their sera of agglutinins against brucella antigen, it is easy to see that overdiagnosis is something that must constantly be guarded against. At present, the serological tests for brucellosis in any patient must not only be interpreted in the light of his symptomatology, but also with cognizance of the serological values obtained in the community in which he lives, and of his epidemiological history.^{1,2} Previous authors have been too ready to accept a history of symptoms obtained by questionnaire as evidence of clinical brucellosis when allied to positive serological tests, resulting in very high reported incidences of the disease in veterinary surgeons.^{6,7} Critical evaluation of each individual is necessary to give an indication of the true incidence of brucellosis in any occupationally exposed group.⁵—I am, etc.,

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** Dr. McDevitt's letter has been shown to Dr. Thrower, who makes the following reply.—Ed., B.M.J.

SIR,—All concerned with brucellosis will be gratified to see the final analysis of the survey made on veterinary surgeons in Northern Ireland. As might be expected, practitioners likely to be in frequent contact with infectious material revealed the effects of such contact in their sera more strikingly than those further removed from infection. After all, human contact with many infectious diseases does not necessarily mean that an infection becomes clinically manifest, and the intermittent nature of brucellosis can make it notoriously difficult to relate serology to symptoms.

There is good reason to believe that in England somewhat similar findings would result from a survey such as that made by Dr. D. G. McDevitt, because, as is generally known, the incidence of overt infections in veterinary surgeons is causing disquiet. The serological reactions to brucella infections in bovines, the natural reservoir of *Br. abortus*, would be more clear-cut than in man but for the widespread use of vaccines, and much attention has been given to this problem since the present campaign started to eliminate brucellosis from the United Kingdom.

Those of us who have seen the human disease in its various forms and its wide variation in symptomatology and acuteness appreciate how difficult it can be at times to make an absolute diagnosis, even with all laboratory aids. This is the chief reason why what can be such a disabling disease has never been notifiable—a pity, because even now no one knows how widespread it really is. It is to be hoped that the situa-

tions being uncovered during the eradication of the disease from bovines is in no way indicative of the position in human beings, in whom missed diagnosis is commoner than overdiagnosis even allowing for the fact that symptoms may be attributed mistakenly to *Br. abortus*. The number of these cases must be infinitely smaller than those in whom the disease is not diagnosed by failure to apply or to persist with whatever tests are available.—I am, etc.,

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Hyperpyrexia after Anaesthesia

SIR,—The case of hyperpyrexia after anaesthesia reported by Drs. Sheila Kenny and D. A. H. Rolfe (21 November, p. 492) differs in many respects from true malignant hyperpyrexia.

The inpatient could not be described as "fit and healthy" at the beginning of anaesthesia as is usual in malignant hyperpyrexia.¹ He was febrile with a large bowel anastomotic leak. No attempt appears to have been made to exclude other causes of hyperpyrexia during anaesthesia by blood culture, etc.² Malignant hyperpyrexia is rare over the age of 45 and this patient would be the oldest so far recorded.

A maximum recorded temperature of 105°F. (40.5°C.) is very low for true malignant hyperpyrexia. The mean maximum temperature in Britt and Kalow's series was 108°F. (42.2°C.).³ Halothane and suxamethonium are the two agents commonly incriminated as triggering malignant hyperpyrexia and neither was used in this case. Subsequent investigation of the patient and his relatives for raised serum creatine phosphokinase levels is not mentioned.

Finally, I would suggest that the lack of muscle rigidity in this case can be explained by the hypothesis that the rise in temperature was not from any abnormal muscle metabolism but from the surgical complication.—I am, etc.,

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Angina and Indomethacin

SIR,—I have noted on occasion that the anti-inflammatory drug indomethacin appears to aggravate anginal pain in some patients with this condition. Because this effect of the drug has not previously been reported in the literature, I carried out a pilot investigation with a view to determining whether in fact indomethacin could bring on or aggravate angina, and whether significant electrocardiographic changes were induced in patients taking this drug. The following is a preliminary report on the trial which is to be fully described elsewhere.

Dr. B. G. Wells, consultant cardiologist at the North Middlesex Hospital, London, kindly analysed the electrocardiograms.

Eleven patients (4 men and 7 women) aged 31 to 74 (median age 59) with various musculoskeletal disorders including two with rheumatoid arthritis, four of whom had a recent history of angina pectoris, had 12-lead electrocardiograms and were then instructed to take two 25 mg. capsules of indomethacin at night. The E.C.G.s were repeated after taking the drug for seven days when the patients were questioned as to development or worsening of anginal pain. Electrocardiograms after exercise were also taken before and after indomethacin in seven patients (it was not considered prudent to record after-exercise E.C.G.s in those who had a past or present history of angina of effort).

One patient who had angina for some months previously developed myocardial infarction four weeks after taking indomethacin. The other three angina patients did not observe any aggravation of cardiac symptoms. Of the seven patients with no history of angina one thought the drug gave her palpitations, but none complained of chest pain. One patient had a normal E.C.G. before indomethacin and developed ischaemic changes of moderate degree after taking the drug, one developed an infarct as already mentioned, five had minor E.C.G. changes of ischaemia before indomethacin and showed some aggravation of ischaemia in E.C.G.s taken while on indomethacin, and four patients showed no E.C.G. changes after taking the drug.

It appears from this short series that indomethacin can aggravate myocardial ischaemia in some patients, and though the occurrence of myocardial infarction in one patient with a history of angina may of course have been fortuitous, the possibility arises that this drug may predispose certain patients to infarction. A controlled trial is now in progress.—I am, etc.,

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Cardiff Infant Inflating Bag

SIR,—The Cardiff Infant Inflating Bag is widely used for neonatal resuscitation. When the description of the bag was first published¹ it was intended that since the bag would be mostly used by those relatively unskilled in resuscitation it would normally be used with a face mask (Rendell-Baker-Soucek mask). This determined the safety factors built into the bag.

We assumed that in those instances when the baby was intubated there would necessarily be a skilled operator present and that the little bag would be squeezed in a controlled manner. We have gained the impression that this does not always follow, and that a further safety factor is required to prevent any excessive pressure which might result from over-enthusiastic compression of the bag when resuscitating an intubated baby. The patient-outlet of the inflating valve has therefore been modified. A D-shaped slot has been cut along the internal bore of this outlet, providing an additional leak when the endotracheal