know will not be questioned rather than try to specify (to the best of their knowledge and belief) the terminal illness and its antecedent causes.

The second major sphere of error lies in the death certificate itself. The coders are bound by two sets of rules. The first is issued by the World Health Organisation and is of worldwide application. Some of the rules appear inappropriate to the needs of England and Wales, but in many developing countries many of those who die are not attended by a doctor. The second is a list of rules drawn up for the coders in the Office of Population Censuses and Surveys itself in order to minimise errors in the national mortality statistics. The coders can code only what is on the certificate, and as they have little medical knowledge they must adhere to the two sets of rules. Herein lies the third way in which error may arise.

The colleges' report suggested that the consultant's name should be stamped on the death certificate and that queries about the cause of death should be referred to him.¹ The Office of Population Censuses and Surveys will be exploring the implementation of this change. The consultant is much less likely to have "moved on" than his preregistration house officer, and not only will his reply be more informative but his interest in correct certification of his patients will doubtless be stimulated. His participation may, indeed, lead to more frequent submission of the "further information" which at present, despite completion of box B on the reverse of the death certificate, is rarely forthcoming. Proposals are being prepared for submission to the Secretary of State on the introduction of new certificates for use in stillbirths and deaths occurring during the first month of life.

Is it too much to hope that deans of undergraduate medical schools will ensure that death certification, the International Classification of Diseases, and the functions of the Office of Population Censuses and Surveys are included in the undergraduate syllabus and that postgraduate deans will promote similar instruction for general practitioners? The Working Party on the Medical Aspects of Death Certification would also like hospital staff to try to obtain necropsies on an additional 20% of patients dying in hospital. The Royal College of Physicians is planning a symposium on death certification for all the royal colleges later this year.

Finally, the Office of Population Censuses and Surveys will be sending to each newly qualified doctor in England and Wales a leaflet explaining how to complete a death certificate and the importance of its accuracy.

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Of cats and women

Congenital toxoplasmosis was first described in 1939; yet it is clinically so rare that we still do not know whether it is preventable or whether treatment is effective. Dr Susan Hall has analysed recent Public Health Laboratory Service returns and has found much lower rates of congenital toxoplasmosis than 10 years ago (p 453). Is this because the true incidence is falling, or are doctors failing to diagnose the condition?

Toxoplasma gondii is a protozoon parasite found throughout the world. The sexual cycle takes place within the intestines of cats, which excrete oocysts during acute infections. The oocysts are ingested by many animals, including mice and birds, and the parasites multiply within tissue cysts in these intermediate hosts. If the animal flesh is eaten by a cat the cycle is completed. Humans are infected either from ingesting oocysts or by eating uncooked meat containing the parasite.

Human infection can be shown by serological methods.¹ The Sabin-Feldman test requires live *T gondii* and is usually performed in Public Health Laboratory Service laboratories. The fluorescent antibody test uses toxoplasma antigen from mouse peritoneal exudates, is simpler, and may be carried out in ordinary laboratories. The two tests probably measure the same antibody, whose titre rises to a maximum about four months after infection and may persist for a year or more. In Britain about 1% of the population show seroconversion each year,²⁻⁴ so that the proportion of the population which is immune increases with age: it is about 30% in young adults. Infection is usually subclinical, though sometimes fever and swollen, tender cervical lymph nodes may persist for some weeks.

Apart from rare overwhelming infections in people with immunological deficiency disorders, toxoplasmosis causes most concern in the context of infections in young children. Relatively more is known about infection during pregnancy,

causing congenital toxoplasmosis, than during infancy. The classic triad of congenital toxoplasmosis, made up of hydrocephalus, choroiditis, and intracranial calcification, is rare; but the results of prospective studies suggest that many more infants, apparently unaffected at birth, later develop isolated clinical features such as choroiditis or neurological problems.

