significant correlations between surface area or weight and B.T.T.

Discussion

Japanese migrants to Hawaii—especially their families, the second-generation Nisei—develop disease patterns similar to Caucasians.[6] The Hawaii-Japanese lose the high gastric-cancer risks of Japan and acquire "Western" malignancies such as those of prostate, breast, and colon.[7] The Hawaii-Japanese have more colonic polyps and diverticuloses than Japanese living in Japan.[8]

It would be expected, then, that the more traditional Issei would have a faster B.T.T. than the Nisei and that the Hawaii-Japanese would have transit-times similar to those of Caucasians. We were surprised to find no differences between the Issei and Nisei, and the Hawaii-Japanese had rapid transit-times comparable with those in rural Africans.[2] The B.T.T. differences between the Japanese and Caucasians could not be explained by education, occupation, body-weight, surface area, or the number of bowel movements per day.

With respect to the Hawaii-Japanese experience, B.T.T.s do not seem to be related to the pathogenesis of colonic disease.

We thank Mr Harry Ito of Kuakini Hospital for engineering the stool-collection equipment; and Dr Edgar Childs, Dr Donald Ikeda, and Dr David Sakuda of the Department of Radiology, Kuakini Hospital, for their technical assistance. This work was supported in part by National Institutes of Health contracts E-71-2170 and PH-43-65-1003-C.

Requests for reprints should be addressed to the Japan-Hawaii Cancer Study, Kuakini Hospital, 347 North Kuakini Street, Honolulu, Hawaii 96817, U.S.A.

REFERENCES

1. Burkitt, D. P. *Cancer*, 1971, **28**, 3.
2. Burkitt, D. P., Walker, A. R. P., Painter, N. S. *Lancet*, 1972, ii, 1408.
3. Worth, R. M., Kagan, A. *J. chron. Dis.* 1970, **23**, 389.
4. Hinton, J. M., Lennard-Jones, J. E., Young, A. C. *Gut*, 1969, **10**, 842.
5. Pryke, E. S., White, H. M. *ibid.* 1970, **11**, 966.
6. Stemmermann, G. N. *Archs envir. Hlth*, 1970, **20**, 266.
7. Doll, R., Muir, C., Waterhouse, J. Cancer in Five Continents; vol. II. Berlin, 1970.
8. Stemmermann, G. N., Yatani, R., *Cancer*, 1974, **31**, 1260.

ASSESSMENT OF COMA AND IMPAIRED CONSCIOUSNESS

A Practical Scale

GRAHAM TEASDALE BRYAN JENNETT

University Department of Neurosurgery, Institute of Neurological Sciences, Glasgow G51 4TF

Summary A clinical scale has been evolved for assessing the depth and duration of impaired consciousness and coma. Three aspects of behaviour are independently measured—motor responsiveness, verbal performance, and eye opening. These can be evaluated consistently by doctors and nurses and recorded on a simple chart which has proved practical both in a neurosurgical unit and in a general hospital. The scale facilitates consultations between general and special units in cases of recent brain damage, and is useful also in defining the duration of prolonged coma.

Introduction

A WIDE range of conditions may be associated with coma or impaired consciousness. Apart from acute brain damage due to traumatic, vascular, or infective lesions, there are metabolic disorders such as hepatic or renal failure, hypoglycæmia or diabetic ketosis, and also drug overdose. In gauging deterioration or improvement in the acute stage of such conditions, as well as in predicting the ultimate outcome, the degree and duration of altered consciousness usually overshadow all other clinical features in importance. It is therefore vital to be able to assess and to record changing states of altered consciousness reliably.

