mendation of the *British National Formulary*. Care in the community was started in different ways by non-government organisations—for example, through the indigenous midwife training in Sudan mentioned by Johnstone and McConnan[1] and the village health worker programmes in India.[4]

Even paediatric hospital practice in Britain is catching up with that in developing countries. Not many years ago in Britain parents were accepted in children's wards with some reluctance, but in developing countries parents have always been expected to be with their children to help with feeding and simple treatments and even to act as human hot-water bottles. In nutrition rehabilitation units in various developing countries, parental participation had a double purpose[5]: as parents helped to prepare nutritious meals from local foods they received a practical training in nutrition. Parents of children in Britain who have been diagnosed recently as having a chronic disease such as diabetes or cystic fibrosis, that requires lifestyle management would benefit from more involvement during their child's stay in hospital. Bengoa's concept of rehabilitation, pioneered in Latin America, is the best way for parents to learn while being involved in the recovery process.[5]

WILLIAM A M CUTTING
Senior lecturer in tropical child health

Department of Child Life and Health,
University of Edinburgh,
Edinburgh EH9 1UW

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Outreach programme in Kenya was based on extensive community participation

EDITOR,—Paul Johnstone and Isobel McConnan suggest that a wealth of knowledge about primary health care exists in developing countries and is now relevant to the NHS.[1] I learnt much from supporting a successful community based health care programme in sub-Saharan Africa for six years.

Chogoria Hospital provides complete medical care for 350 000 people in Meru district of Kenya. Having gained local credibility because of its effective curative services, the hospital developed an outreach programme for maternal and child health services, family planning, and curative medicine in the 1970s. By 1989 there were 30 village health clinics and a community network providing health education and contraceptives to villagers in their homes.[2]

The harnessing of the people's tradition of self help within the primary care programme was vital. The catchment area is served by 42 village health committees, 30 enrolled nurses, 500 volunteer family health workers, and 250 specially trained traditional birth attendants. The village health committees, selected by their communities, represent their constituents' interests and oversee the local clinics and volunteers. These committees and volunteers are active not only in expressing their health needs, prioritising developments, and monitoring the services but also in providing care.

The outreach programme is bound together by a careful monitoring system based on routinely collected service statistics. At all levels the workers' effectiveness at meeting preventive and curative targets is monitored, and help and retraining are given when necessary. Childhood immunisation neared 90%, child mortality decreased, and there was an appreciable decline in fertility associated with a high uptake of family planning services.[3] Fully integrated services, both preventive and curative, were made available (within walking distance) to everyone in the catchment area. They were acceptable to the community and developed to meet its needs and were affordable (or given free in cases of poverty). There was a constant information and education programme in the community, which used all existing public meetings.

At Chogoria in the 1980s the time was ripe for extensive participation by the community in many aspects of their own health care. With which aspects of health care can patients, families, communities, and the wider population be involved in Britain in the 1990s? In my general practice a local community has been involved in assessing and prioritising health needs, using a method from the developing countries.[4] We can learn from the many examples of good practice overseas.

SCOTT A MURRAY
Lecturer

Department of General Practice,
University of Edinburgh,
Edinburgh EH8 9DX

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Authors overestimate role of barefoot doctors in China

EDITOR,—Paul Johnstone and Isobel McConnan are right to draw readers' attention to primary care in developing countries and its relevance for our "primary care led NHS."[1] I wish to make three points.

In drawing attention to the appreciable reduction in childhood mortality in mainland China the authors overestimate the contribution that the barefoot doctors made in the late 1960s and the '70s (including the period of the Cultural Revolution). The considerable health gain made since 1949 can more accurately be attributed to a combination of factors, such as the absence of major national or civil wars, food rationing, the prevention of urban migration, full employment, and increasing equity between social classes and urban and rural areas. Other infrastructural developments also helped—namely, the development of primary education and the more specific public health policies, such as the movements to control four pests in the 1950s and improvements in basic hygiene, clean water, waste disposal, and universal immunisation.[2] The barefoot doctor movement was a potent political symbol used to tackle the rural-urban divide as well as challenge professional medical dominance and show the practical integration of traditional and Western medicine. It is difficult to gauge barefoot doctors' particular contribution, but rural health care always remained dependent on the secondary level hospital doctors, who have received three years' training, and the small county hospitals, local cooperative health insurance schemes, and public health "sanitarians."

