plications in adults, in whom it has been associated with a seronegative arthropathy,[5] which may or may not be accompanied by a rash. It may also produce aplastic crises in patients with chronic haemolytic anaemias such as hereditary spherocytosis and sickle cell disease.[6]

Practitioners concerned in antenatal care should therefore be alert to the possibility of parvovirus infection and its possible complications. Women complaining of a rash or arthropathy during pregnancy or who are in contact with a child with erythema infectiosum should be screened serologically for parvovirus infection. If infection is confirmed the fetus should be monitored by maternal serum α fetoprotein concentrations, which may predict fetal infection, and by ultrasound to detect fetal hydrops. Fetal blood sampling may also be of value in diagnosing fetal anaemia. Parvovirus is an appreciable cause of non-immune fetal hydrops and resultant fetal loss and women at risk should be screened for evidence of infections during pregnancy.

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1 Mortimer PP, Cohen BJ, Buckley MM, *et al.* Human parvovirus and the fetus. *Lancet* 1985;ii:1012.
2 Anand A, Gray ES, Brown T, Clewley JP, Cohen BJ. Human parvovirus infection in pregnancy and hydrops fetlis. *N Engl J Med* 1987;**316**:183-6.
3 Knott PF, Welply GAC, Anderson MJ. Serologically proved intrauterine infection with parvovirus. *Br Med J* 1984;**289**: 1660.
4 Carrington D, Gilmore DH, Whittle MJ, *et al.* Maternal serum alphafetoprotein—a marker of fetal aplastic crises during intrauterine human parvovirus infection. *Lancet* 1987;i:433-5.
5 Schneider AP, Rauhaus AP. Human parvovirus infection and seronegative like arthritis in adults. *Lancet* 1988;i:296.
6 Rao KRP, Patel AR, Anderson MJ, Hodgson J, Jones SE, Patison JR. Infection with parvovirus like virus and aplastic crisis in chronic hemolytic anemia. *Ann Intern Med* 1983;**98**: 930-2.

Cough and angiotensin converting enzyme inhibitors

SIR,—For Dr Christine Bucknall and others (9 January, p 86) to say that cough associated with converting enzyme inhibitors may be a variant of the cough in asthma may cloud the issue of the pathogenesis of this adverse reaction. Our epidemiological data and the observations of others show that there is a significant preponderance of women in reports of cough with captopril and enalapril.[1-3] There is, however, no such sex difference with asthma, which suggests that the mechanism of production of cough with asthma and that with converting enzyme inhibitors are different. Unfortunately, the authors' study did not match controls and cases for sex, three of eight cases and six of nine controls being men.

The question of an association of angiotensin converting enzyme inhibitors with an asthma wheeze is interesting. Dr Bucknall and colleagues included in their study two patients with bronchial asthma who had reported increased wheeze after treatment with enalapril. These patients showed poor control of their asthma before the start of the rechallenge protocol, which started six and 12 weeks after they had stopped taking enalapril, and inhaled steroids had been introduced to control the increased wheeze. Our observations with the cough reaction were that symptoms cleared rapidly on withdrawal, the mean recovery time being 3·4 days for captopril and 5·5 days for enalapril.[1] These facts would suggest that either the exacerbation of wheeze and the cough reaction were produced by different mechanisms or the wheeze was not a reaction and represented a natural progression of bronchial asthma.

We are not convinced that angiotensin converting enzyme inhibitors include among their reactions exacerbation of bronchial asthma. The number of reports of cough so far received at this centre is 167, but we have only eight reports of wheeze or exacerbation of asthma, and we have described three asthmatic patients with cough but no increase in wheeze.[1] One study of seven patients with a cough attributable to converting enzyme inhibitors failed to show either reversible airflow obstruction or altered bronchial reactivity to methacholine.[4] Prostaglandins might have a role in the pathogenesis of the cough reaction,[1] and the observation that sulindac can clear or reduce the cough suggests that their contribution is a major one.[5] This again highlights the difference from bronchial asthma, which is not alleviated by non-steroidal anti-inflammatory drugs.

The incidence of a cough reaction is difficult to assess. Retrospective analyses of highly selected groups of patients—for example, those attending hypertension clinics—could produce an artificially high incidence. The incidence observed by us of 1·1% for captopril and 2·8% for enalapril[1] is probably low because these were reported cases only, but the patient population was unselected and we believe the monitoring method used revealed most clinically important cases. The incidence of cough is probably close to these figures.