Need for a Clinical Scale

Impaired consciousness is an expression of dysfunction in the brain as a whole. This may be due to agents acting diffusely, such as drugs or metabolic imbalance; or to the combination of remote and local effects produced by brain damage which was initially focal. Such focal brain damage may affect some of the responses which are used to assess the level of consciousness, and any scale devised for general use must allow for this possibility. A simpler scale might suffice for metabolic or drug coma, when the likelihood of structural brain damage is small, but in an emergency there may be insufficient information to assign patients confidently to a particular diagnostic category. Moreover, coma of mixed origin is not uncommon, as when head injury is suspected of being associated with ingestion of drugs or alcohol, or with a vascular accident. These seem good reasons for devising a generally applicable scheme of assessment.

Existing Systems

The development of equipment for monitoring various functions in critically ill patients has not altered the need for doctors and nurses to assess the level of consciousness. There is an abundance of alternative terms by which levels of coma or impaired consciousness are described and recorded. Systems for describing patients with impaired consciousness are not consistent.[1-14] Indeed, many clinicians retreat from any formal scheme in favour of a general description of the patient's state, without clear guidelines as to what to describe and how to describe it.

In practice, such unstructured observations commonly result in ambiguities and misunderstandings when information about patients is exchanged and when groups of patients treated by alternative methods are compared, or reported from different centres. There is no general agreement about what terms to use, nor are those in common use interpreted similarly by different workers. Almost every report of patients in coma offers yet another classification. Most divide the spectrum of altered consciousness into a series of steps, which in the reports we reviewed ranged from 3 to 17 and were often described in terms which defied clear definition. Many assume the existence of constellations of clinical features which are unique to each "level", whilst others distinguish between coma and consciousness on the basis of only one aspect of behaviour.

The importance of careful and complete neurological examination in determining the nature and site of the lesion causing coma has been described at length by Fisher[5] and Plum and Posner,[13] who emphasised that tests of brainstem function, not usually included in routine examination, can be useful in the diagnosis of stupor or coma. Neither, however, was primarily concerned with repeated bedside assessment of the degree of conscious impairment, which is the subject of our paper.

Glasgow Coma Scale

To be generally accepted, a system must be practical to use in a wide range of hospitals and by staff without special training. But the search for simplicity must not be the excuse for seeking absolute distinctions where none exist: for that reason no attempt is made to define either consciousness or coma in absolute terms. Indeed, it is conceptually unsound to expect a clear watershed in the continuum between these states. What is required instead is an effective method of describing the various states of impaired consciousness encountered in clinical practice. Moreover, this should not depend on only one type of response because this may, for various reasons, be untestable. The three different aspects of behavioural response which we chose to examine were motor response, verbal response, and eye opening, each being evaluated independently of the other. These feature in many previous reports on coma but not in the formal system we propose. This depends on identifying responses which can be clearly defined, and each of which can be accurately graded according to a rank order that indicates the degree of dysfunction.

Motor Responses

The ease with which motor responses can be elicited in the limbs, together with the wide range of different patterns which can occur, makes motor activity a suitable guide to the functioning state of the central nervous system. Indeed, every one of the reported scales which we reviewed included some aspect of motor responsiveness as a criterion.

Obeying commands is the best response possible, but the observer must take care not to interpret a grasp reflex or postural adjustment as a response to command. The terms "purposeful" and "voluntary" are avoided because we believe that they cannot be judged objectively.

If there is no response to command, a painful stimulus is applied. The significance of the response to pain is not always easy to interpret unless stimulation is applied in a standard way and is maintained until a maximum response is obtained. Initially pressure is applied to the fingernail bed with a pencil; this may result in either flexion or extension at the elbow. If flexion is observed stimulation is then applied to the head and neck and to the trunk to test for localisation. In brain death, a spinal reflex may still cause the legs to flex briskly in response to pain applied locally.[15] For this reason, and because the arms show a wider range of responses, it is wise always to test them, unless local trauma makes this completely impossible.

A localising response indicates that a stimulus at more than one site causes a limb to move so as to attempt to remove it.

A flexor response may vary from rapid withdrawal, associated with abduction of the shoulder, to a slower, stereotyped assumption of the hemiplegic or decorticate posture with adduction of the shoulder. Experienced observers may readily distinguish between normal and abnormal flexion, but for general use in the first few days after brain damage has been sustained it is sufficient to record only that the response is flexor.

