Accepted September 2014

REVIEW

Circulatory shock and peripheral circulatory failure: a historical perspective

A. Lima

Department of Intensive Care, Erasmus MC University Hospital, Rotterdam, the Netherlands

Correspondence

A. Lima – email : a.pintolima@erasmusmc.nl

Keywords – **History, shock, peripheral perfusion, haemodynamic, organ failure**

Abstract

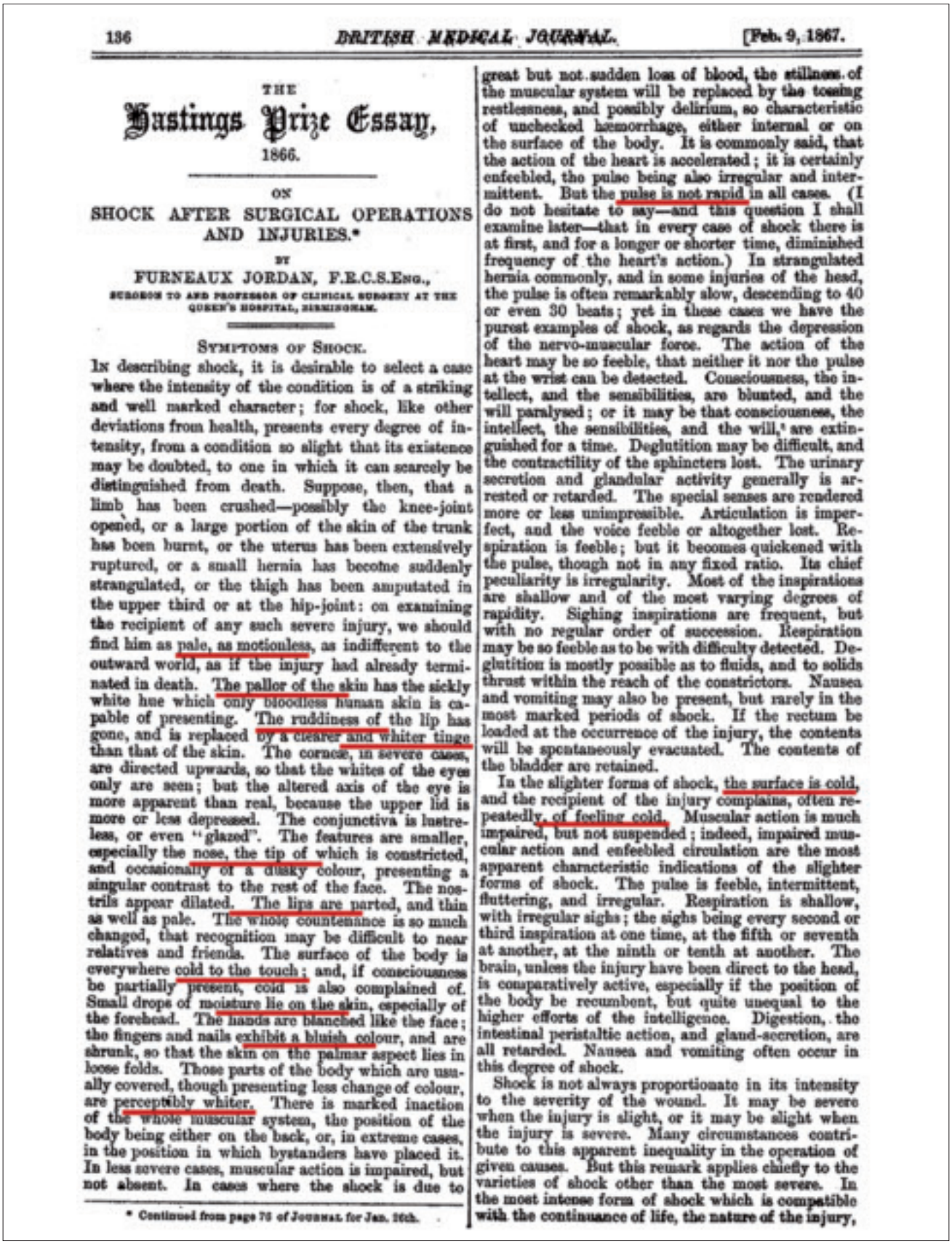
The early descriptions of abnormalities in the peripheral circulation date back to the 18th century during the American Civil War when the British surgeon Jordan Furneaux wrote what is considered to be one of the first elaborate descriptions of abnormalities in the peripheral circulation during shock. However, one of the earliest references to the dynamic component of the peripheral vascular bed is the work of the Danish scientist August Krogh, in the 1920s. His work was followed by a new generation of clinical investigators such as the American physiologist Carl John Wiggers, who introduced the term 'peripheral circulation failure' in his experimental studies of haemorrhagic shock. The introduction of the mercury sphygmomanometer in 1896 and the first performance of right heart catheterisation in 1929 contributed to a great extent to the understanding of the pathophysiology of shock and the associated haemodynamic changes. Over time, pathogenic theories have evolved, providing a better understanding of regulatory mechanisms for the central and peripheral circulation during the state of shock. The connection between hypotension and peripheral vasodilatation in 'vasodilatory shock' was first published in a review by Gilbert in 1960, who was the first to provide evidence of heterogeneous distribution of peripheral blood flow in sepsis. With his experiments in the 1960s, physiologist Arthur Clifton Guyton described the behaviour pattern between peripheral circulation within organs and systemic circulation during acute shock. In the early 1970s, critical care medicine emerged and sophisticated haemodynamic methods of monitoring were introduced, which allowed physicians to understand the relationship between changes in the peripheral circulation and the prognosis of shock. The introduction of gastric tonometry in the 1990s signalled the ability to measure gastric perfusion, and opened a window to systematically assess regional perfusion in patients with shock, proving the concept that the peripheral circulation is the first to deteriorate and the last to reperfuse during cardiovascular collapse. During the last few years, studies have focused on monitoring other vascular beds also susceptible to hypoperfusion, such as skin, subcutaneous tissue and muscle. Progression in the understanding of the role of peripheral circulation in haemodynamic regulation during shock has led to the application of different investigative techniques to continually monitor peripheral circulation in the critically ill patient.

