A commentary in *Transfusion* earlier this year by members of an AABB and College of American Pathologists (CAP) work group urges that it is time to perform RhD genotyping for patients found to have a serologically weak D phenotype. They note that in 2014, the CAP Transfusion Medicine Resource Committee reported survey results from more than 3100 laboratories concerning weak D testing and the use of Rh immune globulin (RhIG). They found a lack of standard practice to approaching this problem. In some labs, a weak D individual is considered RhD-positive, especially if that person is a blood donor. If it is a female of child-bearing age, the person is likely to be called RhD-negative, and if they are pregnant they are considered as a candidate for Rh immune globulin. Most of the labs do not use an indirect antiglobulin test for detection of weak D, thus such a patient is considered to be RhD-negative. But for blood donors and newborns, most did try to identify weak Ds and categorized them as RhD-positive.

These practices are designed to protect RhD-negative persons from accidental immunization to D. Estimates quoted by the authors state that the use of antepartum and postpartum RhIG is 98.4 – 99% successful in preventing such alloimmunization and RhD hemolytic disease of the newborn. But the consequences of not performing weak D analyses are that many unnecessary injections of RhIG are given, as are unnecessary transfusions with RhD-negative red cells, which are always in short supply. The work group reviewed over 140 published reports dealing with these issues and reported their summary findings and recommendations, including a history of the problem and a summary of organizational policies. (See reference.)

Serologically, there are three broad categories of D variants that differ from standard or conventional D: weak D, partial D and DEL. Using conventional typing sera, DEL phenotypes test as D-negative unless adsorption and elution studies are performed. DEL phenotypes are more common in persons of Asian ancestry and do not present serologically as a weak D subtype, so the work group did not make any recommendations concerning this. Weak D phenotypes are the most commonly found D variants in Caucasians of European or American ancestry, and are noted in 0.2% to 1.0% of studies on the issue. Prevalence varies depending on methods and reagents used. In addition, there are several weak D variants, and because of this the work group limited its recommendations to the common American and European variants, weak D types 1, 2, and 3, until there are more available data on the other types, 4.0 and 4.1. The work group estimates that about 3% of pregnant women have a weak D phenotype. Of the 5 million individuals transfused annually in the United States, an estimated 17,500 with a weak D serotype will be typed as RHD-negative. Given the average transfusion rate of 2.7 units per event, RHD genotyping would make an additional 47,000 units of Rh-negative red cells available.

Cover photo: Trypanosoma, a parasitic flagellated protozoan that causes trypanosomiasis (African sleeping sickness and Chagas disease). Visuals Unlimited, Inc.

This issue of PLUS was written by Dr. Robert (Bob) Westphal.
Partial D phenotypes are due to amino acid substitutions in the RhD proteins on the surface membranes of red cells; they lack D epitopes. Many such red cells will agglutinate with anti-D and are interpreted as RhD-positive. This does not distinguish between partial D and weak D, so they are basically handled the same way. The article provides diagrams and algorithms for dealing with resolution of weak D phenotypes using RHD genotyping.

In their conclusion, the working group recommends that RHD genotyping be done whenever a discordant Rh typing result or a serologic weak D phenotype is detected in patients, including pregnant women, newborns and potential transfusion recipients. They point out again that the benefits include fewer unnecessary RhIG injections and more Rh-negative blood for transfusion. Persons who genotype as weak D, type 1, 2, or 3 should be managed as RhD-positive, and they provide an algorithm for resolving weak D phenotype results. Implementing RHD genotype testing would be facilitated if manufacturers offered basic cost-effective tests to identify the most relevant tests. Most hospital lab services and health care facilities won’t have a sufficient number of such samples to support in-house testing, but samples can be forwarded to their reference laboratory; these labs can offer a set of tiered services so that the most clinically relevant genotypes can be detected. It would be a good step towards standardizing this very important aspect of testing for blood transfusion and prevention and treatment of hemolytic disease of the newborn.


Delayed hemolytic transfusion reactions in patients with sickle cell disease are a poorly recognized phenomenon

Those involved in the care of patients with sickle cell disease (SCD) are well aware of the importance of, and dangers from, blood transfusion. The latter include iron overload, transfusion-related infections, and transfusion reactions – especially hemolytic transfusion reactions. Acute hemolytic transfusion reactions are, in general, no longer commonly seen in these patients, or others, for that matter. But delayed hemolytic reactions, DHTRs, are much more common and continue to occur in spite of the increasing trend to avoid alloimmunization by the use of DNA techniques to identify complex red cell types and provide careful selection of donors and units. Physicians from the King’s College University and Hospital in London recently reviewed their 5 year experience with DHTRs in adult patients with SCD.

