Perioperative Patient Blood Management

Pillar One – Anaemia

Ross Kerridge
John Hunter Hospital
Newcastle
Newcastle
Where does our blood go?

- JHH – 2012, 12 286 units red cells

**NOTE:** Excludes blood wastage due to expiry...
Where does our blood go?

- CMN – 2012 – 7313 units red cells

**NOTE:** Excludes blood wastage due to expiry...
Blood Transfusion is ‘exciting’...........

- Implications of Current Research
- New Technology
- Multidisciplinary Complexity
- Variation in Clinical Practice
- Resource Implications
- Emotional Issue
- Political Minefield

AND A MODERN MIRACLE.....
BLOOD TRANSFUSION IS A MODERN MIRACLE.....
BLOOD TRANSFUSION IS A MODERN MIRACLE....
Coming soon to a Hospital near you:

Mindless Blood Transfusion
Hazards of Transfusion

- Mismatched Transfusion
- Infection
- Immunomodulation (TRIM)
- Sepsis
- TRALI
- TACO
- Haemodynamic Hazards of Volutransfusion
- Poorly Managed Massive Transfusion
- etc etc etc

And it isn’t good for you anyway....
Don’t transfuse red blood cells in hemodynamically stable, non-bleeding ICU patients with a hemoglobin concentration greater than 7 g/dL.

Most red blood cell transfusions in the ICU are for benign anemia rather than acute bleeding that causes hemodynamic compromise. For all patient populations in which it has been studied, transfusing red blood cells at a threshold of 7 g/dL is associated with similar or improved survival, fewer complications and reduced costs compared to higher transfusion triggers. More aggressive transfusion may also limit the availability of a scarce resource. It is possible that different thresholds may be appropriate in patients with acute coronary syndromes, although most observational studies suggest harms of aggressive transfusion even among such patients.

Choosing Wisely:
Avoid transfusions of red blood cells for arbitrary hemoglobin or hematocrit thresholds and in the absence of symptoms or active coronary disease, heart failure, or stroke.
What do I need to know about blood transfusion and perioperative bleeding?
Guidelines and overview of patient blood management, correct transfusion practice and consideration of patient iron levels.

Overview of transfusion medicine
Patient blood management guidelines
Obstetric transfusion
Appropriate transfusion practices

Patient blood management
Anaemia and Haemostasis management
Blood conservation

Updated on 22 June 2012
Blood Matters Program

The Blood Matters Program is a Victorian state government program for improving the quality and safety of hospital transfusion care to patients.

Transfusion data collection tool

Blood Matters has collected resources from Victorian Transfusion Nurses and Trainers and developed a data collection template. This template is a resource tool and may assist you to collect transfusion data and graphically present the results for your hospital.

Serious Transfusion Incident Report (STIR) 2009-11

We are pleased to release the third STIR report. A copy of the report has been forwarded to all Victorian Hospitals that are involved in transfusion.

It is also available to download. Please contact Blood Matters for more information.
Stop the Waste! promotional materials now available
Promotional materials to assist in your local wastage reduction campaigns are now...
Order your promotional materials today
Our courses

BloodSafe eLearning Australia courses are designed to improve your clinical practice and patient blood management knowledge. Listed below are our

Transfusion Practice courses

Clinical Transfusion Practice
(including Collecting Blood Specimens and Transporting Blood)

Blood transfusions can save and improve people’s lives, but the process must be performed correctly, using the right blood and ensuring appropriate use and safe practice procedures. It is designed for medical and healthcare professionals who require an understanding of blood transfusions and their treatment.

Clinical Transfusion Practice

Iron Deficiency Anaemia (IDA)

Iron deficiency anaemia is a common condition that causes fatigue and weakness. It is caused by a lack of iron in the body, which can be due to a diet that is low in iron or a condition that causes blood to be lost. This course aims to develop your knowledge of iron deficiency anaemia and its treatment. It is designed for healthcare professionals who require an understanding of iron deficiency anaemia and its management.

Iron Deficiency Anaemia (IDA) App

Medical and Specialties

This course aims to develop your knowledge of blood transfusions and their treatment. It is designed for medical and healthcare professionals who require an understanding of blood transfusions and their safe practice procedures. It is designed for medical and healthcare professionals who require an understanding of blood transfusions and their treatment.

Perioperative

Critical Bleeding

Postpartum Haemorrhage (PPH)

Patient Blood Management (PBM)

Transporting Blood

Collecting Blood Specimens

Clinical Transfusion Practice

Clinical Transfusion Practice

Clinical Transfusion Practice

Clinical Transfusion Practice

Clinical Transfusion Practice
Strategies to preempt and reduce the use of blood products: an Australian perspective

Axel Hofmann\textsuperscript{a,d}, Shannon Farmer\textsuperscript{a,b,d}, and Simon C. Towler\textsuperscript{a,c,d}

Purpose of review
Evidence-based patient blood management (PBM) is aimed at achieving better patient outcomes by relying on a patient’s own blood rather than on donor blood. This review covers the rationale behind PBM, the treatment modalities involved and the drivers to adopt PBM as a new standard of care.

Recent findings
Transfusion rates vary significantly between comparable countries; they also vary between centers for matched patients in standardized elective surgical interventions. Preoperative anemia, perioperative blood loss and liberal transfusion triggers are the main predictors for transfusion and pose risks to the patient. PBM is mitigating these risks by optimizing the patient’s native red cell mass, minimizing blood loss, optimizing the physiological reserve of anemia and preempting transfusions. A growing number of studies...
Patient Blood Management

1st Pillar
Diagnose and manage Anaemia

2nd Pillar
Minimise blood loss
Control bleeding

3rd Pillar
Avoid unnecessary transfusion

Multidisciplinary team approach

James Isbister
The Challenge:- Knowledge into Practice

- Preoperative Patient Optimisation: Anaemia
- Patient Education & Counselling
- Cell Salvage, Blood Collection and Reinfusion
  (Waste Avoidance)
- Massive Haemorrhage
- Point of Care Testing & Thromboelastography
- The Age of Blood
  (Electronic Issue & Remote Release)

- Back to Basics – Simple Hazards of Transfusion
- Appropriate Transfusion Triggers
  – WHY WON’T DOCTORS CHANGE????
- How do we get Doctors to change?
Anaemia and haemostasis:
Perioperative Guidelines

- Preoperative anaemia assessment
- Evaluate surgical patients early
- Optimise patient’s haemoglobin and iron stores
- Administer preoperative iron therapy
- Manage clopidogrel, aspirin, NSAID and warfarin as per guidelines
Why so much fuss about Anaemia???
Preoperative Anaemia:
Common in Adelaide

The prevalence of anaemia, hypochromia and microcytosis in preoperative cardiac surgical patients

O. DAVID*, R. SINHA†, K. ROBINSON‡, D. CARDONE‡
Royal Adelaide Hospital, Department of Anaesthesia, Adelaide, South Australia, Australia

SUMMARY
This retrospective study aimed to determine the prevalence of preoperative anaemia, hypochromia and microcytosis in cardiac surgery patients. Data was analysed for 943 patients (over a two-year period) undergoing coronary artery bypass graft, valve or combined coronary artery bypass graft and valve surgery at a tertiary hospital in South Australia. Overall prevalence of preoperative anaemia was 25.2%, greater in males than females (27.6 vs 19.9%, *P* < 0.01). Of patients with preoperative anaemia, 19.3% had reduced red cell indices (mean corpuscular haemoglobin and/or mean corpuscular volume) compared to 4% of patients without anaemia. The proportion of anaemic patients with low red cell indices was significantly higher in women <50 years and 50-65 years, compared to those >65 years of age (*P* = 0.003). Anaemic patients with low red cell indices had lower preoperative haemoglobin than anaemic patients without low red cell indices (median haemoglobin 112 vs 120 g/L, *P* = 0.008). Compared to non-anaemic patients, anaemic patients had higher transfusion rates (79.8 vs 46.4%, *P* < 0.001), which were greater in those with reduced red cell indices compared to those with normal red cell indices (93.5 vs 76.6%, *P* = 0.01). This study demonstrated a high prevalence of preoperative anaemia, microcytosis and hypochromia in cardiac surgical patients.

