of lithotripsy are therefore 3·8×£87 (inpatient) +£800 (procedure)+£100 (other admission costs, such as investigations and records)=£1231. The costs of one stage percutaneous nephrolithotomy are 5·63×£87 (inpatient)+£408 (procedure)+£100 (other)=£998.

The appendix also omits maintenance, depreciation, rates, electricity, and servicing from the calculations for open surgery and percutaneous nephrolithotomy.

Lithotripsy may not be the "cheapest" procedure: a controlled trial is required which must include an assessment of benefits for the patient (29 March, p 877).

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AUTHOR'S REPLY—I must take issue with Dr Jones on his analysis of daily inpatient costs. Our figures come from the Bloomsbury Health Authority's unit cost statistics for the year ending 31 March 1984. The figures comprise:

Patient care services (medical, dental, and nursing services and supplies)	£87 13
Medical and paramedical supporting services (diagnostic and paramedical treatment)	£18 33
General services (catering, laundry, administration, etc)	£59 06
Total	£164 52
Adjusted total for 1985	£174 00

The "procedures" which are included are medical and diagnostic but not surgical. Nowhere in the analysis are any specific procedures included, nor would one expect there to be as most health authorities find it extremely difficult to cost surgical procedures. This has now been undertaken by the West Midlands Health Authority, whose financial information project aims to computerise the costs of all surgical procedures.

Secondly, the appendix does include the cost of maintenance, depreciation, rates, and services for open surgery and percutaneous nephrolithotomy. Under "theatre time," maintenance is shown as £25/h, which comprises £10 maintenance and £15 depreciation. For the *x* ray department the figures range from £5 to £10/h and are included in units of radiology.

However, it is a sad indictment of the NHS when argument about a major advance in patient treatment is reduced to quibbling over shillings and pence. Undoubtedly the major consequence of extracorporeal shockwave lithotripsy is the saving to the state in sickness benefit for patients who would normally be off work for two to three months. Finally, you have only to ask a patient who has had both open surgery and lithotripsy which treatment he preferred and the answer is obvious. We consider that it would be unethical to subject patients to a controlled clinical trial of lithotripsy at this late stage of its development.

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High density lipoprotein cholesterol is not a major risk factor for ischaemic heart disease in British men

SIR,—There is one aspect of the study by Dr S J Pocock and others (22 February, p 515) that I would like to question.

The study found that serum high density lipoprotein (HDL) cholesterol was lower at entry to the study in those who subsequently developed ischaemic heart disease, as have other studies. However, the authors tended to discount the finding because the differences were small and non-significant after adjusting for age, body mass index, blood pressure, cigarette smoking, and the concentration of non-HDL cholesterol. At first sight this represents the accepted approach of controlling for other recognised risk factors to avoid reporting "spurious" relations. The trouble in this case is that two of the other risk factors cannot be regarded as independent. There is very good evidence that smoking lowers serum HDL cholesterol concentrations in a dose related and reversible manner.[1-3] If HDL concentration has any importance for ischaemic heart disease this would imply that lower HDL values represent one of several mechanisms, through which smoking contributes to the development of ischaemic heart disease.[4] Obesity is also associated with low HDL levels,[1] which could similarly contribute to the increased incidence of ischaemic heart disease in the overweight. In each case HDL would appear to act as one of the intermediate factors through which the so called "risk factors" are effective, though one could argue that the intermediate factors themselves should be seen as the real risk factors in that they represent evidence of risky responses to influences such as smoking and obesity in exposed predisposed subjects.

If smoking and obesity affect ischaemic heart disease in part by reducing serum HDL then adjusting for smoking and obesity will remove a real part of the relation of HDL and ischaemic heart disease along with the "independent" ways in which smoking influences ischaemic heart disease—it will in fact throw the baby out with the bath water. Correlations have been reported between the different intermediate factors affected by smoking which suggest that much of the real effect might go.[4] In that case the net effect of adjusting the risk estimate for ischaemic heart disease for smoking and obesity would be comparable to adjusting the effect of total (or of low density lipoprotein (LDL)) cholesterol for the subject's dietary intake of cholesterol. Adjustment for smoking and obesity cannot be justified by pointing to the fact that the risk estimate for non-HDL cholesterol was not much reduced, since smoking causes a proportionately much smaller increase in LDL cholesterol than decrease in HDL.[5] Smoking also causes increases in serum triglyceride values, and similar logic applies to the authors' earlier study.

