- 1 Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. *N Engl J Med* 1986;315:81-6.
- 2 Blair E, Stanley FJ. Intrapartum asphyxia: a rare cause of cerebral palsy. *J Pediatr* 1988;112:515-9.
- 3 Torfs CP, van den Berg BJ, Oechsli FW, Cummins S. Prenatal and perinatal factors in the aetiology of cerebral palsy. *J Pediatr* 1990;116:615-9.
- 4 Niswander K, Henson G, Elbourne D, Chalmers I, Redman C, Macfarlane A, et al. Adverse outcome of pregnancy and the quality of obstetric care. *Lancet* 1984;ii:827-31.
- 5 Nelson KB. What proportion of cerebral palsy is related to birth asphyxia? *J Pediatr* 1988;12:572-4.
- 6 Blair E, Stanley FJ. Intrauterine growth and spastic cerebral palsy. 1. Association with birth weight for gestational age. *Am J Obstet Gynecol* 1990;162:229-37.
- 7 Naeye RL, Peters EC, Bartholomew M, Landis JR. Origins of cerebral palsy. *Am J Dis Child* 1989;143:1154-61.
- 8 Johnson A, King R. A regional register of early childhood impairments: a discussion paper. *Community Med* 1989;11:352-63.
- 9 Royal College of Obstetricians and Gynaecologists. *Working party on resuscitation of the newborn*. Part 1. London: RCOG, 1989.
- 10 Macdonald D, Grant A, Sheridan-Pereira M, Boylan P, Chalmers I. The Dublin randomized controlled trial of intrapartum fetal heart rate monitoring. *Am J Obstet Gynecol* 1985;152:524-39.
- 11 Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. *Arch Neurol* 1976;33:696-705.
- 12 Breslow NE, Day NE. *Statistical methods in cancer research*. Vol 1. *The analysis of case control studies*. Lyon: International Agency for Research on Cancer Scientific Publications, 1980. (No 32.)
- 13 Schlesselman JJ. *Case-control studies; design, conduct, analysis*. Oxford: Oxford University Press, 1982.
- 14 Epidemiological Resources Incorporated. *EGRET: epidemiological graphics, estimation and testing*. Seattle, Washington: Epidemiology Resources, 1989.
- 15 Office of Population Censuses and Surveys. 1984-1987 infant and perinatal mortality. *OPCS Monitor* 1988;DH3.
- 16 Paneth N, Fox HE. The relationship of Apgar score to neurologic handicap; a survey of physicians. *Obstet Gynecol* 1983;61:547-50.
- 17 Nelson KB. Relationship of intrapartum and delivery room event to longterm neurologic outcome. *Clin Perinatol* 1989;16:995-1007.
- 18 Bryce R, Stanley F, Blair E. The effects of intrapartum care on the risk of impairments in childhood. In: Chalmers I, Enkin M, Keirse MSNC, eds. *Effective care in pregnancy and childbirth*. Oxford: Oxford University Press, 1989:1313-21.
- 19 Myers RE. Two patterns of perinatal brain damage and their conditions of occurrence. *Am J Obstet Gynecol* 1972;116:246-76.
- 20 Stanley FJ, Watson L. Trends in perinatal mortality and cerebral palsy in Western Australia: 1967-1985. *BMJ* 1992;304:1658-64.
- 21 Hagberg B, Hagberg G, Olow I, van Wendt L. The changing panorama of cerebral palsy in Sweden. *Acta Paediatr Scand* 1989;78:283-90.
- 22 Gilles FH. Neural damage: inconsistency during gestation. In: Gilles FH, Leviton A, Dooling EC, eds. *The developing human brain, growth and epidemiologic neuropathology*. Boston: Wright, 1983:227-43.
- 23 Sims ME. Brain injury and intrauterine death. *Am J Obstet Gynecol* 1985;151:721-3.
- 24 Richmond S, Niswander K, Snodgrass A, Wagstaff I. The obstetric management of fetal distress and its association with cerebral palsy. *Obstet Gynecol* (in press).
- 25 Freeman JM, Nelson KB. Intrapartum asphyxia and cerebral palsy. *Pediatrics* 1988;82:240-9.

