Spontaneous Intracerebral Haemorrhage: An Analysis of Factors Affecting Prognosis

A.A. Dixon, R.O. Holness, W.J. Howes and J.B. Garner

ABSTRACT: A retrospective study of 100 patients with spontaneous intracerebral haemorrhage was carried out, to identify clinical factors which have a predictive value for outcome. Numerical equivalents for the admission level of consciousness (the Glasgow Coma Scale), ventricular rupture, partial pressure of oxygen in the blood, the electrocardiogram, clot location, and clot size were combined into equations predicting outcome. The best single parameter for prediction was the Glasgow Coma Scale.

RÉSUMÉ: L'hémorragie intracérébrale spontanée. L'analyse des facteurs influençant la prognose. Une étude rétrospective de 100 patients avec hémorragie cérébrale spontanée a été faite dans le but d'identifier les facteurs cliniques à valeur prédictive en ce qui concerne l'issue. Des équivalents numériques pour le niveau de conscience à la réception (l'échelle de Glasgow), la rupture ventriculaire, la pression partielle de l'oxygène dans le sang, l'électrocardiogramme, la localisation du thrombus et la grosseur du thrombus, ont été combinés en équations afin de prédire l'issue. Le meilleur paramètre prédictif simple était l'échelle de Glasgow.

Can. J. Neurol. Sci. 1985; 12:267-271

Spontaneous intracerebral haemorrhage remains a formidable neurosurgical problem with unanswered questions as to appropriate medical and surgical therapy. The ability to predict clinical outcome is basic to any decision regarding therapy (Sharpiro, 1977). This paper analyzes intracerebral haemorrhage and defines a reliable prognostic scale, by combining multiple clinical factors into a linear discriminant equation.

The prognosis in patients with intracerebral clot is improving (Garraway et al., 1983). This may be partly factitious due to improved diagnosis with computerized tomography (CT) (Hier et al., 1977; Rudick, 1981). Improved non-operative care and surgical techniques have contributed to a lower mortality (Duff et al., 1981; Kaneko et al., 1977). The use of mortality figures, however, does not adequately define outcome. It ignores a group of people who, although alive, persist in an existence unsatisfactory to themselves and to their families (McKissock et al., 1961; Ransohoff et al., 1971). This must be taken into account when determining prognosis.

Multiple factors have been defined in predicting outcome of intracerebral haemorrhage. McKissock demonstrated the importance of level of consciousness in determining outcome (McKissock et al., 1959). This has been confirmed by others (Waga et al., 1983). Other factors which have been examined include clot size, clot location, ventricular rupture, patient age, blood pressure and electrocardiogram (EKG) abnormalities (Doublas et al., 1982; Cerillo et al., 1981).

Any factors which have predictive value should be objective, easily attainable and readily reproducible. Statistical methods have been used in predicting outcome from head injuries (Jennet et al., 1976; Van Dougen et al., 1983).

No group of factors can predict an outcome with 100% certainty and as these head injury studies show, outcome can only be predicted with an associated range of error. This study is an attempt at fulfilling these criteria and minimizing the associated range of error to the most acceptable level.

Clinical Material and Method

Patient Population

One hundred case records and radiographs of patients admitted to the Victoria General Hospital, between 1977 and 1982 inclusive, were retrospectively reviewed. All patients who fulfilled the following criteria for selection were included.

(1) Intracerebral haemorrhage (including cerebellum and brain stem) on CT Scan;
(2) No history of head trauma or hematological disease as a possible cause;
(3) Haemorrhage not secondary to tumor, aneurysm or proven arteriovenous malformation.

Presented in part at the Canadian Congress of Neurological Sciences, Edmonton, June 1984.
Dr. Dixon received the K.G. McKenzie prize of the Canadian Neurosurgery Society for this work.
From the Department of Neurosurgery and Department of Community Health & Epidemiology, Dalhousie University, Halifax.
Received April 25, 1985. Accepted June 10, 1985.
Reprint requests to: Administrative Secretary, Department of Neurosurgery, Victoria General Hospital, Halifax, Nova Scotia, Canada B3H 2Y9.

https://doi.org/10.1017/S0317167100047144 Published online by Cambridge University Press

Clinical Factors

Each of the factors chosen had both a theoretical basis and a practical or historical foundation for inclusion. The $P0_2$ and EKG were chosen as objective tests for medical condition. Subjective criteria, such as past medical history, were studied but felt to be too unreliable for general use. Each of these objective factors was computer coded and their individual and combined significance in their ability to predict the subsequent patient outcome assessed. Over twenty clinical parameters were studied. The following factors were considered the most important in prognosis.