This was a retrospective review of all red cell transfusions in the 637 adult patients with SCD treated in their hospital from 2008-2013. Adults were considered as 16 years or older. Their indications for transfusion were broad. For the acute setting indications, they included: acute chest syndrome, stroke, severe anemia or a 20% drop from baseline, multi-organ failure and severe sepsis. Indications for a chronic transfusion program were: primary and secondary stroke prevention, frequent acute pain when hydroxycarbamide (hydroxyurea) therapy has failed, leg ulceration, intra-hepatic cholestasis, complicated pregnancy, refractory priapism, and pre- and post-solid organ transplantation.

DHTR was defined as a significant drop of >25% in Hgb between 24 hrs and 21 days after a red cell transfusion with new red cell antibodies, or hemoglobinuria, or a 2-fold rise in LDH. During the study period 220 of the 637 adults had at least

Continues on next page
Patients with, and those who care for patients with, hemophilia have been using plasma-derived and recombinant Factors VIII and IX for some time now, and their lack of transmission of infectious diseases, such as HCV, HBV and HIV, have made them the cornerstone of therapy. As well as their use in the treatment of hemophiliac hemorrhage, they have allowed us to plan various schedules of prophylactic therapy such that the spontaneous and mild trauma-induced bleeding, e.g., hemarthroses in joints, can be greatly reduced. This has made an immense difference in the growth and health of these patients from childhood into adulthood and pretty much for the rest of their lives.

A major concern remains though, particularly for young boys and teenage boys for whom compliance is sometimes an issue, especially at the start of prophylactic therapy. The standard half-life (T1/2) in the circulation is about 6-24 hrs in hemophilia A (Factor VIII) and 14-27 hrs in hemophilia B (Factor IX). This is due in part, at least, to the fact that Factor VIII circulates linked to von Willebrand Factor (VWF) which has a fairly rapid clearance rate itself. Therefore, prolonging their action, their effective half-life, would be of considerable benefit to patients and their families, the latter members often responsible for providing infusions to younger children.

There are at least 5 companies who have been working with Factor VIII and 3 with Factor IX. Three technologies currently undergoing clinical trials are most likely to seek and gain approval for human use, and authors from the University of Cardiff, Wales, review these materials and results in a recent article (see reference). The technologies include the addition of polyethylene glycol (PEG), fusion to the Fc portion of IgG1, and fusion to recombinant albumin. The authors review these technologies, their pharmacokinetic studies, and some clinical studies that have been published in peer-reviewed journals. Currently published data show an approximately 1.5 fold increase in Factor VIII levels and a whopping 5 fold increase in Factor IX.

Thus, it may develop that weekly prophylaxis for hemophilia B, Factor IX deficiency, may be feasible, but that a weekly schedule would not be adequate prophylaxis for patients with hemophilia A, Factor VIII deficiency. The driving force of prophylaxis, though, is not convenience but effectiveness: does it prevent bleeding and hemarthroses? One would think that longer-acting agents, even if only slightly so as seems the case with Factor VIII, would improve adherence to preventive therapy. Those of us who have worked in hemophilia centers and treatment programs know how hard that can be, even with adults, but especially so in teens and some parents. But as Hamlet said, “... ‘tis a consummation devoutly to be wished.”

They’re still coming ashore, these bugs: we need a better barrier

A few years ago, many of us had hard numbers at hand indicating the evolution of emerging infectious diseases. There was a book from the Institute of Medicine that documented about 35 “new” infectious diseases... published about 20 years ago, if memory serves. Many more have arrived since, and some of us have lost track. Actually, they are not new at all, just new to the increasingly large number of people populating our planet. The world is not getting smaller, but it is certainly getting closer. With increases in the world population, economic problems and widespread areas of conflict have led to a great deal of suffering, large populations of refugees, and migration of the rural poor to urban areas. In more developed areas of the world, such as the northeast of the United States, migration of the urban affluent to suburbs and exurbs has displaced the “native populations”, such as white-tailed deer, and led to growing concerns about Lyme disease, anaplasmosis, granulocytic ehrlichiosis, babesiosis and other tick-borne diseases. And prions, don’t forget! Many, if not most of these, are transfusion-transmissible.