(Accepted 23 November 1993)

Relation between infection with *Helicobacter pylori* and living conditions in childhood: evidence for person to person transmission in early life

P M Webb, T Knight, S Greaves, A Wilson, D G Newell, J Elder, D Forman

Abstract

Objectives—To relate the prevalence of infection with *Helicobacter pylori* in adults to their living conditions in childhood to identify risk factors for infection.

Design—Prevalence study of IgG antibodies to *H pylori* (>10 µg IgG/ml, determined by enzyme linked immunosorbent assay (ELISA)) and reported living conditions and other socioeconomic factors in childhood.

Setting—Three factories in Stoke on Trent.

Subjects—471 male volunteers aged 18 to 65 years.

Main outcome measures—Seroprevalence and variables in childhood.

Results—Seroprevalence of *H pylori* increased with age (22/74 (29.7%) at <30 years v 29/46 (63%) at 55-65 years; $P < 0.001$ for trend) and was related to manual occupation (14/65 (21.5%) for non-manual v 162/406 (39.9%) for manual; $P = 0.003$). After data were adjusted for age and occupation subjects from large families, whose childhood homes were crowded or who regularly shared a bed in childhood, were significantly more likely to be seropositive (adjusted odds ratio (95% confidence interval) 2.15 (1.41 to 3.30) for crowding and 2.13 (1.38 to 3.30) for sharing a bed), but there was no relation with possession of a bathroom, inside toilet, refrigerator, or household pets in childhood.

Conclusions—Close person to person contact in childhood is an important determinant of seroprevalence of *H pylori* in adulthood, suggesting that the infection is transmitted directly from one person to another and may be commonly acquired in early life.

Introduction

Infection with *Helicobacter pylori* has been established as a major cause of chronic gastritis.¹ It also plays an important part in the development of peptic ulcer disease^{2,3} and has been implicated in the process of gastric carcinogenesis.⁴ Around half of adults may be

infected in developed countries, while this figure could be as high as 90% in developing countries,⁵ yet little is known about the way in which the infection is transmitted. No reservoir of organisms has been identified outside the human stomach, and, although reports have suggested that *H pylori* can be isolated from faecal material⁶ and dental plaque,⁷ these reports have yet to be confirmed.

Infection tends to cluster in families^{8,9} and in people living in crowded^{10,11} or closed environments,¹²⁻¹⁴ suggesting that person to person contact plays an important part in transmission. Infection has also been associated with poor hygiene,¹⁵ and it has been suggested that acquisition may commonly occur in childhood.¹¹

We aimed to relate infection with *H pylori* in adults to their living conditions in childhood by using the presence of circulating antibodies to *H pylori* as a marker of infection. Of particular interest was the effect of sharing a bed in childhood, previously reported by Galpin *et al*,¹⁰ and the possibility that this might explain some of the observed relations with overcrowding and lower socioeconomic status.

Data were collected as part of a larger study of gastritis in Stoke on Trent.

Subjects and methods

Male employees at two factories in Stoke on Trent and men attending a blood donation session at a third factory were invited to participate. Volunteers completed a questionnaire that included detailed questions about their living conditions during two periods of childhood (below 5 years and between 5 and 16 years) before attending an appointment when the information was checked for completeness and a blood sample (10 ml) was collected by venepuncture.

Serum samples were stored below 4°C until they could be transferred to a -40°C freezer at the end of each day. Samples were assayed for antibodies to *H pylori* by using an established enzyme linked immunosorbent assay (ELISA),¹⁶ with a cut off point

Imperial Cancer Research Fund, Cancer Epidemiology Unit, Gibson Building, Radcliffe Infirmary, Oxford OX2 6HE

P M Webb, graduate research student

D Forman, principal scientist

Department of Surgery, Postgraduate Medicine, University of Keele, Stoke on Trent

T Knight, research fellow
S Greaves, research technician
A Wilson, research dietitian
J Elder, professor of surgery

Central Veterinary Laboratories, New Haw, Weybridge KT15 4NB
D G Newell, head of applied and molecular immunology unit

Correspondence to: Ms Webb.

BMJ 1994;308:750-3

of greater than 10 µg IgG/ml indicating positivity. This method has been shown to give a sensitivity of 96% and specificity of 93%.¹⁷

Each subject's occupation and that of their father when the subject was aged about 10 years was classified as non-manual (registrar general's class I, II, or III non-manual) or manual (III manual, IV, or V) according to the classification of occupations (1980) of the Office of Population Censuses and Surveys.¹⁸ To assess the effects of crowding an index was calculated for each subject as the ratio of the number of people in the household when they were young to the number of living rooms in the house.