Glasgow Coma Scale (G.C.S.)

This numerical equivalent of level of consciousness, as described by Teasdale and Jennett, was utilized in all patients (Teasdale et al., 1974). In the early years of the study period, many times a patient was not assigned a G.C.S. number on admission. If not, the patient was assigned a number based on the admission neurological examination.

Blood Pressure

The levels reported on admission are used. In the subsequent classification into normal and abnormal, the level 160/100 was chosen and the patient considered to have an abnormal blood pressure if either the systolic or diastolic blood pressure was elevated.

Partial Pressure Oxygen in Blood (PO_2)

Only half the patients had blood gas recordings on admission. The others had a blood gas recorded during their period of observation. The worst PO_2 was chosen if several were listed. The inspired oxygen level was ignored.

Computerized Tomography (C.T.)

The C.T. Scan of each patient was examined. The EMI 1010 scanner with a 160 x 160 matrix was used. Most scans were done within 24 hours of admission. The clot location, the size of the clot and the presence or absence of ventricular extension were noted. The true size of the clot was calculated by measuring the maximum diameter on the scan and then incorporating the magnification factor of the EMI scanner (Gonzalez et al., 1976; Weisberg, 1979).

Electrocardiogram (EKG)

Only the EKG reports were used. A normal EKG was defined as one read as normal or included only an atrial arrhythmia or atrial ectopics. All other changes were considered abnormal.

Assessment of Outcome

All one hundred patients were assigned an outcome. Patient outcome was determined after a minimum of six months either from the patient's chart or from phoned contact with the patient's family. The Glasgow Recovery Scale (G.R.S.) as described by Jennett and Bond for head injuries was used (Jennett et al., 1975). This was defined here as:—

Good — Capable of a return to the former level of function possibly with a minor deficit.

Moderately Disabled — Independent but disabled.

Severely Disabled — Dependent on others for daily living but capable of social interaction.

Vegetative — No social interaction.

Dead.

To simplify data charting and subsequent prediction, the G.R.S. was divided into Satisfactory and Unsatisfactory outcomes.

CODE	G.R.S.
0 Satisfactory	Good or Moderately Disabled
1 Unsatisfactory	Severely disabled, vegetative, or dead

Statistical Methods

Initially, using the known outcome groups, satisfactory and unsatisfactory, an analysis of the clinical variables in a stepwise manner was carried out to determine if these variables could discriminate between these two groups.

Initially, the variable which best discriminated between the two (i.e. was able to predict the outcome with greatest significance) was chosen. Then the next variable which best discriminated between the two groups, allowing for the discriminating ability of the first variable, was chosen. This stepwise analysis examining all the clinical variables was carried out to the limit of the arbitrarily set level of significance which was $P<.05$. When this was reached, all other factors were discarded from the equation.

These same variables were used to predict the Glasgow Recovery Scale using a multiple regression analysis.

Results

One hundred patients were tabulated, so that all totals represent percentages. Five patients were not utilized in the discriminant analysis, because of absence of a discriminating variable, PO_2 or EKG. All patients were treated in a neurosurgery intensive care unit, if required, with its associated intensive respiratory therapy. No intracranial pressure monitoring was done. Of the 77 survivors from this series, there was a delayed recurrence of intracerebral haemorrhage documented in three. Each occurred after discharge from hospital and in the opposite cerebral hemisphere from the first haemorrhage. The repeat haemorrhages were not included in this study. Pathology reports were available in six lobar clots with amyloid angiopathy found in two. Thirty-nine patients had intracranial surgery. Patients operated on tended to be in better condition and hence had better results. The indications for surgery and the subsequent outcomes of the medical and surgical groups are not separately discussed in the results.

Level of Consciousness

The Glasgow Coma Scale was by far the most important discriminating variable in predicting outcome (Figures 1 and 2). Generally, the admission level of consciousness, as determined by G.C.S. groupings, was much higher than in other clinical series (Paillas et al., 1973; Luessenhop et al., 1967).

No patient admitted in the G.C.S. group 3-5 had a satisfactory outcome; however, an admission grouping of 13-15 did not assure the patient of a satisfactory outcome, as three patients in this category remained severely disabled.

CT Scan Criteria (Figure 2)

The relative frequencies of clot location are comparable to other series (Crowell et al., 1981). The total incidence of lobar

https://doi.org/10.1017/S0317167100047144 Published online by Cambridge University Press

clot was 33%. This has been shown in another series to be associated with a more favorable prognosis (Ropper et al., 1980). Putamen clot was associated with an unfavourable outcome.