Extensor posturing is obviously abnormal and is usually associated with adduction, internal rotation of the shoulder, and pronation of the forearm. The term "decerebrate rigidity" is avoided because it implies a specific physioanatomical correlation.[16]

No response is usually associated with hypotonia and it is important to exclude spinal transection as an explanation for lack of response; and also to be satisfied that an adequate stimulus has been applied.

When recording motor response as an indication of the functional state of the brain as a whole, the best or highest response from any limb is recorded. During a single examination some patients give variable responses, these usually becoming better as the patient becomes more aroused; responses from the right and left limbs may also differ. Any difference between the responsiveness of one limb and another may indicate focal brain damage and for this purpose the worst (most abnormal) response should be noted. But for the purpose of assessing the degree of altered consciousness it is the best response from the best limb that is recorded.

Verbal Responses

Probably the commonest definition of the end of coma, or the recovery of consciousness, is the patient's first understandable utterance; speech figured in nearly all the reported scales which we reviewed. Certainly the return of speech indicates the restoration of a high degree of integration within the nervous system, but continued speechlessness may be due to causes other than depressed consciousness (e.g., tracheostomy or dysphasia).

Orientation implies awareness of the self and the environment. The patient should know who he is, where he is, and why he is there; know the year, the season, and the month. The words "rational" and "sensible" are avoided because they cannot be clearly defined.

Confused conversation is recorded if attention can be held and the patient responds to questions in a conversational manner but the responses indicate varying degrees of disorientation and confusion. It is here that verbatim reporting of the individual patient's responses can be useful.

Inappropriate speech describes intelligible articulation but implies that speech is used only in an exclamatory or random way, usually by shouting and swearing; no sustained conversational exchange is possible.

Incomprehensible speech refers to moaning and groaning but without any recognisable words.

Eye Opening

Spontaneous eye opening, with sleep/wake rhythms, is most highly scored on this part of the scale and it indicates that the arousal mechanisms in the brainstem are active. But arousal does not imply awareness, and we believe it is unwise to try to decide whether a patient is attentive on the basis of eye movements. Patients in the persistent vegetative state,[17] who are subsequently shown to be structurally decorticate, have often been believed by relatives, nurses, and even by doctors to be reacting visually to people around them; probably primitive ocular-following reflexes may be executed at subcortical level.

Eye opening in response to speech is a response to any verbal approach, whether spoken or shouted, not necessarily the command to open the eyes.

Eye opening in response to pain should be tested by a stimulus in the limbs, because the grimacing associated with supraorbital or jaw-angle pressure may cause eye closure.

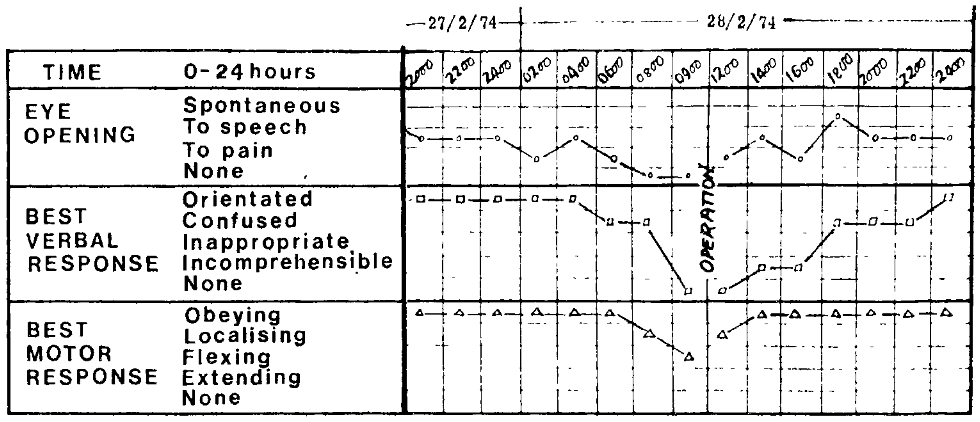

Chart for recording assessment of consciousness.

