habitual lateness will hinder their progress up the greasy pole and be an absolute bar to success in private practice should they have any aspirations in that direction. If trainee surgeons could also be taught that preparing a patient for their knife takes longer than the five minutes they generally allow for changing and scrubbing up knife to skin at 8.30 am would become more of a reality than the myth it now is. But then Mr Datta would have no time to write his delightful articles.

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SIR,—“We never send for the patient until we see the whites of the surgeon's eyes,” the theatre staff have forcibly made clear to me at one of our local hospitals.

Mr Pradip K Datta rightly laments the lack of punctuality with which operations begin and finds his anaesthetic colleagues mainly to blame. The time has come for the profession to reach a consensus on the meaning of the “starting time” of a theatre case and to encourage its realistic use by all concerned.

A difficult aspect of our work as surgeons and anaesthetists is the scheduling of that work in the best interests of all those involved in the safe conduct of an operation: the patient, occasionally parents, ward staff, porters, anaesthetic and theatre nurses, and ourselves. There is, however, no agreement on the meaning of the starting times around which all these participants are required to perform their duties. These times, appended to tolerant noticeboards throughout our hospitals, seem to signify anything from “sending” (even if the patient to be sent for is six floors away) to the moment of knife to skin. Like Mr Datta, I have realised the second definition on only two occasions despite frequent monumental efforts to mobilise the system accordingly. It is one of the delusions of doctors that they alone control such events.

Behind the lack of precision lies a widespread inability to face certain practical facts. Time is required to transport a patient from the ward; to book him or her into the theatre suite; to establish rapport, monitoring, and anaesthesia; and, finally, to wheel the patient into the theatre, lift him across, and settle him under the drapes. The durations of these stages could profitably be measured and recognised in planning theatre time. The dangerous fiction that an anaesthetic is merely a two minute squirt on the way to the knife might then finally be put to rest and the surgeon given a reliable time at which to don his wellingtons.

There is a need for a statement from the Royal College of Surgeons and its faculty of anaesthetists on this matter.

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SIR,—It is regrettable that some statements made by Mr Pradip K Datta, should have been published.

Wick has a fairly small medical community whose members are easy to identify, which is particularly unfortunate as so many of the statements made by Mr Datta are untrue. It is simplistic to assume that a surgical operation starts when the knife strikes the skin. To have a set “knife in” time is a silly objective and certainly one that is way down the list of priorities of any anaesthetist. Getting the patient to operation adequately assessed (after discussion with the patient and colleagues), well prepared, and as stable as possible is surely more important. That this is achieved in daily

surgical practice by all the relevant staff being in the hospital well ahead of time is regarded here in Caithness as self evident.

The suggestion that three minutes is the time that any reliable surgeon would take for a surgical consultation is ridiculous. In any case, even if the person Mr Datta refers to were half an hour late at two patients booked every quarter of an hour this would mean only four patients in the waiting room—surely a small waiting quota. In fact, however, the statement regarding a surgeon leaving home to drive 20 miles to a clinic at 9.30 am is not correct as far as Caithness is concerned.

It is typical of the exaggeration of Mr Datta's claim that he has started his list on time only twice in 19 years.

Usually we associate Mr Datta with light hearted, humorous articles. It is rather a pity that he has overstepped into unkindness on this occasion and that his article has degenerated into an unwarranted assault on his professional colleagues past and present.

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Testing the sense of smell

SIR,—While Dr Victoria Moore-Gillon's leading article (28 March, p 793) provided an admirable introduction to the new olfactory testing kits, she did not address the importance of using the right odours in the clinical testing of olfaction.

Many odours can be detected by trigeminal nerve endings in the nasal cavity. The clinical relevance of this was shown in a simple study in which many of the “standard” odours (peppermint oil, camphor, cloves) failed to detect known lesions of the olfactory (cranial nerve I) pathway.¹ By contrast, these were readily picked up by floral or musky odours—for example, musk ketone, exaltolide, linalyl acetate, coumarin—without any special equipment. The use of these odours also showed an unexpectedly high incidence of hyposmia or anosmia in patients with multiple sclerosis (68%). There was an almost inverse correlation between correct identification of the odours and their value as markers for impaired olfactory sensitivity, reflecting the extremely limited smell vocabulary of most people; this calls into question the importance of correctly identifying the odour in the new kits, as opposed to just detecting something.

If some hospitals do not elect to use the new “scratch and sniff” kits it is high time that they replaced the not only dusty but also inappropriate “smell bottles” with ones containing odours that really do test the first cranial nerve and do so with surprising sensitivity.

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1 Pinching AJ. Clinical testing of olfaction reassessed. *Brain* 1977;100:377-88.

Hypochondriasis: an acceptable diagnosis?

SIR,—Dr Louis Appleby's review of hypochondriasis (4 April, p 857) is a welcome contribution to a problem that goes far beyond psychiatric practice. Though a primary syndrome undoubtedly exists, a purely descriptive basis for this conclusion is insufficient and the role of reassurance is complex. The mechanisms need to be clarified if successful treatments are to be devised.