TABLE I—Numbers (percentages) of subjects seropositive for *Helicobacter pylori* by age and occupation

Age (years)	Subjects with non-manual occupation	Subjects with manual occupation	Total
<30	1/7 (14.3)	21/67 (31.3)	22/74 (29.7)
30-34	3/27 (11.1)	37/127 (29.1)	26/81 (32.1)
35-39			14/73 (19.2)
40-44	5/17 (29.4)	57/129 (44.2)	30/83 (36.1)
45-49			32/63 (50.8)
50-54	5/14 (35.7)	47/83 (56.6)	23/51 (45.1)
55-65			29/46 (63.0)
Total	14/65 (21.5)	162/406 (39.9)	176/471 (37.4)
χ ² For trend	3.4 (P=0.07)	15.8 (P<0.001)	20.6 (P<0.001)

TABLE II—Seroprevalence of *Helicobacter pylori* and socioeconomic variables

Variable	No of subjects	Seropositive subjects			
		No (%)	P value	Age adjusted (%)	P value
Manual occupation:					
No	65	14 (21.5)		22.0	
Yes	406	162 (39.9)	0.003	39.8	0.004
Father's occupation manual:					
No	47	9 (19.1)		19.4	
Yes	393	151 (38.4)	0.007	38.4	0.006
No of siblings:					
0	58	15 (25.9)		27.1	
1	117	35 (29.9)		30.6	
2-3	171	57 (33.3)		33.5	
≥4	125	69 (55.2)	<0.001*	52.6	<0.001*
Crowding (people per room):					
0-0.75	119	38 (31.9)		33.9	
1-1.0	186	54 (29.0)		28.8	
1-1.5	95	47 (49.5)		49.1	
>1.5	39	20 (51.3)	0.001*	49.1	=0.003*
Sharing bed/bedroom:					
Own bedroom	167	48 (28.7)		30.5	
Shared bedroom	135	42 (31.1)		32.8	
Shared bed	140	75 (53.6)	<0.001†	47.8	0.001†
Bathroom:					
No	76	39 (51.3)		42.0	
Yes	376	126 (33.5)	0.004	35.1	0.2
Inside toilet:					
No	98	48 (49.0)		40.3	
Yes	354	118 (33.3)	0.005	35.5	0.3
Refrigerator:					
No	218	102 (46.8)		39.3	
Yes	245	71 (29.0)	<0.001	34.7	0.2
Household pets:					
No	88	32 (36.4)		34.7	
Yes	373	141 (38.2)	0.08	38.2	0.5

*χ² Test for trend.

†χ² Test for heterogeneity.

We used the χ² test to compare the percentage prevalence of infection in different categories of the explanatory variables. Percentages were indirectly adjusted for age by using four age groups (<30, 30-39, 40-49, and 50-65 years), and unconditional logistic regression was used to calculate odds ratios and 95% confidence intervals.

Results

A total of 476 men volunteered for the study. This comprised 258/782 (33%), 138/431 (32%), and 80/113 (71%), of the eligible workforce in each factory. Serum samples were not available from five people so data were analysed for the remaining 471 men.

A total of 176 men (37%) were positive for antibody to *H. pylori*. Seroprevalence varied between the three factories (43% (111/257), 34% (47/138), and 24% (18/76), χ²=10.4, P=0.005), but these differences were of borderline significance after we adjusted for age and manual occupation (χ²=6.2, P=0.044). The data were combined for further analyses.

Subjects were aged between 18 and 65 years, and seroprevalence for *H. pylori* increased significantly with age (table I). Seroprevalence was significantly higher in manual workers than non-manual workers and in subjects whose fathers were manual workers compared with those whose fathers were non-manual workers. These effects were independent and remained significant after we adjusted for age (table II). The difference between occupation groups was already present in the youngest age group (table I), and the yearly increase in seroprevalence was similar in both manual and non-manual workers (0.8% and 0.9% per year, respectively).