Introduction

Shock

The evolution of medical knowledge during history is more recent than advances in art, industry and other sciences. The industrial revolution through the 18th and 19th centuries brought innovations and transformed life in America and Europe, whereas it was widely accepted that diseases were the result of an imbalance in humours, and one of the conventional treatments to bring back good health was draining blood through phlebotomy (bloodletting).[1] Bloodletting was a common 'cure for everything' from ancient times until the 19th century. The practice gained wide acceptance in America in the 18th century with Dr. Benjamin Rush, who treated George Washington for acute laryngitis by draining one litre (nine pounds) of blood in less than 24 hours.[2] George Washington died soon afterwards. At that time, there was no knowledge of the association between loss of blood and circulatory shock. In fact, shock was still an abstract concept, usually described as 'sudden vital depression', 'great nervous depression', or 'final sinking of vitality'. The history of haemodynamic monitoring overlaps with the history of shock and much of the history of shock relates to the history of traumatic shock. The term shock only came into clinical use with Edwin A. Morri, who began to popularise the term by using it in his 1867 American Civil War text, 'A Practical Treatise on Shock After Operations and Injuries'.[3] Since then, the word shock started to be linked with the concept of cardiovascular collapse. In the same year,

Figure 1. One of the first descriptions of abnormalities in peripheral circulation during shock conditions by Dr. Jordan Furneaux. He emphasised the cold, clammy and mottle skin associated with high heart rate (underlined in red)



136 BRITISH MEDICAL JOURNAL. [Feb. 9, 1867.

THE

Hastings Prize Essay,

1866.

ON

SHOCK AFTER SURGICAL OPERATIONS AND INJURIES.*

BY

FURNEAUX JORDAN, F.R.C.S.Eng.,
SURGEON TO AND PROFESSOR OF CLINICAL SURGERY AT THE QUEEN'S HOSPITAL, BIRMINGHAM.

Symptoms of Shock.

