

SHORT REPORT

Predictive value of Glasgow coma scale after brain trauma: change in trend over the past ten years

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Background: Age and the Glasgow Coma Scale (GCS) score on admission are considered important predictors of outcome after traumatic brain injury. We investigated the predictive value of the GCS in a large group of patients whose computerised multimodal bedside monitoring data had been collected over the previous 10 years.

Methods: Data from 358 subjects with head injury, collected between 1992 and 2001, were analysed retrospectively. Patients were grouped according to year of admission. Glasgow Outcome Scores (GOS) were determined at six months. Spearman's correlation coefficients between GCS and GOS scores were calculated for each year.

Results: On average 34 (SD: 7) patients were monitored every year. We found a significant correlation between the GCS and GOS for the first five years (overall 1992–1996: $r = 0.41$; $p < 0.00001$; $n = 183$) and consistent lack of correlations from 1997 onwards (overall 1997–2001: $r = 0.091$; $p = 0.226$; $n = 175$). In contrast, correlations between age and GOS were in both time periods significant and similar ($r = -0.24$ v $r = -0.24$; $p < 0.002$).

Conclusions: The admission GCS lost its predictive value for outcome in this group of patients from 1997 onwards. The predictive value of the GCS should be carefully reconsidered when building prognostic models incorporating multimodality monitoring after head injury.

The Glasgow Coma Scale (GCS) score, since its introduction in 1974,¹ has been frequently used as one of the most important predictors of outcome after head injury,^{2,3} although other variables such as age, abnormal motor responses, CT findings, pupillary abnormalities, and episodes of hypoxia and hypotension, have been subsequently introduced to build more complex and accurate prognostic models.^{4–7} In this context, different authors have pointed out the higher accuracy of the motor score compared with the whole GCS score as predictor of outcome, supporting the importance of splitting the “motor component” of the GCS from the “verbal component” and the “eye component”: the last two are often difficult to evaluate in comatose patients.⁸ However, the GCS remains a key measure in neurological assessment after head injury, and in most studies classification of the severity of the trauma is still based on the admission GCS. A score less than or equal to 8 is the traditional criterion for differentiating between severe and moderate to mild head injury, and patients' management is frequently dependent on this initial classification.

Data derived from invasive measurement of physiological parameters, such as intracranial pressure (ICP) and cerebral perfusion pressure (CPP), have been combined in computerised multimodal monitoring⁹ and systematically used over

the past 10 years in our centre, and they are under review. In this study we concentrate on correlations between outcome and entry data traditionally taken as factors in the prediction models, investigating the time dependence of correlations between GCS, age, and outcome of patients receiving computerised multimodal bedside monitoring.

PATIENTS AND METHODS

We retrospectively reviewed data from 484 head injured patients admitted to our Neurocritical Care Unit (NCCU) between 1992 and 2001. For the purpose of the analysis, only patients included in our multimodal neuromonitoring programme were considered. These were patients whose initial severe neurological condition or secondary deterioration required a neurocritical care environment with continuous monitoring of ICP and invasive arterial blood pressure (ABP), lasting usually longer than 24 hours. It is important to stress that the group of patients studied represented only about half of all admissions following head injury to our NCCU. They were treated according to various protocols which converged in time into unified inpatient guidelines for head injury.¹⁰ Arterial hypotension was promptly treated with fluids and continuous infusion of inotropes. Episodes of increased ICP were controlled with medical treatment whenever possible, including sedation and paralysis, mild hypoventilation, hypothermia, and thiopentone infusion. Surgical treatment was added if persistent or uncontrolled intracranial hypertension developed.

The patients were grouped according to year of admission from 1992 to 2001. The best GCS, after fluid resuscitation and stabilisation of the patient, was considered for the analysis; the GOS score was determined at six months. The Spearman's correlation coefficients between GCS, age, and GOS were calculated for each year.