Table II also shows the association between seroprevalence for *H. pylori* and the other variables studied. Seropositivity was significantly associated with number of siblings, crowding, sharing a bed (but not sharing a bedroom), and with the lack of a bathroom, indoor toilet, or refrigerator in childhood. There was no association with the possession of household pets. After we adjusted for age, the relations with possession of a bathroom, indoor toilet, or refrigerator in childhood became non-significant.

The relation with crowding was not linear; subjects from homes with more than one person per room were significantly more likely to be seropositive (67/134=50.0%) than subjects from less crowded homes (92/305=30.2%). This cut off point, which is also used by the Office of Population Censuses and Surveys to classify homes as overcrowded,¹⁹ was used for further analyses.

Table III shows adjusted odds ratios for the variables that were significantly associated with seropositivity, after adjustment for age, in table II. The effects of number of siblings, overcrowding, and sharing a bed remained highly significant after we adjusted for age

TABLE III—Adjusted odds ratios (95% confidence intervals) for seropositivity for *Helicobacter pylori* and socioeconomic variables

Variable	Odds ratios adjusted for			Reduced models (mutually adjusted)		
	Age*	Age and occupation†	Mutually adjusted‡	Siblings and crowding§	Siblings and sharing a bed§	Crowding and sharing a bed§
Age (per year)	1.05 (1.03 to 1.07)	1.05 (1.03 to 1.07)	1.03 (1.00 to 1.05)	1.03 (1.01 to 1.05)	1.04 (1.01 to 1.06)	1.03 (1.01 to 1.06)
Manual occupation (reference=non-manual)	2.51 (1.33 to 4.73)	2.51 (1.33 to 4.73)	2.21 (1.07 to 4.57)	2.21 (1.16 to 4.22)	2.18 (1.14 to 4.19)	2.07 (1.08 to 3.96)
Father—manual occupation (reference=non-manual)	2.68 (1.25 to 5.74)	2.24 (1.03 to 4.89)	2.05 (0.85 to 4.93)			
Siblings (per sibling) (reference=1 sibling)	1.26 (1.14 to 1.39)	1.26 (1.14 to 1.39)	1.09 (0.94 to 1.27)	1.16 (1.02 to 1.33)	1.21 (1.09 to 1.35)	
Crowding: >1/room (reference=≤1/room)	2.23 (1.46 to 3.40)	2.15 (1.41 to 3.30)	1.54 (0.87 to 2.75)	1.48 (0.86 to 2.54)		1.77 (1.11 to 2.83)
Shared bed (reference=own bed)	2.26 (1.47 to 3.48)	2.13 (1.38 to 3.30)	1.41 (0.83 to 2.40)		1.46 (0.89 to 2.38)	1.60 (0.98 to 2.60)

*Each variable adjusted for age (single year).

†Each variable adjusted for age (single year) and current manual occupation (no/yes).

‡Each variable adjusted for age (single year), current manual occupation (no/yes), and all other variables.

§Each variable adjusted for age (single year), current manual occupation (no/yes), and other variables in model.

and current occupation, while the effect of father's occupation was reduced. Because of the strong collinearity between these variables associated with childhood they all became non-significant when included in a single, fully adjusted model.

In an attempt to overcome this problem the three variables—number of siblings, overcrowding, and sharing a bed—were included in pairs in a multivariate model that also contained age and current occupation (table III). The effect associated with number of siblings remained significant when included with overcrowding or sharing a bed; this could, however, have arisen simply because this variable could be recalled more accurately. Inclusion of overcrowding and sharing a bed in the same model resulted in effects of similar size and significance.

The above results relate to the period when subjects were aged between 5 and 16 years, but similar effects were observed for the period below 5 years.

Discussion

The association between infection with *H pylori* and lower socioeconomic status is well known,²⁰ and recently infection has been related to specific aspects of socioeconomic status, including density of living,^{11,15} sharing a bed,¹⁰ and the lack of a hot water supply¹⁵ during childhood. These variables are closely inter-related, and it has not been possible to identify clearly those that might directly affect transmission of the organism.

In this study *H pylori* seropositivity was related to current and childhood socioeconomic status and to measures of overcrowding and close person to person contact. After adjustment for age there was no association with either a lack of refrigerated food storage or, in agreement with Whitaker *et al.*²¹ but in contrast to Mendall *et al.*¹⁵ with measures of poor hygiene. These latter variables and *H pylori* infection are all strongly correlated with age. It is therefore impossible to say whether the relations were confounded by age or if the age effect itself could have resulted from changes in living conditions over the past 60 years. In either case the persistence of the relations with measures of crowding after adjustment for age suggests that close person to person contact is more important for transmission of *H pylori* than poor hygiene or contaminated food. There was no evidence that infection could be acquired from household pets.