SA Data Linkage Study (Colorectal Surgery)
2821 elective surgical admissions 2008-09 & 09-10

- Pre-op anaemia in 28% overall
  - 20% < 65 years
  - 31% 65-85 years
  - 44% > 85 years

Anaemic:
- 14% MCV < 80
- 32% MCH < 27
Preoperative anaemia

**Table 9: Proportion of patients with pre-operative haemoglobin level below normal range by site and surgery type**

<table>
<thead>
<tr>
<th>DHB</th>
<th>CABG</th>
<th>THR</th>
<th>TAH</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auckland</td>
<td>15%</td>
<td>32%</td>
<td>20%</td>
<td>22%</td>
</tr>
<tr>
<td>Canterbury</td>
<td>25%</td>
<td>18%</td>
<td>14%</td>
<td>19%</td>
</tr>
<tr>
<td>Capital &amp; Coast</td>
<td>5%</td>
<td>5%</td>
<td>15%</td>
<td>8%</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>-</td>
<td>18%</td>
<td>14%</td>
<td>16%</td>
</tr>
<tr>
<td>MidCentral</td>
<td>-</td>
<td>8%</td>
<td>25%</td>
<td>14%</td>
</tr>
<tr>
<td>Otago</td>
<td>25%</td>
<td>14%</td>
<td>5%</td>
<td>15%</td>
</tr>
<tr>
<td>Waikato</td>
<td>5%</td>
<td>15%</td>
<td>15%</td>
<td>12%</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>15%</td>
<td>15%</td>
<td>15%</td>
<td>15%</td>
</tr>
</tbody>
</table>
Preoperative Anaemia:-
*Common in Newcastle*

439 Elective Unilateral TKA & THA

- **15.8% Anaemic preoperatively;**
- **84.5% Anaemic postoperatively.**
- Mean Hb decrease of 25g/L
- Transfusion Rate of 3.9%, strongly predicted by preop anaemia

- Only 31 (7.7%) had Iron studies preop:
  - 1 Severely deficient (Ferritin <30)
  - 12 Ferritin 30-100, of whom 8 were non-anaemic.

n.b. A moderate pre-existing Iron deficiency may impair postoperative erythropoiesis, recovery and rehabilitation.
Risk Associated with Preoperative Anemia in Noncardiac Surgery

A Single-center Cohort Study

W. Scott Beattie, M.D., Ph.D., F.R.C.P.C.,* Keyvan Karkouti, M.D., M.Sc., F.R.C.P.C.;†
Duminda N. Wijeysundera, M.D., F.R.C.P.C.;‡ Gordon Tait, Ph.D.§

• All elective surgery 2003-6 (except CT and Tx)
  • 7670 patients
  • 3047 anaemic (39.8%)
  • 1430 (18.6%) patients transfused
• Mean Hb pre-op 12.7 ± 2.1g/dl
• 68% of transfusions occurred in patients with preop anaemia (30.4% vs 10.6% non-anaemic)
Anaemia also independently predicts mortality.
Anaemia independently predicts mortality

Table 2. Regression Analysis of Anemia Model Predicting Mortality

<table>
<thead>
<tr>
<th>Parameter</th>
<th>OR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amenia (WHO gender defined)</td>
<td>2.43 (1.65–3.60)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age &gt; 70 yr</td>
<td>2.31 (1.64–3.26)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>In-hospital status*</td>
<td>3.51 (2.26–5.44)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>History of CHF</td>
<td>7.99 (4.73–13.5)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Preoperative renal dysfunction†</td>
<td>2.08 (1.22–3.53)</td>
<td>0.0067</td>
</tr>
<tr>
<td>Perioperative medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-Blockers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No β-blockers</td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>1.67 (1.05–2.68)</td>
<td>0.020</td>
</tr>
<tr>
<td>Atenolol or bisoprolol</td>
<td>0.97 (0.63–1.52)</td>
<td>0.198</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>0.56 (0.33–0.95)</td>
<td>0.033</td>
</tr>
<tr>
<td>CCBs</td>
<td>0.57 (0.34–0.96)</td>
<td>0.036</td>
</tr>
<tr>
<td>Any postoperative NSAID‡</td>
<td>0.58 (0.38–0.88)</td>
<td>0.011</td>
</tr>
<tr>
<td>Transfusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No blood products</td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>1–2 units</td>
<td>1.83 (1.20–2.80)</td>
<td>0.032</td>
</tr>
<tr>
<td>3–4 units</td>
<td>2.99 (1.77–5.07)</td>
<td>0.013</td>
</tr>
<tr>
<td>5–10 units</td>
<td>3.19 (1.62–6.32)</td>
<td>0.021</td>
</tr>
<tr>
<td>More than 10 units</td>
<td>3.43 (1.12–10.5)</td>
<td>0.040</td>
</tr>
</tbody>
</table>
Preoperative Anaemia:-

How do I diagnose it?

**Diagnosis and management of iron deficiency anaemia: a clinical update**

Sant-Rayn S Pasricha, Stephen C Flecknoe-Brown, Katrina J Allen, Peter R Gibson, Lawrence P McMahon, John K Olynyk, Simon D Roger, Helen F Savoia, Ramdas Tampi, Amanda R Thomson, Erica M Wood and Kathryn L Robinson*

**ABSTRACT**

- Iron deficiency anaemia (IDA) remains prevalent in Australia and worldwide, especially among high-risk groups.
- IDA may be effectively diagnosed in most cases by full blood examination and serum ferritin level. Serum iron levels should not be used to diagnose iron deficiency.
- Although iron deficiency may be due to physiological demands in growing children, adolescents and pregnant women, the underlying cause(s) should be sought.
- Patients without a clear physiological explanation for iron deficiency (especially men and postmenopausal women) should be evaluated by gastroscopy/colonoscopy to exclude a source of gastrointestinal bleeding, particularly a malignant lesion.
- Patients with IDA should be assessed for coeliac disease.
### Preoperative Anaemia:

