting upon them the same worth and value, seems almost postmodern in a 21st-century sense.) Easley's paper, in its entirety, seems almost anachronistic. It speaks to a medicine of another time, one of individual temperament, constitutional susceptibility, and the idealized notion of a disease without a seat.

While the nervous system was the infrastructure upon which shock propagated, Easley argued that, for practical purposes, death from shock began in the heart, noting that Bichat taught that "death always begins in either the heart, the head, or the lungs." In shock, the heart could fail in one of two subtly different ways: anemia, or asthenia. Anemia was explained as leading to death through a rapid, large-volume loss of blood which deprived the heart "of its accustomed stimulus." Though the heart continued to beat, the circulation failed, not because there was inadequate blood to fill the vessels, but because the reduced quantity of blood led directly to loss of cardiac function. The power of the heart to contract, in Easley's model, comes in some measure from the blood; the failing of the heart could arise from lack of blood to stimulate it.[308]

Far more common than anemia, however, was asthenia, which Easley regarded "indubitably the most frequent cause of death" in cases of shock. Asthenia was weakness and depletion, a failure of the body to mount enough strength to carry out its tasks, and

[308] Ibid., 522-523.

the cardiac asthenia of shock was a nervous phenomenon: "It is now the nervous system that is principally affected, and through it consecutively the heart." Autopsy studies that showed the heart to be congested with blood were proof, Easley wrote, that in shock the heart experiences "total failure of its contractile power." Emotional factors entered into the spiral toward death here – if the nerves could carry a physical impulse to the heart and stop it, they could certainly carry a mental one, as well.[309]

Shock, if it didn't kill outright, could produce a state of delirium, or "prostration with excitement" – a phenomenon also described by Jordan and Gross. It represented a state of partial recovery, where the physical response to shock – the reaction – was insufficiently vital, putting the patient onto a tenuous middle ground. All of the body's organ systems were affected, and could remain weakened for months to years if the patient recovered, but the key player in traumatic delirium became the blood: "The blood itself may suffer an immediate and serious lesion of its integrity as a consequence of violent shock to the nervous system." This is a striking extension of Easley's model that through the workings of the brain and nerves the intangible is made real. The stimulus from injury caused the nerves to change the very character of the blood from a vital fluid into a veritable poison: "We are reminded of what Williams has, with such force, said of death by necraemia …. 'The blood,' he says, 'the natural source of life to the whole body, is itself dead, and

[309] Ibid., 523

spreads death through the frame instead of life.'"[310] Destruction of the blood, naturally, becomes destruction of the organism.

"Pain is of itself destructive"

While the mind played the key role, Easley also felt that direct physical injury could in itself cause shock. In general, physical violence causing great pain was the culprit; tissue destruction played a part, but was neither necessary nor essential to shock. Pain was "of itself destructive," Easley noted, and it was the "acute violence to the peripheral nerves" that produced both pain and shock.[311] Particular mechanisms of injury were often to blame, including gunshot wounds, crushing injury, burns, visceral perforations, and venous air embolism (the introduction of air into a large vein, as could happen in head and neck operations). The common ground between these injuries was their sudden and noxious effect upon the nervous system. Burns rapidly destroyed much peripheral nervous tissue, stimulating an immense pain response and saturating the nerve centers with deleterious signals. Perforations led to caustic irritation of the well-innervated peritoneum, an enormous surface area of membrane lining the inside of the abdominal cavity. Gunshots transferred immense force through the bullet, and also carried the burden of a unique set of emotional circumstances – fear was the most notable factor,

[310] Ibid., 528-530.
[311] Ibid., 432, 512.

provoked by the interpersonal violence as well as the notion that a gunshot wound must by its nature be grievous.[312]

While major physical trauma produced shock, less significant insults were just as capable of sinking a patient. This disproportionality, to an extent, could be explained through the agency of the nervous system and emotions. The effects of fear and anxiety could also have an accumulative effect upon the physical derangement of injury to produce death. The patient, through their unique circumstances, could be particularly susceptible to the injury, for example, or the area injured could in its own right be more susceptible to shock than another.

Susceptible locations in the body could magnify the effect of injury. Easley, like many other writers of the time, offered up a blow to the solar plexus – found in the upper midline of the abdomen just below the tip of the breastbone – as the archetypical shock-producing injury: "It is not incredible that sharp, direct concussion, even without destruction of continuity, should possibly arrest or even destroy organic function."[313] The plexus – which isn't really an anatomical structure, but more of a conceptual point at which structures that interface with the autonomic nervous system are thought to be less protected and thus potentially more exposed to external violence – seemed to capture the imagination of many medical men with respect to shock. Nearly forty years earlier, Charles Bell wrote "a severe blow on the stomach kills instantly" in his volume *Practical*

[312] Ibid., 511-512.
[313] Ibid., 429.

Essays, noting that "the injury is propagated to the source of the respiratory nerves."[314] Samuel Gross concurred, as did T. Lauder Brunton, who noted that shock "readily follows a blow on the abdomen, sometimes even when the blow is by no means severe." By way of evidence, Brunton offered a case of sudden death of a man who had recently lifted a heavy weight collapsing after a gentle tap upon the abdomen. Injury to the testicles enters the literature at many points, as do injuries to the nose and throat; injuries to or pain from midline structures, it seems, had the greatest shock-producing potential of all causes.[315] Easley cited labor and parturition as potential sources of shock; he dispensed with other authors' explanations of "asphyxia" or "paralysis of the heart" and argued instead that labor – through stretching and stressing of the genital organs – produced "an overpowering impression on the nerve centers."[316] Interestingly, the pain of labor's first stage didn't produce shock, nor did the accompanying bleeding, but rather the contractions, stretching, and pushing of the second stage. The second stage of labor – which is defined as the point after the cervix has dilated but before the placenta is delivered, an oblique way of describing the stage at which the infant is delivered – is the stage amenable to surgical intervention by caesarean section, a point which Easley doesn't miss: "Since it has recently got to be so much more than ever in our power to limit the second stage, these deaths are and ought to be extremely rare."[317]

[314] Bell, "On the powers of life to sustain surgical operations," 3-4.
[315] Brunton, "On the pathology and treatment of shock and syncope," 243, 247.
[316] Easley, pp. 432-433.
[317] Ibid., 432.

