Post-ischaemic paraesthesiae in pellagrins

B. S. BOMB, H. K. BEDI, AND L. K. BHATNAGAR

From the Department of Medicine, R.N.T. Medical College, Udaipur, Rajasthan, India

SUMMARY A quantitative assessment of post-ischaemic paraesthesiae has been made in 50 pellagrins and 20 healthy identical controls. The results show a highly significant diminution of the paraesthetic response in pellagrins. In pellagrins having peripheral neuropathy the depression of paraesthesiae was more marked than in those without peripheral neuropathy. There was no consistent relationship between severity of peripheral neuropathy and degree of depression of paraesthetic response.

Post-ischaemic paraesthesia has been used in the evaluation of peripheral nerve function in many diseases including intoxication, vitamin deficiencies, alcoholism, malignant disease, and diabetes mellitus (Poole, 1956b); chronic liver disease (Seneviratne and Peiris, 1970); uraemia (Christensen and Ørskov, 1969); and motor neurone disease (Shahani and Russell, 1969). However, in pellagra, where neurological manifestations are quite common, no such study is available.

Methods

Fifty patients with pellagra admitted to the General Hospital, Udaipur and 20 healthy control subjects of identical age and sex were studied. The diagnosis of pellagra was made on clinical grounds and was confirmed by decreased 24 hour urinary excretion of N¹ methyl nicotinamide (less than 2 mg). The N¹ methyl nicotinamide was estimated by the technique of Sarett (1943).

A careful clinical examination was carried out on all subjects by one of us to assess peripheral nerve functions. To quantify the severity of clinical peripheral neuropathy, scoring was done as described by Seneviratne *et al.* (1973)—that is, each symptom and sign was given a score of one, and on the basis of total scoring the patients were divided into four groups:

- 0: No clinical peripheral neuropathy
- 1-5: Mild peripheral neuropathy
- 6-10: Moderate peripheral neuropathy
- 11-15: Severe peripheral neuropathy

In all subjects vascular occlusion was effected by rapid inflation of an ordinary sphygmomanometer cuff to a pressure of 60 mmHg above the resting systolic blood pressure. This was maintained for 20 minutes and then the cuff pressure was released. All subjects were instructed at the beginning of the vascular occlusion to report the time of onset, nature, and time of cessation of any subjective sensations that they experienced during the ischaemic and post-ischaemic periods. During the post-ischaemic period they were reminded at regular intervals of the need to report the details of any paraesthesiae experienced, special attention being paid to the 'pins and needles' type of post-ischaemic paraesthesia.

A quantitative assessment of the 'pins and needles' paraesthesia was made by determining a post-ischaemic paraesthesiae (PIP) index (Seneviratne *et al.*, 1973). For calculating the PIP index a PIP score was determined depending upon the intensity of paraesthesiae as described below and then this score was multiplied by the duration in minutes of 'pins and needles' paraesthesia. The reasons for selecting 'pins and needles' paraesthesia alone for determining the PIP score were its easy recognition, constancy, reliability, and reproducibility.

PIP SCORE

- 0: No post-ischaemic 'pins and needles' paraesthesia
- 1: Mild 'pins and needles' paraesthesia confined to fingers
- 2: Severe paraesthesiae felt in the fingers and palm, usually accompanied by other reactions of discomfort or distress, such as, grimaces or whistling

¹Address for reprints: Dr B. S. Bomb, H. No. 38, Surana Street, Udaipur-313001, Rajasthan, India.

Accepted 1 October 1976

Results

In the present study, there were 28 male and 22 female patients of 15 to 70 years of age. Most of the cases belonged to the fourth and fifth decades. The duration of illness varied from 15 days to six months. All patients studied were from a rural area and were vegetarian. The main cereal they consumed was maize. Severe peripheral neuropathy was present in five cases, moderate in six cases, and mild in 15 patients. The remaining 24 patients had no manifestation of peripheral neuropathy.

