Responsibility of the Scientist

SIR,—Sir George Pickering (16 October, p. 131) suggests that the widespread current mistrust of science by laymen is unjustified, that it is not science that does harm but the use to which scientific discovery is put, and that we need more science to remedy many of our troubles. I disagree.

I would make two points. First, science is indivisible, and must be judged as a whole; and lay people know this. It is certainly true that the medical branch of science has produced many benefits. It has helped, for instance, to conquer various of the older diseases; but against that must be set the major killing diseases of Western Man, broadly speaking produced by a way of life that science in its other manifestations, together with its sibling, technology, has made possible—lung cancer, ischaemic heart disease, widespread obesity, etc. These are the subject of much earnest scientific research, but none is devoted to querying our entire pathogenic way of life—because to query that would be to query science itself. There must be some decent bounds to scientific scepticism.

To take an even wider view of the science scene: Against our increased expectation of life (now, significantly enough, no longer increasing but falling) and against the "benefits" brought by, for example, the motor car, pesticides, and Concorde must be set over-population, pollution, the destruction of animal species, and our lunatic exploitation of natural resources, as well perhaps as the drug addiction in which some of the young seek a refuge from the world that the scientific approach and its congeners have created and in which our extra years have to be lived out. Science and its fruits must always be judged in this way—as a whole. And why should the layman assume it will change in the future?

My second point is similar. Just as all branches of science form an indivisible unity, so one cannot separate the individual "innocent" scientist from those who use his results—the piece of research from the use to which it is put. *Pace* Sir George, the scientist is not outside society, having private fun. Often, as in the case of the work that led to the first atom bomb, he has a very good idea of where his activities will or may lead. Sometimes, as well as being a scientist, he is a policy maker, or else he becomes one. The transition from the research bench to the board room is very common in industry, and a similar elevation is not unknown in academic fields.

Moreover, what Sir George describes as the scientist's prime goal, satisfaction of his curiosity, means knowledge. This is what society, which finances and encourages and contains the scientist—however "innocent," however non-mission-orientated he may think himself, ultimately expects from his research; for knowledge in the end means power ("*Nam et ipsa scientia potestas est*" was said by Francis Bacon, the founding father of modern science), and it is the acquisition of power that science is really about as far as society is concerned. The "pure" scientists are merely the tips of society's antennae, in no way separate. And in the case of the medical branch of science what seems to be at stake is the acquisition of an absolute power over life and death and disease: the power to prolong life indefinitely, or to take it in some cases, to

eliminate a disease entirely, to defer the ageing process, to transplant any organ, to extract a gene, or to insert a gene. Old-fashioned medicine used to be about something different—the provision of comfort and relief in sickness, and the easing of men into death when that became inevitable. It now uneasily co-exists with scientific medicine, sometimes in the same individual; and uneasily because science is always a means to power.

A Roman centurion, having been stationed for some time in the Libyan desert, wrote on stone: "I have learnt and pondered this thought—in life there are two pursuits, love and power, and no man can have both." Here, I suggest, in the antithesis of love and power, is the great dilemma of medicine today; and, writ a little larger, it is also the dilemma of modern Western Man. In his confrontation of that dilemma I think the non-scientific layman shows more wisdom than Sir George Pickering will allow.—I am, etc.,

S. BRADSHAW

How Caple,
Hereford**Cervical Cytology Form**

SIR,—In your report of the General Medical Services Committee held on 21 October (*Supplement*, 30 October, p. 17) there is a paragraph in which concern is expressed about the passing on of confidential information about patients to the local health authorities, as laid down in paragraph 8 L.H.A.L. 33/71.

We would like to bring to attention another change of policy revealed in H.M.(71)79 and in the revised request/report form. In H.M.(71)79 (para 1) it is clearly stated that it is to be the responsibility of the pathology laboratory to see that the forms are correctly filled in, particularly as regards the National Health Service number, name of patient, and date of birth, and that if the form is sent to the N.H.S. Central Register incompletely filled in it will be returned, not to the originator but to the laboratory. This intention is reflected in the revised form in that there is now (as there was previously) no space for the pathologist's signature in the space provided for the cytology report (previously called the "Pathologist's Report") but the space for his signature is now at the bottom of the form, clearly indicating that he is to take the responsibility for all the information on the form and not just the report.

