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INCIDENCE AND CLINICAL FEATURES OF AUTONOMIC DYSREFLEXIA IN PATIENTS WITH SPINAL CORD INJURY

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Abstract. The histories of 444 patients admitted to this spinal cord injury service were reviewed for the incidence of autonomic dysreflexia (A.D.). Forty-eight per cent of 213 patients with complete cord lesions at T6 or above exhibited A.D. The time of onset post-injury, exciting causes, unusual manifestations of attacks, and the persistence of the condition were studied. These findings and the experience with attempts at prevention by education and by the use of an alpha-adrenergic blocker and a non-adrenergic vasodilating agent are reported.

Key words: Autonomic dysreflexia; Spinal cord injury.

Introduction

AUTONOMIC dysreflexia has been extensively reported and investigated since it was first described in 1890 by Bowley and it is possible that by now everything of importance has been said. Yet it continues to be a problem in the rehabilitation of spinal cord injured patients. We still cannot predict its appearance. Although we know the nervous pathways by which certain stimuli can precipitate an attack, not all patients with apparently similar lesions will respond in the same way, and one patient will exhibit no response to a stimulus which will send another into an acute hypertensive attack.

We have been concerned with two problems in particular. Firstly with the fact that this syndrome is still virtually unknown to the general medical and nursing professions leading to delayed or inappropriate treatment when they are faced with a patient in an acute attack. Secondly with the problem of predicting and preventing frequent repeated attacks of A.D. which can seriously interfere with attempts at rehabilitation and continue to pose a threat to the patient for the rest of his life.

Over the years we have attempted to control chronic attacks by the use of small oral doses of antihypertensive drugs. With the removal from the pharmacopoeia of the ganglionic blocking agents, which have been widely recommended for this purpose and the introduction of newer agents, a re-evaluation of the role of drug therapy seems indicated.

We have made a retrospective review of the histories of all the patients with complete injuries to the spinal cord at the level of T6 or above who were admitted to our Unit since it opened in 1971, and to the Highland View Hospital wards before that time, in the hope of finding some answers which might help us to protect our patients more effectively from the consequences of this condition.

Methodology

Subjects studied

We reviewed the histories of 213 patients (183 men and 30 women), admitted to Highland View Hospital for rehabilitation following spinal cord injury, who had complete neurological lesions at T6 or above. Among these there were 103 (85 men and 18 women), that is 48 per cent of the sample, who had documented episodes of autonomic dysreflexia or 'A.D.' and these constituted our study group.

Definition of A.D.

Autonomic dysreflexia was considered to be present if there was documented evidence of a sudden rise in both systolic and diastolic blood pressures with or without headache and accompanying symptoms of autonomic dysfunction such as sweating, blotching of the skin, goose bumps or dilatation of pupils.

Cystometry

Cystometric examinations and cystograms were performed on 180 of the patients, during which blood pressures were recorded at intervals during filling of the bladder. A.D. was considered to be present if the systolic and diastolic pressures rose by more than 10 mmHg and the examination was stopped if the diastolic pressure reached 90 mmHg.

Antihypertensive drugs

During the period under review we have attempted control of A.D. in patients exhibiting repeated, severe attacks, with one or more of the following drugs: rauwolfia 50 mg t.i.d., hydralazine 10–20 mg t.i.d. or q.i.d., phenoxybenzamine 10–40 mg b.i.d. During severe acute attacks diazoxide 300 mg I.V. was given if the pressure did not drop after the stimulus which precipitated the attack had been removed.

Results

Incidence

Autonomic dysreflexia has developed in 48 per cent of the patients at risk. It was rather more common in our women patients (60 per cent) than in men (46 per cent), but there were only 30 women in the study group, as against 183 men. Although it occurred in patients with cord injury levels from C3 down to and including T6, it was more prevalent in patients with cervical lesions (60 per cent) than in those with thoracic lesions (20 per cent) (Fig. 1).

Time of onset post-injury

Few patients exhibited signs and symptoms of A.D. or had it detected on routine cystometrogams during the first 2 months after their injury, but the majority had manifested themselves by 6 months post-injury, and 92 per cent by the end of the first year (Fig. 2). However, in six cases the first attack did not occur until more than 3 years and, in one case, not until 12 years post-injury (Fig. 3).

