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POINTS OF VIEW: BLOOD TRANSFUSION

Transfusion triggers

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SUMMARY

Transfusion triggers are increasingly accepted in surgery and in the critically ill. There is very little evidence to suggest a restrictive policy is harmful although higher levels might be sensible in those with cardiovascular disease. A considerable tranche of literature shows that blood is bad for a patient but to date no clear mechanism has emerged and there is an argument that needing blood, a surrogate for illness may be as relevant. The impact of anaemia in the postoperative phase has not been evaluated adequately. The triggers lend themselves to non-acute elective situations but where there is acute blood loss and haemodynamic instability a slightly higher threshold, nearer 10 g/dl, allows a margin of safety.

1. Introduction

In the past it was considered essential to maintain a near normal haemoglobin throughout the perioperative period and in patients with critical illness, based on evidence suggesting that there was an haematocrit, 30%, at which oxygen delivery and the flow characteristics of blood intersected.[1] More recently both observational studies and randomised controlled trials have driven the required haematocrit even lower.

The transfusion triggers that are widely used are shown below in Table 1.

The transition to the use of these values has taken time but they are now widely used.[2–6] There is still little evidence to suggest any detriment and so clinicians become more confident and comfortable with their implementation. This has resulted in a significant reduction in both the use and abuse of blood, although the same cannot be said for either platelets or fresh frozen plasma.

It is the intention of this review to look at the accumulation of evidential material both direct and circumstantial that has established these recommendations and to highlight the areas of potential problems for the future both peri-operatively and in critical care.

2. The physiology of haemoglobin and oxygen transport

Before discussing the implications of transfusion it is wise to briefly review the role of blood, or more specifically haemoglobin, in oxygen transport.

Oxygen delivery is measured by the product of cardiac output and bound haemoglobin ($CO \times Hb \times 1.34 \times$ saturation). There is dissolved oxygen but for these purposes it is minimal. Essentially the three variables are the haemoglobin concentration, the saturation (effectively the amount of oxygen bound to each haemoglobin molecule), and the total quantity of both being delivered. This is a global measure but the amount of oxygen actually delivered to cells depends on cellular demand and a host of local environment factors including pH temperature and pCO_2 which determine the extraction ratio, can be as high as 0.7. Alterations in the cardiac output and in the local extraction ratio allow immense flexibility in the amount of oxygen that can be delivered at tissue level. It is important to re-iterate that the amount taken up will be determined by both the tissue requirement and the oxygen supply i.e. the tissues generate the gradient between blood and cells and provided there is an adequate supply, take what is needed and no more.

In acute haemorrhage, assuming functional lungs, the two main factors influencing delivery to the tissues are the cardiac output and the haemoglobin saturation. If the haemoglobin is low an increase in cardiac output will facilitate adequate oxygen delivery.

The Critical Point is where oxygen delivery whether local or global fails to meet demand despite all compensatory measures. At that point anaerobic metabolism will start to increase. This was first demonstrated in a model of exsanguinations by Cain.[7] It is a composite of all the factors involved in both oxygen demand and delivery.

However as long as the global oxygen delivery is above the 'Critical Point' then global oxygen requirements are met. However this may not represent the case at a local level and even when global markers of oxygen utilisation are adequate there can be wide

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Table 1
Transfusion triggers (UK Blood Transfusion Handbook). Please note these are for the stable patient.

Surgery – euvolaemic	7–8 g/dl
Intensive care	8 g/dl
Known or suspected cardiovascular disease	9–10 g/dl

variation and possibly critically decreased oxygen supply to individual organs. For example in a patient with ischaemic heart disease a stenosed coronary artery may preclude adequate flow despite the global appearance being adequate. As myocardial requirements increase the stenosis impedes increased flow and local ischaemia ensues. In that situation the local Critical Point has been reached. Many factors can influence whether a patient reaches the Critical Point (Table 2). It is obvious that for any individual the clinician cannot know where the Critical Point lies nor know how close to the Critical Point a patient can go. What the clinician does know are the factors involved and the overt pathophysiology in an individual patient which are likely to influence their proximity to the Critical Point. It is their responsibility to keep them well above the Critical Point so that oxygenation of any tissue is not compromised.

Figs. 1 and 2 illustrate the Critical point with a falling haemoglobin so oxygen availability and how.

These figures are meant to illustrate proximity to the Critical Point is increased with a lower haemoglobin concomitant with an obvious reduction in the margin of safety.

3. Anaemia

The normal ranges of haemoglobin vary between laboratories but are roughly as seen in Table 3.

The relationship between potential oxygen carriage, haemoglobin and saturation are seen in Table 4. The reduction in safety as the haemoglobin falls is apparent as is the impact of haemoglobin when saturations fall. Clearly there are also significant prognostic implications for patients presenting to theatre or ITU with established anaemia. In the acute major haemorrhage this observation is obvious but there are also significant prognostic implications in chronic anaemia whatever the cause.[8] For example in chronic renal failure it will have implications in terms of haemodynamic compensation and fluid shifts. There are also well-established associations with feelings of well being and more specifically fatigue.