Because of the problems of collinearity it was not possible to separate the effects of number of siblings, overcrowding, and sharing a bed in childhood. Socio-economic status or family size as such, however, cannot be risk factors for infection but are probably markers for some other factor that directly affects transmission. Sharing a bed in childhood could be such a factor as the proximity of two people sharing a bed would allow either oral-oral or faecal-oral transmission of an organism such as *H pylori*.

In a study of healthy volunteer families with children, 68% of partners of index subjects seropositive for *H pylori* were also seropositive compared with only 9% of partners of seronegative subjects.²² In contrast, 17.3% of people attending an infertility clinic were seropositive but in only 6.6% of couples were both partners seropositive,²³ suggesting that children may be important for the transmission of *H pylori* in families. It is possible that children are not only more susceptible to infection than adults but are also able to transmit the infection to non-infected adults.

EFFECT OF AGE

The difference in prevalence between non-manual and manual workers was already apparent in the 20 to 29 year age group, and the change in prevalence above

this age was around 1% a year. This is consistent with data from China, where differences were also seen in the youngest age group (under 5 years) with a constant increase in prevalence of about 1% a year above this age.¹¹ The similar rate of change in adults of different social classes suggests two possibilities. Either risk factors for infection in adults are independent of class and therefore differ from those acting during childhood, or most infections may be acquired before adulthood and the observed increase in seroprevalence with age in adults could be predominantly a cohort effect. The latter possibility is supported by a study that compared the prevalence of antibodies to *H pylori* in stored serum samples collected in 1969, 1979, and 1989.²⁴ Changes in living conditions and trends towards a smaller family size over the past 50 to 60 years could account for the reduction in seroprevalence of *H pylori* in successive cohorts.

We collected data for two periods during childhood in an attempt to identify the age at which children might be most susceptible to *H pylori* infection. There was no difference between the effects observed for the two periods, suggesting that infection can be acquired throughout childhood. This is supported by the observation that subjects who had shared a bed both below the age of 5 and between the ages of 5 and 16 were more likely to be seropositive than those who had shared a bed only during one of the two periods (56.2% *v* 40.0%, *P*=0.06).

BIAS AND CONFOUNDING

In a study reliant on volunteers and the collection of retrospective data, the possibilities of self selection and recall bias cannot be excluded. Subjects were aware that the study would focus on stomach problems but were unaware of whether they were infected with *H pylori*. There was no association between seropositivity for *H pylori* and reported stomach problems, and it seems unlikely that, after adjustment for age, there would be any difference in recall between seropositive and seronegative subjects. The absolute prevalence of infection may differ from the general population, but this would not affect the validity of comparisons made within the population. In addition, the effect of bed sharing remained significant when subjects from the first two factories (where only 33% of the eligible workforce volunteered) were considered separately from those from the third factory (where 71% of a group of blood donors participated), suggesting that this effect is not likely to be restricted to this population.

Although number of siblings, overcrowding, and

Epidemiological implications

- Infection with *Helicobacter pylori* is associated with the development of chronic gastritis, peptic ulcer disease, and, probably, gastric cancer
- Half of the world population may be infected but little is known about how the organism is transmitted
- Subjects from larger families, overcrowded homes, or who had regularly shared a bed during childhood were more likely to be seropositive
- The similar increase in prevalence with age in both manual and non-manual workers was consistent with a cohort effect in which most infections were acquired in childhood
- Very close person to person contact in childhood—for instance, by sharing a bed—may provide a route for the transmission of *H pylori*

sharing a bed were strong risk factors for infection, a quarter of subjects who had no siblings, whose childhood homes were not crowded, and who did not regularly share a bed were seropositive. This implies that there are other risk factors for infection—for instance, when children come into close contact with other children, perhaps in nurseries or schools.