In describing shock, it is desirable to select a case where the intensity of the condition is of a striking and well marked character; for shock, like other deviations from health, presents every degree of intensity, from a condition so slight that its existence may be doubted, to one in which it can scarcely be distinguished from death. Suppose, then, that a limb has been crushed—possibly the knee-joint opened, or a large portion of the skin of the trunk has been burnt, or the uterus has been extensively ruptured, or a small hernia has become suddenly strangulated, or the thigh has been amputated in the upper third or at the hip-joint: on examining the recipient of any such severe injury, we should find him as pale, as motionless, as indifferent to the outward world, as if the injury had already terminated in death. The pallor of the skin has the sickly white hue which only bloodless human skin is capable of presenting. The ruddiness of the lip has gone, and is replaced by a clearer and whiter tinge than that of the skin. The cornæ, in severe cases, are directed upwards, so that the whites of the eyes only are seen; but the altered axis of the eye is more apparent than real, because the upper lid is more or less depressed. The conjunctiva is lustreless, or even "glazed". The features are smaller, especially the nose, the tip of which is constricted, and occasionally of a dusky colour, presenting a singular contrast to the rest of the face. The nostrils appear dilated. The lips are parted, and thin as well as pale. The whole countenance is so much changed, that recognition may be difficult to near relatives and friends. The surface of the body is everywhere cold to the touch; and, if consciousness be partially present, cold is also complained of. Small drops of moisture lie on the skin, especially of the forehead. The hands are blanched like the face; the fingers and nails exhibit a bluish colour, and are shrunk, so that the skin on the palmar aspect lies in loose folds. Those parts of the body which are usually covered, though presenting less change of colour, are perceptibly whiter. There is marked inaction of the whole muscular system, the position of the body being either on the back, or, in extreme cases, in the position in which bystanders have placed it. In less severe cases, muscular action is impaired, but not absent. 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 hæmorrhage, either internal or on the surface of the body. It is commonly said, that the action of the heart is accelerated; it is certainly enfeebled, the pulse being also irregular and intermittent. But the pulse is not rapid in all cases. (I do not hesitate to say—and this question I shall examine later—that in every case of shock there is at first, and for a longer or shorter time, diminished frequency of the heart's action.) In strangulated hernia commonly, and in some injuries of the head, the pulse is often remarkably slow, descending to 40 or even 30 beats; yet in these cases we have the purest examples of shock, as regards the depression of the nervo-muscular force. The action of the heart may be so feeble, that neither it nor the pulse at the wrist can be detected. Consciousness, the intellect, and the sensibilities, are blunted, and the will paralysed; or it may be that consciousness, the intellect, the sensibilities, and the will, are extinguished for a time. Deglutition may be difficult, and the contractility of the sphincters lost. The urinary secretion and glandular activity generally is arrested or retarded. The special senses are rendered more or less unimpressible. Articulation is imperfect, and the voice feeble or altogether lost. Respiration is feeble; but it becomes quickened with the pulse, though not in any fixed ratio. Its chief peculiarity is irregularity. Most of the inspirations are shallow and of the most varying degrees of rapidity. Sighing inspirations are frequent, but with no regular order of succession. Respiration may be so feeble as to be with difficulty detected. Deglutition is mostly possible as to fluids, and to solids thrust within the reach of the constrictors. Nausea and vomiting may also be present, but rarely in the most marked periods of shock. If the rectum be loaded at the occurrence of the injury, the contents will be spontaneously evacuated. The contents of the bladder are retained.

In the slighter forms of shock, the surface is cold, and the recipient of the injury complains, often repeatedly, of feeling cold. Muscular action is much impaired, but not suspended; indeed, impaired muscular action and enfeebled circulation are the most apparent characteristic indications of the slighter forms of shock. The pulse is feeble, intermittent, fluttering, and irregular. Respiration is shallow, with irregular sighs; the sighs being every second or third inspiration at one time, at the fifth or seventh at another, at the ninth or tenth at another. The brain, unless the injury have been direct to the head, is comparatively active, especially if the position of the body be recumbent, but quite unequal to the higher efforts of the intelligence. Digestion, the intestinal peristaltic action, and gland-secretion, are all retarded. Nausea and vomiting often occur in this degree of shock.