In other studies of developing countries that are achieving good health at low cost the primacy of political will to achieve improvements in health and wellbeing is emphasised, as well as educational opportunities for women.[3] The three key findings from experiences in Sri Lanka, Kerala state in India, Costa Rica, and China are an equitable distribution of and access to public health services and health care, a uniformly accessible educational system, and adequate nutrition at all levels of society.

My final point is to clarify that socialism needs to be compared with capitalism as political and economic systems while the medical model needs to be compared with the social model (not socialism) when the determinants of health or disease are being explained.

TONY JEWELL
Consultant in public health medicine

2 Bury Road,
Stapleford,
Cambridge CB2 5BP

1 Johnstone P, McConnan I. Primary health care led NHS: learning from developing countries. *BMJ* 1995;311:891-2. (7 October.)
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Association of *Helicobacter pylori* infection with coronary heart disease

Study shows association between *H pylori* infection and hypertension

EDITOR,—P Patel and colleagues report an association between *Helicobacter pylori* and *Chlamydia pneumoniae* infections and coronary heart disease and plasma fibrinogen concentrations but did not find an association between *H pylori* infection and a history of hypertension.[1] We have studied the relation between *H pylori* infection and cardiovascular risk factors in hypertensive patients. We studied 124 hypertensive patients (61 men, mean age 53·2 (SD 14·7) years), of whom 19 had malignant phase hypertension, and 38 healthy controls (21 men, mean age 52·6 (14·6) years) to see whether they were positive for antibodies to *H pylori* (Oxoid helicobacter antibody kit; results were regarded as positive if *H pylori* IgG was ≥ 8 units/ml).

The prevalence of *H pylori* antibodies was higher in the hypertensive patients (106/124 (85%)) than in the controls (25/38 (66%)) ($\chi^2=7{\cdot}291$, df=1, P=0·007). There was no significant excess of seropositivity among the patients with malignant phase hypertension (13/19 (68%), $\chi^2=0{\cdot}0446$). We could not find any significant differences in mean age, blood pressures, white cell count, plasma viscosity, serum cholesterol, serum triglycerides, or plasma fibrinogen in the hypertensive patients who were positive for *H pylori* antibodies compared with those who were negative. Mean body mass index was, however, significantly higher in the patients who were positive (table 1). There was no

Table 1—*Cardiovascular risk factors in hypertensive patients, according to whether they were negative or positive for antibodies to H pylori. Figures are means (SD)*

	Negative	Positive	P value (unpaired *t* test)
Age (years)	52·3 (17·3)	53·5 (14·2)	0·58
Systolic blood pressure (mm Hg)	194·6 (27·4)	184·3 (29·6)	0·18
Diastolic blood pressure (mm Hg)	1151·1 (25·7)	104·4 (22·4)	0·13
White cell count ($\times 10^9$/l)	7·55 (2·73)	6·06 (3·46)	0·095
Serum cholesterol (mmol/l)	5·5 (1·8)	5·8 (2·5)	0·58
Serum triglycerides (mmol/l)	1·5 (0·7)	1·6 (1·2)	0·57
Plasma fibrinogen (g/l)	3·6 (1·0)	3·3 (1·0)	0·34
Body mass index (kg/m^2)	26·2 (3·2)	28·4 (4·2)	0·019

significant difference in the prevalence of the antibodies between the sexes (51/61 (84%) in the men versus 55/63 (87%) in the women respectively; $\chi^2=0{\cdot}341$) or between the patients with and without left ventricular hypertrophy (defined as a left ventricular mass index >134 g/m^2 in men and >110 g/m^2 in women on echocardiography) (65/78 (83%) versus 41/46 (89%) respectively; $\chi^2=0{\cdot}784$).