In our opinion this is an entirely new policy and we know of no responsible body representing pathologists that has approved of this decision. Taken together with the doubts expressed in the G.M.S. Committee it would appear that this new system has been set up without adequate consultation of those who will be required to work it and we hope that action will be taken to have this whole matter reviewed.—We are, etc.,

B. W. MEADE
M. E. A. POWELLDepartment of Pathology,
Kingston and Long Grove Group Hospitals,
Kingston upon Thames,
Surrey

SIR,—Today I met the new cervical cytology form. It's even worse than usual and I am sure that the dubious benefits of bumbling

bureaucracy as set out in this massive questionnaire will act as a positive deterrent to many of us. For a long time now, general practice has been struggling to keep its chin above the rising tide of paper work and this bulbous horror will add a near final nail to the coffin.

They ask for co-operation and produce that! Fortunately my patients can still attend the near local authority clinics.—I am, etc.,

R. G. WATSON

Dartford,
Kent**Virus-free Blood**

SIR,—We fully agree with the comments of Dr. L. R. I. Baker and colleagues (6 November, p. 360) on the need to minimize transfusion to patients with chronic renal disease, and that when this must be given it should be restricted where possible to liquid-nitrogen-stored, washed red cells derived from donations which have been tested for the presence of the hepatitis-associated antigen and antibody. We also have set up a frozen red cell bank for this purpose in the South West Region.

Although during reconstitution the thawed red cells are washed almost free of leucocytes a small number remains, and it is not yet known whether these are sufficient to sensitize the recipient to HL-A antigens. We are, therefore, as suggested by Akerblom,¹ making use of the frozen red cell bank to lay down donations from potential transplant patients for subsequent auto-transfusion during maintenance dialysis or at the time of kidney transplantation, since this has the added advantage not only of completely eliminating the risk of sensitization to HL-A but also to red cell antigens.—We are etc.,

C. H. BERESFORD
I. D. FRASER
G. H. TOVEYNational Blood Transfusion Service,
Bristol

¹ Akerblom, O., Höglman, C. F., Jameson, S., and Wibell, L., *Lancet*, 1970, 2, 385.

Phenformin, Tetracycline, and Lactic Acidosis

SIR,—Although the role of phenformin as a cause of lactic acidosis remains controversial. I wish to report a case of fatal lactic acidosis which occurred in a hospitalized patient receiving phenformin and tetracycline, to suggest a possible interrelationship of these two drugs.

A 36-year-old female was admitted for control of diabetes which had been detected four years ago. A few days prior to admission she had developed a mild sore throat, cough, and a carbuncle in the right axilla. Urinalysis revealed four plus sugar and four plus acetone. Blood sugar was 221 mg/100 ml. Complete blood count, pH, and serum electrolytes were unremarkable. Twenty units of regular insulin, given on the night of admission, cleared the glycosuria and acetonaemia.

The day after admission the patient was placed on oral tetracycline 500 mg every four hours. Long-acting phenformin 50 mg twice a day was started on the fifth hospital day. Her stay remained uneventful until 2 a.m. of the 12th hospital day when she

awoke and vomited. Prochlorperazine 10 mg was given intramuscularly, which relieved her temporarily. By 5 a.m. she appeared delirious and respirations were 36, pulse 96, and blood pressure 150/90. Venous blood drawn at 7 a.m. revealed sodium 132 mEq/l., potassium 5.6 mEq/l., chloride 89 mEq/l., carbon dioxide content 4.0 mEq/l, pH 6.97, PCO₂ 15.6 mm Hg, and lactic acid 246 mg/100 ml. In spite of infusion of fluids containing dextrose and sodium bicarbonate, therapy with digoxin and hydrocortisone, she died at 5 p.m. Necropsy revealed severe pulmonary oedema and moderate, bilateral chronic pyelonephritis.