Shock is not always proportionate in its intensity to the severity of the wound. It may be severe when the injury is slight, or it may be slight when the injury is severe. Many circumstances contribute to this apparent inequality in the operation of given causes. But this remark applies chiefly to the varieties of shock other than the most severe. In the most intense form of shock which is compatible with the continuance of life, the nature of the injury,

* Continued from page 75 of Journal for Jan. 26th.

a British surgeon, Jordan Furneaux, wrote what is considered to be one of the first elaborate descriptions of abnormalities in the peripheral circulation during shock conditions.[4] In his description, he emphasised the cold, clammy and mottle skin associated with a high heart rate (*figure 1*). The belief held by notable physicians of that time was that those alterations in the peripheral circulation during shock were the result of a disorder of the nervous system, known as 'nervous collapse'. Despite these results, the final studies on neural regulation of cardiovascular function in shock did not occur until 1950s.

The era of modern haemodynamic monitoring begins, in many ways, with two important technological advancements: the ability to measure blood pressure noninvasively and cardiac output. After the introduction of the mercury sphygmomanometer by Scipione Riva-Rocci in 1896, hypotension started to be used to define shock.[5] Riva-Rocci introduced the now familiar instrument that collapses vessels by means of an inflatable cuff, which was widely adopted (*figure 2*). Later on, the work of the German doctor, Werner Forssmann, with his studies of right heart catheterisation, earned him the Nobel Prize in Medicine and Physiology in 1956 (*figure 3*).[6] The primary purpose was to develop a technique for direct delivery of drugs to the heart. His success culminated later on with the development of thermodilution cardiac output measurements with a flow-directed pulmonary artery catheter by Swan and Ganz.[7] The ability to measure blood pressure and cardiac output contributed to a great extent to the understanding of the pathophysiology of shock. Thus, the definition of shock and how it changed over the decades correlated with the advancements in technology used to assess the condition. In the 1940s, the definition of shock was based only on blood volume alterations.[8] In 1950s, the definition was expanded to the concept that the shock condition could become irreversible.[9] In 1960s, low cardiac output started to be used to define shock, and more recently, with current knowledge, the definition of shock is extended to the cellular level (*table 1*).[10-12]

Systemic versus local regulation of circulation

One important step in the understanding of the dynamics of shock was the recognition of the role of the peripheral circulation. In this context, one of the earliest references to the dynamic component of the peripheral vascular bed was the work of the Danish scientist, August Krogh, in the 1920s. He addressed fundamental issues underlying the behaviour of the microvasculature during physiological stimulus. In his line of work, he demonstrated adaptive microvascular adjustments in the muscle during exercise.[13] His investigations suggested a selective increase in the delivery of oxygen to the tissue by mechanisms of recruitment or derecruitment of capillaries with an active blood flow. For this, he was awarded the Nobel Prize in 1920 and he became an internationally well-known biomedical

Figure 2. The ability to measure blood pressure with the mercury sphygmomanometer was first developed by Scipione Riva-Rocci

Figure 3. Werner Forssmann (1904–1979), Berlin, introduced a ureteral catheter into his left basilic vein (1929). Chest X-ray and his paper. Later on his success culminated in the development of thermodilution cardiac output measurements

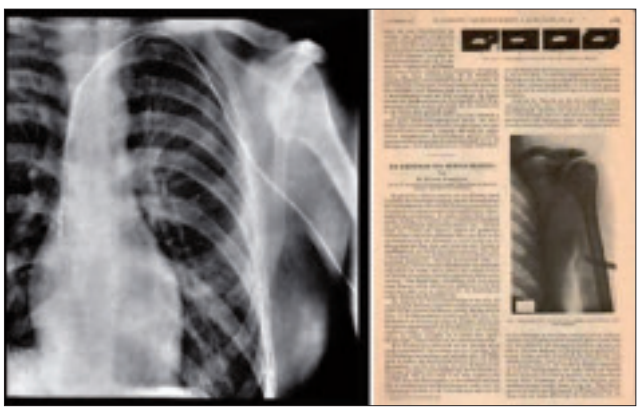


Table 1. Definitions of shock over the last decades. From 'blood volume alterations' to 'inadequate cellular utilisation'