CONCLUSIONS

In conclusion, our results are consistent with the hypothesis that infections with *H pylori* are commonly acquired in childhood and that the increases in prevalence observed with age may be predominantly a cohort effect. They confirm previous associations between seropositivity and socioeconomic status,²⁰ overcrowding,^{11 15} and sharing a bed,¹⁰ suggesting that close person to person contact, such as in sharing a bed in childhood, may increase the risk of infection.

© Crown copyright reserved 1994.

- 1 Dixon MF. *Helicobacter pylori* and chronic gastritis. In: Rathbone BJ, Heatley RV, eds. *Helicobacter pylori and gastroduodenal disease*. 2nd ed. Oxford: Blackwell Scientific Publications, 1992:124-39.
- 2 Wyatt JL. *Helicobacter pylori*, duodenitis and duodenal ulceration. In: Rathbone BJ, Heatley RV, eds. *Helicobacter pylori and gastroduodenal disease*. 2nd ed. Oxford: Blackwell Scientific Publications, 1992:140-9.
- 3 Sobala GM, Axon ATR. *Helicobacter pylori*, gastric ulceration and the postoperative stomach. In: Rathbone BJ, Heatley RV, eds. *Helicobacter pylori and gastroduodenal disease*. 2nd ed. Oxford: Blackwell Scientific Publications, 1992:150-7.
- 4 Forman D. *Helicobacter pylori* infection and gastric carcinogenesis. *European Journal of Gastroenterology and Hepatology* 1992;4(suppl 2):S31-5.
- 5 Megraud F. Epidemiology of *Helicobacter pylori* infection. In: Rathbone BJ, Heatley RV, eds. *Helicobacter pylori and gastroduodenal disease*. 2nd ed. Oxford: Blackwell Scientific Publications, 1992:107-23.
- 6 Thomas JE, Gibson GR, Darboe MK, Dale A, Weaver LT. Isolation of *Helicobacter pylori* from human faeces. *Lancet* 1992;340:1194-5.

- 7 Krajden S, Fuksa M, Anderson J, Kempston J, Boccia A, Petrea C, et al. Examination of human stomach biopsies, saliva and dental plaque for *Campylobacter pylori*. *J Clin Microbiol* 1989;27(6):1397-8.
- 8 Mitchell HM, Bohane TD, Berkowicz J, Hazell SL, Lee A. Antibody to *Campylobacter pylori* in families of index children with gastrointestinal illness due to *C pylori*. *Lancet* 1987;ii:681-2.
- 9 Drumm B, Perez-Perez GI, Blaser MJ, Sherman PM. Intrafamilial clustering of *Helicobacter pylori* infection. *New Engl J Med* 1990;322(6):359-63.
- 10 Galpin OP, Whitaker CJ, Dubiel AJ. *Helicobacter pylori* infection and overcrowding in childhood. *Lancet* 1992;339:619.
- 11 Mitchell HM, Li YY, Hu PJ, Liu Q, Chen M, Du GG, et al. Epidemiology of *Helicobacter pylori* in Southern China: identification of early childhood as the critical period for acquisition. *J Infect Dis* 1992;166(1):149-53.
- 12 Berkowicz J, Lee A. Person-to-person transmission of *Campylobacter pylori*. *Lancet* 1987;ii:680-1.
- 13 Perez-Perez GI, Taylor DN, Bodhidatta L, Wongsrichanalai J, Baze WB, Dunn BE, et al. Seroprevalence of *Helicobacter pylori* infections in Thailand. *J Infect Dis* 1990;161:1237-41.
- 14 Hammermeister I, Janus G, Schamarowski F, Rudolf M, Jacobs E, Kist M. Elevated risk of *Helicobacter pylori* infection in submarine crews. *Eur J Clin Microbiol Infect Dis* 1992;11(1):9-14.
- 15 Mendall MA, Goggin PM, Molineaux N, Levy J, Toosy T, Strachan D, et al. Childhood living conditions and *Helicobacter pylori* seropositivity in adult life. *Lancet* 1992;339:896-7.
- 16 Steer HW, Hawtin PR, Newell DG. An ELISA technique for the serodiagnosis of *Campylobacter pyloridis* infection in patients with gastritis and benign duodenal ulceration. *Serodiagnosis and Immunotherapy* 1987;1:253-9.
- 17 Talley NJ, Newell DG, Ormand JE, Carpenter HA, Wilson WR, Zinsmeister AR, et al. Serodiagnosis of *Helicobacter pylori*: comparison of enzyme-linked immunosorbent assays. *J Clin Microbiol* 1991;29(8):1635-9.
- 18 Office of Population Censuses and Surveys. *Classification of Occupations*. London: Government Statistical Service, 1980.
- 19 Office of Population Censuses and Surveys. *Britain's households. Census guide*. Vol 4. London: HMSO, 1986.
- 20 Taylor DN, Blaser MJ. The epidemiology of *Helicobacter pylori* infection. *Epidemiol Rev* 1991;13:42-59.
- 21 Whitaker CJ, Dubiel AJ, Galpin OP. Social and geographic risk factors in *Helicobacter pylori* infection. *Epidemiol Infect* 1993;111:63-70.
- 22 Malaty HM, Graham DY, Klein PD, Evans DG, Adam E, Evans DJ. Transmission of *Helicobacter pylori* infection. Studies in families of healthy individuals. *Scand J Gastroenterol* 1991;26:927-32.
- 23 Perez-Perez GI, Witkin SS, Decker MD, Blaser MJ. Seroprevalence of *Helicobacter pylori* infection in couples. *J Clin Microbiol* 1991;29(3):642-4.
- 24 Banatvala N, Mayo K, Megraud F, Jennings R, Deeks JJ, Feldman RA. The cohort effect and *Helicobacter pylori*. *J Infect Dis* 1993;168:219-21.