No attempt was made to quantitate the extent of intraventricular rupture. It too, particularly when associated with putamen haemorrhage, was associated with a poor outcome (Figure 4).

Medical Criteria (Figures 2 and 3)

Previous studies have demonstrated the usefulness of EKG and PO_2 in predicting outcome with spontaneous or traumatic intracranial haemorrhage (Douglas et al., 1982; Stablein et al., 1980). It was unusual for a patient with a normal EKG, or a normal PO_2, to have an unsatisfactory outcome.

Although age, blood pressure and clot size were separately significant, these variables were discarded as lacking significance independent of the discriminating variables in the analysis; that is, in combination with these variables their statistical significance was no longer less than 0.05.

Results

Figure 1

Glasgow Recovery Scale Associated with Glasgow Coma Scale

(Glasgow Recovery Scale (P = .001))

		Good	Moderately Disabled	Severely Disabled	Vegetative	Dead
Admission G.C.S. Groups	3-5	0	0	0	1	15
	6-8	2	2	5	0	3
	9-12	6	13	8	0	5
	13-15	21	16	3	0	0
Totals		29	31	16	1	23

Discriminant Analysis

Predicting Satisfactory/Unsatisfactory Outcome

The staged step wise analysis of all variables demonstrated five factors which retained a $P<.05$. These factors along with their code units, in order of importance were:

	Units	Successive P
G.C.S.	3-15	= .001
PO_2	mm Hg	= .005
Ventricular rupture	0 (Absent) 1 (Present)	= .005
Putamen clot	0 (None) 1 (Present)	= .008
EKG	0 (Normal) 1 (Present)	= .012

The next variable was clot size with P = .056 and this and further variables were discarded.

The Equation Predicting Satisfactory Outcome

The linear discriminant equation was calculated as:

Satisfactory outcome predicted with Score >0

Figure 2

Outcomes Associated with Clinical Factors

Clinical Factor		Outcome: Satisfactory	Outcome: Unsatisfactory	Totals
G.C.S.	P = .001	(Number of Patients)		
3- 5		0	16	16
6- 8		4	8	12
9-12		19	13	32
13-15		37	3	40
EKG	P = .001			
0 (Normal)		32	4	36
1 (Abnormal)		28	34	62
Blood pressure (Normal Abnormal)				
0 Normal		33	14	47
1 Abnormal		27	26	53

Clinical Factor		Outcome: Satisfactory	Outcome: Unsatisfactory	Totals
Clot location		(Number of Patients)		
Putamen		14	26	40
Thalamus		10	3	13
Lobar		29	4	33
Ventricular rupture	P=.001			
0 (No)		48	16	64
1 (Yes)		12	24	36
Clot size P = .005				
1. (Less than 2 cm)		5	0	5
2. (2 cm - 3 cm)		23	3	26
3. (>3 cm)		32	37	69

https://doi.org/10.1017/S0317167100047144 Published online by Cambridge University Press

Figure 3
Outcomes Associated with Clinical Factors

Clinical Factor	P	Satisfactory	Unsatisfactory
		(Mean units)	
Age (Years)	P = .005	58.7	66.6
B.P. (mm Hg)			
Systolic	P = .01	162	180
Diastolic	P = .06	94	101
PO_2 (mm Hg)	P = .001	79	68

Figure 4

Outcome in Patients with Putamen Bleed and Ventricular Extension

Outcome P = .001	Number
Good	0
Moderately disabled	1
Severely disabled	5
Vegetative	0
Dead	9
Total	15

To predict satisfactory: (P = 0.005)
[(G.C.S.) (0.17)] + [(PO_2) (0.08)] - [(Ventricular rupture) (1.23)] - [(EKG) (0.75)] - [(Putamen) (0.8)] - 6.52 (Constant)

For example in a patient with:

Putamen clot, G.C.S. = 12, PO_2 = 70,
No ventricular rupture, and EKG abnormal, equation becomes:
[(12) (0.17)] + [(70) (0.08)] - [(0) (1.23)] - [(1) (0.75)] - [(1) (0.80)] - 6.52 = −.43 Predicting an unsatisfactory outcome.

Using this equation classifies correctly 96% of the patients in this clinical series as follows:

	Predicted Satisfactory	Predicted Unsatisfactory
Actual satisfactory (60)	59	1
Actual unsatisfactory (40)	3	37

The correlation of the linear predicting expression with outcome is 0.82.