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1 Coulter DM, Edwards IR. Cough associated with captopril and enalapril. *Br Med J* 1987;**294**:1521-3.
2 Webb D, Benjamin N, Collier J, Robinson B. Enalapril-induced cough. *Lancet* 1986;ii:1094.
3 Inman WHW. Enalapril-induced cough. *Lancet* 1986;ii:1218.
4 Town GI, Hallwright GP, Maling TJB, O'Donnell TV. Angiotensin converting enzyme inhibitors and cough. *N Z Med J* 1987;**100**:161-3.
5 Nicholls MG, Gilchrist NL. Sulindac and cough induced by converting enzyme inhibitors. *Lancet* 1987;ii:872.

Skin reactions and fever with indapamide

SIR,—We appreciate the interest that Drs D Kandela and D Guez (20 February, p 573) have shown in our short report (21 November, p 1313), but we find their letter misleading.

Our paper was a reviewed article and also follows the guidelines formulated by a workshop held under the auspices of Ciba-Geigy.[1] Unfortunately the references of skin reactions to indapamide that they mention do not satisfy these criteria. Even so, we wonder why they did not include them in their product datasheet.

Although we clearly stated that our figures were based on data obtained by voluntary reporting, and that no conclusions could be drawn about the comparative incidence of adverse effects to the different drugs mentioned in the article, Drs Kandela and Guez have wrongly understood that it was our purpose to do so. Their statement about the "highly comparable absolute numbers of cases: 16 and 15 respectively" (of skin reactions to indapamide and chlorthalidone) is irrelevant: chlorthalidone is more widely used in The Netherlands than indapamide, and the relative number of skin reactions to indapamide was much higher than that to chlorthalidone. The same applies to their interpretation of the figures on (hydro) chlorothiazide and frusemide. There is no logic in comparing absolute numbers without comparing prescription figures, and we have some difficulties in understanding their concept "absolute incidence."

Our conclusion than indapamide can cause skin reactions, sometimes serious, still holds, and these reactions were not mentioned in the datasheet. (The reporting of skin reactions to indapamide has increased considerably during the past year, and the World Health Organisation database now holds 266 such reports.)

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HIV, hepatitis B, and sexual behaviour

SIR,—The report by Dr Brian A Evans and others (13 February, p 473) raises disturbing ethical issues, and I was surprised to see no reference to these. Screening for antibodies to the human immunodeficiency virus (HIV) has been the subject of much debate both in your journal and elsewhere.

They state that 1115 women were included in their study and that all were offered the opportunity to ask for an HIV antibody test; 207 accepted the offer, but this means that 908 did not. Surely this means that they were implicitly refusing consent for the test. Despite this the test was carried out on all women.

While the screening test was carried out "independently of and separate from the clinical records," the results were clearly traceable to individual patients as in the case of the one positive result they obtained. This patient was then again counselled. It is difficult to see how they explained this repeat counselling to her, alone of 1115 women, without raising anxiety that there was something wrong. The counsellor presumably knew the result of the first test and would have been encouraging the patient to be tested. Despite this she explicitly declined the test.

Thus the doctors were left with a highly important test result for which permission had been refused. What are they then to do with the result? It has been connected to her case notes. How can the patient be protected from accidental revelation of the result in the future? Is it ethical to withhold the information from the patient when it is critically important to her sexual and reproductive behaviour and her subsequent clinical management?

These impossible dilemmas could have been avoided if patients had not been tested when consent had been withheld.

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SIR,—Dr Brian A Evans and colleagues indicate a low prevalence of antibodies to the human immunodeficiency virus (HIV) in a large cohort of heterosexual women attending a sexually transmitted disease clinic in London. This may at first be reassuring, and the authors conclude that heterosexual women are at low risk in London, but the risk of drawing this conclusion from inadequate, or at times absent, information is perilous.

Those women who were found to be infected were sexual contacts of high risk individuals, and the reasonable conclusion must be that the majority who remained uninfected had sexual partners who were not infected. Transmission was not shown because there was no risk. All this study shows, therefore, is a low prevalence in intravenous drug users and bisexual men—and we know this