RESULTS

Data from 360 cases with complete information on GCS, GOS, and age were considered suitable for the analysis. An average of 34 (7) patients with a mean age of 34 (16) years were monitored annually. The median GCS on admission was 6 (range 3 to 15) and the median GOS was 4 (range 1 to 5). The patient distribution in years was homogeneous: no differences in age (ANOVA), GCS, and GOS (Kruskal–Wallis test) were observed between groups (table 1).

We found a significant positive correlation between the GCS and GOS calculated every year from 1992 to 1996. This correlation, observed for the first five years (overall value from 1992 to 1996: $r = 0.41$; $p < 0.00001$; $n = 183$), was

Abbreviations: GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Score; ICP, intracranial pressure; CPP, cerebral perfusion pressure; NCCU, Neurocritical Care Unit; ABP, arterial blood pressure

Table 1 Distribution of GCS, GOS, age and correlation between GCS and GOS in the period 1992–2001

Year	Number of patients	GCS median (range)	GOS median (range)	Age mean (SD)	GCS and GOS correlation	p Value
1992	29	4 (3–11)	3 (1–5)	30 (17)	0.55	0.019*
1993	39	6 (3–15)	4 (1–5)	32(15)	0.39	0.015*
1994	40	5 (3–15)	4 (1–5)	30 (13)	0.43	0.006*
1995	33	6 (3–12)	4 (1–5)	34 (16)	0.42	0.016*
1996	43	5 (3–14)	3 (1–5)	33 (17)	0.39	0.011*
1997	31	6 (3–14)	4 (1–5)	37 (17)	0.01	0.978
1998	52	7 (3–14)	4 (1–5)	36 (16)	0.21	0.131
1999	30	7 (3–14)	4 (1–5)	30 (15)	–0.16	0.932
2000	27	7 (3–13)	3 (1–5)	38 (18)	0.00	1.000
2001	36	6 (3–12)	4 (1–5)	39 (16)	0.29	0.087

GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Score.
*significant correlation.

consistently lost each year from 1997 onwards (overall value from 1997 to 2001: $r = 0.091$; $p = 0.226$; $n = 175$) (table 1). In contrast, correlations between age and GOS were in both periods of time significant and similar (in both cases, $r = -0.24$; $p < 0.002$). Significant step increases in CPP were observed between 1996 and 1997 (68 ± 15 v 80 ± 11 mm Hg; $p = 1.5 \times 10^{-7}$).

DISCUSSION

The present report of 10 years' data on head injured patients shows a loss in correlation between admission GCS and GOS from 1997 onwards, suggesting a reduction in power of the GCS score as predictor of outcome after brain trauma. In the same group of patients, age remains an important factor in prognostic modelling after head injury.

In this study we have considered only patients included in our multimodal monitoring programme whose data were promptly available. These patients do not represent the entire group of severely head injured patients admitted to our NCCU during the same period of time. They required ICP and ABP monitoring for a comparatively long period, and either their level of consciousness was severely impaired at the time of admission or they showed a progressive deterioration during the following days. Patients who died or who recovered consciousness shortly after admission were not included in the analysis.

As different authors have suggested,⁸ the "motor component" of the GCS score may yield a higher predictive value than the entire GCS score after brain trauma, but difficulties in collecting complete clinical notes did not allow this comparison. This is the obvious limitation of our study, but the overall conclusion remains useful: the traditional link between GCS and GOS is now problematic.

The question arises as to what has changed in the management of head injury from 1992 onwards to influence the relevance of the GCS score on outcome? Marion *et al*⁸ analyse the difficulty of determining the initial GCS in a repeatable and reproducible manner. They identify more aggressive prehospital treatment, involving early sedation and intubation, as a factor obscuring the real GCS assessment. This problem in obtaining a valid neurological examination in the first 24 hours after trauma, as well as progress in clinical management, may have influenced the relevance of the GCS on outcome over the last five years. A step increase in CPP coincided with loss of correlation between the GOS and GCS; is this evidence that success in CPP oriented therapy has severed this link?

We can only speculate as to which element may have caused these changes. Certainly, the predictive value of the GCS should be carefully reconsidered when building

prognostic models after brain trauma or when selecting criteria for patients' inclusion into research protocols for severe head injury.

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