

ABO mismatched platelet transfusions in trauma patients

In the study by Nascimento and colleagues,¹ the patients who received increased plasma and platelet transfusions had greater mortality due to bleeding (22%) than the control group (9%), albeit not statistically significantly so. Nonetheless, it raises the question of whether the plasma and platelet transfusion worsened hemorrhage rather than mitigated it. One possible mechanism is the administration of ABO non-identical plasma and platelets. Preliminary published observational data show that trauma patients receiving ABO non-identical (mismatched) platelets have increased red cell transfusion needs, as do patients who experience serious blood loss during surgery in general.^{2,3} A possible mechanism is that exposure of platelets and endothelial cells to incompatible anti-A and anti-B antibody impairs platelet function, coagulation and hemostasis.⁴ That group O individuals have much lower levels of von Willebrand factor than non-O individuals is also well established.

Have the authors considered analyzing their data according to how much ABO incompatible/non-identical plasma or platelet transfusions were given, or the ABO types of the patients? This variable might have contributed to the increased hemorrhagic mortality seen in the recipients of larger amounts of ostensibly hemostatic blood components.

Neil Blumberg MD

Professor, University of Rochester Medical Center, Rochester, NY

References

- Nascimento B, Callum J, Tien H, et al. Effect of a fixed-ratio (1:1:1) transfusion protocol versus laboratory-results-guided transfusion in patients with severe trauma: a randomized feasibility trial. *CMAJ* 2013;185:E583-9.
- Fialkow LB, Zucchiatti A, Cheng J, et al. ABO non-identical transfusions and red blood cell usage in blunt trauma patients. *Transfusion* 2007;47(Suppl):192A-3A.
- Refaai MA, Fialkow LB, Heal JM, et al. An association of ABO mismatched platelet and cryoprecipitate transfusions with increased red cell transfusion needs and mortality in surgical patients. *Vox Sang* 2011;101:55-60.
- Refaai MA, Carter J, Henrichs KF, et al. Alterations of platelet function and clot formation kinetics following in vitro exposure to anti-A and -B antibodies. *Transfusion* 2013;53:382-93.

CMAJ 2014. DOI:10.1503/cmaj.114-0001

Patient satisfaction

The *CMAJ* commentary by Detsky and Shaul¹ on incentives to improve patient satisfaction is both thought provoking and relevant to day-to-day practice.

Yankelovich² points out that despite better methods of measuring public opinion, little is known about how to improve its quality. Although information has become widely available, it plays only a limited role in shaping public opinion. Opinion polls can be misleading because they do not distinguish between people's immediate reactions and their thoughtful, considered judgments. Patient satisfaction surveys often fail to even measure, let alone correct for, the nature, severity and intractability of the conditions for which care is received. This is a particular problem for teaching hospitals, where the most difficult and complex cases are concentrated.

Undue emphasis on patient satisfaction may make providing services to those most in need impossible for fear of negative repercussions. Added to this is the growing list of for-profit websites that allow anonymous public postings. Many institutions have established patient relations units whose actions are often seen by treating physicians as barriers to following best practices.

At the same time as mandating assessments of patient satisfaction, governments have expressed increasing unwillingness to pay for medically unnecessary measures that would increase satisfaction, such as imaging studies for low-back pain.

Larry M. Picard MD

Wasser Pain Management Centre, Mount Sinai Hospital, Toronto, Ont.

References

- Detsky J, Shaul RZ. Incentives to increase patient satisfaction: Are we doing more harm than good? *CMAJ* 2013;185:1199-1200.

- Yankelovich D. *Coming to public judgment: making democracy work in a complex world*. Syracuse (NY): Syracuse University Press; 1991:258.

CMAJ 2014. DOI:10.1503/cmaj.114-0002

Job strain and lifestyle factors

The article by Kivimäki and colleagues¹ includes several methodological and interpretational errors.

First, we believe the authors should have excluded obesity from their definition of an unhealthy lifestyle. The authors defined an unhealthy lifestyle arbitrarily as a combination of the following four risk factors of coronary artery disease: smoking, heavy drinking, leisure-time physical inactivity and obesity. Although the first three risk factors may be considered lifestyle risk factors, obesity has a multifactorial etiology and cannot be simplistically labelled a lifestyle risk factor. Therefore, the study's results likely overestimate the effect of an "unhealthy lifestyle" on coronary artery disease.

Second, the authors should have made clear that two of the three behavioural risk factors (heavy drinking and leisure-time physical inactivity) did not offset the impact of job strain on coronary artery disease. Figure 1 in the article by Kivimäki and colleagues¹ indicates that there would be no significant reduction in the risk of coronary artery disease from reducing heavy drinking behaviour or physical inactivity among workers with job strain. Rather, it indicates the importance of addressing both job strain and unhealthy behavioural risk factors for prevention of coronary artery disease. Emerging evidence shows that organizational- or task-level interventions for increasing job control and decreasing high job demands can be beneficial for the health of workers and organizations.^{2,3}

Third, the conclusion of the authors ("a healthy lifestyle may substantially reduce disease risk among people with job strain") is misleading; 84.3% of workers with job strain had none or only one of the four risk factors, and in