"Shock is a peripheral circulatory failure resulting from a discrepancy in the size of the vascular bed and the volume of the intravascular fluid." Alfred Blalock, 1940s[8]
"Shock is a syndrome that results from a depression of many functions, but in which reduction of the effective circulating blood volume is of basic importance, and in which impairment of the circulation steadily progresses until it eventuates in a state of irreversible circulatory failure." Carl Wiggers, 1950s[9]
"A clinical condition characterised by signs and symptoms, which arise when the cardiac output is insufficient to fill the arterial tree with blood, under sufficient pressure to provide organs and tissues with adequate blood flow." Simeone, 1960s[10]
"State in which profound and widespread reduction of effective tissue perfusion leads first to reversible, and then if prolonged, to irreversible cellular injury." Kumar and Parrillo, 2001[11]
"Clinical expression of circulatory failure that results in inadequate cellular oxygen utilisation." De Backer and J-L Vincent, 2013[12]

scientist during the first decade of the 20th century. August Krogh's studies not only provided one of the fundamental principles of the blood-tissue perfusion relationship, but also attracted a new generation of clinical investigators who ameliorated human studies in circulatory function. From the 1930s and 1940s, physiology was beginning its growth as a science and investigations of the phenomena of shock and variations of local blood flow in the peripheral circulation became the focus of scientists' attention. During this period, many investigators showed that the terminal network of microscopic-sized vessels represents an organic unit essential in the maintenance of tissue perfusion. But it was in 1950 that the American physiologist, Carl John Wiggers, in his experimental studies of haemorrhagic shock, introduced the term 'peripheral circulation failure' by demonstrating the association between acute reduction of circulating blood volume and impairment of perfusion at the tissue level.[14] His studies were followed by a broad spectrum of basic physiological questions that were posed with respect to the role of the peripheral circulation under circulatory shock conditions.

Although in the early 1900s the scientific community had already realised there were other origins of shock than trauma, it was only in the 1920s and 1940s that it became recognised that intravenous administration of endotoxins (e.g., typhoid toxin) could produce hypotension. Some doctors even suggested a therapeutic use for endotoxin derived from P. aeruginosa to treat malignant hypertension, fortunately a treatment further rejected by the medical establishment.[15] The connection between hypotension and peripheral vasodilatation in vasodilatory shock was first published in a review by Gilbert in 1960.[16] It was clear from his review that local vascular changes and general cardiovascular alterations were both part of the endotoxin challenge. He suggested that changes in systemic vascular resistance could not provide information about local vascular resistance changes and described this phenomenon as "dilation in one (vascular) bed might be accompanied by constriction elsewhere"; he was one of the first to provide evidence of heterogeneous distribution of blood flow in sepsis. This phenomenon was later confirmed with the use of intravital microscopy with experimental shock models in the 1960s, when the different behaviour between peripheral circulation within organs and systemic circulation during acute shock situations became apparent.[17] This overlap in systemic versus local regulation led to a shift in thinking concerning separate sets of regulatory mechanisms for central and peripheral circulation during shock conditions. In this regard, the work of the physiologist, Arthur Clifton Guyton, was perhaps one of the most important scientific contributions in this field. Guyton is most famous for his experiments in the 1950s and 1960s, which studied the physiology of cardiac output and its relationship with the peripheral circulation. He elaborated on the *Frank-Starling law of the heart* by showing that it is not only the function of the heart which controls cardiac output but instead that cardiac output is controlled by various factors in the peripheral circulatory system which regulate the return of blood to the heart. It was Guyton who formulated the concept that the venous return is approximately proportional to the mean circulatory filling pressure minus the right atrial pressure; this pressure difference is called the pressure gradient for venous return.[18] The clinical relevance of such studies became apparent in the 1970s and 1980s when investigators recognised that resuscitation is based on knowledge of fundamental physiological variables not only of the central but also of the peripheral circulation.