Practical Applications of the Scale

Different observers were able to elicit the responses in this scale with a high degree of consistency, and the likelihood of ambiguous reporting appears to be small. This was demonstrated by having several doctors and nurses examine the same group of patients. Disagreements were rare.[18] This was in pronounced contrast to what happened when the observers were asked instead to judge only whether patients were conscious or unconscious; one in five observers then disagreed with the majority opinion. This 20% disagreement-rate compared with rates of 20–35% which have been reported in various different clinical situations,[19] whilst in one study extensor plantar responses showed only 50% consistency when observations were repeated.[20]

One or other components of this scale may be untestable, and this fact can be recorded. Limbs may be immobilised by splints for fractures, tracheostomy may preclude speech, and eyelid swelling or bilateral third-nerve lesions make eye opening impossible. In the rare " locked-in syndrome," a patient with totally inactive limbs may obey commands to move the eyes and may even be able to signal his needs.[21]

The nurses in our intensive-care unit have willingly adopted this method of formalising observations which they previously used to record as a descriptive comment. They now plot them on a chart (see accompanying figure) somewhat similar in format, but not content, to one proposed by Bouzarth,[1] and which also provides for conventional recording of temperature, pulse and respiration, of the pupil size in mm., and of focal motor signs. This method has already been adopted successfully for making observations on head injuries in a neighbouring general hospital. In such hospitals patients with head injuries form a considerable proportion of acute surgical admissions, and observations there depend on medical and nursing staff who have no special experience of neurology and neurosurgery.

Discussion

Apart from its practical use in the management of recently brain-damaged patients, this scale allows the duration of coma to be defined more precisely, in terms of how long different levels of responsiveness have persisted. There is evidence that this is a crucial criterion when it comes to predicting the ultimate outcome of coma, particularly after head injury.[22] It would make it possible also to examine critically claims for good recovery after weeks or months " in coma," by enabling the alleged coma to be more accurately assessed. In such cases as we have scrutinised, it has been clear, even retrospectively, that there had been evidence of much earlier recovery, on at least one component of the coma scale, than had been recognised. By resolving the problem of defining " prolonged coma " the scale also makes it possible to distinguish between the various states which this term embraces, such as akinetic mutism and the persistent vegetative state.[17]

Some may have reservations about a system which seems to undervalue the niceties of a full neurological examination. It is no part of our case to deny the value of a detailed appraisal of the patient as a whole, and of neurological function in particular, in reaching a diagnosis about the cause of coma, or in determining the probable site of brain damage. However, repeated observations of conscious level are usually made by relatively inexperienced junior doctors or nurses; these staff are not only few in number but they change frequently even during the course of a day. There are therefore good reasons for restricting routine observations to the minimum, and for choosing those which can be reliably recorded and understood by a range of different staff.

We are grateful to many colleagues for their assistance in developing this scale; particularly Dr Fred Plum of New York, Dr Reinder Braakman of Rotterdam, and Dr David Shaw of Newcastle upon Tyne, in whose units its practical value has also been confirmed. We thank the consultants of the division of neurosurgery, Glasgow, for their cooperation. This scale was devised as part of a study of severe head injuries supported by the National Fund for Research into Crippling Diseases.

Requests for reprints should be addressed to B. J.

REFERENCES

1. Bouzarth, W. F. *J. Trauma,* 1968, **8**, 29.
2. Bozza Marribini, M. L. *Acta neurochir.* 1964, **12**, 352.
3. Bricolo, A. *Minerva neurochir.* 1965, **9**, 150.
4. Fischgold, H., Mathis, P. *Electronecephalogr. clin. Neurophysiol.* 1959, suppl. 11.