The incidence of congenital toxoplasmosis is difficult to state precisely because of this variable presentation and also because the level of human infection varies between urban and rural settings and across countries. In Britain about two or three women in every 1000 become infected during pregnancy, as judged by seroconversion. Nevertheless, we do not know accurately in how many of these pregnancies the babies become infected or how many of these children are clinically affected. In a London population of 3187 women studied by Ruoss and Bourne² seven women developed toxoplasmosis—but only one infant had a positive dye test, and none developed clinical congenital toxoplasmosis. In a Scottish study of 5576 pregnancies at risk Williams and her colleagues³ found two children with clinical toxoplasmosis (and a third with positive serological findings) from 15 women who had been infected during pregnancy. In the United States Kimball *et al*⁵ found two infected children in 2765 pregnancies, neither with clinical toxoplasmosis; and Alford *et al*⁶ screened 7500 children at birth, finding 10 with evidence of infection but only one with clinical toxoplasmosis.

Other information about congenital toxoplasmosis has come from referral centres where mothers or infants are investigated because a clinician has suspected the infection. A large series in Paris⁷ has been analysed according to the trimester of the mother's infection. The data suggest that clinical signs are more common with infection early in pregnancy and that about 30% of infections of the mother are transmitted to the child. But because the series combined screened with referred women and the infection rate was much higher than in British studies these data are a poor indication of the incidence in Britain.

Hall's review (p 453) of the Public Health Laboratory Service reports received by the Communicable Disease Surveillance Centre has the advantage of being national (for England, Wales, and Northern Ireland) but the disadvantage of collating information from laboratories rather than clinical care. The incidence might have been underestimated in either of two ways: by clinicians not recognising the possibility of infection in symptomatic children or by the laboratories not reporting low titre, but true, infections to the Communicable Disease Surveillance Centre. This recent laboratory identification rate was much lower than in a survey 10 years ago which used similar methods. At face value, this difference would suggest a fall in incidence; but it may simply reflect a change in clinical awareness. Either way, the current levels of reporting are much lower than the estimates of incidence from prospective studies.

What are the opportunities for prevention of congenital toxoplasmosis? There is no effective live vaccine. Fewer than one third of women of reproductive age in Britain are seropositive, and reducing the rate of infection in pregnant women will prove difficult unless society changes its attitudes towards cats. The alternative is secondary prevention by serological screening and early diagnosis of infection during pregnancy.⁸ This might be costly, and there has been no controlled trial to show whether treatment (with spiramycin) is effective; such a trial would need surveillance of many thousands of pregnant women to gain a reliable result.

Congenital toxoplasmosis is a rare cause of mental handicap

—being recorded in only one in 500 of all affected people—whereas Down's syndrome and birth injury together account for over half of all cases.⁹ We could assess the potential value of screening better if we knew more about the clinical course of toxoplasma infection in pregnancy. Paediatricians can help by keeping a high level of alertness and giving as full a clinical description as possible when seeking serological confirmation. The Public Health Laboratory Service needs to standardise the test dilutions at which the diagnosis is regarded as positive. And clinicians can help confirm—or reject—the diagnosis by remembering to submit follow up sera for testing.

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Myocardial infarction and the normal coronary arteriogram

Myocardial ischaemia occurs when there is an unfavourable balance between coronary blood flow and the metabolic requirements of the myocardium. If the obstruction to flow is sufficiently prolonged it will lead to myocardial infarction. Almost all patients with a myocardial infarct have severe obstructive coronary arterial lesions, and coronary arteriography in the early hours of infarction often shows superadded thrombus that may completely occlude the vessel. Among the few exceptions are the patients with a myocardial infarction occasionally seen in whom the coronary arteriogram fails to show evidence of coronary obstructive lesions. Although pathological data would suggest that some of these so called normal coronary arteries do indeed have diffuse atheromatous narrowing,¹ there is no doubt that myocardial infarction can occur in the absence of gross narrowing of the arteries.

Such myocardial infarction with angiographically normal or near normal coronary arteries is rare. The prevalence varies from 1% to 12% of patients according to the mode of investigation and the population studied.^{2,3} Recent prospective studies show that in patients surviving an acute myocardial infarction normal coronary arteries are found in about 1-3% of cases.^{4,5}