Monitoring of the peripheral circulation

In 1958, in an attempt to perform real-time measurements of vital signs and alarms in a four-bed unit called the 'Shock Ward', Dr. Weil introduced continuous monitoring of the electrocardiogram, blood pressure, pulse rate, respiratory rate, and other vital signs complemented by arterial and central venous pressures, urine output and by peripheral temperatures. With the initial emphasis on myocardial infarction complicated by cardiogenic shock, it was the first prototype of an intensive care unit with continuous monitoring, which later became a 42-bed intensive care unit at the University of Southern California. His service pioneered routines of bedside monitoring and measuring devices, including the earliest use of arterial and central venous catheters.[19] In the 1960s, respiratory and haemodynamic measurements were complemented by laboratory measurements, including lactate and blood gases analysis.

Critical care medicine has emerged as an independent multidisciplinary speciality with the first organisation created by Drs. Safar, Shoemaker and Weil in 1967, which evolved into the Society of Critical Care Medicine. Over the years that followed, increasingly sophisticated haemodynamic and respiratory methods of monitoring were introduced. Weil was one of the first investigators to study the relationship between peripheral circulation measured with skin temperature and prognosis of low flow shock. In 1969, in a cohort of 100 patients (44 non-survivors) admitted with the diagnosis of cardiogenic or hypovolaemic shock, he showed that the likelihood of death was high when the skin temperature measured on the big toe was persistently low (< 27°C) during the first three hours of admission.[20] He also found a high correlation between skin temperature and cardiac output. Weil suggested that skin temperature measurements in low flow shock could be an alternative approach to overcome the technical difficulty of measuring cardiac output, which by that time was still problematic at the bedside.

New concepts have evolved which have defined organ or systemic failures. Clinical reports of patients who developed multiple organ failure after trauma, shock or sepsis started

to emerge describing the factors contributing to organ dysfunction. In the 1970s, systemic organ perfusion was assessed indirectly at the bedside by measuring the degree of derangement in global variables, such as cardiac output, lactic acidosis and base deficit, and resuscitation of the critically ill was based on normalisation of these haemodynamic values. In the 1980s, some studies proposed increasing cardiac output or oxygen delivery to 'supranormal' values, an alternative approach aimed at adequate organ utilisation.[21,22] In the 1990s, it became clear that even though global haemodynamic variables may be normalised, there could be regions with inadequate oxygenation at the tissue level, suggesting the importance of assessment of regional oxygenation at the organ level. Of all of the tissue-specific vascular beds that could be monitored, the gut mucosa, and specifically the gastric mucosa, was more commonly appropriated due to its easy assessment. Gastric tonometry was then effectively introduced into clinical practice for assessing the adequacy of local gastrointestinal perfusion, and this represents an important landmark in the history of peripheral circulation monitoring.[23-26] Because gastric tonometry has demonstrated its prognostic value, the field of regional noninvasive monitoring has gained wide interest, and studies have begun to address the importance of monitoring peripheral vascular beds that are more susceptible to hypoperfusion. This scenario has led to growing interest in noninvasive methods designed to evaluate regional perfusion in peripheral tissues as a valuable adjunct to standard global parameters to predict or diagnose ongoing tissue hypoperfusion. During the last few years, the advent of techniques enabling the continuous monitoring of peripheral circulation has shifted the clinical focus to maintaining normal perfusion in non-vital organs, such as the skin, subcutaneous tissue and muscle. Today, noninvasive monitoring of the peripheral circulation is based on the measurement of physiological variables directly or through signal processing (with the use of a data processor), and these methods do not involve intravascular catheters, transoesophageal probe insertion, blood component analysis or penetration of the skin.[27] Direct means of assessing the peripheral circulation are limited to the clinical evaluation and measurements of body temperature gradients. Processed noninvasive variables include transcutaneous oximetry and optical monitoring. Transcutaneous oximetry is based on the electrochemical properties of noble metals to measure the oxygen content of the tissue, whereas optical monitoring utilises the optical properties of haemoglobin to measure the partial pressure of oxygen and haemoglobin saturation. Recent progress in understanding the role of the peripheral circulation in haemodynamic regulation during shock has led to the application of these investigative techniques to continually monitor peripheral circulation in critically ill patients. However, the true clinical implications of this strategy may be better defined in future clinical trials of peripheral perfusion target resuscitation. Noninvasive monitoring of the peripheral circulation is growing, and it is the hope that simple direct measures and instruments can continue to provide new knowledge for the resuscitation of critically ill patients.