**How do I diagnose it?**

#### Table: Interpreting laboratory blood test results to assess iron status

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Haemoglobin</th>
<th>Mean cell volume and mean cell haemoglobin</th>
<th>Serum ferritin μg/L</th>
<th>Transferrin or total iron binding capacity</th>
<th>Transferrin saturation†</th>
<th>Soluble transferrin receptor</th>
<th>Serum iron‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue iron deficiency without anaemia</td>
<td>Normal</td>
<td>Normal or low</td>
<td>&lt; 15–30</td>
<td>Normal or high</td>
<td>Low-normal or low</td>
<td>High-normal or high</td>
<td>Low</td>
</tr>
<tr>
<td>Iron deficiency anaemia (IDA)</td>
<td>Low</td>
<td>Low (or normal in early IDA)</td>
<td>&lt; 15–30 adult</td>
<td>High</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Anaemia of chronic disease or inflammation</td>
<td>Low</td>
<td>Normal (may be mildly low)</td>
<td>Normal or elevated (elevated ferritin does not imply elevated iron stores)</td>
<td>Normal</td>
<td>Low</td>
<td>Normal</td>
<td>Low</td>
</tr>
<tr>
<td>IDA with coexistent chronic disease or inflammation</td>
<td>Low</td>
<td>Low</td>
<td>Low or normal, but usually &lt; 60–100 μg/L</td>
<td>Normal or high</td>
<td>Low</td>
<td>Normal</td>
<td>Low</td>
</tr>
<tr>
<td>Thalassaemia minor§</td>
<td>Low (or normal)</td>
<td>Low (or normal)</td>
<td>Normal or elevated</td>
<td>Normal</td>
<td>Normal or elevated</td>
<td>Normal or elevated</td>
<td>Normal</td>
</tr>
<tr>
<td>Iron overload</td>
<td>Normal</td>
<td>Normal</td>
<td>Elevated (correlates with body iron stores)</td>
<td>Normal to low</td>
<td>Normal</td>
<td>High</td>
<td>Normal to elevated</td>
</tr>
</tbody>
</table>

*Compared with laboratory reference range for age, sex and gestation if applicable. † Ideally performed on fasting morning sample.

‡ Serum iron is markedly labile with a significant diurnal variation, is low in both iron deficiency and inflammation, and should not be used to diagnose iron deficiency.

§ Includes β-thalassaemia minor and single or two alpha gene deletion thalassaemia minor. A thalassaemic condition and iron deficiency may coexist, particularly in pregnancy.
Preoperative Anaemia: How do I diagnose it?

Consistent with IDA
Review clinical findings for possible underlying pathology and sources of overt and occult blood loss (e.g., GI, genitourinary, nose, mouth, blood donation) *

Preschool children
- Consider:
  - Inadequate dietary iron
  - Inadequate complementary foods (e.g., excess milk ingestion)
  - Cows milk allergy
  - Rapid/rebound growth, former low birth weight
  - Coeliac disease
  - Parasitic infection
  - GI blood loss

Older children
- Consider:
  - Inadequate dietary iron (especially if vegetarian)
  - Rapid/rebound growth
  - Coeliac disease
  - Parasitic infection
  - GI blood loss

Adolescent and premenopausal women
- Consider:
  - Inadequate iron intake
  - Blood loss (e.g., menstruation, GI, haemostatic defect)
  - Coeliac disease
  - Parasitic infection

Risk factors for GI pathology:
- GI symptoms
- Family history of colorectal cancer
- Age ≥ 50 years
- Refractory, recurrent or unexplained IDA

Treatment (see Box 4):
- Oral iron (usually 100–200 mg elemental iron per day**) for at least 3 months after normalisation of Hb
- IV iron for selected patients (see Box 6)
- Optimise dietary iron (secondary prevention) and address underlying cause

Exclude:
- GI blood loss (e.g., neoplasm)
- Coeliac disease
- Investigations:
  - Gastroscopy/colonoscopy
  - Coeliac screening
  - Others as directed by clinical findings and context

Adult men and postmenopausal women
- Consider:
  - Inadequate dietary iron
  - Overt or occult blood loss

Treatment (see Box 4):
- Oral iron liquid (3–6 mg/kg elemental iron per day) for at least 2–3 months after normalisation of Hb
- Optimise dietary iron content
Preoperative Anaemia:- How do I diagnose it?

**2 Iron deficiency anaemia (IDA): assessment and management***

- Hb below laboratory reference range for age, sex, gestation (full history and physical examination is essential in all cases)

**Anaemia**

- Serum ferritin <15–30 μg/L in adults†
- Serum ferritin <10–12 μg/L in children (see Box 3):
  - **Confirmed IDA‡**
  - MCV may be normal in early IDA or with coexisting vitamin B₁₂ or folate deficiency

  **Serum ferritin >15–30 μg/L but <100 μg/L (see Box 3):**
  - Possible IDA†
  - Iron deficiency is not excluded in the presence of inflammation, chronic disease or high CRP

  **Consider clinical context and seek advice**
  - Expert interpretation of blood film
  - Consider testing CRP, sTfR
  - Clinical context may warrant presumptive investigation and management as IDA
  - Consider discussion with haematologist or clinical pathologist

**Serum ferritin >100 μg/L: IDA unlikely**

- Identify alternative cause(s) of anaemia
- FID may still be present in ACD or CKD if Tfsat<20%
- Diagnosis of FID may be important for directing therapy with IV iron and ESAs

**Not consistent with IDA**

- Identify alternative cause(s) of anaemia

**Consistent with IDA**

- Review clinical findings for possible underlying pathology and sources of overt and occult blood loss (eg, GI, genitourinary, nose, mouth, blood donation)*
Preoperative Anaemia: There’s an App for that!

app for diagnosis & management IDA
www.bloodsafelearning.org.au

Is anaemia present? ✶

Is haemoglobin below laboratory reference range for age, sex and gestation?

Yes  No

Full history and examination is essential in all cases.

What is the ferritin level?

- Serum ferritin <15-30 mcg/L in adults ✶ or <10-12 mcg/L in children ✶
- Serum ferritin >15-30 mcg/L but <100 mcg/L
- Serum ferritin >100 mcg/L


• Spahn DR. Anemia and patient blood management in hip and knee surgery: a systematic review of the literature. Anesthesiology 2010; 113: 482-95


Preoperative Anaemia:- How do I diagnose it?

Preoperative haemoglobin assessment and optimisation template

This template is for patients undergoing procedures in which substantial blood loss is anticipated such as cardiac surgery, major orthopaedic, vascular and general surgery. Specific details, including reference ranges and therapies, may need adaptation for local needs, expertise or patient groups.