International travel and commerce have also played a role, witness the importation of Aedes albopictus mosquitoes from Asia into the Americas, this insect being even more tolerant of cooler climates than its cousin, A. aegypti, the carrier of yellow fever and chikungunya. This latter virus has rapidly spread from Indian Ocean countries to southern Europe, the Caribbean and other parts of America very recently. From this vantage point, one can begin to get a feel of the enormity of the problem. Those of us involved with transfusion medicine have seen a common scenario over and over. From earlier years, through “non-A, non-B hepatitis” and HIV in the early 1980s to CMV, HCV, Lyme disease, babesiosis, West Nile virus, T. cruzi, dengue, chikungunya... and they’re still coming ashore!

The authors of a recent Perspective article in the New England Journal of Medicine (see reference) point out that we in the blood banking field have in the past—and today—relied on a reactive approach to all this. First, a “new” infection is noted in humans; then, we see it in transfused patients. Then a lot of work in identifying, characterizing, and developing and testing diagnostic methods takes place; meanwhile many patients get infected. Once the tests are validated and implemented there are additional concerns, but at least we’ve started to deal with the problem. In addition to time, this also takes money, and the cost of these problems/solutions to the health care budget is enormous.

The authors believe, and firmly state the case, that the already documented (and in some cases FDA and CE approved) methods currently available for platelet and plasma product pathogen inactivation be mandated. Seven years ago (2008) an advisory committee on blood and tissue safety forwarded to the Secretary of Health and Human Services a statement testifying to the efficacy and safety of such methods and asked for “a commitment and concerted effort to add this technology as a broadly applicable safeguard.” But nothing has happened. In their article, the authors describe the several methods approved and in development for these technologies. Almost all involve UV light exposure, which cannot be used for red cell-containing products. But this goal is attainable, say the authors, and should become a priority for research in our field.

Hit the beaches, everyone!

For several years now, TRALI (Transfusion-Related Acute Lung Injury) has been the most common cause of fatality following allogeneic blood transfusion. It looks a lot like Transfusion Associated Circulatory Overload (TACO), but it is not due to cardiogenic causes or circulatory overload. TACO shows evidence of ventricular failure and increased left atrial pressure.

Usually, symptoms of TRALI occur within 6 hrs. post-transfusion and include shortness of breath, hypoxemia, fever, pulmonary edema with normal left atrial pressure, and hypotension. It is the subject of two editorials and several articles in the May, 2015, edition of Transfusion. Although descriptions in the 1950s and 1960s had described cases described as allergic pulmonary edema, leuko-agglutinin transfusion reaction, pulmonary hypersensitivity and non-cardiogenic pulmonary edema, TRALI was not so-named and recognized as a distinct syndrome until 1983 by Popovsky and others and was the subject of a major review in this journal (Transfusion) in 1985.

The agreed hallmark of TRALI was the presence of the development of bilateral pulmonary infiltrates with attendant hypoxemia but without evidence of circulatory overload as evidenced by increased left atrial pressure. Also, it has been shown experimentally that there is a major association of TRALI with leuko-agglutinating antibodies in the transfused product, often HLA or human neutrophil antibodies which may be present in the plasma of parous women or other previously sensitized donors. In 2004, a consensus conference in Toronto recommended that TRALI be defined as “a new episode of acute lung injury (ALI) that occurs during or within 6 hrs of a completed transfusion”. That panel also recommended that if this ALI was temporally related to another known cause of ALI it should be known as “possible TRALI”, or pTRALI. All of the authors discussing TRALI in this issue make clear that there is much that remains unknown, or unclear, including just when the acronym “TRALI” should be used.

Dr. Popovsky points out that since the 1990s there have been two main mechanisms examined for TRALI causation: the antibody-mediated model, as noted above; and, the two-hit model, in which a patient is “predisposed” to TRALI by way of cardiac surgery, hematologic malignancy, mechanical ventilation or some other physiologic insult that leads to increased stickiness of neutrophils—to one another and to pulmonary endothelial cells. Transfusion of antibodies, bioactive lipids or cytokines, and probably other agents, may then lead to overcoming the threshold that causes the neutrophil aggregation, a very specific microscopic pathological finding in TRALI cases.

