Trends in Anaesthesia and Critical Care 5 (2015) 89–92

Contents lists available at ScienceDirect

Trends in Anaesthesia and Critical Care

journal homepage: www.elsevier.com/locate/tacc

LESSON FROM CLINICAL PRACTICE

Vital signs after haemorrhage – Caution is appropriate

William Schultz, Ian McConachie*

Department of Anesthesia and Perioperative Medicine, Western University London, Ontario, Canada

Keywords:
Haemorrhage
Trauma
Vital signs
Heart rate
Blood pressure

SUMMARY

Clinicians rely on a patient's vital signs, especially heart rate and blood pressure to assess the magnitude of haemorrhage in patients presenting with shock due to traumatic injury or other causes. Conventional wisdom is that HR rises proportionally to the volume of blood loss. This conventional wisdom is useful but can be misleading, especially in the absence of significant tissue trauma. The physiological responses to haemorrhage are complex and patients who have suffered significant haemorrhage may not present with tachycardia.

© 2015 Elsevier Ltd. All rights reserved.

Clinicians rely on a patient's vital signs, especially heart rate (HR) and blood pressure (BP) to assess the magnitude of haemorrhage in patients presenting with shock due to traumatic injury or other causes. Various systems of assessment have been proposed – the most widely adopted of which is the ATLS system (Advanced Trauma Life Support) developed by the American College of Surgeons.[1] ATLS guidelines suggest that blood loss can be grouped into 4 classes which are associated with different physiological responses. Progression through each stage, from <15% loss of blood volume to >40% loss of blood volume, will result in progressive increases in heart rate, decreases in blood pressure and pulse pressure, increases in respiratory rate, and decreases in level of consciousness.

These classically taught changes in vital signs with progressive haemorrhage form the backbone of clinical assessment of haemorrhage for many practicing physicians and are used by physicians to triage patients, to guide initial therapy and evaluate response to resuscitation.[2–4]

Thus, conventional wisdom is that HR rises proportionally to the volume of blood loss but BP is initially maintained by vasoconstriction. This conventional wisdom is useful in the main, but can be misleading.

1. False sense of security from vital signs?

A recent case at our academic institution is that of a 15 year old male who sustained a single 1 inch penetrating wound in the chest wall causing a haemothorax requiring emergent sternotomy and ligation of the bleeding vessel. Apart from some superficial cuts to the arms there were no other injuries. On presentation to the Operating Room the patient's vital signs were essentially normal with, in particular, a HR in the 60 s. At thoracotomy, the estimated blood loss was at least 2.5–3 L. According to ATLS guidelines, this degree of blood loss (approximating 40% of blood volume) would be expected to result in the patient's HR being >120/min. In retrospect, the normal vital signs and, in particular, the low-normal HR, had resulted in significant underestimation of the volume of blood loss (and also inadequate resuscitation prior to anaesthesia). This case has provided a valuable opportunity for experiential learning among anaesthesia trainees, and has spurred us to review the human physiologic response to acute haemorrhage, both with and without concomitant trauma, with the aim of improving future management of similar cases.

2. Physiological responses to haemorrhage

Following haemorrhage, the body attempts to compensate for blood loss. The end-goal of these compensation attempts is to maintain oxygen delivery (DO_2) to vital organs such that organ function is maintained. Classically, in response to a decrease in circulating blood volume, compensatory mechanisms include an increase in heart rate initiated by arterial baroreceptors located in the aortic arch and carotid bodies. These receptors sense a decrease in circulating blood volume, and decrease their tonic inhibition of the sympathetic nervous system. As such, the cardiovascular system, which in a resting state is predominantly governed by the parasympathetic system, receives an increased influence of the sympathetic system.[5] The result is an increased heart rate such that, although the blood volume available for oxygenation is less, it is

* Corresponding author. Tel.: +1 519 685 8500x55115.
E-mail address: ian.mcconachie@sjhc.london.on.ca (I. McConachie).

http://dx.doi.org/10.1016/j.tacc.2015.04.001
2210-8440/© 2015 Elsevier Ltd. All rights reserved.

circulated faster, and thus delivers oxygen at the same rate to the tissues. Vasoconstriction attempts to maintain BP. Further compensation is mediated by the tissues themselves, which if delivery of oxygen by the circulating blood volume falls, can extract a greater fraction of oxygen and maintain their metabolic functions.[6–8] While these changes are aimed at preserving organ function, neuroendocrine changes are aimed at restoring the organism's normal homeostasis. Sympathetic stimulation of the renin-angiotensin-aldosterone system is aimed at restoring the organism's circulating volume, as is an upregulation of vasopressin release. Further mechanisms include an upregulation of the Hypothalamus Pituitary Adrenal axis, resulting in an increase in cortisol production. Cortisol helps to osmotically maintain circulating blood volume. It is also involved in a complex regulation of blood glucose mediated via the sympathetic nervous system and the endocrine system, resulting in an increase in blood glucose supply to the tissues.[9] Tissue hypoperfusion and ischaemia leads to cytokine response and an inflammatory response. Although the cytokine release is an inflammatory response important in the body's ability to fight infection and for the wound healing process, exaggerated cytokine responses in relation to overwhelming trauma contribute to the systemic inflammatory response syndrome. It is debatable whether these exaggerated responses are wholly protective towards the organism.