**Preoperative tests**
- Full blood count
- Iron studies including ferritin
- CRP and renal function

**Is the patient anaemic?**
- Hb <130 g/L (male) or
- Hb <120 g/L (female)

**NO**
- Ferritin <30 mcg/L
  - No anaemia: ferritin <100 mcg/L
    - Consider iron therapy if anticipated postoperative Hb decrease is >30 g/L
    - Determine cause and need for GI investigations if ferritin is suggestive of iron deficiency <30 mcg/L
  - Iron deficiency anaemia
    - Evaluate possible causes based on clinical findings
    - Discuss with gastroenterologist regarding GI investigations and their timing in relation to surgery
    - Commence iron therapy

**YES**
- Ferritin 30–100 mcg/L
  - Possible iron deficiency
    - Consider clinical context
    - Consider haematology advice or, in the presence of chronic kidney disease, renal advice
    - Discuss with gastroenterologist regarding GI investigations and their timing in relation to surgery
    - Commence iron therapy

- Ferritin >100 mcg/L
  - Possible anaemia of chronic disease or inflammation, or other causes
    - Consider clinical context
    - Review renal function, MCV/MCH and blood film
    - Check B12/folate levels and reticulocyte count
    - Check liver and thyroid function
    - Seek haematology advice or, in the presence of chronic kidney disease, renal advice
Diagnosis of Iron Deficiency: Can be confusing

Pathology Notes

Requested by: DR ROSS KERRIDGE
Report To:
John Hunter Hospital
JHH Pre-Procedural Clinic

Diagnosis of Iron Deficiency: Can be confusing

Clinical Notes on Request Form: Hypertension

Pathology Notes

Report To:
John Hunter Hospital
JHH Pre-Procedural Clinic

Clinical Notes on Request Form: Hypertension

Analyte Units Ref Range
Blood Blood

Ferritin: ug/L (30 - 300) 119 28 L
Iron: umol/L (11 - 30) 8 L 14
Transferrin Sat'n: % (20 - 50) 12 L 16 L
Transferrin: g/L (1.7 - 3.6) 2.6 3.4

Comments


Red Cell Folate Comment

11 Dec 19 Feb 23 Feb 24 Jun
2014 2015 2015 2015
20:00 22:35 13:45 11:10
182821048164330685171205136178555937

Analyte Units Ref Range
Blood Blood Blood Blood

Full Blood Count

White Cells: 10⁹/L (4.0 - 11.0) 9.6 9.9 8.8 5.9
Red Cell Count: 10¹²/L (4.50 - 6.50) 5.25 4.81 5.03 4.73
Haemoglobin: g/L (130 - 180) 155 137 145 150
Haematocrit: L/L (0.380 - 0.490) 0.471 0.411 0.443 0.440
MCV: fl (80.0 - 100.0) 89.7 85.5 88.1 93.2
MCH: pg (27.0 - 32.0) 29.5 28.5 28.9 31.6
MCHC: g/L (310 - 360) 328 333 328 340
RDW: % (9.0 - 14.5) 12.6 12.6 13.2 19.6 H
Platelets: 10⁹/L (150 - 400) 196 245 246 212
MPV: fl (7.2 - 11.1) 10.2 10.4 10.6 9.4
Neutrophils: 10⁹/L (1.8 - 7.7) 6.4 7.1 6.2 4.5
Lymphocytes: 10⁹/L (1.0 - 4.0) 2.2 1.7 1.8 0.6 L
Monocytes: 10⁹/L (0.1 - 0.8) 0.6 0.9 H 0.4 0.5
Eosinophils: 10⁹/L (< 0.6) 0.2 0.1 0.4 0.2
Basophils: 10⁹/L (< 0.3) 0.2 0.1 0.0 0.0
Preoperative Iron Deficiency Anaemia:-
How do I treat it?

Oral Iron....

Effective? ....
Oral Iron – Side Effects

Fig. 1. Tolerability of intravenous ferric carboxymaltose in patients with iron-deficiency anaemia. Descriptive incidence of possible or probable drug-related adverse events occurring in ≥1% of patients receiving either ferric carboxymaltose administered as a 15-min infusion once weekly until each patient had received his or her calculated total iron replacement dose (maximum single weekly iron dose 1000 mg or 15 mg/kg in those weighing <66 kg) or oral ferrous sulfate (equivalent to 65 mg of iron three times daily or 100 mg of iron twice daily for 6–12 wk) in nine randomized, open-label, multicentre trials (available as an abstract plus poster).[^34]
6 Indications for intravenous (IV) iron therapy in patients with iron deficiency anaemia (IDA)

IV iron should be considered in patients with confirmed IDA* and one or more of the following:

- Demonstrated intolerance, non-compliance or lack of efficacy with oral iron, despite modification of dose, timing and frequency;
- Pregnancy (beyond the first trimester) and postpartum, for the above reasons or to avoid imminent decompensation/transfusion (eg, in women who present late and/or display severe anaemia);
- Intestinal malabsorption (eg, inflammatory bowel disease);
- Ongoing iron (ie, blood) losses that exceed absorptive capacity;
- A clinical need for a rapid iron supply (ie, in patients where optimisation of erythroid response is important to prevent physiological decompensation/ transfusion);
- Chronic renal impairment receiving concomitant erythropoietin-stimulating agent therapy.

* Prescribed in consultation with an expert in the use of IV iron and the relevant patient group.
Parenteral Iron?

- Iron Polymaltose
- Iron Carboxymaltose
- Iron Sucrose

(Other Formulations used elsewhere)
Parenteral Iron Formulations

Ionic Iron is highly reactive and hence toxic.

The body stores iron bound within Ferritin.

Parenteral Iron formulations must be bound in a complex that is stable in physiological conditions.

The stability of the complex determines the total safe dose.

Earlier formulations (e.g. dextran) were notoriously allergenic.
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

Hb response to Fe carboxymaltose

![Graph showing mean Hb change (g/L) over weeks from start of treatment for Ferric carboxymaltose and Oral iron.](http://www.biomedcentral.com/1471-2326/11/4)

Moore et al. BMC Blood Disorders 2011, 11:4  
http://www.biomedcentral.com/1471-2326/11/4
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

IV Iron in colorectal blood loss

Optimal Iron Replacement for Colorectal Cancer-Induced Anaemia

The Open Colorectal Cancer Journal, 2010, Volume 3

Fig. (1). Total Hb rise over the course of treatment with Venofer®, CosmoFer® and oral iron.
Preoperative Iron Deficiency Anaemia:—
Should I treat it with intravenous Iron?

PROTECT Trial

![Graph showing the percentage of non-anaemic patients over time with two treatments: placebo and iron isomaltoside 1000 infusion.](http://onlinelibrary.wiley.com/doi/10.1111/vox.12278/full#vox12278-fig-0004)

Non-anaemic: male: haemoglobin $\geq 13$ g/dl, females haemoglobin $\geq 12$ g/dl
Fisher's exact test: NS = not significant

$P < 0.05$
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

Effect of a patient blood management programme on preoperative anaemia, transfusion rate, and outcome after primary hip or knee arthroplasty: a quality improvement cycle


1 Airedale NHS Foundation Trust, Steeton, UK
2 School of Health Studies, University of Bradford, Bradford, UK
* Corresponding author: Department of Anaesthesia, Leeds General Infirmary, Great George Street, Leeds LS1 3EX, UK.
E-mail: alvvnkotec@doctors.net.uk

Fig 6 Hb response in patients offered preoperative treatment. Scatter plot showing individuals’ Hb concentration at surgical decision to list for arthroplasty, against the Hb change achieved with treatment.
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

Table 6  Before-and-after comparisons. Continuous data expressed as median (IQR). †P=0.02; *P<0.01; **P<0.001