Easley's treatment of hemorrhage and shock is somewhat confusing and contradictory. Like his contemporaries, he had difficulty reconciling a relationship between the two phenomena. Although he stated "when considerable in quantity, and frequently when it is not so [hemorrhage] is a fruitful and general cause of shock," he also noted that "it is also true that fatal shock may supervene upon injuries unattended by hemorrhage of any considerable amount."[318] The condition of the wound causing the hemorrhage seemed to be more important – lacerations, comminuted fractures, dirty wounds all were more shock-prone than simple loss of blood. The speed at which blood was lost was also a factor, particularly if a large volume of blood was lost at once.[319] As noted above, blood loss like this could produce what Easley termed death by anemia, which was a more purely physical failing (and, in a nebulous way, different from proper shock). Another thing that hemorrhage could do, though, was predispose to a sort of exhaustion rather than to circulatory collapse by taking away physiologic reserve. A bleeding man lost strength; strength was important to surviving the neurologic failure of shock and starting to rally toward recovery. In a man already weakened by constitutional factors, hemorrhage could be the foothold that allowed shock to supervene on an injury: "Often a man, especially if his hold on life be a feeble one, perishes from the shock and exhaustion of a rapid loss of blood when the wound from which the hemorrhage proceeds is simple and unimportant." In support of this contention, Easley offered a case: A man is stabbed in the arm, the brachial artery is divided, and jets of blood spatter the room. Though "every effort" was immediately made to revive the patient, he died within fifteen minutes

[318] Ibid., 433, 509.
[319] Ibid., 434.

of injury. The cause? "The man's antecedents were poor, his morals very bad, and no doubt he perished as much from shock, alarm, as from the actual amount of blood abstracted."[320] The blood seems to lack a fixed volume; the idea of exsanguination seems less pressing in this explanation than the idea of a man – and a morally questionable one at that – literally scared to death by the sight of his own blood. "In the major operations patients sink oftener from shock than from loss of blood," Easley observed.[321] Shock and alarm – echoes of LeDran's "shock and agitation, communicated to the whole animal machine" – are separate entities from hemorrhage, and furthermore they are what kill injured patients more regularly than mere loss of blood.

Shock from a physical cause was predicated on the notion of nervous failure through a sort of overload. To Easley, shock at its core was a phenomenon of enervation; its onset, course, severity, and prognosis defined chiefly by the effect of the injury to the nervous system, modified at the top level by the patient's interpretation of and attitude toward the situation. Shock could be initiated by internal or external factors, chiefly through violence – though violence in the sense of a sudden, stressful change of state, rather than strictly the application of some external physical force. Shock was propagated through the nerves, from peripheral to central, following the great nerve trunks that supplied the viscera. Shock exhausted the central nervous system, suspending the body's ability to continue living through almost a process of attenuation rather than a specific mechanism – the vital force that kept the organism unified and intact waned, dissipated, and faltered;

[320] Ibid.,. 434-435.
[321] Ibid., 510.

fading to black rather than failing stepwise and in sequence. The emotional state and the individual's demographics could act as a sort of insulation to this barrage of stimulus; fear, questionable morals, a weak (or weakened) constitution could all amplify the effect of injury. The physical and the mental/emotional responses complemented one another, creating from a seemingly localized, simple phenomenon a means for total dissolution of the organism.[322]

Shock as metaphor, metaphor as disease

Easley's clinical portrait of shock is familiar and resembles the contemporary literature. "The diagnosis of shock requires little remark," Easley wrote, "it is not likely to be mistaken, and is generally, if not always, instantly manifested on the cause producing it."[323] Interestingly, given his prefatory laments on how misunderstood shock was among medical men, Easley clearly felt they knew it when they saw it. A weak pulse, loss of lustre in the eyes, gasping respirations, depressed alertness, prostration, and vomiting – in the setting of an inciting factor – all portended shock. Easley lacked interest in his contemporaries' attempts to quantify and categorize shock. To him, clinical diagnosis was sufficient, and no meaningful numerical data would facilitate the diagnosis. What would one measure, after all, when shock's appearance was so reliable and its inner workings so sublime?

[322] Easley, 431; Brunton, 242.
[323] Easley, 516.

Instead of attempting to elucidate shock in an evolving language of pathophysiology, the Easley shock model relies on certain empirical observations about shock – the recognizable clinical particulars, such as its succession upon injury or the bloodless aspect of the suffering patient – put into a theoretical whole using a set of ideas and assumptions about the function of the human organism which cannot be tested or falsified. Easley posited a human structure in which the mind and nervous system – both in an unconscious, reflexive sense and in a conscious, moral-emotional sense – drive physiology. He drew a trace of evidence from autopsy and pathologic study – primarily to discuss the enervated, overfilled heart of asthenic shock – but otherwise based the remainder of his analysis on theoretical and metaphorical constructions of how man must necessarily work. According to Janet Oppenheim, metaphor can serve an important intellectual function in phenomena that are otherwise medically inexplicable. Metaphor creates "an illusion of theoretical completeness," bridging gaps in knowledge and organizing the world in a framework that feels more intuitive and logically sound.[324]

Easley's framework fit in well with contemporaneous ideas of how the nervous system functioned. The idea of a finite, exhaustible nerve force in the body – often referred to as the *vis nervosa* – was well-established by the late 19th century, and the doctrine of this force allowed for whole-body effects to arise from particular, localized issues.[325] Drawing a close parallel to Easley's emotional/moral shock schematic was neurasthenia,

[324] Oppenheim, *Shattered Nerves*, 80-85.
[325] Oppenheim, *Shattered Nerves*, 79-82.

another disease of nervous exhaustion and imbalance which traced its rise to medical prominence through these same years in the late 19th century.

Neurasthenia can be viewed as the archetypical functional disease. Much like shock, neurasthenia was a common, much-discussed ailment which was a purely clinical entity. It existed in the realm of the subjective only, producing symptoms and complaints, but few diagnostic signs of its own and no pathologic changes to allow for its ready classification in the roster of diseases. George M. Beard noted that "unlike the existence of surgical and acute and inflammatory diseases, the phenomena of which the physician can see and feel," the underlying physiologic alterations in neurasthenia did not "appeal directly to the eye or ear or touch, and are in fact quite out of the range of all modern appliances to supplement the defects of the senses, as the ophthalmoscope and laryngoscope, or even the spectroscope."[326] The symptoms of neurasthenia, like shock, can be protean; and to Beard they represented depletion and exhaustion of nervous energy; "a lack of force in the disordered nervous system."[327]

A prominent commonality between shock and neurasthenia was the lack of pathologic change in its subjects. Neurasthenia thus produced some of the same consternation for medical writers as did shock. Beard argued, though, that the disease was one of pure function; no organic lesion was necessary to produce the disease. Rather, any findings identified were secondary phenomena caused by the failure of nervous power itself. As in

[326] Beard, *Practical Treatise*, 26-27.
[327] Ibid., 41, 51.