Presenting symptoms

The classical symptoms of a pounding headache usually accompanied by sweating and blotching of the skin of the face and thorax was noted in the majority of our patients (78 out of 88 documented cases). There were nine instances

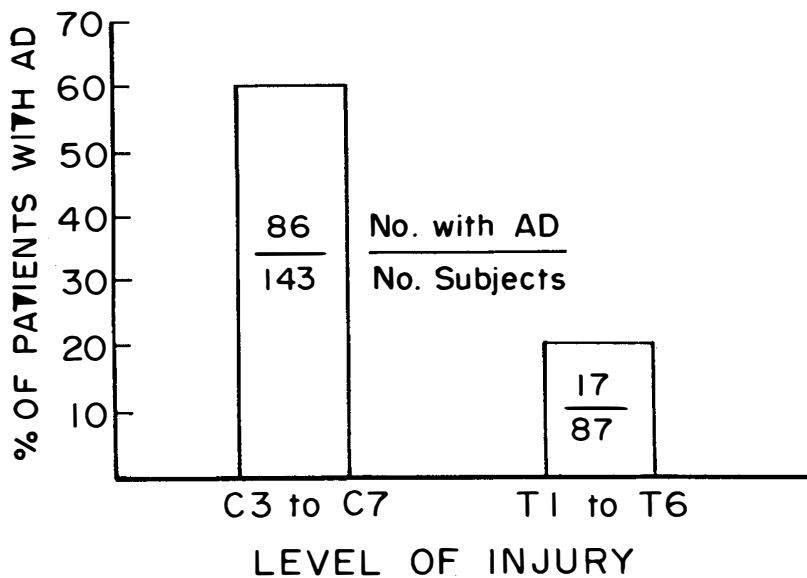


FIG. 1

Comparison of incidence of A.D. in patients with cervical and patients with thoracic lesions.

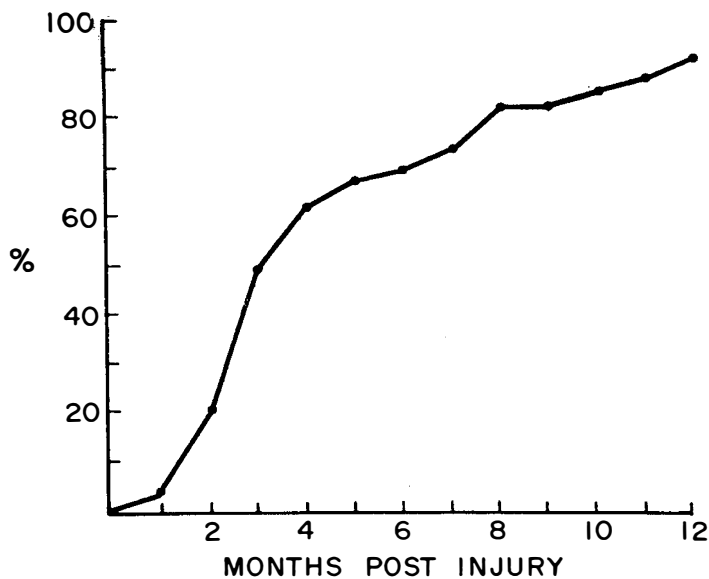


FIG. 2

Cumulative percentages of patients with A.D. who developed symptoms within 12 months after injury.

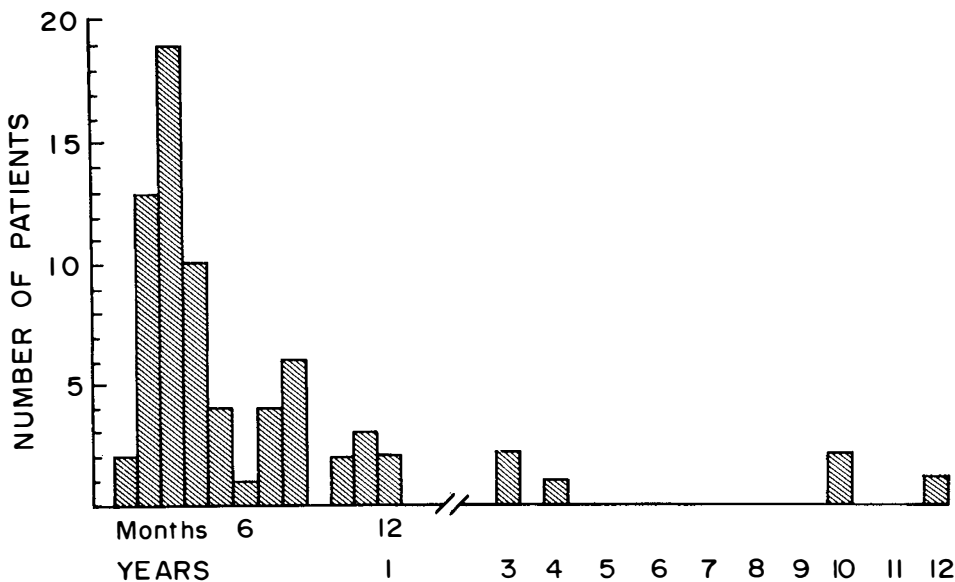


FIG. 3

Time interval between spinal cord injury and onset of autonomic dysreflexia in 72 patients.

however where the presenting symptoms were such diverse manifestations of cerebral hypertension as aphasia, blindness, convulsions and coma, dyspnoea and nausea; symptoms which do not immediately suggest a primary problem of a blocked catheter or constipation to the average doctor or nurse. However, failure to recognise this connection can result, and has resulted in disaster. One patient with convulsions died while being treated as a case of status epilepticus.

