packed with British graduates as ours are with those of the underdeveloped and under-doctored countries?

Dr. Baber regrets that Ivan Illich (and I) cannot provide the "practical solution." I would find one highly suspect, but of set purpose the piece I wrote began "La condition humaine," the title of a 1920s novel by André Malraux about Chinese revolutionary activity, a novel that ended on a note of disillusionment. But look, I was trying to imply, at China today: it is the most hopeful country in the world. Dr. Millar asks "Can Affad do what it wants in Iraq and uproot through the unknown grass again with Eve, ignorant even of their nakedness?" Facedown-ness aside, I suspect—and so, I find, do many young people—that something as radical as that is needed. When we knew little," he says, in defence of science, "we were not so far from the animals." There speaks Homo sapiens, self-styled and self-condemned. By what right, unless one that our "ability to destroy an entire species confers upon us, do we look down upon the "animals"? Are we not animals? What other creature has ever touched the depths that the human animal has scourged? Dr. Millar says it is not science and technology that I am to blame "but our own faults." I agree. But he and Dr. Hunter quite miss my point: until the last few decades man did not have, in the service of his unique human greed, envy, and hatred, weapons whose use might end the species. Man has not changed, but his weapons have: ergo man must now change or else.

Finally, I go guessing in general with Dr. Hunter's suggestion that "each challenge must be met and tackled in a spirit of hope, for it will do no good to despair." But there are items I find myself unable to tackle in any spirit of hope: the thermostat, nuclear weapon about to explode, for instance, and starved children when there is simply no food to give them. Or, rather, my hope then would not be of the temporal kind.—I am, etc.,

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Trasylol for Pneumocystis

SIR,—Dr. M. L. Lewis (21 September, p. 741) expresses concern about the statement in our letter (20 July, p. 133) that aprotinin (Trasyol) was "harmless" with an "absence of side effects." I agree that neither aprotinin nor any other drug should be regarded as completely harmless, though I believe that the drug was "harmless" to be understood as relative.

Dr. Lewis presents two cases of pneumocystis in which he associated the use of aprotinin with a deterioration in the course of the disease. I would like to comment on his conclusions with respect to the assumed aggravation of consumption coagulopathy or hypercoagulability because the full spectrum of activity of the drug was not fully consi dered.

Aprotinin is undoubtedly a very powerful inhibitor of plasmin, which to many haematologists would appear to be a contra-indication for aprotinin in consumption coagulopathy. Other proteins are, however, inhibited by aprotinin, including those that play an important role in the clotting cascade.1-4 Kallikrein has recently been added to this list (to be published in this issue of the journal), and this provides a better understanding of the antithrombotic action of aprotinin.4 5 It is misleading to consider the action of aprotinin on the coagulation and lysin systems in terms of plasmin inhibition. Experimental evidence has shown that fibrin deposits are reduced in both size and number under aprotinin treatment in shock states associated with disseminated intravascular coagulation. Further, it has been demonstrated that clotting time is prolonged rather than shortened in conditions where activation of clotting takes place. Regarding the shock lung syndrome, there is again evidence that it is either prevented or favourably influenced by aprotinin.6 Though aprotinin does not aid the dissolution of established fibrin deposits, it may well help to prevent their further formation.

Finally, I would agree with Dr. Lewis that both streptokinase and heparin may have a role here, but in this complex situation, I feel it would be wrong to advocate their clinical use until experiments along the lines of Blümil have proved that the risks of further activating proteolysis do not outweigh any possible benefits.—I am, etc.,

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Prevention of Exercise-induced Asthma by Indoramin

Sir,—Professor S. Bianco and others (5 October, p. 18) claimed to have shown that the alpha-adrenoceptor blocking drug indoramin inhibits exercise-induced asthma (E.I.A.) and that this supports the concept of abnormal receptor response as the cause of E.I.A. Before this can be accepted I believe that it is important to point out that their data are open to question.

Indoramin is clearly a potent bronchodilator, as they have shown, but this means that the exercise test after the drug had been given was begun with airways of greater calibre than in the control experiments. In this situation it is difficult to compare control and drug tests, but it is certainly not acceptable to claim that the drug blocks E.I.A. because conductance never fell below the level in the control (undilated) tests. When expressed as a percentage of the post-drug value the fall in conductance after exercise in their subjects ranged from 0 to 69%. In the control tests the percentage fall ranged from 38% to 81%. Analysed in this way it would appear that six of their patients...