shock, in neurasthenia we see the capacity of the nervous system to allow the emotional to be made physical. Blushing, argued Beard, was proof that emotion could become physically, involuntarily manifest, as could the dyspepsia associated with nervousness. The heart was particularly susceptible to mental formations: "It is so powerfully affected by the mind, so intimately under the influence of emotions, that I have almost abandoned the habit of examining the pulses," Beard wrote, noting that nothing was to be learned from such an examination. The pulse, in neurasthenia, is wholly under the influence of the mind; the mental state drives circulatory physiology so completely that there was no point in attempting to quantify its effect. The diagnosis rested on the presence of subjective "irritable heart," not a numerical count.[328] Some elements of quantification could be found in discussions of the disease; perhaps these served as a means of attempting legitimation in a medical paradigm that favored and embraced the objective. Beard noted that laboratory alterations could be found in neurasthenia – nonspecific and subtle things, such as alterations in the shape of the red blood cells and appearance of microscopic amounts of protein in the urine (again, further manifestations of emotion as physical consequence). Later revisions of Beard's text included the use of the sphygmograph to establish prognosis and progression of treatment.[329]

Beard argued that since the symptoms of neurasthenia were so nonspecific and broad only a well-honed diagnostic sense could elucidate the diagnosis out of the physiologic cross-talk going on in the patient's complaints. The clinician's mind must be "so well

[328] Ibid., 72-74, 84.
[329] Ibid., 87, 102-103.

disciplined and trained that there shall be no over-estimate of the relative importance of special facts, and no overlooking of any phenomena, however trifling."[330] The ability to glean subtle cues from the complaints of the patient harkens back, much like Easley's shock, to an older medicine of type, character, and nature. The objective, in neurasthenia, becomes secondary to finding the thread of diagnosis in the subjective. The concepts of depleted nervous force, disordered reflexes, and disunity of the body through the medium of the central nervous system create a metaphorical scaffold that, in the words of Oppenheim, "[bestows] an air of precision" upon a disease which cannot be articulated in the scientific – objective – terms of the day. Easley's shock model, as we have seen, uses a similar strategy of dealing in metaphor to lend scientific credence to a phenomenon that cannot be explained by the extant disease paradigm.

[330] Ibid., 24.

Chapter 6 – A momentary pause in the act of survival

Shock in the 19th century was a conundrum. It was ever-present, easily recognized, and clearly dangerous. Yet, despite its apparent ubiquity, it was difficult to explain, categorize, and understand. The disease paradigm that emerged in the early 19th century hinged upon identification of pathological changes in the tissues of a patient's body; shock produced no such defining feature. This required, to some extent, for shock to be articulated in a combination of scientific language and metaphor. Shock became one of an array of functional neurological conditions, a result of overstimulation of a set of innate reflexes and anatomic connections that either destroyed or exhausted the energy reserves of the central nervous system and prevented it from carrying out its assigned duty of holding the systems making up the organism in harmony.

Investigations into shock in the 19th century follow Kuhn's model of normal science, in that they attempt to describe, refine, and reinforce this central paradigm of shock as a neurologic dysfunction. There are not attempts to rupture this paradigm, even in the branch of physicians – along the lines of Jordan, or Woolsey, or Kinnaman – who chose to investigate and express shock in the increasingly quantitative, laboratory-physiologic, and numerical language of medicine as it entered the Progressive era. No researcher raised the argument that the central dysfunction in shock was anything other than exhaustion or disruption of the central nervous system, a lifting away of the central mediating, controlling forces of life with resultant physiological derangement and dissolution of the organism. Even when these writers began to express shock in new

terms – a phenomenon with a certain pulse wave, or with a drop in temperature, or with a loss of serum alkali – the goal was to refine description, recognition, and communication of shock, not to change the basic understanding of it.

The story of shock in the early 20th century is portrayed in many settings as the transition from a clinical and descriptive understanding to a scientific and quantifiable approach with resultant change in the shock model that bridged antiquated and modern constructions of the condition.[331] Central to this apparent change is the fact that by the early 20th century the dominant, defining clinical characteristic of shock became low blood pressure. It fit well with the Progressive era's epistemological demands for precision, measurement, and reproducibility in the production of knowledge – blood pressure was easily determined, had little inter-observer variability, and was seen as a more concrete demonstration of physiologic derangement than were the prior generation's more clinical, nuanced, and ephemeral notions of shock. Ultimately, 20th-century shock would become constructed as a state of physiologic compensation wherein the suffering patient's autonomic reflexes and neurohormonal responses attempted to restore cellular homeostasis in the face of some injurious stimulus – blood loss, infection, thromboembolism, neurologic trauma, visceral rupture – often manifest by the familiar alterations of consciousness, rapid heartbeat and breathing, and cooling of the skin. These features, considered signs of a failing organism to 19th-century practitioners, would become instead evidence instead of an unseen, inner struggle to survive.

[331] Examples can be found in Wiggers, *Physiology of Shock*, 1-25; Thal, et al., *Shock*, 5-34; English. *Shock*.

Merely assigning a new, measurable diagnostic feature to shock did not create this change in identity, however. The works of G.W. Crile, in particular, are cited as laying the groundwork for breaking shock free of its pre-modern conception and re-making it in modern terms. A close reading of Crile's theories, however, suggests that even until the time of World War I the neurological underpinnings of the shock paradigm remained a persistent epistemological obstacle to any change in the conceptualization of shock. Shock as described in the early Progressive era is at its core essentially the same shock as it was known before the Civil War, only articulated with a different grammar.

20th-century science, 19th-century disease

George Washington Crile is the physician most associated with expressing shock as a function of the blood pressure. Crile, educated and trained in the late 19th century, considered himself a "physiologic surgeon" who applied laboratory principles to clinical practice.[332] As has been described in English's study, Crile developed an interest in shock early in his medical career and in the 1890s investigated the effect of shock upon the arterial blood pressure. He determined that low blood pressure – arterial hypotension – was a reliably measurable finding in shock; further, he felt that the clinical manifestations of shock were secondary to this fall in pressure. Crile posited three potential reasons for this finding. First, that some loss of blood volume occurred, thus leaving the circulation

[332] English, *Shock*, 47-82.

under-filled and thus under-pressured. But, given that shock could occur in the absence of hemorrhage, Crile argued that simple hydraulics could not be the cause. A second potential explanation was failure of the heart itself, a situation in which the pump could not generate a sufficient pressure head to keep the vascular system charged. This also seemed to be disproven by animal experiments, in which the heart could be demonstrated to fill and pump effectively when blood was completely drained away and the heart re-filled with salt solution. Crile concluded that the only remaining explanation was that the vascular system itself must have failed. No mere passive conduits, the arteries and veins could dilate and constrict in response to stimuli; thus if the vessels dilated inappropriately, the capacity of the entire vascular system would increase, forcing the blood pressure in the now-underfilled circulatory tree to fall.[333]

Crile's explanation for this failure of the pipes, so to speak, sounds familiar in the setting of the shock literature: "As a result of these investigations we concluded that shock was *the result of exhaustion* and since, from the surgeon's point of view, the most vital phenomenon accompanying shock was a low blood-pressure, we concluded at the time that the most important effect of traumatism was impairment of the vasomotor mechanism."[334] Though he viewed the circulation as the effector system of shock, Crile's shock paradigm remained staunchly neurological.