Bradycardia is often considered to be a hallmark of reflex hypertension, especially if the lesion is between the first and sixth thoracic segments (Bors & Comarr, 1971). This is not however a consistent finding, and is dependent on whether the pulse rate is recorded while the pressure is rising, at which time the transmission from the baroreceptors triggering the vagal response will be intense, or when the pressure is at a steady high or already dropping, at which time the transmissions may cease (Guyton, 1975). In our cases where the pulse was recorded (29 cases), there were equal numbers, including those with cervical lesions, with bradycardia and tachycardia, but the point at which the pulse was recorded was not noted.

Stimuli causing onset of attacks

The stimuli which most commonly precipitated attacks in our patients, as in all others reported, were those originating in the bladder (84 cases) resulting from spasms or distension, or from manifestations such as catheter irrigations or insertions, or from cystometry (Table I).

Over distension of the bowel, or rectal stimulation during bowel routines, were primarily responsible for causing attacks of A.D. in 14 patients, the next largest group. In a few patients, attacks were precipitated by lesions of the skin such as paronychia, ingrowing toe nails, or the development of pressure sores.

We were interested to observe three patients in whom attacks were quite

TABLE I
Precipitating causes of A.D.

	No. of cases
<i>Bladder</i> distension or catheter insertion	84
<i>Bowel</i> distension or stimulation	14
Infections—Urinary	3
Skin (paronychia)	1
<i>Psychological</i> stress	3

clearly and repeatedly precipitated by psychological stress factors, in the absence of any other stimuli, although at other times they were also sensitive to bladder stimuli.

Persistence

In 62 cases available for follow-up more than 1 year post injury, A.D. attacks were still being experienced by 57 (92 per cent) even up to as long as 20 years after their original injury and rehabilitation (Table II). In the majority of cases, the attacks became less frequent, but the severity of the occasional attacks did not noticeably diminish, and could still present as emergencies.

TABLE II
Persistence of A.D.

	No. of patients
No recorded attacks after 1 year post-injury	5
Persistence of attacks—	57
2–6 years post-injury	30
7–11 years post-injury	16
12–20 years post-injury	11

Use of hypertensive drugs

The first oral drug with which we attempted to control chronic repeated attacks in 1974 was rauwolfia 50 mg t.i.d., suggested to us by Dr John Yeo of Sydney. Rauwolfia has a central effect and can cause depression although we didn't find this a problem, and it also acts at the transmitter site. The incidence of side effects, particularly stuffy nose and diarrhoea experienced by our patients led to discontinuation of this therapy after a trial in only ten patients. Because hydralazine acts directly on the arteriolar smooth muscle producing relaxation and dilatation of the peripheral vessels, this seemed to be an ideal drug for treatment of this condition because it would thereby be free of the side effects resulting from the more generalised sympatholytic activity of drugs affecting the post ganglionic or transmitter sites (Young, 1963). We have used it in 26 patients with very variable results. There seems to be a narrow therapeutic range between having no demonstrable effect and putting the patient into hypotensive attacks. If a very susceptible

patient is to be exposed to a known stimulus such as a catheter change, the administration of hydralazine 20 mg about 1 hour before, has been found effective in one of our patients in preventing an attack from developing, but a dose of this size repeated on a chronic basis has regularly produced hypotensive episodes in four other patients.

Phenoxybenzamine, which is an alpha adrenergic blocker, has been used in 26 patients, particularly in those patients who have no catheters, as it is reputed to be effective also in relaxing the internal urethral sphincter thereby improving voiding. In four patients who carried large residual urines the dosage was gradually raised, at the suggestion of the urologist, to 80 mgm a day, that is to four times the usual dose. In spite of this the patients continued to exhibit autonomic dysreflexia on cystometric examination, and one had an acute attack secondary to bowel stimulation. Two of the patients complained of being very sleepy on this dose, and the voiding patterns did not improve. Whether or not phenoxybenzamine is really effective in the standard dosage in preventing chronic attacks in some patients is still not clear to us.

Diazoxide, which has a similar action to hydralazine, is very effective, by the parenteral route for the treatment of severe attacks which do not respond to general conservative measures.