Multiple Regression

Equation Predicting Glasgow Recovery Scale

The same variables were used to predict the G.R.S. number. These corresponded to 1 (Good) 2 (Moderately disabled) 3 (Severely disabled) 4 (Vegetative) 5 (Dead)

The successive variables chosen were:

Factor	Units	P
G.C.S.	3-15	.001
PO_2	mm Hg	.001
Ventricular rupture	0 (Absent) 1 (Present)	.001
Clot size	1 (less than 2 cm) 2 (2-3 cm) 3 (Greater than 3 cm)	.036
EKG	0 (Normal) 1 (Abnormal)	.028

The resultant equation becomes (P = .001)
G.R.S. (1-5) = - [(G.C.S.) (0.18)] - [(PO_2) (0.04)] + [(Ventricular rupture) (0.61)] + [(Clot size) (0.35)] + [(EKG) (0.43)] + 6.23

For example with:

G.C.S. 10 PO_2 70 ventricular rupture Clot size greater than 3 cm Abnormal EKG

Calculated G.R.S. becomes:

− [(10) (0.18)] − [(70) (0.04)] + [(1) (0.61)] + [(3) (0.35)] + [(1) (0.43)] + 6.23
= 3.72 Corresponding to a score between severely disabled (3) and vegetative (4).

Discussion

The significance of this study is the demonstration of how certain clinical parameters affect outcome in intracerebral haemorrhage. Predicting prognosis in a group of patients with this disorder can be done accurately. A numerical scale reflecting level of consciousness is crucial in the development of a predictive equation for outcome. Patients in coma did uniformly poorly while patients admitted alert usually, but not always, did well.

Most intracerebral clots tend to expand inward. Extension into the ventricle is more common than through cortex. It would take a larger clot from the putamen to enter ventricle than one from the thalamus. If clot size has a bearing on outcome, then one would expect ventricular rupture with putamen haemorrhage to have a worse prognosis than thalamic clot. In this study, ventricular extension in general was associated with a worse outcome. In putaminal bleeds, ventricular haemorrhage was associated with a particularly poor outcome. A recent study suggested that thalamic clot extending into the ventricle was also associated with a poorer outcome (Kwak et al., 1983).

Prognosis corresponded well to clot size. The larger the clot, no matter where the location, the poorer the outcome. The 3 cm diameter size seems to be a crucial one, for both supratentorial and cerebellar haemorrhage. Separate studies have shown a tendency for clinical deterioration or a poorer prognosis when this diameter is reached (Kwak et al., 1983; Crowell et al., 1981). Other authors have reported a correlation between clot volume and patient outcome (Ropper et al., 1980). This study combined all locations of haemorrhage together and then defined relevant characteristics. Each clot location has anatomic relationships which make this inaccurate. The size of clot for example is far more crucial with a pontine haemorrhage than one into the occipital lobe. Lobar clot has a much better outcome than other locations (Ropper et al., 1980).

The medical condition of the patient has an important bearing on prognosis. There is some controversy as to how important age is, all other factors being equal.

There is a tendency for a poorer outcome in the aged, especially over seventy years (Kwak et al., 1983). The clinical outcome in this group of patients was better than in most series (McKissock et al., 1961; Paillas et al., 1973; Luessenhop et al., 1967). This is due to several factors. A greater proportion of patients were in the higher C.G.S. groups and there was a higher percentage of lobar clot. The use of the CT scan in all cases improved the diagnosis of smaller clots, expected to have a better prognosis. The criteria for entry into the study excluded patients with coagulopathy, tumor and aneurysm, etc.

https://doi.org/10.1017/S0317167100047144 Published online by Cambridge University Press

No matter how strong a statistical correlation between variables, this still does not imply a cause and effect relationship. All results should be interpreted with this caution and the understanding that, for this reason, extrapolation may only have limited usefulness.

Conclusion

Whether it be considered the art or science of medicine, statistical analysis and prediction of outcome for any illness forms the basis for most clinical approaches to that disease. Easily available clinical parameters can be combined into a predictive equation for outcome of intracerebral clot.

Acknowledgements

We would like to thank Ms. C. Wilkinson and Mrs. M. Jarvis for secretarial assistance.

References

Cerillo A, Vizioli L, Falivene R, Bernini FP, Tedeschi G (1981) Intracerebral Hemorrhage: An attempt at statistical assessment for operability. Neurochiurgia 24: 163-169.

Crowell RM, Ojemann RG (1981) Surgery for Brain Hemorrhage From Cerebrovascular Diseases. Edited Moossy J, Reinwith OM. New York Raven Press 233-254.

Daniel WW (1978) Biostatistics: A Foundation for Analysis in the Health Sciences. 2nd Edition John Wiley and Sons.