[333] English, *Shock*, 88-90.
[334] George W. Crile, William E. Lower, *Anoci-Association* (Philadelphia: WB Saunders and Company, 1914), 19. Italics in original.

Crile conceived of the human organism as being in a balanced tension between beneficial and harmful stimuli. He termed these competing forces *bene-association* and *noci-association*. This draws a parallel to a Brunonian sthenos/asthenos physiology, with a wounding noci-association producing involuntary, reflexive reactions in the organism to lead to its correction: "Every adequate stimulus awakens an ontogenetic or phylogenetic memory – or *association*, and the nerve mechanism evolved by countless similar experiences in the life of the individual or of his race makes the appropriate response."[335] The goal of every living thing, argued Crile, was to reach a state wherein the noxious stimuli were eliminated, which he termed *anoci-association.* The reasons for this were not purely ones of comfort. As the response arc for a given noci-association involved activation of a motor system reaction, continued noxious exposures – or exposure to a sufficiently strong stimulus – could, through continued discharge of physiologic energy, produce exhaustion, which was synonymous with shock. "In other words," wrote Crile, "shock is the result of the *excessive conversion of potential into kinetic energy in response to adequate stimuli*."[336]

Crile termed this the "kinetic theory of shock."[337] To read his explanation is to read a markedly different monograph than the one written by Easley. The language is scientific and physiologic, there are no references to literature or scripture, there are accompanying photo plates demonstrating the subtle pathologic changes Crile argued were present in the

[335] Crile, *Anoci-Association*, 29.
[336] Ibid., 30-31. Italics in original.
[337] Ibid., 31.

endocrine and nervous tissues of the patient suffering from shock. Crile is engaging shock in the language and epistemology of clinical medicine as it transitions into the Progressive era, and he is describing shock in the terms that defined disease, using heretofore elusive evidence. Data are quantifiable and measured, laboratory experimentation has become the means of producing and proving theories, and pathologic changes are presented to fix the identity of the disease in the tissues.

Yet Crile conceived of a shock that was not that much different from that described by the generation which preceded him. "In each individual at a given time there is a limited amount of potential energy stored in the brain, the suprarenals, and the liver," he noted.[338] If this potential energy is converted too rapidly into kinetic, cells are exhausted and damaged, the vasomotor centers fail, the hormonal axis that maintains blood pressure and cardiac function is depleted. If this conversion of energy happens as a result of some physical action (such as running, or fleeing, or fighting) it is "exhaustion," if it happens as a result of discharge of energy unrelated to obvious, physical work – such as the energy release accompanying a sudden blow, or that caused by fear or strong emotion – it is "shock."[339]

Crile's paradigm allows for a role of the emotions to persist in shock. By constructing a physiology in which reflexive responses to powerful stimuli deplete the energy of the

[338] Ibid., 31. The suprarenals are the adrenal glands, which produce hormones associated with involuntary "fight or flight" responses.
[339] Ibid., 31-32.

organism, he maintains a central role for the nervous system in the unification and control of bodily systems. While the nerves now do not change the character of the blood to cause systemic dissolution and collapse (an older contention, which Crile disproved experimentally), they still are the conduit through which an excessive discharge of energy cripples the animal machine.[340] The emotions are linked, phylogenetically, to the most ancient and basic survival drive. The fear of danger and the desire for self-preservation are so powerful, argues Crile, that they can produce physical alterations in the body that culminate in shock: "So powerful has this instinct to flee from anything which endangers the safety of the individual become that distant dangers even, or the mere memory of them, may cause all the phenomena associated with the activity once experienced by the individual or his ancestors when escaping from a present danger."[341] The potential for shock is evolutionarily hardwired into man via the agency of the central nervous system. Its existence seems almost a necessary consequence of an organism's ability to protect itself and survive in the wild.

Crile backed up his contentions, fittingly, not with references to Byron but with laboratory data. He reported on the physical changes manifest in rabbits experimentally frightened by dogs – rapid heartbeat and respirations, a rise in temperature, prostration. These elements exist, he argued, even if the rabbits are restrained and not allowed to use their muscles (so as not to expend their strength physically). Further, he argued that the

[340] Ibid., 72-76. Recall Easley's discussion of necraemia for one concrete example. Crile addressed whether the blood developed a poisoned character experimentally, and found no evidence to support the claim.
[341] Ibid., 94.

rabbits were "more exhausted" than the dogs who frightened them, which were allowed to expend muscular energy in the experiment – though how he quantifies depth of exhaustion is unclear. He concluded, though, that "fear, therefore, and above all fear associated with trauma may drain the dischargeable nervous energy of the body to the lowest depths and as a consequence, produce the greatest possible exhaustion, even to the point of death." Fear additionally lowered the "brain threshold," inhibiting the mind's ability to insulate itself from noxious stimuli and allowing the effects of all further traumas to be magnified.[342]

Crile cited anger and worry as the other two destructive emotional states; the former as it was a phylogenetic precursor to violence (the body must ramp up its muscles, alertness, and energy to fight), the latter for its futility. Worry "is a chronic state of attempt to escape from some threatening evil or of futile efforts to combat the cause of some anticipated disaster."[343] He attributed to worry a broad and protean array of ailments, almost reminiscent of the sort of clinical picture one would expect from neurasthenia. The emotions, Crile felt, were "activations of the entire motor system" to perform some function, either coded in the individual's memory or, more deeply, in some sort of greater unconscious species memory. The emotions thus could translate into direct physical effect. "The effect of the emotions on the body mechanism may be compared to that produced upon the mechanism of an automobile if its engines are kept running at full speed while the machine is stationary," he wrote. "The whole machine will be shaken and

[342] Ibid., 94-95.
[343] Ibid., 95-96.

weakened."[344] The physicality of LeDran's *secousse* has come full-circle; in kinetic theory Crile has created a shock that can reverberate through the whole of the animal machine with no inciting agent beyond a thought.

