improvement in gastritis histologically,¹²⁻¹⁴ which has been confirmed in this study. Moreover, recent reports suggest that treating patients with non-ulcer dyspepsia associated with *H pylori* with agents known to be active against the organism leads to appreciable improvement in symptoms.^{15,16} None of these studies, however, have concentrated on the effect of eradication of *H pylori* on symptoms of dyspepsia, and thus they cannot adequately explain the role of *H pylori* in the disorder.

In this study only patients found to have *H pylori* on culture and histological examination were analysed, and eradication of *H pylori* or otherwise was recorded four weeks after completion of treatment. This allows the effect of eradication of *H pylori* alone on symptoms of dyspepsia to be compared directly in a large group of patients from a single institution.

H pylori infection was eradicated after treatment in 41 of 83 patients, and, as might be expected from other treatment studies, a significant improvement in symptom score was noted. However, a similar proportion of patients (51%) remained infected after treatment and also experienced significant improvement in symptoms. Clearly, colonisation of the gastric antrum by *H pylori* is unlikely to be responsible for the symptoms experienced by this group of patients, and our findings strongly suggest that the improvement occurred by some other mechanism. It is also unlikely that this response occurred owing to a local effort of bismuth on the gastric mucosa as the antibiotics alone proved to be equally effective in reducing symptom scores. A placebo effect is thus likely, but this cannot be proved in the setting of this study.

As expected, eradication of *H pylori* and improvement in gastritis histologically were significantly correlated. However, improvement in symptoms proved to be independent of improvement of gastritis. Patients whose gastritis remained unchanged with treatment showed an improvement in symptoms similar to that of those whose gastritis improved. Thus the presence of chronic active gastritis, although strongly related to the presence of *H pylori* infection does not seem to bear a significant relation to the presence of symptoms.

In conclusion, we confirmed the close association of

H pylori infection with chronic active gastritis and that eradication of *H pylori* leads to improvement of this gastritis histologically. There seems to be no relation, however, between eradication of *H pylori* or such improvement of gastritis and improvement of symptoms of non-ulcer dyspepsia. Thus, although *H pylori* may have an important role in the pathogenesis of peptic ulcer disease, this short term study suggests that it is unlikely to do so in non-ulcer dyspepsia. Clearly, long term follow up of these patients is indicated to identify whether any future benefit of eradication of *H pylori* becomes apparent.

- 1 Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1984;i:1311-4.
- 2 Marshall B, Armstrong J, McGreiche D, Glancy R. Attempts to fulfil Koch's postulates for pyloric campylobacter. *Med J Aust* 1985;142:436-9.
- 3 Goodwin C, Armstrong J, Marshall B. Campylobacter pyloridis gastritis and peptic ulceration. *J Clin Pathol* 1986;7:206.
- 4 Coghlan JG, Gilligan D, Humphries H, McKenna D, Dooley C, Sweeney E, et al. Campylobacter pylori and recurrence of duodenal ulcers: a 12 month follow up study. *Lancet* 1987;ii:1109-11.
- 5 Adami HO, Aghasizadeh S, Lööf L, Nyberg A, Nyrén O, et al. The clinical diagnosis of "gastritis." *Scand J Gastroenterol* 1984;19:216-9.
- 6 Heatley RV, Rathbone BJ. Dyspepsia: a dilemma for doctors. *Lancet* 1987;ii:779-82.
- 7 Management of dyspepsia: report of a working party. *Lancet* 1988;i:576-9.
- 8 Marshall BJ. Campylobacter pylori: addressing the controversies. In Menge H, Gregor M, Tytgat GNJ, eds. *Campylobacter pylori*. Berlin: Springer-Verlag, 1988:235-45.
- 9 Barthel JS, Westblom TU, Havey AD, Gonzales F, Everett DE. Gastritis and campylobacter pylori in healthy asymptomatic volunteers. *Arch Intern Med* 1988;148:1149-51.
- 10 Morris A, Nicholson G, Lloyd G, Haines D, Rogers A, Taylor D. Seroprevalence of campylobacter pyloridis. *N Z Med J* 1986;99:657-9.
- 11 Morris A, Nicholson G. Ingestion of campylobacter pyloridis causes gastritis and a raised fasting pH. *Am J Gastroenterol* 1987;82:192-9.
- 12 Loffeld RJLF, Potters HVJP, Strobberingh E, Flendrig JA, van Spreenwel JP, Arends JW. Campylobacter associated gastritis in patients with non-ulcer dyspepsia: a double blind placebo controlled trial with colloidal bismuth subcitrate. *Gut* 1989;30:1206-12.
- 13 Rokkas T, Pursey C, Uzoehina E, Dorrington L, Simmons NA, Filipe MI, et al. Non ulcer dyspepsia and short term De-Nol therapy: a placebo controlled trial with particular reference to the role of campylobacter pylori. *Gut* 1988;29:1386-91.
- 14 Raus EAJ, Langenberg W, Houthoff HJ, Zanen HC, Tytgat GN. Campylobacter pyloridis associated chronic active antral gastritis. *Gastroenterology* 1988;94:33-40.
- 15 Borody T, Hennessy W, Daskalopoulos G, Carrick J, Hazell S. Double blind trial of De-Nol in non ulcer dyspepsia associated with campylobacter pyloridis gastritis [abstract 919]. *Gastroenterology* 1987;92 (suppl):1324.
- 16 Lambert JR, Dunn K, Borromeo M, Korman MC, Hanskey J. Campylobacter pylori: a role in non-ulcer dyspepsia. *Scand J Gastroenterol* 1989;160 (suppl):7-13.