The patient had been receiving three grammes daily of tetracycline for 10 days and phenformin 100 mg daily for seven days, when suddenly a catastrophic episode intervened under observation in the hospital. Although spontaneous lactic acidosis occurs in diabetics not on phenformin, the frequency of case reports of lactic acidosis in patients on phenformin has prompted suggestions of a causal relationship. Tetracycline may have played a role in this patient by increasing the blood levels of phenformin through its catabolic effects in a patient with impaired renal function.—I am, etc.,

CHARLES K. TASHIMA

Saint Francis Hospital,
University of Hawaii School of Medicine,
Honolulu, Hawaii

Ganglion Cysts of Bones

SIR,—In discussing ganglion cysts of bones you state that the pathogenesis of ganglia is obscure (13 November, p. 380).

In 1938 Wilson, Eyre-Brook, and Francis¹ suggested that semimembranous bursae (which contain ganglion-like fluid) might become distended by reason of a valve-like opening into the knee joint. A similar aetiology for ganglia seemed likely but, as you rightly point out, defects in the joint capsule are difficult to substantiate by dissection and injection of ganglia with radio-opaque material fails to show a communication with the joint.²

However, failure to demonstrate flow from ganglion to joint is just what one would expect with a valve-like mechanism; what is needed is evidence of one way flow from joint to ganglion. This has now been convincingly demonstrated by Andrén and Eiken,³ who have been able to show communication between the wrist and adjacent ganglia in 37 out of 59 cases investigated by injecting radio-opaque fluid into the wrist joint under pressure.

There are strong theoretical arguments in favour of ganglia being "blow outs" through valve-like openings in joint capsules and tendon sheaths. They occur in characteristic anatomical sites, they are aggravated by movements and relieved by rest, they recur if excised without operating on the valve opening, and they often exhibit remarkable ramifications and burrow into tendons, nerves, and bones.

For many years I have operated successfully on ganglia on the basis of a valve aetiology. Cure can be always obtained by either closing the opening into the capsule or tendon sheath or, if this cannot be demonstrated, by excising the area of capsule underlying the ganglion. In suitable cases I am now taking this treatment to its logical conclusion by leaving the ganglia in situ and

operating only on the valve mechanism.—I am, etc.,

A. W. FOWLER

Bridgend General Hospital,
Bridgend, Glam

¹ Wilson, P. D., Eyre-Brook, A. L., and Francis, J. D., *The Journal of Bone and Joint Surgery*, 1938, **20**, 963.

² McEvedy, B. V., *British Journal of Surgery*, 1962, **49**, 585.

³ Andrén, L., and Eiken, O., *The Journal of Bone and Joint Surgery*, 1971, **53-A**, 299.

Submandibular Gland and Lingual Nerve

SIR,—The necessity for identification of the lingual nerve is a leitmotif in descriptions of removal of the submandibular gland in textbooks of operative surgery. That this particularly applies to removal of the gland for calculi is obvious from the facts of the anatomy of the lesion, the two sites at which calculi calling for the operation impact being at the hilum of the gland as it lies on the hyoglossus just below the lingual nerve, and in the posterior part of the extra-glandular duct at or close to the point at which it is crossed by the lingual nerve. The nerve is 3-4 mm wide, and only rarely are adhesions so dense as to render its identification unduly difficult. In recent years, however, the idea has obtained a certain currency in Britain that it is unnecessary to identify the lingual nerve if the gland is being removed for calculi. The object of this letter is to emphasize that this is a dangerous and mistaken idea, and to try to trace how it has arisen.

