Letters to the Editor

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Nosocomial anaemia

Aronson et al.1 reported their findings on the important long-term prognostic significance of the drop in haemoglobin levels that had developed during hospital stay, in patients with acute myocardial infarction in the intensive coronary care unit (they included only survivors at discharge, so we do not have details on that correlation in the deceased). They discussed the possible causes of that drop and mentioned about bleeding due to pharmacological interventions or invasive procedures.

They did not mention, however, (nor did the editor for that matter)2, diagnostic phlebotomy as another possible and important contributor to blood loss which can play a role in causing anaemia in the hospital. The authors did not indicate if a policy of minimizing diagnostic blood loss is practiced in their unit. This kind of iatrogenic blood loss is recognized and much discussed, occurring mostly in the premises of intensive care units. In a recent paper on ventilated patients,3 10% of patients had more than 500 cc of blood withdrawn. However, it was shown that in the general medical service too, the amount of blood withdrawn may be significant and that the amount correlated well with the drop observed in haemoglobin during the hospital stay.4

Given the evidence gathered on the possible role of phlebotomy in causing ‘nosocomial anaemia’ and on the presumed contributory role of that anaemia to prognosis worsening, it seems that the goals of patient safety and quality of care dictate for measures of blood conservation in this domain to be taken and enforced everywhere. The first step is indeed the awareness of the hospital team, and possible measures are, for example, restraining of the frequency of sampling to the minimum necessary, frequent checks for superfluous ‘routines’, the use of small volume blood containers, etc.5

References
5. Meir Liron
Yakum 60972
Israel
Tel: +972 9 9524515
Fax: +972 9 9524637
E-mail address: mliron@yakum.co.il

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Nosocomial anaemia: reply

We appreciate Dr Liron’s comments regarding our article. Because the focus of our study was to examine the relationship between changes in haemoglobin during hospital course and post-hospital discharge outcome, we excluded patients who died during the index hospitalization. This allowed that every patient would have three haemoglobin measurements (baseline, nadir and discharge).1 It also excluded cases in which severe bleeding during hospital course may have contributed directly to mortality.

Blood loss due to diagnostic phlebotomy has been mainly described in ventilated surgical intensive care patients, particularly with arterial lines.2,3 The quoted article of patients who had more than 500 mL blood withdrawn refers to four patients who were hospitalized for 36–65 days.2 However, blood loss due to diagnostic phlebotomy is much lower in general medical wards,3–5 including medical intensive care units.4 The mean volume of blood drawn per day has been reported to be 16–42 mL in the intensive care setting3,4,6 and 4–12 mL in general wards.1 In a study of 2654 patients in general wards and intensive care units, diagnostic blood loss of 200 mL or less occurred in 95% of patients during hospital stay.1 In our study, the median length of hospital stay was 7 days (inter-quartile range 6–10), with about half of the time spent in the intensive care unit.

Previous studies in patients with acute coronary syndromes have shown haemoglobin drops that were even greater than those observed in our study. In patients with acute myocardial infarction, Thakk-Johnson et al. observed haemoglobin decrease of ≥ 3.0 g/dL in 22% of patients receiving thrombolysis during the first 24 h of hospitalization.7 They concluded that phlebotomy loss accounted for only a fraction of the haemoglobin decline in their patients. In a large contemporary population of patients with non-ST-segment elevation acute coronary syndromes, median haematocrit declined from 41 to 35% during hospital stay in patients not receiving blood transfusion and from 35 to 26% in patients receiving blood transfusion.8

The anaemia that developed in many of our patients during their hospital course can be clearly described as nosocomial anaemia. However, we believe that laboratory-related blood loss was not a major contributor to the development of anaemia in our patient population. Thrombolytic, anti-thrombotic, and antiplatelet therapy along with coronary revascularization largely account for the magnitude of haemoglobin drop observed in our study.

Notwithstanding, we agree with Dr Liron that diagnostic blood loss may contribute to the development of anaemia in hospitalized patients. Blood testing should be performed with a clear indication, using the minimal sample volume required. In addition, judicious dosing of anti-thrombotic medications, proper selection of patients for invasive procedures, and the use of safer anticoagulant agents may prevent bleeding complications and anaemia and improve patient outcome.