Our work supports the view that anxiety, bodily symptoms, and illness related behaviour (of which reassurance seeking is just one example) are functionally interrelated in the maintenance of hypochondriasis.^{1,2} In particular, illness behaviours contribute to increased perception and misinterpretation of and preoccupation with bodily symptoms. This mechanism closely resembles the self defeating and self maintaining behaviour of severe obsessional ritualisers. In hypochondriacal patients the illness related behaviours lead to short term relief of anxiety but long term increase in fear and preoccupation and the need to seek further reassurance.

We agree that some health workers have a negative view of hypochondriacal patients, often based on the patients' failure to respond to repeated reassurance. Paradoxically, repeated discussion of potentially anxiety provoking information exacerbates the patients' fears despite initial relief. New and relevant information given with great care is, however, helpful when it accurately explains the patient's frightening symptoms rather than attempting to argue the absence of feared illness. Dr Appleby suggests that detailed reassurance becomes more effective with time and reduces anxiety. This view contrasts with the very definition of the problem—that is, the failure of patients to respond to reassurance³—unless it is assumed that doctors do not give detailed reassurance to hypochondriacal patients. Reassurance based on inappropriate investigations, physical examinations, complex communications, and repeated clinic appointments may be extremely detailed but must not be confused with giving accurate, new, and relevant information that the patient understands.²

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1 Warwick HMC, Salkovskis PM. Reassurance. *Br Med J* 1985; 290:1028.

2 Salkovskis PM, Warwick HMC. Morbid preoccupations, health anxiety and reassurance: a cognitive behavioural approach to hypochondriasis. *Behav Res Ther* 1986;24:597-602.

3 American Psychiatric Association Committee on Nomenclature and Statistics. *Diagnostic and statistical manual of mental disorders*. 3rd ed. Washington, DC: American Psychiatric Association, 1980.

Inadequacy of oleic acid in erythrocytes as a marker of malignancies

SIR,—We support the findings of Dr N Lawson and coworkers (21 March, p 769) and Dr O Soreide and colleagues (28th February, p 549) regarding the inadequacy of the stearic to oleic acid ratio in erythrocytes as a marker of malignancies, as proposed by Wood *et al*,¹ who suggested that this ratio might be used not only as a diagnostic marker in patients with colorectal cancer in particular but also to monitor recurrence.² We fully endorse the comments of Dr Lawson and colleagues about the methods of the two other groups of workers.

There are, however, further points to consider, including the influence of short term dietary intake as well as age and sex on the erythrocyte fatty acid membrane profile.^{3,4} We are investigating these factors in patients with colorectal cancer, and our results will soon be published in full. The following results were presented to the Surgical Research Society last year.⁵ We found that the erythrocyte stearic to oleic acid ratio in 21 patients with colorectal cancer (median 0.94; interquartile range 0.88-1.0) was similar to that in 21 patients with benign disease (0.92; 0.87-0.96). Unlike those in

other studies our controls were patients who did not have acute medical conditions or deranged liver function, and they were matched individually for age and sex with the cancer patients. Furthermore, patients receiving special diets, such as vegetarians or patients receiving low fat diets because of gall stones, were excluded. Though there was no difference in the erythrocyte stearic to oleic acid ratio between the groups, we found that the palmitic to stearic acid ratio was significantly different between the groups (cancer patients: 1.11; 1.02-1.28 *v* controls: 1.41; 1.31-1.53; *p* 0.005, Mann-Whitney U two tailed test).

Dr Sørreide and coworkers raised the possibility of an association between age and the erythrocyte stearic to oleic acid ratio. Their scatter graph was, however, a composite of two groups: a fairly young, healthy "reference" group and an older hospital "control" group (many with acute medical disorders or altered dietary lipid intake). This must undermine any statistical correlation that was thereby shown. These authors did not mention an association between age and this ratio in the group with malignancies. In our own preliminary work we have found no statistical association between age and the stearic to oleic acid ratio in either the control or colorectal cancer group. We did, however, find a significant negative correlation between age and the palmitic to stearic acid ratio in the colorectal cancer group (0.02 < *p* < 0.05) but not in the control group.

Though the evidence against the use of the stearic to oleic acid ratio as a diagnostic marker in patients with solid malignancies is mounting,⁵ our preliminary studies suggest a different erythrocyte membrane fatty acid profile in patients with colorectal cancer, which requires further investigation.

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Childhood leukaemia in relation to nuclear establishments

SIR,—Dr Eve Roman and colleagues (7 March, *p* 597) do not discuss the reliability of their estimates of the child population in small areas. Substantial changes may occur over 14 years, not only in the total ward population (adjusted for by their method) but also in the age structure within a ward.¹ As few as 40 or 50 children aged 0-4 years live in some electoral wards, and so it would take only a few young families moving to the M4's "silicon corridor" for the calculation of expected numbers of cases to be seriously awry. The assumption of constant age structure within wards might be tested by comparing the assumed ward populations for 1972 with the 1971 census results.