(Accepted 12 November 1993)

Taping the patella medially: a new treatment for osteoarthritis of the knee joint?

Janet Cushnaghan, Conor McCarthy, Paul Dieppe

Abstract

Objective—To test the hypothesis that medial taping of the patella reduces the symptoms of osteoarthritis of the knee when the patellofemoral joint is affected.

Design—Randomised, single blind, crossover trial of three different forms of taping of the knee joint. Each tape (medial, lateral, or neutral) was applied for four days, with three days of no treatment between tape positions.

Subjects—14 patients with established, symptomatic osteoarthritis of the knee and both clinical and radiographic evidence of patellofemoral compartment disease.

Main outcome measures—Daily visual analogue scale ratings for pain; patients' rating of change with each treatment; and tape preference.

Results—Medial taping of the patella was significantly better than the neutral or lateral taping for pain scores, symptom change, and patient preference. The medial tape resulted in a 25% reduction in knee pain.

Conclusion—Patella taping is a simple, safe, cheap way of providing short term pain relief in patients with osteoarthritis of the patellofemoral joint.

Introduction

Osteoarthritis of the knee is a major cause of pain and disability in older people.^{1 2} There is no specific treatment to modify the disease; current treatment is

aimed at reducing symptoms and improving function.³ Analgesic and anti-inflammatory drugs are used widely, in spite of potential side effects and doubts about their efficacy.^{3 5} Some forms of physical therapy, such as quadriceps exercises, are known to be of benefit,⁶ and walking aids can improve function. Surgical procedures, particularly knee joint prostheses, are increasingly being used. However, most current treatments are both expensive and potentially dangerous.

Simple, inexpensive treatment is needed for common disorders such as knee osteoarthritis, which is not life threatening but can cause years of pain and handicap for a large number of people in the community. Inexpensive interventions that give patients some control over their symptoms are particularly attractive. If effective, they could reduce the financial burden of these patients as well as improving their quality of life.

Recent reports have emphasised the importance of the patellofemoral compartment in knee osteoarthritis.^{7 8} Disease of this part of the joint can cause severe pain, particularly when the patient is using stairs, squatting, or kneeling.⁹ Malalignment of the patella, with consequential abnormal force distribution on the lateral facet, is thought to be the cause of these symptoms. Taping of the patella to pull it medially, followed by quadriceps exercises, has recently been recommended for the treatment of young people with anterior knee pain arising from the patellofemoral joint (chondromalacia patellae).^{10 11} However, data from controlled clinical trials to support such recommendations have not been published. The aims of this study were, firstly, to evaluate the symptomatic benefit

University of Bristol
Rheumatology Unit, Bristol
Royal Infirmary, Bristol
BS2 8HW
Janet Cushnaghan, clinical
research assistant
Conor McCarthy, research
registrar
Paul Dieppe, ARC professor
of rheumatology

Correspondence to:
Mrs Cushnaghan.

BMJ 1994;308:753-5