(Accepted 16 August 1991)

Relation between Glasgow coma scale and the gag reflex

C Moulton, A Pennycook, R Makower

Accident and Emergency Department, Glasgow Royal Infirmary, Glasgow G4 0SF
C Moulton, FFARCS, registrar
A Pennycook, FRCSGLAS, registrar
R Makower, FRCSed, registrar

Correspondence to:
Dr Moulton.

BMJ 1991;303:1240-1

The airway is the foremost concern of those who treat acutely ill or traumatised patients. Avoiding hypoxia due to airway obstruction and the aspiration of vomitus are always priorities, especially in the accident and emergency department. Airway patency is normally maintained by a variety of protective and regulatory reflexes.

In the comatose patient (defined as a Glasgow coma score ≤ 8) these vital mechanisms may be compromised. The situation at higher levels of consciousness is unreported. In this prospective study we aimed to clarify the relation between the Glasgow coma scale and the gag reflex, which is the most easily and commonly tested airway reflex and the absence of which is recognised as one of the criteria for brain stem death.^{1,2}

Patients, methods, and results

The Glasgow coma score, gag reflex, and clinical details were collected for 111 patients (treated con-

secutively by the authors) requiring neurological observation in the accident and emergency department. The gag reflex was tested by pharyngeal stimulation and the response was recorded as being normal (light touch), attenuated (more vigorous stimulation required), or absent.

The table compares the Glasgow coma score and gag reflex in patients whose presentation was related to exposure to various pharmacological substances and in patients with a non-pharmacological cause for cerebral depression, usually head injuries. Both groups might have taken alcohol.

The gag reflex may be significantly attenuated, or absent, at all levels of the Glasgow coma scale. In the comatose patient (score ≤ 8) it is unlikely that the gag reflex will be normal. In more conscious patients (score > 8) the gag reflex was depressed in 64% (32/50) of patients who had been exposed to drugs but in only 8% (2/24) of head injured patients ($p < 0.05$, χ^2 test).

Comment

The Glasgow coma scale is widely accepted as an easily and accurately reproducible method of assessing conscious level. Many patients have a depressed conscious level of mixed origin, and separation of the individual components is impossible.³ A Glasgow coma score of 15 does not, however, distinguish between the

	Pharmacological cause for cerebral depression			Non-pharmacological cause for cerebral depression		
	Absent reflex	Attenuated reflex	Normal reflex	Absent reflex	Attenuated reflex	Normal reflex
Glasgow coma score:						
15	3	5	5			6
14		6	5			6
13	3	5	5			3
12	3	2	3		1	2
11		1				1
10	1	1				2
9		2			1	2
8	2	4	1			2
7	1	3			2	1
6	1	1			2	
5	2					
4	1	1		2	1	
3	5	1		3	1	
Range (Glasgow coma score)	3-15	3-15	8-15	3-4	3-12	7-15

alert, lively patient and the drowsy, lethargic one. Most of the patients with an impaired gag reflex at this level were drowsy and lethargic, and most had been exposed to a narcotic analgesic. Many of the others with a Glasgow coma score of 14 or 15 and an impaired gag reflex had been exposed to tranquillisers.

It is important to realise that the gag reflex and possibly the other protective reflexes of the airway may be compromised in the apparently conscious patient. This may not signify an increased risk of aspirational injury, but most standard textbooks make the assumption that it does.⁴ We believe that the gag reflex should be assessed independently of conscious level in the accident and emergency department and used as an indicator of an "at risk" airway.