My other point is whether it is appropriate to use the same pattern of analysis for HDL and LDL cholesterol. HDL concentration is relevant to the incidence of ischaemic heart disease only in populations,[6] and probably individuals, when LDL concentrations are high. I therefore question the validity of standard management as continuous variables when both are concerned either as single variables or expressed as a ratio. Perhaps comparisons of the effect of HDL should also be performed separately in patients with high LDL values before serious doubt is cast on the role of HDL cholesterol.

On the present evidence this paper does not show that HDL cholesterol is not a major risk factor for ischaemic heart disease, though this may in fact be the case. I suspect that the authors may well be right when they suggest that it may be a subfraction of HDL cholesterol that is important and that the present method of estimation still represents a fairly unrefined epidemiological tool.

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1 Garrison RJ, Kannel WB, Feinlab M, Castelli W, McNamara PM, Padgett SJ. Cigarette smoking and HDL cholesterol. *Atherosclerosis* 1978;**30**:17-25.
2 Criqui MH, Wallace RB, Heiss G, Mishkel M, Schonfield G, Jones GTL. Cigarette smoking and plasma HDL cholesterol. *Circulation* 1980;**62**(suppl 4):70-6.
3 Stubbe I. Eskilsson J, Nilsson Ehle P. HDL concentrations increase after stopping smoking. *Br Med J* 1982;**284**:1511-13.
4 Anthony HM. Reactive changes in the blood of smokers and the development of arterial diseases and COPD. *Reviews on Environmental Health* (in press).
5 Goldbourt V, Medalie JH. Characteristics of smokers, non-smokers and ex-smokers among 10,000 adult males in Israel. *Am J Epidemiol* 1977;**105**:76-86.
6 Aravanus C. The classic risk factors for CHD: experience in Europe. *Prev Med* 1983;**12**:16-9.

AUTHOR'S REPLY—Dr Anthony raises some interesting points concerning the relation between HDL cholesterol and other risk factors. Our own data also show a lower mean level of HDL cholesterol in cigarette smokers.[1] However, the association is not very strong: only 1% of the variance of HDL cholesterol is attributed to smoking, so that an individual's HDL cholesterol concentration is primarily determined by other factors. If we adjust for HDL cholesterol when analysing the association between cigarette smoking and risk of major ischaemic heart disease smokers still have a threefold increase in risk compared with non-smokers. That is, we see no evidence for the effect of smoking being via HDL cholesterol. A similar conclusion applies to serum triglyceride concentrations.

As we have shown previously,[2] the impact of obesity on risk of major ischaemic heart disease seems to be explained by the increases in mean blood pressure and mean serum total cholesterol (or non-HDL cholesterol) found in obese men. Thus adjustment for obesity does not affect the estimation of HDL cholesterol's contribution to risk.

Dr Anthony's suggestion for looking at the risk contribution of HDL cholesterol at different levels of LDL cholesterol is well worth pursuing. However, it would require a large number of patients with major ischaemic heart disease to achieve reliable estimation of effects in such subgroups. We hope to be able to present information on this issue in the near future, since our current data on over 300 cases are being processed at present. It should be noted that our British men have considerably higher mean serum concentrations of total cholesterol, and hence probably LDL cholesterol, than those in the other major studies of HDL cholesterol in Framingham and Israel.

Overall, we stand by our previous conclusion that HDL cholesterol is a far less important risk factor than the established factors such as cigarette smoking, blood pressure, and serum total cholesterol.

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1 Thelle DS, Shaper AG, Whitehead TP, Bullock DG, Ashby D, Patel I. Blood lipids in middle aged British men. *Br Heart J* 1983;**49**:205-13.
2 Shaper AG, Pocock SJ, Walker M, Phillips AN, Whitehead TP, MacFarlane PW. Risk factors for ischaemic heart disease: the prospective phase of the British Regional Heart Study. *J Epidemiol Community Health* 1985;**39**:197-209.

SIR,—The analyses by Professor V Wynn and his colleagues (12 April, p 1013) attempt to control for total cholesterol and then ask whether high density lipoprotein (HDL) cholesterol is more important than non-HDL cholesterol as a correlate of coronary heart disease. This is, however, a completely nonsensical statistical question: indeed, given an exact measure of total cholesterol, HDL and non-HDL cholesterol would necessarily be of