References

1. Parapia LA. History of bloodletting by phlebotomy. Br J Haematol. 2008;143:490-5.
2. Manji RA, Wood KE, Kumar A. The history and evolution of circulatory shock. Crit Care Clin. 2009;25:1-29, vii.
3. Morris EA. A practical treatise on shock after operations and injuries. Philadelphia: J.B. Lippincott & Co, 1868.
4. Jordan F. On Shock after Surgical Operations and Injuries. Br Med J. 1867;1:136-7.
5. Riva-Rocci S. Un nuovo sfigmemanometro. Gazetta Medica di Torino. 1896;47:981-6.
6. Bourassa MG. The history of cardiac catheterization. Can J Cardiol. 2005;21:1011-4.
7. Swan HJ, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. N Engl J Med. 1970;283:447-51.
8. Blalock A. Acute circulatory failure as exemplified by shock and hemorrhage. Surg Gynecol & Obst. 1934;58:551-66.
9. Wiggers CJ. The physiology of shock. Harvard University Press, 1950.
10. Simeone FA. Some issues in problems of shock. Fed Proc. 1961;20:3-11.
11. Kumar A, Parillo JE. Shock: pathophysiology, classification and approach to management. In: Parrillo JE, Parrillo JE, Bone RC (eds): Critical care medicine: Principles of diagnosis and management. Mosby: Toronto, 1995, pp 371-420.
12. Vincent JL, De Backer D. Circulatory shock. N Engl J Med. 2013;369:1726-34.
13. Krogh A. The number and distribution of capillaries in the muscles with calculations of oxygen pressure necessary for supplying the tissue. J Physiol London. 1919;52:409-15.
14. Wiggers CJ, Ingraham RC, Dillie J. Hemorrhagic-hypotension shock in locally anesthetized dogs. Am J Phys. 1945;143:126-33.
15. Dapremont C, Thomas RF, Johnson JB. Observations on the effects of pyrogens in the treatment of patients with hypertension. J Natl Med Assoc. 1951;43:300-4.
16. Gilbert RP. Mechanisms of the hemodynamic effects of endotoxin. Physiol Rev. 1960;40:245-79.
17. Hinshaw LB. Sepsis/septic shock: participation of the microcirculation: an abbreviated review. Crit Care Med. 1996;24:1072-8.
18. Guyton AC, Lindsey AW, Kaufmann BN. Effect of mean circulatory filling pressure and other peripheral circulatory factors on cardiac output. Am J Physiol. 1955;180:463-8.
19. Weil MH, Tang W. From intensive care to critical care medicine: a historical perspective. Am J Respir Crit Care Med. 2011;183:1451-3.
20. Joly HR, Weil MH. Temperature of the great toe as an indication of the severity of shock. Circulation. 1969;39:131-8.
21. Vincent JL. The International Sepsis Forum's frontiers in sepsis: High cardiac output should be maintained in severe sepsis. Crit Care. 2003;7:276-8.
22. Sharma VK, Dellinger RP. The International Sepsis Forum's frontiers in sepsis: High cardiac output should not be maintained in severe sepsis. Crit Care. 2003;7:272-5.
23. Doglio GR, Pusajo JF, Egurrola MA, et al. Gastric mucosal pH as a prognostic index of mortality in critically ill patients. Critical Care Medicine. 1991;19:1037-40.
24. Gutierrez G, Palizas F, Doglio G, et al. Gastric intramucosal pH as a therapeutic index of tissue oxygenation in critically ill patients. Lancet. 1992;339:195-9.
25. Marik PE. Gastric intramucosal pH. A better predictor of multiorgan dysfunction syndrome and death than oxygen-derived variables in patients with sepsis. Chest. 1993;104:225-9.
26. Maynard N, Bihari D, Beale R, Smithies M, Baldock G, Mason R, McColl I. Assessment of splanchnic oxygenation by gastric tonometry in patients with acute circulatory failure. JAMA. 1993;270:1203-10.
27. Lima A, Bakker J. Noninvasive monitoring of peripheral perfusion. Intensive Care Med. 2005;31:1316-26.