Organ Donation and Dual Advocacy

TO THE EDITOR: In a Perspective article in this issue of the Journal, Truog provides his views on organ donation, suggesting that the pendulum between the need for transplantable organs and the rights of potential donors’ families “has swung too far in the direction of procuring organs.” Truog asserts that consent for organ donation is obtained “at the expense of commitments that are fundamental to the patient–physician relationship.”

We disagree with this description because it does not reflect current consent practices. In practice, there is a confluence between the ethical commitments that Truog outlines: “that the desires of people who want to donate organs are respected” and “that the consent process is informed and voluntary.” The New England Organ Bank (NEOB) and most other organ-procurement organizations (OPOs) have supplanted the “presumptive” consent approach criticized by Truog with a consent process of “dual advocacy.” This approach considers the interests of both the donor family and persons awaiting a transplant. It is based on the fact that most people, given the opportunity, will choose to help others. By supporting the family’s right to make a choice that is based on complete information, including the positive impact that the gift will have on others and the solace derived from organ donation, dual advocacy recognizes that those requesting donation must also consider the needs of the donor family. This approach relies on observations and data indicating that the involvement of an OPO professional is critical to success, that health care practitioners cannot reliably predict which families will want to donate, and that apologetic approaches to seeking consent are more likely to result in refusals.

In addition, Truog implies that the consent process for medical research should be used as a model for obtaining consent to organ donation. The research consent process is designed to protect human subjects, not their families, from the risk of participating in research; there is no risk of harm to the donor after death. Moreover, Truog’s portrayal of donation after cardiac death is inconsistent with current practices, which have been refined through accumulated experience. In the NEOB service area, the donor’s family usually is given a choice for the location of the withdrawal of treatment, catheters are seldom placed before death, and no vasodilators are administered.

A recent Gallup survey showed that almost 95% of the U.S. public supports organ donation, yet the current consent rate for donation hovers around 65 to 70%. This gap suggests that families are not in fact being browbeaten but rather that the goal of the Department of Health and Human Services of achieving 75% donation rates is anchored in a realistic reflection of public attitudes.

As an organization that has coordinated more than 5500 organ donations, we are grateful for the opportunity to work with organ donors and their families to save lives and sustain hope.
families in an open and compassionate manner in order to maximize the benefits of donation, both to them and to transplant recipients.

Richard S. Luskin, M.P.A.
Alexandra K. Glazier, J.D., M.P.H.
Francis L. Delmonico, M.D.
New England Organ Bank
Newton, MA 02458
rlusk@neob.org

Dr. Serebruany reports being listed as a coinventor on and receiving compensation for a U.S. patent application for prasugrel and receiving grant support from Eli Lilly and Sanofi–Bristol-Myers Squibb and advisory fees from Sanofi–Bristol-Myers Squibb. No other potential conflict of interest relevant to this letter was reported.


**Prasugrel versus Clopidogrel**

TO THE EDITOR: Wiviott et al. (Nov. 15 issue) report the pivotal results of the Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel–Thrombolysis in Myocardial Infarction (TRITON–TIMI) 38. To better assess the advantage of prasugrel over clopidogrel with respect to the benefit:risk ratio, two important clinical issues should be clarified. Since the numbers of deaths were nearly identical in the prasugrel group (154 deaths) and the clopidogrel group (155 deaths), as were the numbers of strokes, and since for each death from cardiovascular causes prevented by the use of prasugrel, one additional episode of fatal bleeding occurs, the efficacy benefit of prasugrel in TRITON–TIMI 38 is driven by nonfatal myocardial infarction (with 475 events in the prasugrel group vs. 620 events in the clopidogrel group). However, it is not clear whether the numbers reported by Wiviott et al. represent real clinical scenarios or just the increase in cardiac ischemic biomarkers that is so common during successful reperfusion. It is also important to report how many documented cases of stent thrombosis occurred in each treatment group, and why there was no difference in mortality between the two groups, rather than just to provide the combined number of patients with “definite or probable thrombosis.” Considering that it may be a very close call for the regulatory approval of prasugrel, these clarifications are urgently needed for assessment of the drug’s potential efficacy.

Victor Serebruany, M.D., Ph.D.
Johns Hopkins University
Baltimore, MD 21204
heartdrug@aol.com

TO THE EDITOR: In TRITON–TIMI 38, Wiviott et al. found a reduction in the incidence of myocardial infarction but an increase in major bleeding with prasugrel as compared with clopidogrel. The study design involved a 300-mg loading dose of clopidogrel; in the majority of patients (74%), the loading dose of either study drug was given after the first coronary guidewire was placed and during percutaneous coronary intervention (PCI) or within 1 hour after PCI, although it is well known that a 300-mg dose of clopidogrel achieves good clinical efficacy only after 8 to 12 hours. We and others have found, in randomized trials, that a 600-mg loading dose of clopidogrel before PCI (which is associated with maximal platelet inhibition within 2 hours) significantly reduces the risk of periprocedural myocardial infarction, as compared with a 300-mg loading dose, with no additional risk of bleeding. Wiviott et al. report that data supporting the 600-mg dose “have been inconsistent”; however, the results of randomized trials have consistently supported the 600-mg loading regimen. Since the definition of myocardial infarction in TRITON–TIMI 38 includes an increase in the level of creatine kinase MB fraction to twice the upper limit of the normal range after intervention, the use of prasugrel might involve trading a reduction in the risk of small myocardial...