Dr. Webert notes that even though red cell units have little remaining plasma, they are still seen as clear causes of TRALI. The current limitation on the use of plasma from parous women has had a clear effect on the incidence of TRALI; however, there remain many cases of non-immune TRALI, or pTRALI, so the search for other mechanisms and further ameliorating actions must continue. She appears to come out on the side of “splitting”, versus “lumping”, making the definition(s) of TRALI more specific so that larger clinical studies might focus on other causes, other risk factors. See the other articles in this issue of PLUS for more discussion on these points. For literary fun, see the poem “Stopping by woods on a snowy night”, by Robert Frost.

Popovsky MA. Transfusion-related acute lung injury: three decades of progress but miles to go before we sleep. Transfusion 2015; 55: 930-934.

So ... what’s up with this TRALI stuff?

It’s healthy to note that things are not always what they seem, even though “lumping” facts together sometimes makes it easier to keep track of them. Thus, the “splitters” may seem to be nit-picking intellectuals who just want to make things harder for us to organize our world. The splitters say, basically, “There are no easy solutions, just more problems.” Splitting improves scientific accuracy, generally.

Dr. Pearl Toy and colleagues carried out a nested case control study on a cohort of patients previously examined in a TRALI study of 2006-2009 and published in 2012. They prospectively identified 145 patients with TRALI and randomly selected 163 transfused controls over the same 4 yr period from the two medical centers involved in the prior study. They used the recently discussed definition of pTRALI (possible TRALI), that is a new acute lung injury (ALI) developing during or within 6 hrs of a transfusion and with a clear temporal relationship to an alternative risk factor for ALI, and looked at the original cohort of patients from the 2012 study. In the 145 pTRALI cases, clear temporal relationships were found between development of ALI and sepsis, pneumonia, aspiration multiple fractures and pancreatitis.

They noted that evidence in the literature strongly suggests that the two entities (TRALI and pTRALI) are quite different. The incidence of TRALI has decreased with the move towards male-predominant plasma, but pTRALI has not. Similarly, the presence of HLA or HNA antibodies has decreased in TRALI blood recipients, but not pTRALI. Also, pTRALI has had worse clinical outcomes and higher mortality rates similar to those of ARDS (acute respiratory distress syndrome).

In this selected group, originally TRALI now pTRALI, this study shows that transfusion factors were not strongly, independently, associated with pTRALI. These negatively associated factors included infusion of female plasma or whole blood, total number of transfusion products and total number of red cell or whole blood units received.

In contrast, there were positive associations (p < 0.0024 in all cases) between pTRALI and chronic alcohol abuse, current smoker vs never- or former—smoker, presence of shock before transfusion, and a positive fluid balance. All of these are known to increase the risk and manifestations of ARDS. These data clearly help explain the fact that the incidence of TRALI but not pTRALI decreased with the move towards greater male plasma use. They suggest that TRALI is donor white cell-related, but pTRALI is mostly related to recipient ARDS factors.

It seems that these two entities have different causes, different treatment requirements and different outcomes, given the reported incidence of death in cases of TRALI vs ARDS or pTRALI. In these studies, the investigators found that multiple transfusions in themselves did not seem to be an independent risk factor for ARDS, although receipt of antibody-containing plasma was. In his editorial, Dr. Popovskiy points out that the best way to reduce the incidence of TRALI, used here in the generic sense, I believe, is to follow the rule of “rights” when transfusing blood products: the right product, the right dose, the right time and the right patient.

True for all transfusions, all patients, right?

In the PLUS article in this issue about pathogen reduction, it was noted that the movement of people is bringing emerging blood-borne infections to non-endemic areas, emphasizing our closer, if not smaller, world. An example of this was recently noted in *Transfusion*. The authors reminded us that the 20 cases of *Trypanosoma cruzi* associated with transfusion in North America and in Spain were all related to platelet or whole blood transfusion. Neither of these regions is known to be endemic for Chagas’ disease, caused by *T. cruzi*. From 2011 to 2013, the authors carried out a prospective study in a group of donors on the island of Majorca, Spain, who had emigrated from Chagas-endemic areas. Using real-time PCR, they looked at the parasitic load in leukocyte-reduced plasma and in apheresis platelets, comparing these results with peripheral whole blood. The blood samples studied were all from seropositive donors who had detectable parasitemia in their circulating peripheral blood.