The use of anaemia as a screening tool should alert the clinician to potential co-morbidity but the issues regarding correcting that anaemia are less clear. Some studies suggest that there is no demonstrable benefit in correcting anaemia pre-operatively while others indicate that at low haemoglobin, and certainly at less than 6 g/dl there is an increased risk.[9] It has been established in anaesthesia that the elderly tolerate isovolaemic haemodilution at least to 8.8 g/dl well. Too much is also a problem. Early postoperative graft occlusion in vascular surgery has shown that a high

Table 2
Factors that may result in a patient being potentially closer to the critical point than normal.

1. Hypoxaemia from any cause
2. Hypovolaemia
3. Fixed cardiac output with LV impairment
4. Anaemia
5. Ischaemic heart disease
6. Arterial stenosis and vascular disease
7. Increased metabolic activity – increased oxygen consumption

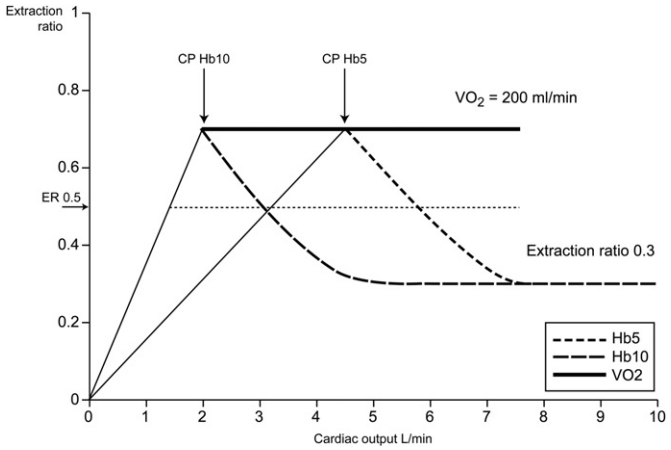

Fig. 1. The relationship between haemoglobin, cardiac output and the Critical Point. The lines represent the value when oxygen delivery of 200 ml/min is at the Critical Point. As haemoglobin falls the cardiac output must rise to stay above the critical point. This is modified if oxygen extraction rises (dotted line). Note that it is only at a haemoglobin of less than 5 when the cardiac output rises.

haemoglobin, greater than 12 g/dl, equates to a worse outcome.[10] A sound compromise might be 10 g/dl which is the value that was previously demonstrated to balance optimal viscosity and flow.

The focus tend to be on the physiology of anaemia which can be used to define exactly what is required in terms of oxygen delivery. If oxygen delivery is the only criterion, then a haemoglobin of 5 g/dl in a euvolaemic patient at normal pH and with their own blood, is more than adequate.[38] However there is no safety margin at all if the status quo alters.

This is confirmed by animal studies, human volunteer studies and in observational reports of Jehovah's witnesses.[11–14] In most patients very low haemoglobin values can be tolerated without obvious detriment. The available evidence suggests that the margin of safety is reduced if there is cardiovascular disease as adverse events seem to start increase below 10 g/dl but more impressively below 8 g/dl.[14] It would appear prudent that the haemoglobin should be higher and a value of 9–10 g/dl has been recommended.

There is inevitably a tension between the epidemiological studies that associate anaemia with prognosis and the physiological studies that suggest anaemia is functionally reasonable unless there is cardiovascular disease. The obvious implication being that

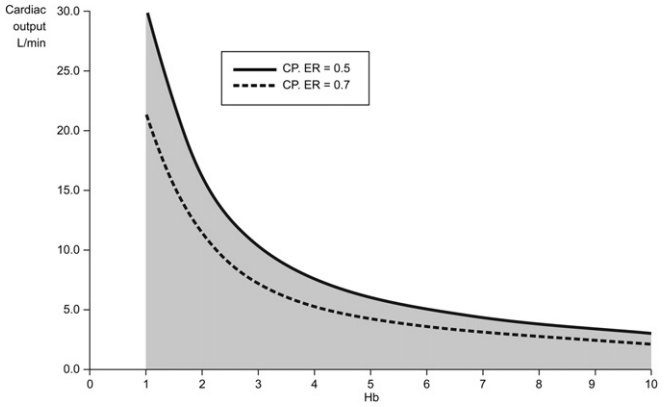

Fig. 2. The relationship between a reduced cardiac output, the Critical point and haemoglobin. At a set oxygen consumption of 200 ml/min as the cardiac output falls the extraction ratio rises. With a haemoglobin of 10 g/dl the Critical Point is not reached until the cardiac output is less than 3 L/min while at a haemoglobin of 5 g/dl the Critical Point is reached at 5 L/min.