HIV infection and AIDS. This is to discriminate on the basis of HIV.

Fourth, by allowing people who are HIV positive to enter our country we are not importing an incurable infection. We already have HIV infection in Canada. A preoccupation with testing immigrants for HIV antibodies and enacting laws to this effect is probably linked to many factors, including symbolism, disidentification, the need to take action and adoption of a politically safe approach. But most of all it may symbolize that AIDS is "out there", not "in here" and that we can take effective action to prevent its entry. This is one way to disidentify from AIDS, but it is destructive, not constructive, in terms of inhibiting the spread of HIV.

To turn to Dr. Frew's letter, first, I certainly did not intend to give any impression that the issues raised by HIV infection and AIDS should be looked at only from the aspect of the person infected with HIV. Both individuals and the community have justifiable claims. Second, Frew seems to state that "the unwitting victims of this condition" are people other than those infected with HIV. This is difficult to understand unless he is implying that there are "guilty" and "innocent" victims of HIV. Such reasoning is destructive of efforts both to inhibit transmission of HIV and to deal appropriately with people affected by HIV.

The rest of his arguments appear to confuse several concepts and are also difficult to interpret. Frew seems to address (a) compulsory testing, (b) rights not to know that one is HIV positive (that is, rights not to have test results disclosed to one against one's will), (c) confidentiality, possibly including whether society has a right to know a person's HIV status, and (d) discrimination in testing.

There are two underlying issues that need to be addressed in order to formulate responses to these concerns: first, which approach will best reduce transmission of HIV; and, second, which approach respects human rights the most, because all of the concepts mentioned raise questions of fundamental human rights. Fortunately, respect for people as individuals and for their human rights is most likely to inhibit the spread of HIV.[1] That is, at the level of principle and policy there is not a conflict between achieving both of these aims. There could, of course, be individual cases in which this is not true. These should be treated as exceptions to the approach adopted in general and governed by measures that are clearly characterized as exceptional. To the extent that Frew suggests some other course of action I would argue strongly that he is wrong.

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Congenital dislocation of the hip in Canadian Indian populations

Dr. R. Brian Lowry, Nancy Y. Thunem and Stacey Anderson-Redick are to be congratulated for their comprehensive study of congenital anomalies in Alberta (*Can Med Assoc J* 1989; 141: 1155-1159).

It is of great interest that the prevalence of congenital dislocation of the hip (CDH) is *less* among the aboriginal populations of Alberta, British Columbia and Western Australia than it is among whites, whereas in Saskatchewan,[1] Manitoba[2] and northwestern Ontario[3] the situation is reversed. Six Indian communities in northern Saskatchewan had a prevalence rate exceeding 10/1000 in 1967,[1] and at Island Lake, Man., Walker[2] recorded a world-record rate of 337/1000.

This difference in CDH prevalence between the two provinces farthest to the west and the two adjacent provinces is striking. Swaddling of infants, with the legs adducted and extended, has been almost universal among the Cree, Saulteaux, Ojibwa and Chipewyan Indians of Saskatchewan and Manitoba. This cultural practice certainly "brings out" any overt or latent genetic predisposition to CDH, and Indian infants do not show the higher incidence of CDH among firstborn infants and among infants delivered in the breech position observed in studies of white infants.[1] Cultural and genetic differences between the two Canadian Indian populations deserve attention.

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[Dr. Lowry replies:]

Dr. Houston's comments on the prevalence of CDH in Saskatchewan and Manitoba are of great interest. His observations suggest that there are both cultural and genetic differences between the Canadian Indian populations in the various provinces.

Throughout the 1960s and

early 1970s I conducted extensive studies of cleft lip and palate among the Indians of British Columbia, in the course of which I visited almost every reserve and area in the province. Although I was not specifically looking for CDH it is my impression that swaddling and the use of cradleboards were not common.

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Prophylactic transfusion of platelets and plasma in trauma care

It is generally considered important in every instance of transfusion to minimize the number of donor exposures. In their recent review article "Current concepts in trauma: 1. Principles and directions for development" (*Can Med Assoc J* 1989; 141: 529–533) Dr. Robert Y. McMurtry, Dr. William R. Nelson and Michael R.P. de la Roche recommend prophylactic transfusion of platelets and fresh frozen plasma to reduce the risk of coagulopathy when massive transfusion is required.

There are no good data to support this approach. Recent consensus conferences on the use of fresh frozen plasma[1] and platelet concentrates[2] clearly opposed the prophylactic use of these blood components in massive transfusion situations.

A more rational approach is to give the appropriate blood component to a patient with clinical evidence of microvascular bleeding in whom there is documented significant thrombocytopenia (a platelet count less than 50×10^9/L) or significant depletion of coagulation factors (e.g., a fibrinogen level less than 1.0 g/L). Prophylactic transfusion may be justified in some rural hospitals in which rapid platelet counts and coagulation screens are unavailable, but this is not usually the case in large urban centres.

The risk–benefit ratio of transfusion must always be considered, particularly since non-A, non-B hepatitis develops in 9.2% of Canadian transfusion recipients.[3]

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[Dr. McMurtry and the physician responsible for the protocol quoted in the article respond:]

Drs. Ferguson and Wadsworth raise the valid point that the risk of transfusion-derived infections such as non-A, non-B hepatitis increases with the number of units transfused. This risk is likely to diminish this year with the introduction of testing for the agent of hepatitis C by the Red Cross Blood Transfusion Service. Nevertheless, the long-term morbidity and mortality risks from such sources must be weighed against the possible risks of withholding blood component therapy.

The consensus reports cited by Ferguson and Wadsworth do not explicitly review the published information, and in spite of the consensus there remains a body of opinion that an aggressive approach is warranted in patients requiring *rapid* massive transfusion.[1-3] Although, as Ferguson and Wadsworth state, there are no good data to support this approach, there are really no good data to refute it either.

When rapid massive transfusion (i.e., more than 20 units) is being given it is extremely difficult to provide laboratory data on hemostasis in a timely fashion, even in major centres that are fully staffed. We agree that when massive transfusion is being given over several hours it is rational to monitor the need and the effects of blood component therapy. However, we were talking about the massive transfusion support that is rapidly needed in resuscitation.

It is uncertain with the present evidence whether it is better to try to prevent microvascular bleeding and run the risk of transfusion-transmitted disease or to wait until microvascular bleeding develops and treat it. There are no good outcome studies addressing the question of whether one should treat according to a protocol or wait until bleeding occurs or certain laboratory criteria are met before treating. There are also no good studies of the incidence of transfusion-transmitted disease in these patients. Perhaps we need a clinical trial of prophylaxis versus criteria-triggered treatment and a follow-up study.

In the only reported controlled study assessing platelet prophylaxis during massive transfusion Reed and colleagues[4] found that it had no value. However, the study prescribed prophylaxis after 12 units of blood had been transfused, whereas the evidence suggests that clinically significant thrombocytopenia occurs at or after transfusion of 20 units;[5,6] thus, Reed and colleagues may have masked differences by transfusing too soon or unnecessarily. In addition (as pointed out by