Discussion and Conclusions

Kurnick (1956) in his discussion of autonomic dysreflexia quotes an incidence of 85 per cent in patients with lesions at T6 and above, and Guttman and Whitteridge (1947) found it in all the patients whom they specifically investigated for this condition, but in both instances the size of the samples was either not stated or was small. From our own observations of more than 200 patients it would seem that only about half of the patients at risk will develop attacks. Why the other half do not, we do not know. It would seem that the chances of developing A.D. are greater in patients with cervical lesions than in those with upper thoracic lesions but we could find no other factor such as age, sex or quality of care received which might help us predict whether or not a patient would be likely to develop A.D. We have been routinely performing cystometric examinations on all patients before starting the bladder retraining programme. This meant that cystometrograms have been obtained as soon as possible after admission. As a result of the finding that most patients do not develop A.D. until 4 to 6 months after injury, we are now repeating this test on all patients at intervals of 1-2 months, if the original examination reveals no change in the blood pressure during bladder filling. It is obviously preferable that the patient should experience the first attack of A.D. in a controlled situation so that the blood pressure does not go too high and he or she will be made aware of the signs and symptoms. Since we find that any patient with a lesion at T6 or above may develop A.D. even many years after injury, it is essential that all patients be educated during rehabilitation concerning the signs and symptoms, and what to do if they experience an attack. Moreover, with the increasing number of spinal cord injured persons now living and working in areas remote from the specialised units it becomes of greater importance to educate also the members of the general medical and nursing professions concerning this condition, since they may fail to recognise it in an emergency situation, and treat it inappropriately. To guard against such an event, we give all our A.D.-prone patients a small medical alert wallet card for the information of physicians or emergency room personnel whom they may have to contact during an attack.

Those of us working in large medical centres and in medical schools obviously have a responsibility to see that teachers and students of medicine know of this condition, its diagnosis and appropriate treatment. The diversity of possible presenting symptoms needs to be stressed, including the fact that bradycardia may not always be present. Any spinal cord injury patient presenting as an emergency or with strange symptoms should probably be suspected of having autonomic dysreflexia until proved otherwise.

Antihypertensive drugs presently available although very useful in the treatment of the acute attack when given intravenously do not appear to be so effective in preventing chronic attacks when used orally in doses small enough to avoid orthostatic hypotension. Controlled prospective studies are needed with antihypertensive agents which act specifically on the arteriolar smooth muscle.

At present the mainstay of therapy is still prevention, by attention to bowel and bladder care and the prevention of infections below the level of the lesion.

SUMMARY

In a survey of 213 patients with complete lesions of the spinal cord at and above T6 it was found that 48 per cent of them suffered from autonomic dysreflexia.

The incidence was higher in patients with cervical lesions than in those with upper thoracic injuries, but no other difference was noted between those affected and those not affected.

The condition usually manifested itself within a few months of injury but occasionally not until many years later. It appeared that affected patients could continue to experience attacks of the same severity following rehabilitation.

The value of preventive therapy with oral antihypertensive drugs was still unclear. An important and urgent problem is the education of the general medical and nursing professions so that this condition will be more readily recognised and appropriately treated.

RÉSUMÉ

En étudiant 213 malades avec la moelle traumatisée à un niveau supérieur à D7, on a constaté que 48% d'entre eux souffraient de l'hyperréflexie autonome.

La fréquence de cette complication était plus grande parmi les malades avec des lésions cervicales que parmi ceux avec des lésions dorsales supérieures, mais on n'a constaté aucune autre différence entre ceux qui étaient affectés et ceux qui n'en étaient pas.

La maladie se manifestait d'habitude pendant les premiers mois suivant l'accident, mais quelquefois le délai était de quelques années. Il paraît que les malades affectés continuaient à avoir des accès de la même gravité après la réhabilitation.

La valeur du traitement préventif avec des médicaments antihypertensifs par voie orale n'était pas encore claire. Un problème important et urgent est celui de l'éducation des professions médicales et des infirmières en général, pour que cet état soit reconnu plus promptement et traité correctement.

ZUSAMMENFASSUNG

In 213 Patienten mit Rückenmarksschädigung (vollständige Lähmung), deren Verletzung oberhalb T6 lag, war autonome Hyper-reflexia in 48% vorzufinden.

Das Vorkommen war grösser in Patienten mit cervikalen Verletzungen als in Patienten mit hohen thorakalen Verletzungen, sonst aber war kein Unterschied zu finden zwischen Patienten mit, und Patienten ohne autonome Hyperreflexia.

Der Zustand offenbart sich gewöhnlich innerhalb weniger Monate nach der Verletzung, aber manchmal nicht bis mehrere Jahre später. Anscheinend können diese Patienten Anfälle derselben Schwere auf unabsehbare Zeit nach ihrer Rehabilitierung haben.

Der Wert der vorbeugenden Behandlung mit antihypertensiven Medikamenten p.o. bleibt immer noch unklar. Ein wichtiges und dringliches Problem ist die Instruktion von Ärzten und Krankenschwestern, so dass die autonome Hyperreflexia besser erkannt und behandelt werden kann.

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