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female:male ratio</td>
<td>412:305</td>
<td>155:126</td>
</tr>
<tr>
<td>TKR:THR ratio</td>
<td>356:361</td>
<td>123:158</td>
</tr>
<tr>
<td>ASA score</td>
<td>2 (2–2)</td>
<td>2 (2–3)</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>72 (65–78)</td>
<td>74* (66–80)</td>
</tr>
<tr>
<td>Anaemia prevalence at decision for surgery</td>
<td>166/684</td>
<td>73/281</td>
</tr>
<tr>
<td>Nadir Hb in transfused patients (g dl⁻¹)</td>
<td>7.8 (7.2–8.7)</td>
<td>7.6 (7.3–9.2)</td>
</tr>
<tr>
<td>Discharge Hb (g dl⁻¹)</td>
<td>10.4 (9.5–11.4)</td>
<td>10.4 (9.4–11.0)</td>
</tr>
<tr>
<td>Hb loss: THR (g dl⁻¹)</td>
<td>3.8 (2.9–4.9)</td>
<td>3.1** (1.9–4.6)</td>
</tr>
<tr>
<td>Hb loss: TKR (g dl⁻¹)</td>
<td>3.1 (1.9–4.6)</td>
<td>2.6 (2.0–3.3)</td>
</tr>
<tr>
<td>Received ABT: THR</td>
<td>83/361</td>
<td>12**/158</td>
</tr>
<tr>
<td>Received ABT: TKR</td>
<td>24/356</td>
<td>0**/123</td>
</tr>
<tr>
<td>Length of stay (days): THR</td>
<td>6 (5–8)</td>
<td>5** (3–7)</td>
</tr>
<tr>
<td>Length of stay (days): TKR</td>
<td>6 (5–8)</td>
<td>4** (3–6)</td>
</tr>
<tr>
<td>Readmitted within 30 days</td>
<td>49/717</td>
<td>12/281</td>
</tr>
<tr>
<td>Readmitted within 90 days</td>
<td>97/717</td>
<td>23†/281</td>
</tr>
</tbody>
</table>
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

Safety and efficacy of intravenous iron therapy in reducing requirement for allogeneic blood transfusion: systematic review and meta-analysis of randomised clinical trials

Edward Litton staff specialist clinical senior lecturer, Jing Xiao registrar, Kwok M Ho staff specialist associate professor

1Department of Intensive Care Medicine, Royal Perth Hospital, Perth, Western Australia 6000; 2School of Medicine and Pharmacology, University of Western Australia, Perth, Western Australia, 6009; 3School of Population Health, University of Western Australia, Perth, Western Australia, 6009

What is already known on this topic
Intravenous iron therapy is effective at increasing haemoglobin concentration in patients with iron deficiency anaemia

What this study adds
Intravenous iron therapy can effectively reduce the need for red blood cell transfusion across a range of acute care settings
The benefit of intravenous iron in increasing haemoglobin concentration was seen when compared with both oral iron and no iron supplementation and was more effective when used with erythropoietin stimulating agents and in patients with lower baseline plasma ferritin concentrations
Preoperative Iron Deficiency Anaemia: Should I treat it with intravenous Iron?

Red cell transfusion risk is reduced

<table>
<thead>
<tr>
<th>Study</th>
<th>No of events/total</th>
<th>Relative risk (95% CI)</th>
<th>Weight (%)</th>
<th>Relative risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV iron vs oral iron</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Al 2005</td>
<td>0/45</td>
<td>0.3 (0.01 to 7.97)</td>
<td>0.3</td>
<td>0.3 (0.01 to 7.97)</td>
</tr>
<tr>
<td>Auerbach 2006</td>
<td>9/78</td>
<td>3.7 (0.39 to 2.12)</td>
<td>3.7</td>
<td>3.7 (0.39 to 2.12)</td>
</tr>
<tr>
<td>Auerbach 2010</td>
<td>41/116</td>
<td>17.6 (0.65 to 1.25)</td>
<td>17.6</td>
<td>17.6 (0.65 to 1.25)</td>
</tr>
<tr>
<td>Bayoumeu 2002</td>
<td>0/24</td>
<td>0.3 (0.01 to 7.48)</td>
<td>0.3</td>
<td>0.3 (0.01 to 7.48)</td>
</tr>
<tr>
<td>Breymann 2008</td>
<td>1/227</td>
<td>0.3 (0.06 to 37.82)</td>
<td>0.3</td>
<td>0.3 (0.06 to 37.82)</td>
</tr>
<tr>
<td>Dangsuwan 2010</td>
<td>5/22</td>
<td>3.8 (0.16 to 0.82)</td>
<td>3.8</td>
<td>3.8 (0.16 to 0.82)</td>
</tr>
<tr>
<td>Froessler 2013</td>
<td>1/101</td>
<td>0.6 (0.03 to 3.03)</td>
<td>0.6</td>
<td>0.6 (0.03 to 3.03)</td>
</tr>
<tr>
<td>Garrido-Martin 2012</td>
<td>20/54</td>
<td>11.7 (0.47 to 1.13)</td>
<td>11.7</td>
<td>11.7 (0.47 to 1.13)</td>
</tr>
<tr>
<td>Henry 2007</td>
<td>11/63</td>
<td>5.7 (0.55 to 2.12)</td>
<td>5.7</td>
<td>5.7 (0.55 to 2.12)</td>
</tr>
<tr>
<td>Kochhar 2012</td>
<td>0/50</td>
<td>0.3 (0.01 to 7.99)</td>
<td>0.3</td>
<td>0.3 (0.01 to 7.99)</td>
</tr>
<tr>
<td>Meyer 1996</td>
<td>0/21</td>
<td>0.3 (0.01 to 3.93)</td>
<td>0.3</td>
<td>0.3 (0.01 to 3.93)</td>
</tr>
<tr>
<td>Steensma 2011</td>
<td>20/164</td>
<td>9.5 (0.56 to 1.52)</td>
<td>9.5</td>
<td>9.5 (0.56 to 1.52)</td>
</tr>
<tr>
<td>Weisbach 1999</td>
<td>6/30</td>
<td>2.3 (0.80 to 7.23)</td>
<td>2.3</td>
<td>2.3 (0.80 to 7.23)</td>
</tr>
<tr>
<td>Westad 2008</td>
<td>4/59</td>
<td>2.3 (0.14 to 1.28)</td>
<td>2.3</td>
<td>2.3 (0.14 to 1.28)</td>
</tr>
<tr>
<td>Subtotal: =0.45, I²=0%</td>
<td>118/1054</td>
<td>58.6 (0.67 to 1.00)</td>
<td>58.6</td>
<td>58.6 (0.67 to 1.00)</td>
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<tr>
<td>IV iron vs no iron</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Edwards 2009</td>
<td>0/34</td>
<td>0.3 (0.01 to 3.08)</td>
<td>0.3</td>
<td>0.3 (0.01 to 3.08)</td>
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<tr>
<td>Hedens 2007</td>
<td>2/33</td>
<td>0.5 (0.20 to 21.63)</td>
<td>0.5</td>
<td>0.5 (0.20 to 21.63)</td>
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<tr>
<td>Karkouti 2006</td>
<td>4/21</td>
<td>2.0 (0.15 to 1.52)</td>
<td>2.0</td>
<td>2.0 (0.15 to 1.52)</td>
</tr>
<tr>
<td>Klin 2007</td>
<td>12/30</td>
<td>9.8 (0.38 to 1.01)</td>
<td>9.8</td>
<td>9.8 (0.38 to 1.01)</td>
</tr>
<tr>
<td>Madjebara 2004</td>
<td>17/80</td>
<td>5.1 (0.46 to 1.93)</td>
<td>5.1</td>
<td>5.1 (0.46 to 1.93)</td>
</tr>
<tr>
<td>Na 2011</td>
<td>11/54</td>
<td>7.3 (0.21 to 0.68)</td>
<td>7.3</td>
<td>7.3 (0.21 to 0.68)</td>
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<tr>
<td>Pedrazzoli 2008</td>
<td>2/73</td>
<td>1.1 (0.08 to 2.08)</td>
<td>1.1</td>
<td>1.1 (0.08 to 2.08)</td>
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<tr>
<td>Serrano-Tenas 2011</td>
<td>33/100</td>
<td>15.3 (0.56 to 1.16)</td>
<td>15.3</td>
<td>15.3 (0.56 to 1.16)</td>
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<tr>
<td>Subtotal: =0.33, I²=14%</td>
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<td>41.4</td>
<td>41.4 (0.49 to 0.85)</td>
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<tr>
<td>Subtotal: =0.34, I²=9%</td>
<td>199/1479</td>
<td>100.0 (0.62 to 0.88)</td>
<td>100.0</td>
<td>100.0 (0.62 to 0.88)</td>
</tr>
</tbody>
</table>
**Preoperative Iron Deficiency Anaemia:- Should I treat it with intravenous Iron?**