During the study, 1201 blood donors who originated from Chagas-endemic areas were tested for *T. cruzi* antibodies. All donors with positive serology who had not previously been treated for the parasite, and who consented, were analyzed for parasite load in their peripheral blood and blood components, namely plasma and platelets by apheresis. All donors were permanently deferred as donors for transfusion.

Of the total of 1201 donors, 23 were seropositive by the three serologic tests used. This represents 1.91% of the total donor population. In the Bolivian segment of this cohort, 106 people, 17 or 16% were seropositive, the highest of any subgroup. (This is not surprising since previous studies have noted almost 50% positivity in some groups of Bolivians in Bolivia.) Parasitic DNA was detected in peripheral whole blood in 14 of the 23 seropositive donors, using PCR. All 18 of the 18 platelet samples were positive by PCR, and all 3 replicates of each sample were positive. None of the 18 tested plasma samples were positive. The peripheral whole blood obtained the same day as the samples were all positive with a mean parasitemia of 0.42/ml. This compares to parasite loads that were undetectable in plasma and 5.33/ml in platelets.

The authors clearly make their point that these data likely explain the increased transfusion-transmission risk of Chagas’ disease in platelet transfusions as reported in published cases. Given the size of the parasite, as noted in peripheral blood smears, it seems possible that they are either captured in the apheresis process separately, or are prone to stick with the platelets.

Two more on TRALI

Workers in the Netherlands have been exploring thoughts similar to what we’ve discussed regarding TRALI (see first reference) and transfused mediators of neutrophil activation. They note that the antileukocyte antibodies implicated in TRALI, anti-HNA or anti-HLA, must be of high affinity and activating potential to lead to TRALI in otherwise non-predisposed blood recipients, and that our current methods of avoiding alloimmununized or alloexposed donors have led to the realization that there are other transfused mediators of neutrophil activation that are important, such as biologically active lipids and peptides and even cell debris that can act as biological response modifiers. These are thought to cause TRALI in patients who are predisposed to it, for any number of reasons; thus, the “two-hit model”, much as noted in the previous article looking at the work of Toy and colleagues. But they argue that there may be more than “two hits” as discussed above, such as the patient’s predisposing factors. This is an example of continued “lumping”, and ignores the fact that there are individual predisposing conditions that should be examined independently.

Multicausality of other diseases is an established fact for which several models have been developed, and the authors evaluate two of these in common use to examine this question with regard to TRALI, the threshold model and the sufficient cause model, the latter more abstract than the first. They each have decided strengths and evident limitations, but the authors believe them to be better than the two-hit model for describing the multicausal nature of TRALI.

In the threshold model, the propensity for a TRALI reaction is determined by exceeding a threshold from various underlying events: pneumonia, say, or chemotherapy, which have certain damaging effects on pulmonary vasculature and neutrophil reactivity, and they may vary from person to person for a number of reasons. But the accumulation of these factors, and how long each is operative, can (or not) place the patient’s risk for TRALI over that threshold.

The sufficient cause model, state the authors, is a general model of causality that is not just applicable to disease. They use diagrams to describe both of these models, noting that any of the causes might be what pushes the patient over the threshold, but that it is not always possible to know which one it is. Additionally, one or more of the component causes might disappear before the event threshold is reached, and the sufficient cause model does not always allow for duration effects to be realized. The threshold model thus seems best, although the authors go on to demonstrate that sometimes combining the two models is superior. This discussion is elaborated in the paper (see first reference).

In a Commentary on the topics in this journal, Warkentin and colleagues suggest that there are lessons to be learned regarding the etiology and treatment/prevention of TRALI by looking at the condition called heparin-induced thrombocytopenia (HIT). They recommend looking at TRALI from the perspective of detectability or non-detectability of leukoreactive alloantibodies, sort of an immune vs a non-immune TRALI. The implication is that if none are present in the donor unit, it is something else that is causing the apparent TRALI, or ALI.

In the case of HIT, earlier classifications were called non-immune HIT (HIT-1), in which no antibodies were found and immune HIT (HIT-2), in which they were. Distinguishing immune HIT where HIT antibodies were found from non-immune HIT, where no HIT antibodies were found, helped to focus basic research on the clinical syndromes but also on looking at the mechanism of disease, the direct proaggregetary effects of heparin on platelets that could also help explain the effects of heparin in non-immune HIT.