This preliminary study suggests a relation between *H pylori* infection and hypertension. We did not, however, find any associations with cardiovascular risk factors (including the presence of left ventricular hypertrophy) except for a weak relation with body mass index. Our findings are consistent with a study from a general practice in Gloucester, which showed a significant relation between *H pylori* infection and hypertension.[2] Our group has also reported data suggesting an association between the presence of antibodies to *C pneumoniae* and hypertension.[3] Thus our study and that of Barnes *et al*[2] are at variance with that of Patel and colleagues as there seem to be significant relations between both *H pylori* and *C pneumoniae* infections and hypertension.

We thank Ms Pauline Lowe, Dr J Zarifis, and Dr A Blann for technical help.

GREGORY Y H LIP
Lecturer in medicine
RICHARD WISE
Professor of microbiology
GARETH BEEVERS
Professor of medicine
Departments of Medicine and Microbiology,
University of Birmingham,
City Hospital,
Birmingham B18 7QH

1 Patel P, Kendall MA, Carrington D, Strachan DP, Leatham E, Molineaux N, *et al.* Association of Helicobacter pylori and Chlamydia pneumoniae infections with coronary heart disease and cardiovascular risk factors. *BMJ* 1995;311:711-4. (16 September.)
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Study confirms previous findings

EDITOR,—P Patel and colleagues' study substantiates our observation of an association between infection with *Helicobacter pylori* and coronary heart disease.[1] The first report from the group[2] prompted our retrospective serological study among patients presenting to the emergency care unit of our hospital.[3] Currently we are recruiting all patients admitted to the coronary unit of our hospital with acute myocardial infarction during the time that one of us (MTLR) is on duty there. Patients recruited from 1 July to 30 September 1995 are analysed here. In addition to being tested for antibodies, all patients were assessed for the presence of *H pylori* infection with the carbon-13 urea breath test, which was performed according to the European standard protocol.[4] Volunteer blood donors attending the blood bank of our hospital served as controls.

Twenty four (89%) of the 27 patients with myocardial infarction and 291 (47%) of the 619 blood donors were found to be infected with *H pylori*. As only two women had been admitted to the coronary unit (both of whom were positive for *H pylori* infection) we assessed only the male patients and compared them with blood donors of similar age. Table 1 gives the results; the Mantel-Haenszel weighted odds ratio was 4·4 (95% confidence interval 1·2 to 20·6).

The preliminary findings from this population case-control study confirm previous reports of an increased risk of acute myocardial infarction in men infected with *H pylori*. In our final analysis several potentially confounding variables will be taken into account. We believe, however, that a randomised study is needed to determine more clearly the effect of eradicating *H pylori* infection in patients at high risk of myocardial infarction.

Table 1—*Prevalence of H pylori infection among men with myocardial infarction and controls aged 40 and over. Figures are numbers (percentages)*

Age (years)	Patients with myocardial infarction	Controls
40-49	6/6 (100)	80/142 (56)
50-59	5/7 (71)	85/135 (63)
60-69	8/9 (89)	18/33 (55)
≥70	3/3 (100)	—
≥40	22/25 (88)	183/310 (59)

ANTONIO PONZETTO
Scientific coordinator,
department of experimental gastroenterology
MARIA TERESA LA ROVERE
Assistant professor, division of cardiology
PATRIZIA SANSEVERINO
Biologist, department of microbiology
Ospedale Molinette,
Turin,
Italy
FRANCO BAZZOLI
Professor of gastroenterology
University of Bologna,
Bologna,
Italy

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Association may not be causal

EDITOR,—P Patel and colleagues report a causal relation between infection with *Helicobacter pylori* and the presence of ischaemic heart disease.[1] We do not believe that this assertion is supported by the evidence.