Crile's approach to dealing with shock in the surgical setting cements the fact that he understood it to be a primarily neurological issue. If shock were purely an issue of pain and fear, then anesthesia should prevent shock from occurring as a result of an operation, as consciousness was suspended and the emotional elements of a patient's care were removed from the equation for a time. Easley, in fact, had argued "the moral element of the case must be eliminated at any cost" in some cases of shock accompanied by anxiety by making the patient "oblivious to the situation" with opium.[345] It was well-known, though, that anesthesia did not have this protective effect – surgical shock still occurred, despite good anesthesia, adequate pain control, and cautious hemostasis and dissection. Crile reasoned that while the patient had been rendered unconscious, their nerves still carried out their prescribed functions; in other words, the noci-associative stimuli of operation still traveled centripetally and still generated their potentially pathologic downstream response at an unconscious level. Simple lack of awareness on the patient's standpoint did not influence the function of the nerves. If these noxious stimuli could instead be blocked from reaching the brain, he reasoned, then the corresponding reflex arc which produced the kinetic changes in the cells should not be able to take place. Conveniently, this hypothesis was easy to test: The aminoester and aminoamide local

[344] Ibid., 96.
[345] Easley, "A study of shock," 537.

anesthetic agents, particularly cocaine, were known to block signal transmission along nerve trunks.

Writing in 1902, Harvey Cushing described an approach to preventing operative shock that he stated was inspired by Crile's research. He hewed to the Crile etiologic model of vasomotor exhaustion: "In order to produce shock, the impulses resulting from [traumatism] must have acted reflexly upon the vasomotor mechanism in the medulla in such a way as to occasion a marked fall in blood-pressure. This diminution of arterial tension is the most characteristic symptom of shock."[346] Cocaine "effectually blocks the transmission of all centripetal or sensory impulses," and thus if given before incision should block the effect on the nervous system, and thus be able to prevent shock.[347] He sought to prove his contention using a comparison of two of his operative cases, both of them forequarter amputations for cancer in which the arm, shoulder blade, pectoral and shoulder muscles, and collarbone were removed. These operations involved division of the cords of the brachial plexus – a meshwork of thick nerves that arise from the spinal cord in the neck and supply the shoulder and arm – which was well-known to produce the shock-defining physical signs of low blood pressure and fast heart rate. By ablating the nervous impulses activating the brain's nociceptors with cocaine, Cushing asserted, the cardiovascular manifestations of shock would not occur. [348]

[346] Harvey Cushing, "On the avoidance of shock in major amputations by cocainization of large nerve-trunks preliminary to their division," *Annals of Surgery* 36 (1902):321.
[347] Cushing, "On the avoidance of shock," 322.
[348] Ibid., 322-326.

In the first case he described the operation in a 41-year-old woman under ether anesthesia, and noted that when the brachial plexus was divided the heart rate accelerated to 150 beats per minute, the pulse became weak and "thready," and the patient developed shock lasting a full 24 hours.[349] By comparison, he reported on the same operation on a 32-year-old man performed with ether anesthetic and direct injection of the brachial plexus with cocaine. Despite incurring significant hemorrhage from the large artery under the clavicle, "after preliminary cocainization of the brachial plexus, the bundle of nerves was severed; the extremity with clavicle and scapula was removed, the dry wound closed without drainage, and no shock resulted from the operation."[350] The ether chart, in fact, showed the heart rate slowing after cocainization, which Cushing took as at least proof of concept, if not outright proof of the theory: "The facts remain that injuries of most diverse nature to peripheral nerves may, especially in some physical states, produce reflexly a fall in blood-pressure; that this loss of vascular tone, when it endures, is the most characteristic feature of shock, the symptom-complex of which is largely due to this one factor; that local anaesthetization of a nerve-trunk will block the transmission of the centripetal impulses which otherwise might bring about this reflex loss of vascular tone."[351]

[349] Ibid., 322-324.
[350] Ibid., 325-327.
[351] Ibid., 345.

A momentary pause in the act of survival

To summarize, Crile understood shock to be the exhaustion of vasomotor control through exposure to a sufficient stress over a sufficient time. This occurred because the stressful stimulus created a reflexive response which, while generating some physiologic benefit to allow the organism's survival (rapid heartbeat, widened pupils, faster respirations, increased blood sugar, etc.) in a fight-or-flight situation, also drained some unmeasurable amount of a finite reserve of energy. When this reserve became depleted, shock was the outcome.

A key argument made by English in his study of Crile is that the surgeon was the first researcher to envision shock in terms of its potential to keep the injured organism alive – a survival-oriented paradigm, rather than one that approached shock as a pathologic process leading to death: "Crile changed the surgical idea of shock from the process of dying to the struggle for existence."[352] Further, he contends that Crile was the first researcher to truly attempt to understand shock through researches in the laboratory, and that the use of arterial hypotension as a new unifying element in shock that Crile's version of shock was novel in its incorporation of the circulatory, respiratory, nervous, musculoskeletal, and endocrine systems into a unified whole, all participating in the body's drive for survival.[353]

[352] English, *Shock*, 177.
[353] English, *Shock*, 92, 177-178.

While Crile considered shock the by-product of survival-based reflex response, this does not equate to him creating a new vision of shock as a survivable event. Crile held to a model of nervous depletion and exhaustion as the underlying etiology of shock. But, as we have seen, this fits into a nearly two-century-old epistemology. While Crile explained vascular constriction as the corrective response by which the organism attempted to maintain blood pressure, the fact that this protective reflex necessarily led to exhaustion and consequent physiologic collapse is essentially unchanged from prior ideas of shock. Survivability is not equivalent to survival-oriented. Protective reflexes are protective only in the shortest of terms, in this model; the very things the organism does to maintain its integrity become the engines of the organism's ultimate failure and dissolution. There is a finite, exhaustible supply of energy that the survival mechanisms deplete.

Crile expressed his ideas regarding exhaustion on both concrete and abstract levels. His earlier work is of the classical laboratory/physiologic variety. His papers meticulously detail experimental design, execution, and results. He uses physiologic diagrams and data, schematic depictions of his equipment, and photomicrographs of histologic slides to demonstrate pathologic changes in the nervous and endocrine cells of animals in shock.[354] He also delved into the more philosophical, though, and in the Crile epistemology there is persistence of the use of the exhaustibility metaphor to describe shock. As English discusses, Crile's work relies more heavily on the metaphorical as his

[354] See, for example, George W. Crile, *An Experimental Research into Surgical Shock* (Philadelphia: J.B. Lippincott, 1899); also George W. Crile, William E. Lower, *Surgical Shock and Shockless Operation through Anoci-Association*, ed. Amy F. Rowland (Philadelphia: WB Saunders and Company, 1920).

career progresses and he tries to create a more unifying, overarching shock theory. His inclination to create a more reductionist, universalist physiological model is possibly best-illustrated in his 1915 work *A Mechanistic View of War and Peace*. In this monograph, the human body becomes extrapolated into the macrocosm of the Great War, with armaments, bullets, explosives, vehicles, fuel, and the other *materiel* of combat assuming the roles for nations that the individual would have for things like adrenaline, thyroid hormone, cortisol, and blood.[355] Metaphor, as noted by Oppenheim, is a means of adding precision and tangibility to phenomena that lack these features. It is a holdover of a 19th-century logical and epistemological model – recall the physiological episteme of E.T. Easley, for example – and Crile, in trying to link evolution, teleology, physiology, and society into a grand narrative of shock continues in this older tradition.