References
7. Thakk-Johnson ME, Sharkey SW. Impact of thrombolytic therapy on haemoglobin change.

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Doron Aronson
Department of Cardiology
Rambam Medical Center
Bat Galim
PO Box 9602
Haifa 31096
Israel
E-mail address: daronson@rambam.health.gov.il

Walther Markiewicz
Department of Cardiology
Rambam Medical Center
Bat Galim
PO Box 9602
Haifa 31096
Israel

Haim Hammerman
Department of Cardiology
Rambam Medical Center
Bat Galim
PO Box 9602
Haifa 31096
Israel

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Carotid intima-media thickness and coronary atherosclerosis: weak or strong relations?

We would like to respond to a recent article by Bots et al.1 We agree with the authors’ conclusion that there is a relationship between carotid intima-media thickness (CIMT) with coronary atherosclerosis. However, significant progress has been made in the analysis of IMT and lesions since the time many of these studies were conducted. Furthermore, the predictability of event risk has evolved from large epidemiological prediction to individual prediction.

In the beginning, this group recognized the importance of IMT by itself to predict stroke and myocardial infarction on a population scale.2 Nevertheless, in 2007, the analysis method has clearly evolved since the quoted studies were performed. For example, the authors previously published an article on a related topic3 and the letter to the editor by Barth et al.4 addressed similar issues. The response to that letter by Bots et al. stated that ‘our CIMT measurement predicts future disease in a magnitude similar to that of population based studies that use either manual tracings or automated edge detection tracings.’ A fully automated individualized analysis method is now possible and may, given a long-term sequential database, lead to an individual predictability that was not previously available.

Additionally, the fact that the authors are not dealing with all aspects of carotid ultrasound and coronary angiography and the incomplete use of the literature in their meta-analysis5 may explain, in part, their conclusions. Coronary angiography focuses on the lumen and is generally performed in symptomatic/advanced disease populations, whereas with IMT HeartScan, lesion detection and tissue typing are usually performed in an asymptomatic population. Only considering what is happening in the lumen to assess the disease and not the wall is debatable. Further, the importance of lesion detection as indicated by Spence6 further underscores that, although in large population studies manual or automated edge detection tracings may demonstrate a relationship, it fails to assess lesions or plaque composition, resulting in low confidence of event predictability on an individual basis. Measuring the area of such lesions, particularly when assessing progression, is much more informative than measuring the thickness alone, because plaque progresses along the carotid artery ~2.4 times faster than it thickens.7 In a prospective study,8 a risk score based on age, blood pressure, smoking, and cholesterol predicted only 32% of patients with vascular events over a 5-year period, whereas 77% of events occurred among patients in the top quartile of plaque area.

Finally, the clinical relevance and long-term follow-up in different ethnic and age groups of IMT measurements in combination with plaque formation underscores the importance of current advances in IMT technology.5,6 Our large database can reliably predict on an individual basis the likelihood of a cardiovascular complication within several years if no intervention is performed. The SHAPE report highlights the importance of an initial IMT measurement for clinical follow-up.7 Quantitative IMT in combination with lesion detection and plaque composition assessment is used widely in clinical settings with great predictability on an individual basis for cardiovascular outcomes.

References

Jacques D. Barth
Department of Family Medicine
Keck School of Medicine
University of Southern California
12335 Santa Monica Boulevard Suite 200
Los Angeles CA 90025
USA
Tel: +1 310 566 7050
Fax: +1 310 566 7057
E-mail address: jbarth@usc.edu

Christian K. Roberts
Department of Preventive Medicine
Center for Obesity and Metabolic Health
Keck School of Medicine
University of Southern California
CA, USA
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Discrepancy in guidelines for the prevention of thrombo-embolism in patients with prosthetic heart valves

We have read with interest and became aware of diverse therapeutic approaches in several recently published guidelines, referring to patients with prosthetic heart valves and atrial fibrillation.

In the latest European guidelines on the management of valvular heart disease,1 lifelong oral anticoagulation is recommended for all patients with mechanical valves and for those patients with bioprostheses who have additional indications for anticoagulation such as atrial fibrillation, heart failure, or impaired left ventricular function. Indications for the addition of antiplatelet therapy include concomitant arterial disease, in