Douglas MA, Haerey AF (1982) Long Term Prognosis of Hypertensive Intracerebral Hemorrhage. Stroke 13: 488-491.

Duff TA, Ayeni S, Levin AB, Javid M (1981) Non Surgical Management of Spontaneous Intracerebral Hemorrhage. Neurosurgery 9: 387-393.

Garraway WM, Whisnant JP, Druro I (1983) Changing Pattern of Survival Following Stroke. Stroke 14: 699-703.

Gonzalez CF, Grossman CB, Palacios E (1976) Computed Brain and Orbital Tomography. 1st Edition New York John Wiley and Sons 1-13.

Hier DB, Davis KR, Richardson EP, and Mohr JP (1977) Hypertensive Putaminal Hemorrhage. Ann. Neurol. 1: 152-159.

Jennett B, Bond M (1975) Assessment of Outcome After Severe Brain Damage. A practical scale. Lancet 1: 480-484.

Jennett B, Teasdale G, Braakman R, Minderhoud J, Knill-Jones R (1976) Predicting Outcome in Individual Patients After Head Injury. Lancet 1:1031-1034.

Kaneko M, Koba T, Yokoyama T (1977) Early Surgical Treatment for Hypertensive Intracerebral Hemorrhage. J. Neurosurgery 46: 579-583.

Kwak R, Dadoya S, Suzuki T (1983) Factors Affecting Prognosis in Thalamic Hemorrhage. Stroke 14: 493-500.

Luessenhop AJ, Sheulin WA, Ferrero AA, McCullough DC, Barone BM (1967) Surgical Management of Intracerebral Hemorrhage. J. Neurosurg. 27: 419-427.

McKissock W, Richardson A, Taylor J (1961) Primary Intracerebral Hemorrhage: A Controlled Trial of Surgical and Conservative Treatment in 180 Unselected Cases. Lancet 2: 221-226.

McKissock W, Richardson A, Walsh L (1959) Primary Intracerebral Hemorrhage: Results of Surgical Treatment in 244 Consecutive Cases. Lancet 2: 683-686.

Naroyan RK, Greenberg RP, Miller JD, et al. (1981) Improved Confidence of Outcome Prediction in Severe Head Injury. J. Neurosurgery 54: 751-762.

Paillas JE, Alliez B (1973) Surgical Treatment of Spontaneous Intracerebral Hemorrhage. J. Neurosurg. 39: 145-151.

Plum F, Posner JP. Contemporary Neurology Series. Diagnosis of Stupor and Coma. 1st Edition Philadelphia: FA Davis 966: 2.

Ransohoff J, Denby B, Kricheff I (1971) Spontaneous Intracerebral Hemorrhage. Clinical Neurosurgery 18: 247-266.

Ropper AH, Davis KR (1980) Lobar Cerebral Hemorrhage: Acute Clinical Syndromes in 26 Cases. Ann. Neurol. 8: 141-147.

Rudick RA (1981) Asymptomatic Intracerebral Hematoma as an Incidental Finding. Arch. Neurol. 38: 396.

Sano K, Yoshida S (1980) Cerebellar Hematomas — Indications and Prognosis in: Spontaneous Intracerebral Hematomas. Advances in Diagnosis and Therapy. Edited Pia, HW, Languard C, Berlin, Springer Verlag 1980; 348-360.

Shapiro AR (1977) The Evaluation of Clinical Predictions. A Clinical Method and Initial Application. NEJM 296: 1509-1514.

Stablein DM, Miller JD, Choi SC, Becker DP (1980) Statistical Methods for Determining Prognosis in Severe Head Injury. Neurosurgery 6: 243-248.

Steiner L, Bergvall U, Zqetnow N (1975) Quantitative Estimation of Intracerebral and Intraventricular Hematoma by Computerized Tomography. Acta. Radiol. (Stockholm) Suppl. 346: 143-154.

Teasdale G, Jennett B (1974) Assessment of Coma and Impaired Consciousness. A Practical Scale. Lancet 2: 81-84.

Van Dougen KJ, Braakman R, Gelpke GJ (1983) The Prognostic Value of Computerized Tomography in Comatose Head Injured Patients. J. Neurosurgery 59: 951-957.

Waga S, Yamamoto Y (1983) Hypertensive Putaminal Hemorrhage: Treatment and Results. Is Surgical Treatment Superior to Conservative One? Stroke 14: 480-485.

Weisberg LA (1979) Computed Tomography in Intracranial Hemorrhage. Arch. Neurol. 36: 422-426.

https://doi.org/10.1017/S0317167100047144 Published online by Cambridge University Press