3. The difference between “simple” haemorrhage and “traumatic” haemorrhage

‘Simple’ haemorrhage, where the subject has brisk removal of blood, but minimal tissue trauma is commonly seen with penetrating trauma (minimal tissue injury as opposed to blunt trauma), gastrointestinal bleeding, and ruptured ectopic pregnancy. This is different from haemorrhage accompanied by significant tissue injury (be that from traumatic injury or major surgical incision). Simple haemorrhage is tolerated much better by the vital organs. With significant tissue injury the fall in BP is less than that caused by equivalent blood loss without tissue injury – but at the potential expense of reduced organ blood flow.[10] In addition, tissue injury and nociception impair the body's ability to maintain oxygen consumption (VO_2) by increased oxygen extraction resulting in acidemia and increasing plasma lactate and cortisol levels.[6,7]

3.1. Physiological mechanisms

The baroreceptor reflex maintains arterial blood pressure in early stages of shock. This is achieved via stretch receptors in the aortic arch and carotid body which tonically inhibit the sympathetic nervous system. They respond to a decrease in arterial stretch, and reflexively decrease their inhibition of the sympathetic system.[9] This reflex is impaired by significant tissue trauma (but not haemorrhage), occurs within 3 h of injury and may persist for at least 2 weeks.[11] Although the mechanisms by which this occurs remain unclear, animal studies suggest that the reflex is impaired by changes in the brainstem rather than in the afferent or efferent limbs of the reflex.[12] The resultant relatively fixed tachycardia from tissue injury, but not from haemorrhage, is a further confounder of the simplistic ATLS classification of responses to haemorrhage.

Furthermore, although VO_2 is maintained in phase 1 of simple haemorrhage, VO_2 in haemorrhage with concomitant tissue trauma is significantly lower.[6–8] In addition to a decreased VO_2, it appears that alterations in organ blood flow distribution in traumatic injury patients lead to poorer outcomes. Evidence exists that in simple haemorrhage, blood is prioritized to vital organs.[13] However, in haemorrhage with significant tissue injury, blood is shunted preferentially to skeletal muscle (a tissue which tolerates hypoxia quite well) and away from the gut (which tolerates hypoxia poorly), leading to increased risk of gut ischaemia, and bacterial translocation resulting in sepsis.[14]

4. Response of vital signs in haemorrhage

Significant data from both humans and animal models exists which details the heart rate response to haemorrhage as biphasic, or at extremes triphasic.

The first phase of shock occurs with a loss of up to 30% of blood volume. It results in the classically taught autonomic response to decreased circulating volume. As blood volume is lost, baroreceptors cause an increase in arterial tone and blood pressure is maintained. A withdrawal of vagal activity and an increase in sympathetic discharge cause an increase in heart rate, and further help to maintain arterial blood pressure. Although DO_2 may decrease, the tissues are able to extract more oxygen, and thus VO_2 remains normal. The clinical result is a mild tachycardia with arterial blood pressure maintained, and vital organs perfused adequately.[5–8,15–17]

When blood loss progresses beyond approximately 30% of blood volume, a second phase of hemorrhagic shock is seen, referred to as the sympatho-inhibitory phase. During this phase, C-fibres in the left ventricle send afferent signals to the parasympathetic nervous system, whose efferent fibres then increase their output, resulting in inhibition of the sympathetic nervous system. The result is a normal or decreased heart rate, and the arterial pressure beginning to trend downwards.[5,14–18] At this point, DO_2 to the organs decreases to the point where VO_2 is no longer able to be maintained simply through increased extraction.[6–8] This clinical picture contrasts starkly with that predicted from ATLS guidelines, of a very tachycardic patient presenting with this degree of haemorrhage.

A third phase of shock is present in some experimental models, where at extremes of haemorrhagic shock, once the animal has passed through the sympatho-inhibitory phase, very significant tachycardia is seen in conjunction with significantly compromised arterial blood pressure and poor vital organ perfusion. This appears to represent a pre-terminal state of shock.[15]

5. Limitations of vital signs

Others have investigated the degree to which patients with traumatic injury progress through the ATLS classifications of hemorrhagic shock, and found that not all patients with hemorrhagic shock present with tachycardia, and that those who do, often do not present with the degree of tachycardia ascribed to the estimated blood loss. This has led some to feel that although ATLS provides a useful universal guideline for the teaching of trauma management, its guidelines when used in the absence of further evaluation may lead to significant underestimations of blood loss from haemorrhage.[2–4]

Evidence of the poor correlation between heart rate and the degree of blood loss from penetrating injuries has been documented by Vayer et al. in the military setting, and Snyder et al. in the civilian setting. They caution that a lack of tachycardic response to penetrating abdominal injury should not be taken as a sign that significant blood loss has not occurred.[19,20] Indeed, the inability to mount a tachycardic reponse to severe haemorrhage is associated with a poor outcome in patients presenting with exsanguination – chiefly as a result of penetrating injuries.[21] Additionally, in patients with major thoracoabdominal haemorrhage, normal vital signs did not accurately predict the absence of life threatening blood loss (except in cases of extreme blood loss).[22]

Using patterns of change of vital signs (such as HR) in inpatients is increasingly utilized to assess overall clinical status and identify

Download English Version:

https://daneshyari.com/en/article/2772548

Download Persian Version:

https://daneshyari.com/article/2772548

Daneshyari.com