References continued overleaf

SEASONAL VARIATION OF HISTOLOGICAL OSTEOMALACIA IN FEMORAL-NECK FRACTURES

J. E. AARON J. C. GALLAGHER
B. E. C. NORDIN

Medical Research Council Mineral Metabolism Unit, General Infirmary, Leeds 1

Summary In a series of 134 iliac-crest biopsies from unselected cases of fracture of the proximal femur, the proportion with evidence of osteomalacia varied with the season. The highest frequency of abnormal calcification fronts (43%) was observed in February to April and the lowest (15%) in August to October. The highest frequency of abnormal osteoid-covered surfaces (47%) was observed in April to June and the lowest (13%) in October to December. The overall frequency of osteomalacia in femoral-neck-fracture cases in Leeds seems to be about 37%. It is concluded that variation in hours of sunshine is responsible for a seasonal variation in osteomalacia in these cases and, possibly, in the elderly population as a whole.

WE have previously reported[1] histological evidence of osteomalacia in about 25% of patients with femoral-neck fractures in the Leeds area. In view of the reports of a seasonal variation in plasma concentrations of 25-hydroxycholecalciferol (25-H.C.C., 25-OHD_3) in the United Kingdom,[2,3] we have now re-examined this material for evidence of seasonal variation in histological osteomalacia.

The material comprises 134 unselected iliac-crest biopsy specimens from cases of fractured neck of femur admitted to the orthopædic department of the General Infirmary, Leeds, in the years 1969–73. The biopsied cases represent about 20% of the admissions during this period.

The histological procedures have already been described.[1] We simply grouped the biopsy specimens according to the month in which they were taken, usually at operation or very shortly after the fracture. Our criteria of normality[1] were up to 24% osteoid-covered surfaces and over 60% osteoid surfaces with calcification fronts. Preliminary examination of the data showed that the largest proportion of specimens with excess osteoid-covered surfaces were seen in April, May, and June, and the largest proportion with a subnormal percentage of calcification fronts were observed in February, March, and April. The data were then analysed in 3-monthly periods, starting in January for osteoid surfaces and in November for calcification fronts.

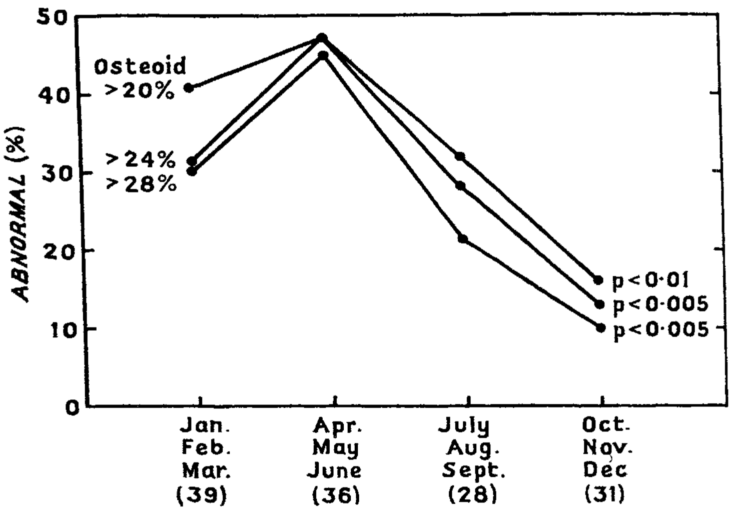

Fig. 1—Frequency of excessive osteoid-covered surfaces grouped in 3-monthly periods.

The three lines are based on three different upper normal limits for the percentage of osteoid-covered surfaces. The number of specimens in each 3-month period is indicated. The P values refer to the difference between the second and fourth quarter.

With osteoid-covered surfaces (fig. 1), there is a clear seasonal trend. In the second quarter of the year, 17 of 36 biopsies (47%) showed more than 24% of osteoid-covered surfaces, whereas in the fourth quarter this abnormality was only observed in 4 out of 31 (13%). In the first and third quarters the frequencies were 12 of 39 (30%) and 8 of 28 (28%), respectively. This difference is highly significant. This seasonal variation is not influenced by lowering the upper normal limit of percent osteoid-covered surfaces to 20% or raising it to 28%.

