necessarily imply that "where intervention is frequent clinical standards are necessarily low." All this was discussed in our pamphlet with much supporting evidence, amply quoted.

It is not difficult to deny our conclusions in a leading article claiming highly dubious premises to be the basis for our argument. What we argued was quite simply that where large variations in surgery rates exist it is important to know whether or not any of the variation is caused by essentially inappropriate (that is, non-medical) considerations. If it is, then it is important to know whether the higher rates usually associated with fee-for-service medicine are more appropriate than the lower rates of the NHS or prepaid practice. That is why we devoted some space to evaluation of these issues by clinical trials and quoted some examples where high rates may not be appropriate. We also discussed some work attempting an evaluation of the costs, risks, and benefits of surgery² to throw some further light on this difficult problem. Unfortunately there is no space here to go through the arguments, which anyway suffer too much from simplistic polarisations.

We also stressed that the process of evaluation was rarely easy for a whole series of reasons to do with the organisation of medicine and for legitimate scientific or ethical considerations. An example of one of the difficulties is provided by your quotation of one recent study³ of the relationship between rates and "clinical standards." It is, however, one among many studies looking at the variations in rates of surgery and one among a few studies of the relationships between rates and standards. To suggest that this study is puzzling implicitly recognises the validity of our argument, for it finds no negative association between rates of tonsillectomy (TA) in Manitoba and clinical standards. The proportion of these operations which had a particular set of clinical indications was no lower in areas with a higher rate. However, it is the core of your evidence suggesting our inference to be weak. We actually cited much more evidence pointing in the opposite direction, particularly for dentistry as well as diagnostic tests and surgery. We could not then discuss this paper because it was not published.

However, it ought to be noted that since interest in surgical rates has received more publicity several things have happened to TA rates in North America. In Canada these rates have dropped by over 30% since 1968⁴ and Manitoba, during the period of the above study, had the second lowest rates of all Canadian provinces (these are using total population as a denominator but are age-standardised). The lowest province, Newfoundland, happened to have the lowest rates for all the other seven common procedures which were reported. Most important, however, there has been a more dramatic reduction in the variation of these rates over the same time. A study in Vermont, USA,⁵ shows that since information on rates has been reliably collected and fed back to clinicians not only has the TA rate declined by 46% over the state but the variation between hospital service region of residence has decreased by a factor of nearly four between 1969 and 1973 measured by the standard deviation of the rates. Obviously many factors may contribute to this reduction, not least of which is the estimate that the highest rate in 1969 left only 37% of children⁶ with any tonsils. The fact remains that for whatever reason the variation in rate has reduced in Canada too and in Manitoba it was less than the variation in Vermont even in 1973.

It is a basic principle of good experimental design that to test an aetiological hypothesis of a given factor on another one is ill advised to take as one's sample a set of observations known to vary less than does the "population" for which one would want to make an inference. For example, if one wants to investigate the possible role of dietary fibre

intake on the prevalence of diseases of the colon one would try not to rely too heavily on information based on a group of people known to have, relative to the entire population, similar dietary habits. What we were discussing were those otherwise comparable areas for which the variations in levels of practice were higher than any variation in legitimate index of medical need could explain, of which there are many. TA rates in Manitoba in 1973 did not happen to be among them and the results of that study are not, in the end, all that puzzling.

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¹ Radical Statistics Health Group, In *Defence of the NHS*. London, RSHG, 1977.

² *Costs, Risks, and Benefits of Surgery*, ed J Bunker, B Barnes, and F Mosteller. London, Oxford University Press, 1977.

³ Roos, N P, Roos, L L, and Hanteleff, P D, *New England Journal of Medicine*, 1977, **297**, 360.

⁴ Vayda, E, Morison, M, and Anderson, G, *Canadian Journal of Surgery*, 1976, **19**, 235.

⁵ Wennberg, J, et al, *Pediatrics*, 1977, **59**, 821.

⁶ Wennberg, J, and Gittelsohn, A, *Science*, 1973, **182**, 1102.

Dietary cholesterol and atherosclerosis

SIR,—In their survey of diet in relation to coronary heart disease (19 November, p 1307) Professor J N Morris and his colleagues found that "analysis of dietary cholesterol against coronary events showed no association at all." In this connection I wonder how many of your readers could give the author and date of the following quotation.

