Relation between Glasgow coma score and gag reflex in 111 patients requiring neurological observation in accident and emergency department

	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.

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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.

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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 toxaemia, 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 toxaemia." 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 case[4] 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

our twins had completed a detailed survey of hair and eye colour and height and had reported that when they were children parents and teachers had had difficulty telling them apart. Such surveys are reliable for determining zygosity.[3]

The present study indicates that caution should be exercised before accepting a single gene hypothesis for pre-eclampsia. Pre-eclampsia is not a maternal autosomal recessive condition because in recessive inheritance penetrance of the homozygote should be near 100%. We cannot rule out dominant inheritance with incomplete penetrance, but the upper limit for this penetrance is about 31%.

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Safety of thrombolysis in association with cardiopulmonary resuscitation

Stephen J Cross, Hai Shiang Lee, John M Rawles, Kevin Jennings

Cardiac Department, Aberdeen Royal Infirmary, Aberdeen AB9 2ZD
Stephen J Cross, MRCP, *registrar*
Hai Shiang Lee, MRCP, *registrar*
Kevin Jennings, FRCP, *consultant*

Department of Medicine and Therapeutics, University of Aberdeen, Aberdeen
John M Rawles, FRCP, *consultant*

Correspondence to:
Dr Cross.

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Thrombolysis is now standard first line treatment for acute myocardial infarction: infarct size is decreased[1] and mortality reduced.[2] Owing to the temporary systemic fibrinolytic state induced by this treatment thrombolytic agents are not recommended after prolonged or traumatic cardiopulmonary resuscitation.[3,4] Therefore patients who would potentially benefit from thrombolysis may be denied it if they have an arrest before or shortly after admission to hospital and require more than cardioversion for resuscitation. For example, patients with primary ventricular fibrillation after myocardial infarction have larger infarcts than those who do not[5] and might therefore benefit from thrombolytic treatment.

We looked for bleeding complications in a consecutive series of patients presenting with acute myocardial infarction who had a cardiac arrest before or shortly after receiving thrombolysis.

Patients, methods, and results

Thirty nine patients presented to Aberdeen Royal Infirmary during 1990 with acute myocardial infarction treated by thrombolysis and had an early (within 24 hours) cardiac arrest not associated with cardiogenic shock. Myocardial infarction was diagnosed according to standard World Health Organisation criteria in those who survived and from the history and electrocardiogram in those who died.

The patients were divided into three groups (table). Patients in group I had a cardiac arrest before receiving thrombolysis; four of them had chest compression with cardioversion and the rest cardioversion alone. There were no bleeding complications. One patient died a week later; postmortem examination showed extensive myocardial infarction with biventricular failure.

Patients in groups II and III had a cardiac arrest within 24 hours after receiving thrombolysis. Those in group II could not be resuscitated despite chest compression in all cases, and cardioversion when appropriate. No bleeding complications were reported at the time. Patients in group III were successfully resuscitated. Chest compression was performed in two patients, the others having cardioversion alone. The only bleeding complication was a chest wall haematoma after a precordial blow in a patient who did not have chest compression; the haematoma resolved completely.

When group II and group III were compared, group II had significantly more arrests secondary to electromechanical dissociation and asystole ($p<0.05$) and significantly fewer from ventricular fibrillation and tachycardia ($p<0.01$) (χ^2 tests).

Characteristics of three groups of patients who presented with acute myocardial infarction and had cardiac arrest before or shortly after thrombolysis

	Cardiac arrest before thrombolysis	Cardiac arrest after thrombolysis	
		Resuscitation unsuccessful	Resuscitation successful
No of patients	10	18	11
Mean age (years)	64·8	69·1	62·7
Age range (years)	51-79	56-79	47-73
Sex (M/F)	6/4	11/7	7/4
Diagnosis (No of patients):			
Electromechanical dissociation		7	
Asystole	1	6	2
Ventricular fibrillation	6	4	7
Ventricular tachycardia	2		1
Unknown	1	1	1

Comment

Although thrombolysis may be associated with haemorrhagic complications, we found no clinically significant bleeding complications in patients receiving such treatment either before or after cardiopulmonary resuscitation. All but one of the patients who received thrombolysis after resuscitation (group I) survived to leave hospital. Four of them had had chest compression without any adverse effects, and postmortem examination of the only patient who died in this group showed no evidence of internal bleeding. The only bleeding complication in the series, in a patient who received thrombolysis before resuscitation, was a chest wall haematoma from a precordial blow, and this completely resolved.

Cardiac rupture may be a complication of thrombolysis and may explain the high incidence of apparent rupture in our series. Most of our patients had a cardiac arrest while their heart rhythms were being monitored; the hearts of those with asystole and electromechanical dissociation (and presumed rupture) would be expected to have ruptured before the start of resuscitation.

Although our study was small, the results suggest that cardiopulmonary resuscitation is not a clear contraindication to thrombolysis.

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