Crile's laboratory work fits Kuhn's definition of normal science in that it seeks to refine or explain particular nuances of a scientific question without addressing or challenging assumptions about the nature of the question itself. In Crile's case, the interest is in chemically, physiologically, and pathologically detailing how arterial hypotension fits into the already extant shock paradigm of exhaustion. And in this way, Crile's laboratory work does not much differ from that of the researchers in the mid- to late 19th century who preceded him (such as Jordan, or Wagstaffe, as we have discussed previously), who

[355] English, *Shock*, p. 177. George W Crile, *A Mechanistic View of War and Peace*, ed. Amy F Rowland (New York: MacMillan, 1915). In this work, Crile views war as an ancient human endeavor, phylogenetically encoded as a sort of race memory among humans. The work delves into eugenics a bit, lamenting war's capacity to destroy the fittest members of the combatants' races, and approaches war metaphorically as opposing single animals whose inner "physiology" of men and materiel determine success or failure.

really sought ways to recognize, communicate and express shock rather than shake the foundations of its understanding.

Hypotension in Crile's system ultimately functioned metaphorically, as it represented central nervous system and neurohormonal axis failure and exhaustion. In this way, it is not much different from the trifling sadness of countenance that foretold shock to Samuel Gross, or the lusterless eyes and bloodless skin commented upon by Hermann Fischer. But, as it could be measured and quantified, hypotension better-satisfied the 20th-century demand for more concrete, precise, and scientific knowledge. It was a means of re-casting the identity of shock in a new epistemological model while essentially maintaining the same underlying model of depletion, exhaustion, and failure. Exhaustion of the body, even through purportedly protective reflexes, is still exhaustion of the body; shock expressed as hypotension remains shock as systemic dissolution and failure, despite the new grammar with which it could be expressed.

Despite data that supported the vasomotor exhaustion hypothesis, Crile's theories of shock did not meet with universal acceptance. Physiologists, in particular, took issue with his methodology and findings. Some argued that vasomotor tone was still demonstrable in cases of shock, others noted that they could not reproduce Crile's results or even produce complete neuromotor exhaustion – the very basis of the theory – in experimental animals.[356]

[356] English, *Shock*, 130-140.

Other clinical writers took issue with the vasomotor hypothesis by arguing that the mechanics and hydraulics of loss of tone didn't correspond to observed evidence. "The most popular theory at present, however would appear to be that the essential factor in the production of the condition of shock is a fall of blood pressure," wrote British surgeon John Malcolm in 1905, "and that this fall of blood pressure is brought about by a relaxation of the whole vascular system. This theory is, I think, founded chiefly on Dr. G.W. Crile's experiments."[357] But, Malcolm argued, this flew in the face not only of his personal experience, but of experimental evidence as well. He cited laboratory data that showed increased blood flow in vessels released from vasomotor control, and further data showing that arteries were constricted in shock rather than dilated, as one would expect with Crile's explanation.[358] Shock patients were pale, Malcolm argued, and "if the vaso-motor centre is exhausted in shock some explanation is required of the fact that the whole surface of the body is not suffused with blood" – in other words, if the control of blood vessel tension and diameter was released and the blood vessels relaxed, the shock patient should be flushed and pink, not pale and bloodless.[359] The clinical appearance in shock, argued Malcolm, suggested vascular constriction and not dilation. Vascular constriction, he argued, was the *response* to stress stimuli, not the effect of exhaustion in the face of same. Constriction shunted blood from the periphery to the core of the body, and led to the failing pulses and rapid heart rate of shock: "I was led to believe that the phenomena

[357] Malcolm, "A lecture," 573.
[358] Ibid., 573.
[359] Ibid., 573.

of shock are due to a contraction of the arteries by noting that evidence of anything like a dilatation of the vessels generally, or in exposed parts of the splanchnic area alone, is usually wanting during life, even in conditions of the most severe shock."[360]

Several important things emerge from the Crile/Malcolm disagreement that point to the paradigm shift that would follow the war and recharacterize shock. Crile, as proxy for the idea of neurological depletion and exhaustion, viewed shock as the end result of repeated withdrawals from a bank of energy that had finite supply: "There will be finally a condition produced in which no stimulation even of a fresh nerve-trunk will produce a rise in the blood-pressure; in other words, it would seem that the pressor action may be exhausted in a given nerve-trunk by repeated stimulation of different nerve-trunks, the latter being general, the former local, in the sphere of its influence."[361] Crile saw the human as beholden to the laws of nature, thus any stimulus that would require activation of a flight/fight response would require increased power from the heart and a boost in the blood pressure to prepare the remainder of the system for action: "*The result of action is reaction; of rest is restoration."*[362] Once the ability to sustain this almost Newtonian action/response was lost, shock was the result. Shock was collapse, exhaustion, failure of the system. Shock was the manifestation of dissolution, of the organism ceasing.

[360] Ibid., 576.
[361] Crile, *An Experimental Research*, 152.
[362] Ibid., 154. Emphasis in original.

This contrasted with the notion of shock as described by writers like Malcolm, who argued that the vasomotor center produced persistent, even increasing constriction of the blood vessels in the face of shock; this would be its survival-centered mechanism. Vascular constriction redistributed blood within the circulatory system, forcing it toward the core of the body, the veins, and the central organs. This had the effect in injury of stopping peripheral bleeding and conserving heat, at the cost of reducing blood flow to vital structures.[363] In this case, exhaustion isn't the core factor in shock, but rather dysregulation – too much vascular tone, leading to too little blood flow as a side effect of a physiologic response. The organism hasn't paid out the last of its reserve of physiologic strength, but rather has lost the ability to control its reflexive responses. Shock is not exhaustion, but disorder.