Errors in estimating small area population denominators contributed to a previous mistaken

conclusion that nitrate in drinking water caused stomach cancer²; it would be a shame if the otherwise excellent paper by Dr Roman and co-workers were similarly flawed.

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- 1 Jessop EG. Electoral ward mortality analysis in Newcastle upon Tyne. *Public Health* 1983;97:285-9.
- 2 Davies JM. Stomach cancer mortality in Worktop and other Nottinghamshire mining towns. *Br J Cancer* 1980;41:438-45.

AUTHORS' REPLY.—The reliability of our estimates of population size, and hence of the expected number of cases of leukaemia, may be assessed by comparison with figures in the Office of Population Censuses and Surveys' report on cancer incidence and mortality in the vicinity of nuclear installations.¹ The Office of Population Censuses and Surveys used different methods from ours in estimating the population at risk and did use information from the 1971 census, as Dr Jessop suggests. It also considered larger geographical areas than we did and obtained comparable population data from the 1971 and 1981 censuses. In contrast, we could obtain only electoral ward population data from the 1981 census as ward boundary changes in 1974 made it impossible to use the 1971 census data.

Of the 143 electoral wards examined in our study, 75 (52%) are contained within the boundaries of the seven local authorities described in the Office of Population Censuses and Surveys' report as having at least one third of their population living within 10 miles of the Atomic Weapons Research Establishment at Aldermaston (41 of those 75 wards were also contained within the 10 km circles described in our report). The table compares data published by the Office of Population Censuses and Surveys for the seven local authorities over the 10 years 1971-80 with those that we calculated for the corresponding 75 wards over the nine years 1972-80. The fact that the Office of Population Censuses and Surveys' estimate of the number of children aged 0-9 years expected to develop leukaemia is similar to ours implies that its estimate of the population is also similar to ours. Furthermore, the Office of Population Censuses and Surveys' data support our finding of an excess incidence of childhood leukaemia in the vicinity of Aldermaston.²

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Incidence of leukaemia in children aged 0-9 in the local authority areas defined by the Office of Population Censuses and Surveys as having at least one third of their population living within 10 miles of the Atomic Weapons Research Establishment at Aldermaston

Data source	Time period	No of children with leukaemia		Incidence ratio* (95% confidence interval)
		Observed	Expected	
OPCS	1971-80	35	23.9	1.5 (1.0 to 2.0)
Roman <i>et al</i>	1972-80	31	22.1	1.4 (1.0 to 2.1)

*Ratio of observed to expected registrations.

SIR,—Dr Robin Russell Jones (28 March, *p* 835) cites an 11% increase in the incidence of leukaemia among children living in the vicinity of nuclear installations from data presented in a recent Office of Population Censuses and Surveys' report.¹ He does not mention that the authors discussed apparent biases in the cancer registration data and that there was no comparable general effect in the mortality figures for those living within an approximate 10 mile radius,^{2,3} though rates were raised for those living in the vicinity of specific installations.¹

Dr Russell Jones's reference to "extraordinary methods" used in the report (in order to group districts by distance from installations) suggests a misunderstanding of the scope of the study. It was undertaken using the only sets of data that are readily available for such a large scale investigation: the computer files of cancer registrations and deaths that have been prepared over the years by the Office of Population Censuses and Surveys. Between 1959 and 1980, the period covered by the study, address of residence at diagnosis or death was coded according to the local authority areas that existed before boundary reorganisation in 1974 (rural and urban districts and municipal, county, and London boroughs). Many of the rural districts covered fairly large areas, and the distribution of population in these areas was taken into consideration when districts were categorised according to the proportions of the population living within six, eight, and 10 miles of an installation.

The decision to take account of population distribution meant that, because of the dilution effect of more distant communities, some districts that included an installation or had communities living very close to an installation fell into the outer distance zones—that is, an individual district might contain an installation and yet have most of its population living more than eight miles away from the installation (an anomaly that is discussed in the report¹). The advantage of the method is that for the scientific core of the study (the consideration of cancer patterns around installations grouped by similarity of environmental impact or by length of time since the installation was opened) it permits a more accurate summation of populations that live with increasing proximity to nuclear installations and, therefore, the search for a dose-response effect. Results by individual district are also presented in the report (with the raw data available from the Office of Population Censuses and Surveys on microfiche or magnetic tape), which allow both the study of local patterns and the calculation of registration and mortality ratios for any combination of districts that other investigators might consider to be more appropriate.

During the course of work on the Office of Population Censuses and Surveys' study¹ increased incidences of or death rates for leukaemia in children have been reported in the close vicinity of certain installations in Britain using wards or parishes or postcodes as the basic unit of study.⁴ These local occurrences are being investigated further.^{1,5,6} A similar fine scale investigation (including all installations, all age groups, and all malignancies, as in the report from the Office of