They go on to suggest to the TRALI research community that the immune and non-immune cases be clearly distinguished, and that TRALI be limited to cases in which passive leukocyte antibody transmission can be identified, much as originally proposed by Popovsky and Moore. Or designations could be even further split: TRALI-Ia in cases with HNA antibodies; TRALI-Ib for HLA Class I alloantibodies; TRALI-Ic for HLA Class II alloantibodies. TRALI-II could be used for those cases in which no such alloantibodies can be identified, and a non-immune causality thus suspected.

Clearly, as suggested by Dr. Popovsky in his words from the Frost poem, we have a long way and many miles to go before we sleep.


When should we give platelets…and how many?

In the last 15+ years, perhaps longer in some instances, we have seen a movement towards administering fewer transfused blood components than previously, lowering the threshold for intervention. Ten grams of Hgb is now rarely used as a “transfusion trigger”, nor is a platelet count of 20,000/µl. There have been a number of reports for both products that have moved our decision points for both therapeutic and prophylactic intervention. We’ve recognized that the “normal” levels of circulating blood cells we’re used to aren’t very effective—or sometimes attainable—goals for successful therapy.

A group of Transfusion Medicine experts, representing the AABB Platelet Transfusion Guidelines Panel, recently undertook a systematic review of evidence-based studies on platelet transfusion in order to synthesize recommendations for the most common situations in which platelet transfusions are considered. They developed a list of clinical questions based on a panel of experts in transfusion medicine, surgery, hematology, anesthesiology, intensive care and guidelines development. This group provided the authors with directions for the review which was conducted using standards from the Cochrane Collaboration.*

The study group looked at randomized controlled trials and at observational studies, including prospective and retrospective cohort studies, case control studies, and those with no control arm. If more than two randomized, controlled studies addressed the question, no observational studies were used. Evidence from randomized control studies was favored since most observational studies have transfusion-indication bias inherent in them. They searched the Cochrane Central, PubMed and Web of Science data bases from their inception until September, 2014. References in these publications were also examined for pertinent studies.

From the 1594 studies so identified, 17 randomized, controlled trials and 55 observational studies were included in the review. The quality of the controlled trials was “moderate”, as judged by the low number of events for some outcomes or other reporting bias. Methodologic quality of the observational studies was judged to be low due to selection bias, low numbers and poor standardization among other things. In the article, well worth your review, they list the 12 questions that they posed for data evaluation, and the very specific responses they developed from the collected information. Briefly:

1. Prophylactically transfusing platelets decreased the overall incidence of clinically significant bleeding, except in a small subgroup of autologously transplanted patients; however, it did increase the number of platelet transfusions given, and it was not associated with a difference in all-cause mortality.

2. In looking at a transfusion “trigger” of 10,000 platelets/µl, compared to 20,000/µl, for prophylactic platelet transfusion, there was no significant difference in the incidence of significant bleeding (see article for definition); however, there was—as one would expect—a greater number of platelets used, and a greater cost.
3. There were no significant differences in the incidence of significant bleeding, all-cause mortality, or bleeding-related mortality when comparing high dose (2.2–2.6 x 10^11/m^2) vs low-dose (1.1-1.3 x 10^11/m^2) transfusions. Because dosage was reported differently in different studies, the authors had to convert the different dose metrics into the number of platelets/m^2.

4. Studies in children undergoing lumbar puncture with acute lymphoblastic leukemia and thrombocytopenia (1450 pts. total) showed a spinal hematoma incidence of 0. In adults there were 2 cases in 86 total patients. The authors believe this is a rare event, and that lumbar puncture may not be associated with bleeding.

There were eight other interesting questions addressed, including the use of prophylactic platelets in cardiac surgery, traumatic brain injury or non-traumatic cerebral hemorrhage, general surgery, ITP, TTP, DIC, paracentesis, and thoracentesis. The article does not contain a summary of harms since adverse effects were reported sporadically in the studies, and most of these events are already known. Overall, the study group was surprised by the small quantity and low quality of available information. It would be helpful, they suggest, if registries could be established in which outcomes of studies involving consecutive patients undergoing a given procedure could be established.

The reader is encouraged to review these findings in some detail. This is a very important piece of work, and the authors and their supporting committees have done a terrific service with a difficult task. Congratulations to them!


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