Risk of infection in patients who received intravenous iron?

<table>
<thead>
<tr>
<th>Study</th>
<th>Treatment</th>
<th>Control</th>
<th>Relative risk (95% CI)</th>
<th>Weight (%)</th>
<th>Relative risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV iron v oral iron</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benacakova 2009</td>
<td>9/130</td>
<td>1/130</td>
<td>0.9</td>
<td>9.00</td>
<td>(1.16 to 70.03)</td>
</tr>
<tr>
<td>Breymann 2008</td>
<td>19/227</td>
<td>4/117</td>
<td>3.1</td>
<td>2.45</td>
<td>(8.85 to 7.03)</td>
</tr>
<tr>
<td>Heny 2007</td>
<td>9/63</td>
<td>23/124</td>
<td>5.8</td>
<td>0.77</td>
<td>(0.38 to 1.56)</td>
</tr>
<tr>
<td>Kulig 2008</td>
<td>18/137</td>
<td>6/63</td>
<td>4.2</td>
<td>1.38</td>
<td>(0.38 to 3.31)</td>
</tr>
<tr>
<td>Lindgren 2009</td>
<td>12/45</td>
<td>4/46</td>
<td>3.1</td>
<td>3.07</td>
<td>(1.07 to 8.80)</td>
</tr>
<tr>
<td>Meyer 1996</td>
<td>2/21</td>
<td>2/21</td>
<td>1.1</td>
<td>1.00</td>
<td>(0.16 to 6.45)</td>
</tr>
<tr>
<td>Pollack 2001</td>
<td>2/10</td>
<td>4/19</td>
<td>1.6</td>
<td>0.95</td>
<td>(0.21 to 4.32)</td>
</tr>
<tr>
<td>Qunibi 2013</td>
<td>20/147</td>
<td>8/103</td>
<td>5.1</td>
<td>1.75</td>
<td>(0.80 to 3.82)</td>
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<td>Schröder 2005</td>
<td>2/22</td>
<td>0/24</td>
<td>0.5</td>
<td>5.43</td>
<td>(0.28 to 107.33)</td>
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<td>Steensma 2011</td>
<td>28/164</td>
<td>11/163</td>
<td>6.4</td>
<td>2.53</td>
<td>(1.30 to 4.91)</td>
</tr>
<tr>
<td>Van Wyck 2007</td>
<td>24/174</td>
<td>22/178</td>
<td>8.4</td>
<td>1.12</td>
<td>(0.65 to 1.91)</td>
</tr>
<tr>
<td>Van Wyck 2005</td>
<td>2/79</td>
<td>0/82</td>
<td>0.4</td>
<td>5.19</td>
<td>(0.25 to 106.38)</td>
</tr>
<tr>
<td>Khalafallah 2010</td>
<td>0/92</td>
<td>0/91</td>
<td>0</td>
<td>(Excluded)</td>
<td></td>
</tr>
<tr>
<td>Schilder 1994</td>
<td>0/30</td>
<td>0/30</td>
<td>0</td>
<td>(Excluded)</td>
<td></td>
</tr>
<tr>
<td>Singh 1998</td>
<td>0/50</td>
<td>0/50</td>
<td>0</td>
<td>(Excluded)</td>
<td></td>
</tr>
<tr>
<td>Subtotal: P=0.196, I²=25.3%</td>
<td>147/1381</td>
<td>85/1241</td>
<td>40.7</td>
<td>1.63</td>
<td>(1.16 to 2.29)</td>
</tr>
<tr>
<td>IV iron v no iron</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allen 2011</td>
<td>3/24</td>
<td>2/21</td>
<td>1.3</td>
<td>1.31</td>
<td>(0.24 to 7.12)</td>
</tr>
<tr>
<td>Anker 2009</td>
<td>50/304</td>
<td>24/155</td>
<td>10.3</td>
<td>1.06</td>
<td>(0.68 to 1.66)</td>
</tr>
<tr>
<td>Basilt 2008</td>
<td>43/203</td>
<td>37/193</td>
<td>10.3</td>
<td>1.10</td>
<td>(0.75 to 1.64)</td>
</tr>
<tr>
<td>Coyne 2002</td>
<td>8/68</td>
<td>10/66</td>
<td>4.3</td>
<td>0.78</td>
<td>(0.33 to 1.85)</td>
</tr>
<tr>
<td>Evstatiev 2013</td>
<td>12/103</td>
<td>8/99</td>
<td>4.4</td>
<td>1.41</td>
<td>(0.60 to 3.31)</td>
</tr>
<tr>
<td>Friel 1995</td>
<td>9/14</td>
<td>7/12</td>
<td>7.1</td>
<td>1.10</td>
<td>(0.59 to 2.04)</td>
</tr>
<tr>
<td>Grote 2009</td>
<td>1/29</td>
<td>0/31</td>
<td>0.4</td>
<td>3.20</td>
<td>(0.14 to 75.55)</td>
</tr>
<tr>
<td>Hedenus 2007</td>
<td>18/33</td>
<td>6/34</td>
<td>5.0</td>
<td>3.09</td>
<td>(1.40 to 6.81)</td>
</tr>
<tr>
<td>Krayenbuehl 2011</td>
<td>10/43</td>
<td>6/47</td>
<td>3.9</td>
<td>1.82</td>
<td>(0.72 to 4.59)</td>
</tr>
<tr>
<td>Okonko 2008</td>
<td>0/24</td>
<td>1/11</td>
<td>0.4</td>
<td>0.16</td>
<td>(0.01 to 3.64)</td>
</tr>
<tr>
<td>Serrano-Trenas 2011</td>
<td>16/100</td>
<td>13/100</td>
<td>6.2</td>
<td>1.23</td>
<td>(0.63 to 2.42)</td>
</tr>
<tr>
<td>Singh 2006</td>
<td>9/75</td>
<td>9/46</td>
<td>4.5</td>
<td>0.61</td>
<td>(0.26 to 1.3)</td>
</tr>
<tr>
<td>Subtotal: P=0.345, I²=10.2%</td>
<td>179/1022</td>
<td>123/815</td>
<td>59.3</td>
<td>1.17</td>
<td>(0.94 to 1.47)</td>
</tr>
<tr>
<td>Subtotal: P=0.156, I²=22.7%</td>
<td>326/2413</td>
<td>208/2056</td>
<td>100.0</td>
<td>1.34</td>
<td>(1.10 to 1.64)</td>
</tr>
</tbody>
</table>