In each of the models is the interesting notion that normal physiology, driven by sufficient stimuli, could actually produce the death of the organism through over-compensation; that a normal response could become pathological, and there was no counterbalancing process to check its propagation. The animal machine either burned through its fuel reserves and sputtered out, or kept clamping down so tenaciously that instead the circulation seized up. While one could argue that shock in the neurologic exhaustion model is really shock of dysregulation – central nervous action over- or under-inhibiting normal body function – this isn't the key point. The idea of normal processes producing exhaustion and collapse, which was Crile's paradigm, would be

[363] Malcolm, "A lecture," 576-579.

challenged by one which writers like Malcolm were beginning to articulate in the early 20th century. In this construction, shock transitions from an event, a sort of physiological tipping point beyond which the organism begins to collapse, into a state characterized by the organism attempting to maintain normal physiological function under increasingly difficult circumstances. This is the underpinning of an epistemologic break that would ultimately open over the course of the next 20 years; and is probably best seen in the work of Walter Bradford Cannon and his colleagues.

Acidosis, bicarbonate reserve, and WB Cannon

Walter B. Cannon was a physiologist on the faculty of Harvard, and his entry into the story of shock comes through his involvement in the US Army Medical Corps during World War I. Cannon was invited by the US government to contribute his expertise to studying shock on the battlefield, and prioritized finding an effective therapy for shock, rather than an etiology.[364] Interestingly, his approach to shock – defining it as the measurable, detectable result of a physiological dysfunction arising from altered cellular oxygen metabolism – is a far better candidate for creating the epistemological tension that would ultimately shift the shock paradigm from its 19th-century to its 20th-century enunciation than Crile's exhaustion-based model. Shock, in the Cannon model, is the byproduct of a global physiological derangement, not the source of the derangement itself.

[364] Benison, Barger, Wolfe, "Walter B. Cannon and the mystery of shock," 217.

Cannon and his co-researchers understood that to investigate shock was to enter into particularly difficult territory: "That investigation of shock has been difficult and baffling is indicated by the number and variety of theories put forth to account for it. The difficulties lie not only in the obscure character of shock itself, but also in complications introduced by attendant conditions, such as hemorrhage and sepsis."[365] Shock was frequently found in association with low arterial blood pressure, and was most severe when the systolic pressures were less than 70 millimeters of mercury. The exact etiology of this fall in pressure was uncertain, and Cannon's team members elected not to base their research on discerning a concrete cause for it. Instead, they took the approach that for the hypotension to be meaningful, there had to be a biological consequence to the patient. Shock was not pure hypotension, there needed to be associated biochemical changes that represented the ill effects of the hypotension upon the system of a wounded man. These could be seen through clinical expertise and the trained eye, but more practically and physiologically speaking, they were best described using measured, laboratory values.[366]

The first major laboratory anomaly Cannon described was hemoconcentration, which was commonly found in peripheral blood drawn from shock patients. This was measured through the hematocrit, a laboratory finding that describes the percentage of the blood that is solid components (red blood cells, white blood cells, platelets, etc.) as compared to

[365] Cannon, Fraser, Hooper, "Some alterations," 526.
[366] Ibid., 527.

liquid plasma. To a large extent, Cannon argued that this related to a common environmental issue among the wounded soldiers being brought through his casualty clearing station – they were cold. "In all probability the low temperature typical of patients in shock is an important factor in producing the increased corpuscular content of the capillaries." The capillaries are considered the "resistance vessels" of the circulation, where flow is the slowest and the blood can stagnate, a phenomenon exacerbated by cold.[367] Cold was known to alter the rheostatic properties of blood, making it more viscous and resistant to flow as temperature fell. Loss of bodily fluid – chiefly through sweating during exertion before the time of injury – could further affect blood viscosity and predispose to capillary sludging; if the dampness caused by sweat was left unattended and the patient became cold, a sort of mutually reinforcing feedback loop was established.[368]

Cannon argued that the stagnation of blood in the capillaries reduced the amount of circulating blood available to support the blood pressure, which led to a further worsening of flow. Comparison of the hematocrit between blood drawn from a capillary and from a vein would be confirmatory – the vein (a large, more freely flowing vessel) would not exhibit the same degree of stagnation and concentration as the blood in a capillary; further, the greater the discrepancy between the two values, "the more profound the shock."[369] This anomaly was not seen in cases of uncomplicated hemorrhage in which

[367] Ibid., 527-528.
[368] Cannon, "A consideration of the nature of wound shock," 615.
[369] Cannon, Fraser, Hooper, "Some alterations," 531.

the circulation continued to function effectively; thus, a fairly simple laboratory test could give insight into differentiating shock from simple blood loss.[370]

Another significant laboratory finding was acidosis. The presence of acidosis – measured either as lowered pH or lowered bicarbonate ion in the blood – had been described in association with shock in the early 20th century.[371] The accumulation of biologic acids in the blood is reflective of impaired cellular respiration – in other words, tissues that didn't receive sufficient oxygen would produce acids as a byproduct of secondary, inefficient metabolic pathways. The impaired circulation of shock inhibited oxygen delivery to tissues, and acidosis was the result, the severity of which was proprortional to the severity of shock: "In general, the lower the blood pressure the lower the alkaline reserve, that is, the greater the acidosis."[372] The falling blood pH then stimulated the brain to increase the respiratory rate, lowering the carbon dioxide content of the blood which, in turn, would help buffer the accumulation of acid and bring the pH more toward normal. Again, Cannon noted proportionality – respiratory rate "increased as the alkali reserve fell," becoming "more marked as the limit of the reserve was more nearly approached."[373] The rapid breathing associated with shock, then, was a reflexive response of a physiologic system trying to correct a worsening acid-base imbalance, and not a consequence of altered neurological control over the central nervous system's respiratory centers. Also,

[370] Ibid., 529.
[371] W.B. Cannon, "Acidosis in cases of shock, hemorrhage and gas infection," *Journal of the American Medical Association* 70 (1918):531.
[372] Cannon, "Acidosis," 531.
[373] Ibid., 533.

Cannon again noted a difference here between simple hemorrhage and shock: while both diminished the alkaline reserve, hemorrhage produced less of an effect than shock – unless, of course, the hemorrhage was profound enough to generate shock itself.[374]

Cold, acidosis, and hemoconcentration acted reciprocally on one another, creating what Cannon termed "vicious circles" of shock: "In all probability a number of vicious circles would be started which, if not interrupted, would lead to an aggravation of the already existent abnormal state, and which would account for the progressive nature of fatal shock."[375] Fluid loss through sweating or hemorrhage stole plasma volume, reducing blood pressure and making the patient cold. Cold, concentrated blood would sludge into peripheral capillary beds, reducing further the amount of blood available to circulate. Poor circulation (as measured by low blood pressure) led to poor oxygen delivery to tissues, producing more acidosis and depleting the body's buffering capacity. Poor cardiac output also led to poor perfusion of the kidneys, which help to create the alkali buffer in the blood, exacerbating the deepening acidosis created by the flagging circulation. Acidosis, in turn, contributed further to worsened blood viscosity, further reducing the cardiac output. And, as output fell, more blood stagnated, more heat was lost, and more acid was produced. This interlocking set of processes created positive feedback loops that reinforced one another, leading to progression of shock.[376] Cannon's hypothesis stands in contrast to the older shock notion of systemic collapse based on