The calcification-front data are shown in fig. 2. With a lower normal limit of 60% of calcification

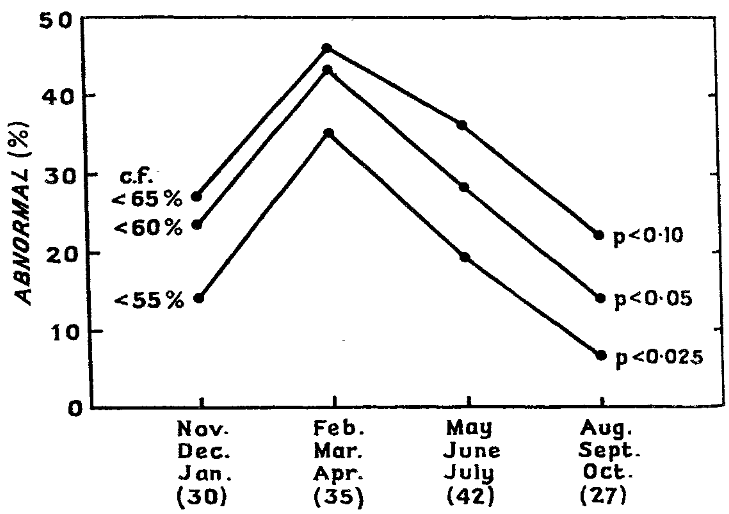

Fig. 2—Frequency of deficient calcification fronts grouped in 3-monthly periods.

The three lines are based on three different lower normal limits of percentage calcification fronts. The number of specimens in each 3-month period is indicated. The P values refer to the difference between the second and fourth quarter.

MR TEASDALE, PROF. JENNETT: REFERENCES—*continued*

5. Fisher, C. M. *Acta neurol. scand.* 1969, suppl. 36.
6. Mansuy, L., Lecuire, J., Jouvet, M. *Proc. 3rd int. Congr. neurol. Surg.: Excerpta med. int. Congr. Ser.* 1966, no. 110, p. 411.
7. Matthew, H., Lawson, A. H. H. *Q. Jl Med.* 1966, **35**, 539.
8. Medical Research Council. A Glossary of Psychological Terms Commonly Used in Cases of Head Injury. H.M. Stationery Office, 1941.
9. Mollaret, P., Goulon, M. *Rev. Neurol.* 1959, **101**, 3.
10. Rowbotham, G. F. Acute Injuries of the Head. Edinburgh, 1964.
11. Ommaya, A. K., Sadowsky, D. *J. Trauma*, 1966, **6**, 605.
12. Overgaard, J., Christensen, S., Haase, J., Hvid-Hansen, O., Hein, O., Land, A. M., Pedersen, K. K., Tweed, W. A. *Lancet*, 1973, ii, 631.
13. Plum, F., Posner, J. B. The Diagnosis of Stupor and Coma. Philadelphia, 1972.
14. Powiertowski, H., Huber, Z. *Proc. 3rd int. Congr. neurol. Surg.: Excerpta med. int. Congr. Ser.* 1966, no. 110, p. 209.
15. Ivan, L. P. *Neurology*, 1973, **23**, 650.
16. Feldman, M. H. *Archs Neurol.* 1971, **25**, 501.
17. Jennett, B., Plum, F. *Lancet*, 1972, i, 734.
18. Teasdale, G. M., Knill-Jones, R. P. Unpublished.
19. Conn, H. O. *New Engl. J. Med.* 1965, **272**, 830.
20. McCance, C., Watt, J. A., Hall, D. J. *J. chron. Dis.* 1968, **21**, 369.
21. Feldman, M. H. *Neurology*, 1971, **21**, 459.
22. Jennett, B. *Clin. Neurosurg.* 1972, **19**, 200.