**Litton E et al. BMJ 2013;347**
Safety and efficacy of intravenous iron therapy in reducing requirement for allogeneic blood transfusion: systematic review and meta-analysis of randomised clinical trials

Iron and risk of infection

The reduction in allogeneic red blood cell transfusion must be considered alongside our finding of an increased risk of all cause infections after intravenous iron therapy. Free iron has been shown to potentiate bacterial growth in vitro. Clinical evidence on the association between intravenous iron therapy and infection, however, has been inconclusive, with no increase in infection observed with intravenous iron therapy in patients undergoing dialysis or in patients after surgery or in a mouse model of critical care anaemia. This discrepancy might be explained by the study design and sample size. Intravenous iron therapy was associated with an increased risk of infection.
considered alongside our finding of an increased risk of all-cause infections after intravenous iron therapy. Free iron has been shown to potentiate bacterial growth in vitro. Clinical evidence on the association between intravenous iron therapy and
Iron is needed for bacterial growth making its bioavailability an important factor in controlling infection.\[25\]

Blood plasma as a result carries iron tightly bound to transferrin, which is taken up by cells by endocytosing transferrin, thus preventing its access to bacteria.\[26\]

Between 15 and 20 percent of the protein content in human milk consists of lactoferrin\[27\] that binds iron. As a comparison, in cow's milk, this is only 2 percent. As a result, breast fed babies have fewer infections.\[26\]

Lactoferrin is also concentrated in tears, saliva and at wounds to bind iron to limit bacterial growth.

Egg white contains 12% conalbumin to withhold it from bacteria that get through the egg shell (for this reason prior to antibiotics, egg white was used to treat infections).\[28\]
To reduce bacterial growth, plasma concentrations of iron are lowered in a variety of systemic inflammatory states due to increased production of hepcidin which is mainly released by the liver in response to increased production of pro-inflammatory cytokines such as Interleukin-6.

This functional iron deficiency will resolve once the source of inflammation is rectified, however if not resolved it can progress to Anaemia of Chronic Inflammation.

The underlying inflammation can be caused by fever, Inflammatory Bowel Disease, infections, Chronic Heart Failure (CHF), carcinomas and following surgery.
Reflecting this link between iron bioavailability and bacterial growth, the taking of oral iron supplements causes a relative over abundance of iron that can alter the types of bacteria that are present within the gut.

There have been concerns regarding parenteral iron being administered whilst bacteremia is present, although this has not been borne out in clinical practice.

A moderate iron deficiency, in contrast, can provide protection against acute infection, especially against organisms that reside within hepatocytes and macrophages such as Malaria and TB. This is mainly beneficial in regions with a high prevalence of these diseases and where standard treatment is unavailable.
HEPCIDIN

- 25aa Hormone that appears to be the key regulator of Iron entry into mammalian cells
- First discovered in 2000. Synthesised in the Liver; (hence Hep)
- Appeared to be Bactericidal (hence –cidin)
- Reduces dietary iron absorption by reducing iron transport across enterocytes of the gut mucosa; reduces iron exit from macrophages, the main site of iron storage; and it reduces iron exit from the liver. (All actions reduce free iron that could be available to bacteria.)
- This is accomplished by inhibiting the transmembrane iron transporter ferroportin.

(Adapted from Wikipedia)
considered alongside our finding of an increased risk of all-cause infections after intravenous iron therapy. Free iron has been shown to potentiate bacterial growth in vitro. Clinical evidence on the association between intravenous iron therapy and...

**What is this all about??????**

*Not Sure ...*

*Probably not all that clinically relevant.....*

*But may be worth being cautious about giving IV iron during bacteraemia*
Other Hazards of i.v. Iron
Which Parenteral Iron?
### Table 2: Treatment-emergent adverse events experienced by ≥ 1% of subjects in either treatment phase during the first 24 h and 7-d treatment periods (completer population)

<table>
<thead>
<tr>
<th>Adverse event</th>
<th>1st 24 h</th>
<th>7 d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ferric carboxymaltose (N=559)</td>
<td>Placebo (N=559)</td>
</tr>
<tr>
<td>At least 1 treatment-emergent adverse event</td>
<td>84 (15%)</td>
<td>64 (11.4%)</td>
</tr>
<tr>
<td>Gastrointestinal disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>30 (5.4%)</td>
<td>19 (3.4%)</td>
</tr>
<tr>
<td>Nausea</td>
<td>17 (3.0%)</td>
<td>8 (1.4%)</td>
</tr>
<tr>
<td>General disorders and administration site conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pyrexia</td>
<td>2 (0.4%)</td>
<td>0</td>
</tr>
<tr>
<td>Fatigue</td>
<td>5 (0.9%)</td>
<td>0</td>
</tr>
<tr>
<td>Infections and infestations</td>
<td>2 (0.4%)</td>
<td>5 (0.9%)</td>
</tr>
<tr>
<td>ALT increased</td>
<td>2 (0.4%)</td>
<td>5 (0.9%)</td>
</tr>
<tr>
<td>AST increased</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Bailie GR, Mason NA, Valaoras TG. Safety and tolerability of intravenous ferric carboxymaltose in patients with iron deficiency anemia. Hemodialysis International 2010;14:47–54

*Department of Cardiology and Haematology, Medical University of Bologna, and Department of Medicine, Fee di Roma.
“Uncertainty about when and how to administer IV iron preparations is an important contributor to underutilisation in patients with indications.”

Total dose infusion of iron polymaltose complex is recommended only when the intramuscular route is assessed for risks; doses up to 1250 mg of iron polymaltose can be diluted in 250 mL of 0.9% sodium chloride and given by rapid infusion.

**Test dose:** 25 mg over 20 minutes (hourly rate will vary depending on dose – see table below). If no reaction then proceed to ongoing infusion rate.