[374] Ibid., 532-533.
[375] Cannon, "A consideration of the nature of wound shock," 616.
[376] Cannon, "A consideration of the nature of wound shock," 616.

exhaustion. The loss of motive force (be it nerve-force, reflex, neurohormonal function, vasopressor responses, etc.) created the cascading failure of multiple body systems based on the notion that some central physiologic engine could no longer function, and with loss of the energy holding the system together it decayed in an entropic fashion into shock and death. In the Cannon model, the physiologic derangements of the system attempting to continue functioning in a normal fashion (through anaerobic cellular processes producing more and more organic acid buildup, vascular constriction preserving some blood flow at the expense of ever-increasing cardiac work) create an accumulating physiologic debt that, at some point, can no longer be paid back. Shock is the cost of the system maintaining unification, not the price paid as it dissolves.

This strikes at a central point of departure in Cannon's work from Crile's: One thing that did not occur in shock, Cannon argued, was the failure of the vasopressor apparatus of the central nervous system or the function of the suprarenal hormonal axis. "The concept has been gradually developed that shock consists essentially of exhaustion of cells in the brain, the liver, and the suprarenal glands," Cannon wrote, introducing it as an idea "long ago expressed by Mitchell, Keen and Morehouse" and "elaborated by Crile," who believed that "the most vital effect of shock is 'the impairment of the vasomotor mechanism.'"[377] These failings of the body's regulatory and stimulatory pathways, as we have discussed, were thought to produce the characteristic failing blood pressure of shock.

[377] Ibid., 612.

The problem with this notion, to Cannon, was that there was no evidence to corroborate it. Low blood pressure, he argued, was not proof of vasomotor failure – blood pressure could fall through simple hemorrhage, for example, and this was not shock. He argued that there was ample clinical evidence that vasomotor function was preserved in shock, describing operative cases in the casualty clearing station where men in shock did not bleed during operations because their circulation was so tightly clamped down. He cited laboratory studies that also supported his claim, and concluded that "the vasomotor center should be regarded as an agent whose functions are extremely stable and whose capabilities for continued service are its most outstanding feature.[378]

If the vasomotor center could still function despite physiological stress, was suprarenal failure at the heart of shock instead? No, argued Cannon. "The epinephrin content of the glands in fatal cases of shock is not notably reduced," he wrote. "The suprarenal glands are, if anything, overactive rather than exhausted."[379] The notion that the vasopressor system and the adrenal glands failed, Cannon felt, was related to faulty evidence: "The evidence for exhaustion which has been advanced by Crile and his coworkers is mainly histologic, and is based on examination of nerve cells taken from shocked animals." Histologic preparations, he argued, were subject to technical failure both in their handling and in their interpretation, leaving open the possibility that Crile's pathologic findings

[378] Ibid., 612. Cannon's argument here draws parallel to Malcolm's critique of Crile's shock as vasodilation mechanism.
[379] Ibid., 612.

were related to his laboratory process, not to shock. In fact, Cannon asserted, "the suggestion is reasonable that any cell alterations that may occur in shock are the resultant of the low blood pressure rather than its cause."[380] This argument attacked Crile's research at a fundamental level by chipping away at the pathological evidence of shock's identity, the elusive gold standard of identifying and characterizing shock for which medicine had sought so assiduously for almost a hundred years. The origins and identification of shock, as it turned out, were not fixable on a microscope slide and told through some pathognomonic change in tissues; they were instead a constellation of findings, some physical/clinical, some objective/numeric, that fit into a larger overall physiological picture.

It is in this physiological picture that possibly the most important feature of Cannon's shock emerges. In this construction, the physiological alterations that allow for diagnosis of shock are actually the byproducts of shock, not its etiologies. Depletion of the alkali reserve is not the same as depletion of the vasomotor response in Crile's model, for the reason that the acidosis of shock is produced by the impaired circulation and resultant oxygen starvation of tissues, whereas vasomotor collapse was a point at which a bodily system could no longer regulate its reflexive responses, with shock as the result. Cannon's model is a true departure, epistemologically speaking, from the shock of the preceding century. Both models view shock as an expected physiologic response to stress, an attempt by the organism to arrest the progress of injury and restore physiologic

[380] Ibid., 613.

homeostasis. The preservation of core-body temperature and the arrest of hemorrhage through increased vascular tone, at the physiologic expense of falling blood pressure, is at its root a means by which the organism's physiology attempts to preserve the life of the organism. But shock as elucidated by Crile understood these ancient, innate survival mandates as withdrawals from a finite, fixed resource pool – ultimately, the organism would exhaust its ability to compensate, the survival responses would fail, and shock would ensue in a grand cascade of dissolution, spiraling to death. Even articulated in the language of the Progressive years, this is shock that is at its heart no different from that of Samuel L Gross.

In the Cannon model, however, shock becomes more about the clinically evident results of an organism attempting to right a listing physiologic ship, rather than the means by which the ship itself sinks. The survival mechanisms are sufficiently powered to carry out their duties until the death of the organism; the effects of shock are more truly the results of the organism laboring under increasingly austere physiologic conditions than they are of some central failing. While ultimately the blood flow alterations produced by acidosis, reflex vascular constriction, and cold would produce a physiologic milieu which the organism could not survive, the important thing is that shock in this construction is not about this tipping point, it is everything leading up to it. It is not exhaustion of the body's resources that produce shock, instead it is a failing at the level of the cellular metabolism that reflects a mismatch of oxygen need and delivery, with subsequent alterations in the body's other systems produced through the organism's attempts at compensation.

In the 19th century, John Collins Warren described shock as a momentary pause in the act of death; articulating a worldview in which some manner of physical or emotional trauma exploited human physiology to snuff out the powers of life. By a century later, however, the same collection of clinical phenomena grouped together as shock – pallor, pulselessness, inanition, depression, low blood pressure – represented the opposite end of the physiologic spectrum. "Shock," wrote a surgical review from the 1970s, "is, in essence, a story of survival, a struggle by the organism in adverse environment to preserve the life of its most vital tissues."[381] The key change in shock that occurred between the 19th and 20th centuries was less one of how the phenomenon was explained, or studied, or treated than it was of how it was conceived. This simple change in theme – an exchange of a death-centered physiology for a survival-centered physiology – was ultimately the epistemological shift that changed the shock paradigm.

[381] Thal, et al, *Shock*, 17.

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