packed with British graduates as ours are with those of the underdeveloped and underdoctored countries?

Dr. Baber regrets that Ivan Illich (and I) cannot offer a "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 Adam now replace the fruit and tiptoe through the unmown grass again with Eve, ignorant even of their nakedness?" Facetiousness 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 even touched the depths that the human animal has scoured? Dr. Millar says it is not science and technology that are 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 along 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—a thermonuclear weapon about to explode, for instance, and starving 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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Prevention of Exercise-induced Asthma by Indoramin

SIR,—Professor S. Bianco and others (5 October, p. 18) claim 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 a different interpretation.

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

had significant reduction of E.I.A. by the drug and five had almost as much or more E.I.A. compared with control studies.

However, there are further complicating factors in this type of work, such as the major diminution of E.I.A. by placebos¹ and the variability of the response to exercise, especially when a bicycle is used.² No placebos were used in the control studies of Professor Bianco and his colleagues and this must diminish the validity of their conclusions. Perhaps this accounts for the fact that Sly *et al.*³ were unable to prevent E.I.A. with alpha-blockade when proper controls were used. The present authors do not provide any data on the reproducibility of their tests, but in eight of the 11 subjects the resting conductance was lower on the drug-test day before the drug and in the one subject tested twice without drug the fall in conductance appears to have been 60% on one day and 44% on another. This same subject had a 69% fall with 50 mg of indoramin but only 14% with 60 mg of the drug—a quite extraordinary dose-related phenomenon.

While not denying that effective alpha-blockade may cause bronchodilatation in the asthmatic, and may to some extent influence the severity of E.I.A., I do not believe that the study by Professor Bianco and colleagues can justify their statement that indoramin "has been found to prevent the occurrence of exercise-induced bronchoconstriction" or that their work provides any evidence for the pathogenesis of E.I.A.—I am, etc.,

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Trasylol for Pancreatitis

SIR,—Dr. M. L. Lewis (21 September, p. 741) expresses concern about the statement in your leading article (20 July, p. 133) that aprotinin (Trasylol) 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 concept "harmless" was understood to be relative.

Dr. Lewis presents two cases of pancreatitis 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 considered.

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 proteinases 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 list⁵ and helps to give a better understanding of the antithrombotic action of aprotinin.⁶⁻⁹ It is misleading to consider the action of aprotinin on the coagulation and lysis systems in terms of plasmin inhibition alone. 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.¹⁰ Furthermore, it has been demonstrated^{11,12} 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.¹² 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 place, but in this complex situation I feel it would be wrong to advocate their clinical use until experiments along the lines of Blümel¹³ have proved that the risks of further activating proteolysis do not outweigh any possible benefits.—I am, etc.,

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Treatment of Casualties in General Practice

SIR,—A few months ago I was summoned on a Sunday evening from my home to the surgery to suture a small patient who had severely cut her finger. The nearest casualty department is 13 miles (21 km) in either direction. During the course of 30 minutes I cleaned the wound, ascertaining there was no tendon damage, and used an ampoule of sterile suture material. I applied a dressing kept in position by Tubegauze, administered the child's booster of tetanus toxoid, and arranged for our practice sister to remove the sutures after the appropriate time. I tendered a prescription on E.C.10 for the suture material and Tubegauze, which has been returned to me, each item stamped twice by the pricing bureau in Durham.

I should like to therefore give notice that in future I shall think very hard before providing any minor casualty service whatsoever and shall limit my item of service to applying a piece of gauze and strip of Elastoplast and arranging transport to the nearest casualty department. I do not regard injuries such as the one described above as the proper responsibility of a hospital casualty department. In other parts of the world they