Firstly, their findings were based on a cross sectional survey, and it is difficult to separate cause and effect from such surveys. This means that a person's status with regard to exposure to these infections at the time that he or she was included in the study may have had little to do with the exposure status at the time that the coronary heart disease process began. Coronary heart disease has a high prevalence in the population and may have its origins early in a person's life. The seroprevalence of antibodies to *H pylori* is also high and may be closely linked to childhood living conditions or adult social class. Unless a temporal relation can be established, the cause of the coronary heart disease remains uncertain.

Secondly, we found a reverse association from

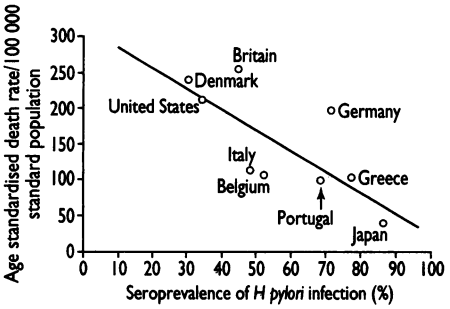

Fig 1—*Relation between prevalence of H pylori infection and standardised death rate from ischaemic heart disease in nine countries*

that claimed by the authors when we compared the seroprevalence of antibodies to *H pylori* reported by the EUROGAST Study Group[2] with the age standardised death rate from ischaemic heart disease in nine developed nations.[3] Figure 1 shows that deaths from ischaemic heart disease were negatively associated with the seroprevalence of antibodies to H pylori ($r=-0{\cdot}73$, $P<0{\cdot}05$).

The causation of disease is complex. Patel and colleagues found an association between *H pylori* infection and ischaemic heart disease, but a temporal relation has not been shown and the finding has not been consistent. We remain unconvinced that this association is causal.

QUENTIN D SANDIFER
Senior registrar in public health medicine
SU VUI LO
Consultant in public health medicine
Department of Public Health Medicine and Strategic Planning,
South Glamorgan Health Authority,
Cardiff CF1 3NW
GARETH CROMPTON
Professor of public health medicine
University of Wales College of Medicine,
Centre for Applied Public Health Medicine,
Cardiff CF1 3NW

1 Patel P, Mendall MA, Carrington D, Strachan DP, Leatham E, Molineaux N, *et al.* Association of Helicobacter pylori and Chlamydia pneumoniae infections with coronary heart disease and cardiovascular risk factors. *BMJ* 1995;311:711-4. (16 September.)
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Eradication of the infection on grounds of cardiovascular risk is not supported by current evidence

EDITOR,—P Patel and colleagues report having found an association between *Helicobacter pylori* infection and coronary heart disease in a cross sectional study.[1] As the association based on history alone failed to reach significance when adjusted for other risk factors this finding is based largely on the interpretation of resting electrocardiograms. The authors fail to provide data on the predictive value of left bundle branch block, T wave inversion, and ST depression in a similar population or to explore the sensitivity of their result to these values.

Furthermore, insufficient detail of the logistic regression is given for readers to determine which groups were contrasted and how the group with both *H pylori* and *Chlamydiae pneumoniae* infections was treated in the analysis. If the latter group was included in the groups with a single infection the results could be misleading and adjustment for the other infection by an additive covariate may be inadequate. Also, generalised linear interactive modelling is known to give inaccurate results for logistic regression when there are few results in a particular group. There were only 11 patients in the group with electrocardiographic abnormalities but no *H pylori* infection and at least 14 covariates to be adjusted for simultaneously.

We have evaluated a near patient test for *H pylori* infection (Helico G test) in six general practices in Birmingham.[2] A total of 311 patients were tested, having been recruited from patients aged over 50 who were attending the practices; their medical histories were recorded and validated against their general practice records. The prevalence of angina or myocardial infarction was 13% (41/311). Twenty six (63%) of the 41 patients with a history of ischaemic heart disease were positive for *H pylori*, compared with 198 (73%) of the 270 without a history of the disease. Our results, based on history alone, therefore do not support the findings of Patel and colleagues.

Patel and colleagues have attempted to provide evidence of a causal link. When two common conditions that have known confounding variables[3]