Its proximate source would appear to be a misinterpretation of an account of the operation in Farquharson's *Textbook of Operative Surgery*,¹ a popular work among candidates for the final Fellowship. A short paragraph describing the operation terminates with the sentence: "Related to the deep surface of the gland, between it and the hyoglossus, are the hypoglossal and lingual nerves; these should be identified and safeguarded." This is clear and categorical, but the next paragraph continues: "In inflammatory and calculous disease, only the superficial part of the gland requires to be removed. In malignant disease the deep part of the gland extending forwards between the mylohyoid and hyoglossus should be dissected out and included in the excision."

This latter paragraph is an almost literal transposition of a similar paragraph in the first edition of Aird's *A Companion to Surgical Studies*,² which seems to be the original source. The paragraph is capable of being misinterpreted in two ways. The first is in relation to the word "superficial." The "superficial part of the submandibular gland" as anatomically defined is the whole of the gland with the exception of the small portion running forwards deep to the mylohyoid, which is anatomically defined as "the deep part." In both Aird and Farquharson such anatomical details as are given make it clear that they are using the terms as anatomically defined, and that they are not advising a blind cutting through the substance of the superficial part. Again, careful reading of the paragraph in which the warning on the lingual nerve occurs makes it obvious that it is removal of only the anatomical superficial part which is being described, no mention of the deep part being made or implied.

I am writing to the late Mr. Farquharson's

publishers suggesting that in the next edition the pseudo-qualifying paragraph in question should be omitted. Quite apart from its potentialities for misinterpretation, it is on the one hand unsound on the treatment of calculi, and on the other irrelevant to the realities of malignant disease. Exposure, and preferably also removal, of the deep part of the gland is necessary to deal adequately with calculi impacted in the posterior part of the extra-glandular duct, and is desirable, because of the increased access thus provided, for calculi impacted at the hilum of the gland.

In the treatment of the rare malignant disease of the submandibular gland, preservation of the lingual nerve becomes of secondary importance. In fact, since the commonest variety primarily affecting the gland is adenoid cystic carcinoma which has a predilection for spreading along nerve sheaths, resection of the nerve may actually be indicated.—I am, etc.,

DAVID PATEY

London W.1

¹ Farquharson, E. L., *Textbook of Operative Surgery*, 4th edn. Edinburgh, Livingstone, 1969, p. 408.

² Aird, I., *A Companion to Surgical Studies*. Edinburgh, Livingstone, 1949, p. 255.

Antileprosy Drugs

SIR,—May I comment on the letters of my good friends, Dr. A. S. Garrett (30 October, p. 300), and Dr. W. H. Jopling (6 November, p. 366) regarding the occurrence of psychosis in leprosy patients under treatment with dapsone? As so frequently happens, when two knowledgeable and sincere persons disagree, both may be right.

It is undeniable that despite social pressures and attitudes, and despite the emotional shock occasioned by the diagnosis of leprosy, psychotic manifestations were quite rare in the developing countries in the pre-sulphone days among leprosy patients undergoing treatment in institutions or at rural dispensaries with the pure hydroncarpus or chaulmoogra oil or their derivatives or esters (iodized or not), or with such salts as sodium gynocardate. It is also true that the widespread use of dapsone at daily doses of 300 or 200 mg (as advocated by Lowe and others), was accompanied by a high incidence of dose-related toxic phenomena in the skin (notably exfoliative dermatitis) and other organs, and of acute psychotic episodes. When, in the earlier '50's, the dose of dapsone in general use was reduced to 100 mg a day, the frequency of acute psychosis diminished. These observations were made in several countries, and in typically rural areas where patients were not taking other drugs or confined in institutions.

A very small proportion of patients, probably less than 1 in 1,000, under treatment for leprosy with even lower doses of dapsone, suffer from insomnia, delusions, and hallucinations, sometimes with manic or suicidal tendencies. When dapsone is stopped the mental state slowly returns to normal. If dapsone is given again—even in very small doses—the psychotic symptoms recur; if a placebo of similar appearance is given, nothing happens.

The 12 leprosy patients reported by Jopling¹ as having mental disturbances (including six who "developed signs of frank schizophrenia, requiring admission to a mental hospital") among 125 admissions to