It is often recognised that the comatose patient may require endotracheal intubation to protect the airway as well as to provide ventilatory support. We think that too little consideration may be given to the risks of airway compromise in the more conscious patient. Proper nursing care in the lateral position with effective, constant observation is imperative and gastric intubation to empty the stomach and reduce the risk of aspiration should also be considered.

1 Conference of Medical Royal Colleges and their faculties in the UK. Diagnosis of brain stem death. *BMJ* 1976;iii:1187-8.
2 Pallis C. ABC of brain stem death. *BMJ* 1982;285:1641-4.
3 Teasdale G, Jennett B. Assessment of coma and impaired consciousness. *Lancet* 1974;ii:81-4.
4 Yates DW, Redmond AD. *Lecture notes on accident and emergency medicine*. Oxford: Blackwell Scientific, 1985.

(Accepted 13 June 1991)

Pre-eclampsia: discordance among identical twins

James G Thornton, Joseph L Onwude

Department of Obstetrics and Gynaecology, St James's University Hospital, Leeds LS9 7TF
James G Thornton, MD, senior lecturer
Joseph L Onwude, MRCOG, senior registrar

Correspondence and requests for reprints to: Dr Thornton.

BMJ 1991;303:1241-2

Studies of relatives of patients with eclampsia in which only patients with proteinuria in their first pregnancies were included have shown a distribution of affected cases compatible with mendelian genetic models acting in the mother.^{1,2} These were compatible with both a single recessive gene with gene frequency of around 0.25 and with a dominant gene with penetrance of 0.5 and frequency of 0.14. This pattern is, however, not specific to conditions inherited in mendelian fashion, and other evidence such as twin studies should be sought before accepting a genetic aetiology.

Methods and results

Ninety nine adult female identical twin pairs who had both had at least one child were identified through the Birmingham twin registry in England and sent a postal questionnaire. They were asked if they had had either toxæmia, hypertension, pre-eclampsia, or eclampsia in their first viable pregnancy. Pairs replying in the negative were recorded as concordant unaffected. Further inquiry was made from the delivery hospitals and general practitioners if either twin recalled any of the above complications. Pre-eclampsia was defined as a blood pressure of more than 140/90 mm Hg on at least two occasions with proteinuria (0.25 g/l) when the booking and postnatal blood pressure were normal. Zygosity was based on the results of a standard twin questionnaire.³

The table summarises the results. All six cases with proteinuric hypertension were discordant, although in one pair non-proteinuric hypertension probably occurred in the other twin. Twin 1 had definite proteinuric pre-eclampsia but delivery records were not available for twin 2, who recalled that her blood pressure was raised throughout the later part of pregnancy but that she did not have proteinuria. It is now impossible to verify the diagnosis and we excluded

this pair from analysis. In another pair proteinuria was not documented in the scanty records available, but they did record that the affected twin was delivered at 33 weeks because of "fulminating pre-eclamptic toxæmia." Hospital details were not available for the unaffected twin, but she clearly recalled that the pregnancy and delivery had been uncomplicated. We included this pair as discordant. In summary, therefore, all five cases identified with adequate records of pre-eclampsia with proteinuria in their first pregnancy were discordant with their cotwin. No concordant affected twin pairs were identified.

Concordance for hypertension or pre-eclampsia in 108 pairs of adult identical twins

	No
Both unaffected	68
Both affected with non-proteinuric hypertension	4
Discordant for non-proteinuric hypertension	7
Discordant for proteinuric pre-eclampsia	5
Both affected with proteinuric pre-eclampsia	0
Proteinuric pre-eclampsia plus non-proteinuric hypertension (see text)	1
Incomplete records	14
Total	99

Comment

For analysis we combined these five discordant cases with our single previously reported discordant case⁴ and with four discordant pairs reported by Thompson and Fraser.⁵ No recent well documented report of any concordant affected pairs has been identified. For the present series (0/5 concordant) the 95% confidence interval for the proportion of concordant affected cases derived from tables of the binomial distribution is 0% to 52%, and when previous reports are included (0/10 concordant) the confidence interval is 0% to 31%. The penetrance of the putative gene, defined as the percentage of concordant affected cases out of all affected cases, should lie in this range.

The zygosity of the identical twin pairs may be disputed as HLA, blood grouping, and other polymorphism analyses were not performed on these five cases; however, Thompson and Fraser have confirmed zygosity by blood group.⁵ Nevertheless, all