**Ongoing Infusion:** 120 mL/hour, or if required infusion may be given up to 250 mL/hour.

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Test dose (25mg run over 20 min)</th>
<th>Ongoing infusion</th>
<th>Concentration</th>
<th>Test dose (25mg run over 20 min)</th>
<th>Ongoing infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>750 mg/500 mL</td>
<td>50 mL/hr</td>
<td>120 mL/hr</td>
<td>750 mg/250 mL</td>
<td>25 mL/hr</td>
<td>120-250 mL/hr</td>
</tr>
<tr>
<td>1000 mg/500 mL</td>
<td>37 mL/hr</td>
<td>120 mL/hr</td>
<td>1000 mg/250 mL</td>
<td>19 mL/hr</td>
<td>120-250 mL/hr</td>
</tr>
<tr>
<td>1250 mg/500 mL</td>
<td>30 mL/hr</td>
<td>120 mL/hr</td>
<td>1250 mg/250 mL</td>
<td>15 mL/hr</td>
<td>120-250 mL/hr</td>
</tr>
<tr>
<td>1500 mg/500 mL</td>
<td>25 mL/hr</td>
<td>120 mL/hr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1750 mg/500 mL</td>
<td>21 mL/hr</td>
<td>120 mL/hr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000 mg/500 mL</td>
<td>19 mL/hr</td>
<td>120 mL/hr</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>2250 mg/500 mL</td>
<td>17 mL/hr</td>
<td>120 mL/hr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2500 mg/500 mL</td>
<td>15 mL/hr</td>
<td>120 mL/hr</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Livestrong?

Is EPO necessary?
A single dose of erythropoietin reduces perioperative transfusions in cardiac surgery: results of a prospective single-blind randomized controlled trial

Luca Welcert, Beatrice Rondinelli, Ricardo Bello, Mauro Falco, Alessandro Bellisario, Daniele Maselli, Franco Turani, Ruggero De Paulis, and Luca Pirelli

BACKGROUND: We conducted a prospective single-blind randomized study to assess whether a single 80,000 IU dose of human recombinant erythropoietin (HRE), given just 2 days before cardiac surgery, could be effective in reducing perioperative allogeneic red blood cell transfusion (aRBCt).

Major complex surgical procedures may be associated with substantial intra- and postoperative blood loss. A significant number of patients who develop anemia due to blood loss will require allogeneic red blood cell transfusion (aRBCt). This is more likely if preoperative anemia is persistent and severe. The objective of the present study was to determine whether a single 80,000 IU dose of HRE administered 2 days before cardiac surgery is effective in reducing the incidence of aRBCt without increasing adverse events.

CONCLUSION: In anemic patients (Hb < 13 g/dL), a single high dose of HRE administered 2 days before cardiac surgery is effective in reducing the incidence of aRBCt without increasing adverse events.
Iron has other effects not associated with haemoglobin.
Key Points about Anaemia

- Preoperative anaemia should be corrected before blood-losing elective surgery
- Iron deficiency is common
- Ferritin levels are not usually done
- Iron deficiency (absolute or functional) is the most treatable cause of anaemia
- Preoperative red cell transfusion should NOT be the treatment for Iron deficiency in a “stable” patient
- Raising the Hb, reduces transfusion
- The effect of oral therapy is slow compared to parenteral iron.
- Many patients are intolerant of oral iron
- Post op oral iron has limited absorption
- IV Iron is safe
- Normalisation of Iron stores may have other benefits
All we ever wanted to know about perioperative bleeding

Donat R. Spahn and Rolf Rossaint

European Journal of Anaesthesiology 2013, 30:267–269

This Invited Commentary accompanies the following article:

In this issue of the Journal, the European Society of Anaesthesiology’s (ESA) guidelines on the management of severe perioperative bleeding are published. These guidelines are part of the ESA’s efforts to increase patient safety as declared in the ‘Helsinki Declaration on Patient Safety in Anaesthesiology’, and they are the result of more than 2 years of hard work by the task force led by Sibylle Kozek-Langenecker who was mandated by the ESA in 2010.

interest in perioperative bleeding and coagulation. For these individuals, these guidelines should be the basis upon which they create algorithms on how to treat patients in specific situations at their hospitals as well as for educational initiatives. Such local educational programmes and algorithms are vital to transform the knowledge provided within the guidelines into outcome benefits for patients suffering from major bleeding. This is indeed possible as the example of Gorlinger et al. shows. They created such algorithms (which were based on near patient testing with rotational thromboelastometry) for the individual, specific treatment of coagulation derangements with coagulation factor concentrates and labile blood products. Moreover, they analysed the outcome of patients undergoing cardiac surgery at the Essen University Hospital before and after algorithm implementation between 2004 and 2009. Not only were they able to substantially decrease the number of transfusions of red blood cells and fresh frozen plasma, they also managed to reduce by 50% the incidence of massive transfusion, take-backs and thrombotic complications.
Autologous blood, (pre-donated) is risk free...

MYTH BUSTED

Pre-donated autologous transfusion is not risk free and there are a variety of adverse events associated with this practice.

Use of autologous blood still carries equal, if not greater, risk of bacterial contamination. There are three reasons for this:

- autologous donor criteria are generally more flexible than allogenic donor acceptance
- it is possible that autologous donor screening, at the point of donation is less precise and rigorous
- the typically longer storage interval of autologous RBC units maximises the opportunity for bacterial proliferation

Complications of Autologous Transfusion

- Blood wastage
- Errors and accidents
- Donor reactions
- Heterogenic anemia
- Bactemia
- Volume overload

Autologous donations may cost the patient $200 or more, per unit collected.

For more information about the risk of autologous transfusion go to:
Patient Blood Management
A 68-Year-Old Woman Contemplating Autologous Blood Donation Before Elective Surgery

Lynne Uhl, MD, Discussant

**DR TESS: Ms C** is a 68-year-old woman who presented with progressive right knee pain and swelling. She first developed pain and swelling in her right knee in 2003 and was diagnosed as having osteoarthritis. She underwent arthroscopy and bursectomy in 2006, but in the last few years, she has experienced worsening of her pain as well as significant physical limitations. Joint injections with steroids have resulted in little improvement, and now she is planning to undergo knee replacement surgery in 8 weeks.

Ms C’s medical history includes osteoarthritis, hypertension, hyperthyroidism, hypercholesterolemia, and uterine fibroids in addition to the arthroscopy in 2006. Her medications include hydrochlorothiazide, levothyroxine, simvastatin, and aspirin. She has no known drug allergies.

On examination, Ms C is a healthy-appearing woman with normal vital signs. Her physical examination results were normal except for pain on palpation of her medial right knee, an antalgic gait, and difficulty with toe and heel walking due to pain. A routine complete blood cell count revealed a white blood cell count of 7900/μL, hemoglobin level of 15.1 g/dL, mean corpuscular volume of 92 fl, red blood cell (RBC) distribution width of 13.4%, and platelet count of 290 × 10^9/μL.

Globally, more than 81 million units of red blood cells are transfused annually. Of the 15 million red blood cell components transfused annually in the United States, approximately 40% are transfused to patients undergoing elective surgical procedures. Because of concerns about limited blood availability as well as risks of transfusion-related adverse events, blood products should be used judiciously. Using the case of Ms C, a 68-year-old woman considering autologous blood donation prior to knee replacement surgery, the concept of patient blood management is discussed. This approach entails a complete evaluation of the patient in the preoperative period to assess for bleeding risks and anemia, with a goal to optimize a patient’s condition prior to surgery; use of various strategies in the operative period to mitigate the need for allogeneic blood transfusion; and meticulous postoperative care to again avoid the need for blood transfusion.

*JAMA. 2011;306(17):1902-1910*  
www.jama.com

about the do’s and the don’ts of transfusion and explain the risks and options to them.