"Among other factors that may play an important part in the pathogenesis of atherosclerosis attention has recently often been drawn to the importance of an increased amount of cholesterol in the organism in general and in the blood in particular. As is well known, an attempt has even been made to set up a theory of the origin of atherosclerosis that rests solely on hypercholesterolaemia.

"The most important facts that form the basis of this and similar theories are the following. Firstly, one often finds a more or less raised blood cholesterol content in cases of human atherosclerosis. Secondly, the wall of the atherosclerotic aorta always contains an increased amount of cholesterol or of its compounds, which apparently play a large part in the origin and development of the atherosclerotic changes. Thirdly and last, it is possible to produce typical atherosclerosis in rabbits by prolonged feeding of cholesterol-rich foods or of pure cholesterol.

"The two first of these arguments do not have the force of direct and incontrovertible proof since hypercholesterolaemia, although often accompanying the obvious cases of atherosclerosis, does not appear to be an absolutely constant finding in atherosclerosis. Cholesterol deposits in the aortic wall as well as hypercholesterolaemia may also be looked upon simply as a concomitant phenomenon in atherosclerosis. Moreover, the occurrence of cholesterol in the aortic wall may be considered, as has already been done by several writers, simply as being derived from breakdown products of the already pathologically altered components of the aortic wall.

"In contrast, the third of the above-mentioned arguments can to some extent be accepted as a direct proof for the 'cholesterol aetiology' of atherosclerosis. If it is in fact possible to produce typical atherosclerotic changes in the aorta of rabbits by the administration of pure cholesterol without any other harmful factor, then it seems that this is proof that the development of atherosclerosis depends entirely on the content of cholesterol or of its compounds.

"It is, however, possible to find important objections to a direct transfer of these experimental results to man. Firstly, in order to produce experimental atherosclerosis by feeding rabbits with

cholesterol colossal doses are needed. In association with this the cholesterol content of the blood in these rabbits reaches enormous values. On the other hand human beings never consume these quantities of cholesterol and never does the amount of cholesterol in the blood of human beings, even in the severest degree of hypercholesterolaemia, reach such enormous values as in animals that have been fed with cholesterol for a long time. . . ."

This was written by Anitschkow in a paper published in 1914¹ which I have translated from the German. It appeared one year after his classic paper² describing the first experimental production of atheroma, by feeding rabbits with cholesterol.

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¹ Anitschkow, N, *Deutsche medizinische Wochenschrift*, 1914, **24**, 1215.

² Anitschkow, N, and Chalutow, S S, *Zentralblatt für allgemeine Pathologie und pathologische Anatomie*, 1913, **24**, 1.

Treating dissenters

SIR,—Dr Edward Hare in his article on dissenters (29 October, p 1136) refers to the historical background of psychiatric abuse in Russia and accepts that it has become more common in that country in the past 25 years. This psychiatric abuse as practised both in the past and at the present time consists essentially in diagnosing an individual held to be politically undesirable as mentally ill when this is not the case and then proceeding to deprive him of his liberty by incarcerating him for an indefinite period in a special or prison mental hospital. There can be surely no greater abuse of medicine than this and it can be perpetrated only with the active co-operation of the psychiatrist. It must be seen to be what it is—namely, an act of political repression masquerading as medical treatment.

This is tacitly admitted when Dr Hare states that Soviet politicians might argue that they are acting with as much humanity as possible in a collectivist society against persons whose behaviour constitutes a serious threat to the stability of government. To suggest that this action of quite unwarranted commitment to a mental hospital is a humane alternative to a defined term in prison merely adds insult to injury. And the further comment of Dr Hare that although treatment is in some cases against the will of the individual, yet it is no different from that given to a patient with the same diagnosis ignores the fact that he should not have been forced there in the first place and is no great source of consolation to the victim convinced of his own sanity.

Such dissidents, Dr Hare feels, behave as they do either because of a fearless and noble love of truth or from an obstinate determination to speak one's mind. Is it possible that it could be for both reasons? This obsessive persistence, as he terms it, in the face of warnings and threats could well be applied to the men of destiny and the martyrs of history down the ages and his apologia for the Soviet psychiatrists acting under pressure from the State could equally well apply to the infamous Nazi doctors of the concentration camps.

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SIR,—Dr Edward Hare's summary under the heading "Evidence of guilt" (29 October, p 1136) is clear and helpful. However, under