deal of historical analysis can be undertaken while the introduction of the new NHS patient numbering system, which will solve the problem, is awaited.

The software to achieve this is fairly straightforward, and I will provide a copy of it to anyone who is interested.

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1 Radical Statistics Health Group. NHS "indicators of success": what do they tell us? *BMJ* 1995;310:1045-50. (22 April.)

Incidence of gastrointestinal bleeding in Italy is similar to that in Britain

EDITOR,—I was interested in the paper on the audit of acute upper gastrointestinal haemorrhage in the United Kingdom.¹ As current knowledge of the epidemiology of acute upper gastrointestinal haemorrhage comes from English speaking countries, I wish to draw attention to the results of a prospective, population based survey conducted in the Treviso area (north east Italy) a decade ago.² Treviso hospital is a general hospital with 1264 beds serving a population of about 270 000 inhabitants (232 000 aged over 15). During 27 months, from 1 October 1983 to 31 December 1985, 526 emergency admissions for acute upper gastrointestinal haemorrhage were recorded, while bleeding occurred in 61 patients already in hospital.

The table compares some of the findings of our unselected study with those of T A Rockall and colleagues. There are impressive similarities in the

Comparison between two studies on acute upper gastrointestinal haemorrhage. Figures are numbers (percentage) of patients

	Rockall and colleagues ¹	Loperfido et al ²
Year	1993	1983-5
Adult population	12 500 000	232 000
No of cases	4185	587
Mean age (years)	66	61
Haemorrhage in inpatients	584 (14)	61 (10)
Peptic ulcer	1448/4137 (35)	347/587 (59)
Varices	180/4137 (4)	81/587 (14)
Negative diagnosis	1014/4137 (25)	28/587 (5)
Overall incidence (No/100 000/year)	103	112
Mortality in emergency admissions	365/3473 (11)	68/526 (13)
Mortality in inpatients	191/578 (33)	23/61 (38)

overall incidence of acute upper gastrointestinal haemorrhage, proportion of cases of bleeding occurring in inpatients, and mortality. On the other hand, the distribution of sources of bleeding differs between the two populations investigated, the main difference concerning the high proportion of cases of bleeding in which the cause was undetermined in the English series (25% v 5% in our study). Since digestive endoscopy for acute upper gastrointestinal haemorrhage seems to have become routine, the finding that a quarter of the sources of bleeding were undiagnosed in the English series is surprising, even when we take into account that cases of bleeding of undetermined cause carried a high mortality (20%) and included similar proportions of emergency admissions and cases of haemorrhage in inpatients. The design of the large English study, involving 74 hospitals, and the advanced age of most of the patients may help to explain this peculiarity. Nevertheless, data on the performance of emergency endoscopy and on variability among areas might be an important clue.

Overall mortality in patients admitted after the onset of acute upper gastrointestinal haemor-

rhage outside hospital fell in our area from 12.9% (68/526) in 1983-5 to 3.8% (10/262) in 1993, and, specifically, mortality from bleeding oesophagogastric varices fell from 43% (35/81) to 11% (4/38) and mortality from bleeding peptic ulcer from 7.3% (23/313) to 1.3% (2/158). On the other hand, our experience agrees with the common one that the prognosis of upper gastrointestinal haemorrhage in inpatients, a very high risk group, is still poor.³

One question arising from the findings of the English series is how to transfer the advances in terms of improved outcome that have been achieved (at least for emergency admissions) at specialised centres into general hospital practice, even if the proportion of elderly patients continues to increase everywhere.

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1 Rockall TA, Logan RFA, Devlin HB, Northfield TC, on behalf of the steering committee and members of the national audit of acute upper gastrointestinal haemorrhage. Incidence of and mortality from acute upper gastrointestinal haemorrhage in the United Kingdom. *BMJ* 1995;311:222-6. (22 July.)

2 Loperfido S, Monica F, Vallenari A, Demattè P, Fabi F, Di Falco G, et al. Epidemiologia dell'emorragia del tratto digestivo superiore in una provincia italiana. *Giornale Italiano di Endoscopia Digestiva* 1988;11:45-56.

3 Loperfido S, Monica F, Maifreni L, Paccagnella A, Famà R, Dal Pos R, et al. Bleeding peptic ulcer occurring in hospitalized patients: analysis of predictive and risk factors and comparison with out-of-hospital onset of hemorrhage. *Dig Dis Sci* 1994;39: 698-705.

Vitamin D receptor genotype and bone mineral density

Evidence conflicts on link

EDITOR,—Two linkage studies of vitamin D receptor genotype and bone mineral density have reported different results. In Australian twins the vitamin D receptor gene was in strong linkage with bone mineral density.¹ In contrast, in American twins we found no evidence of linkage of the vitamin D receptor gene with bone mineral density.² We were therefore interested in the study in British twins to see if it sheds light on these contrasting findings.³ Unfortunately, it does not.

Hip bone mineral density (n=20 pairs) and spinal bone mineral density (n=23 pairs), measured by dual energy x ray absorptiometry, in relation to TaqI polymorphism (T=presence of restriction site) in discordant adult female dizygotic twins. Twin pairs are connected by a line. Mean absolute differences between pairs and their significance, tested by analysis of variance, are also shown

Despite the fact that there were no significant ($P < 0.05$) differences between dizygotic intraclass correlations in twins concordant and discordant for the vitamin D receptor gene at any of five skeletal sites, the authors conclude that bone mineral density is linked to the gene. The dizygotic intraclass correlation for bone mineral density of the spine, the site with strongest linkage in Australian twins, was 0.36 (SE 0.12) in concordant and 0.33 (0.14) in discordant British dizygotic twins. Furthermore, the direction of the difference between discordant dizygotic twins is not shown, and thus it is uncertain which allele associates with low or high bone mineral density. In dizygotic twins as individuals, the genotype tt has significantly lower adjusted bone mineral density than Tt or TT. However, 45% of these dizygotic twins had used oestrogen for a median of 24 months, and subjects of genotype tt (that is, those with the lowest bone mineral density) had lowest use (32%) and shortest median duration of use (12 months) of oestrogen, which in postmenopausal women leads to lower bone mineral density. Bone mineral density was corrected for use and duration of use of oestrogen, but it is not clear how this was, or indeed if it can be, done. It would be important to see an analysis without twins who had used oestrogen and to explain how data were corrected for use and duration of use of oestrogen.

The authors argue that their results contrast with ours because the number of discordant dizygotic twins in our study was small. Since our publication on 39 dizygotic pairs, 14 of whom were discordant for TaqI,² we have studied more dizygotic premenopausal twins not taking oestrogen, bringing our total number of dizygotic pairs to 53, 23 of whom are discordant for the vitamin D receptor gene. The larger sample supports our original conclusion of no linkage between the gene and bone mineral density. The dizygotic intraclass correlations for bone mineral density are higher, but not significantly, in discordant dizygotic pairs (dizygotic intraclass correlation: spine=0.59, hip=0.53) than in concordant dizygotic pairs (spine=0.44, hip=0.42). Moreover, the direction of differences in bone mineral density within twin pairs is random in relation to the vitamin D receptor gene (figure).

In summary, the British twin study shows no evidence of significant linkage between the vitamin D receptor gene and bone mineral density and corroborates both our original and our updated