The mean PIP index in the control group was 26.4 ± 7.2 , while in pellagrins it was 12.3 ± 5.4 (Table 1). The mean PIP indices in pellagrins without neuropathy and in those with neuropathy were 15.4 ± 4.6 and 6.2 ± 3.8 respectively. Comparison of the results obtained from healthy control subjects with that of the pellagrins, and of pellagrins with and without peripheral neuropathy shows a very highly significant ($p < 0.001$) difference of PIP indices between these groups.

There does not seem to be a consistent relationship between the severity of peripheral neuropathy and the degree of diminution of the PIP index (Table 2). Thus, among the 16 pellagrins who had a PIP index between 1 to 5, there were four subjects who had no clinical evidence of peripheral neuropathy, while among the seven pellagrins who had a PIP index of more than 15 (near normal) there were three who had evidence of mild or moderate peripheral neuropathy.

Table 1 PIP indices in controls, pellagrins with neuropathy, and pellagrins without neuropathy

	No.	Mean	Range	Standard deviation
Healthy controls	20	26.4	16-34	± 7.2
Pellagrins	50	12.3	2.8-26.2	± 5.4
Significance of difference of PIP index between controls and pellagrins, <i>t</i> test (<i>p</i>)		< 0.001		
Peripheral neuropathy				
Pellagrins without	24	15.4	3.6-26.2	± 4.6
Pellagrins with	26	6.2	2.8-10.0	± 3.8
Significance of difference of PIP index between pellagrins without and pellagrins with peripheral neuropathy, <i>t</i> test (<i>p</i>)		< 0.001		

Discussion

Of 50 pellagrins studied, 26 (52%) had mild to severe peripheral neuropathy. This observation is in agreement with that of Spies *et al.* (1937) who

Table 2 PIP indices in pellagrins according to severity of peripheral neuropathy

PIP index	Number of subjects	Neuropathy			
		Nil (24)	Mild (15)	Moderate (6)	Severe (5)
1-5	16	4	6	3	3
6-10	15	7	5	1	2
11-15	12	9	2	1	0
> 15	7	4	2	1	0

have reported an incidence of 62%, while it is contradictory to the experience of Shah *et al.* (1971) who are of the opinion that peripheral neuropathy is uncommon in pellagra. This discrepancy may be due to differing criteria for the diagnosis of peripheral neuropathy. For example, Shah *et al.* (1971) did not consider that paraesthesiae (present in 64.3% of their cases) was evidence of peripheral neuropathy. In the present study, we have considered spontaneous paraesthesiae to be evidence of peripheral neuropathy.

In control subjects the PIP index ranged from 16 to 34 with a mean of 26.4. These figures are similar to those of Seneviratne *et al.* (1973). In pellagrins the PIP index ranged from 3.8 to 26.2 with a mean of 12.3. The difference between the PIP indices of the control and the pellagrin group is very highly significant, only seven of the 50 pellagrins having an index that lay within the range of normal values. The results also show that pellagrins with peripheral neuropathy tend to have significantly lower indices than those without it, but there was no consistent relationship between the severity of peripheral neuropathy and depression of post-ischaemic paraesthesiae. There is no available study in the literature on post-ischaemic paraesthesiae in pellagrins.

The results obtained in control subjects confirm the observation of Poole (1956a) and Seneviratne *et al.* (1973) that paraesthesiae can consistently be elicited in healthy subjects. The diminished response of post-ischaemic paraesthesia in 43 of 50 pellagrins further indicates that peripheral nerve dysfunction is very common in pellagrins. The observation that there is no consistent relationship between the severity of clinical peripheral neuropathy and depression of post-ischaemic paraesthesiae response supports the experimental observations of Seneviratne and Peiris (1968) and Gregersen and Pilgaard (1971) that increased resistance to ischaemia does not necessarily follow demyelination and, conversely, that nerves which exhibit no clinical or conventional electrophysiological evidence of dysfunction may exhibit increased resistance to inactivation by ischaemia.

We wish to thank the subjects for their co-operation and Professor M. L. Gupta, Principal and Controller, R.N.T. Medical College, Udaipur, for his kind permission to publish this work. We are also grateful to other physicians at General Hospital, Udaipur, for the opportunity to study the patients under their care.

References

- Christensen, N. J., and Ørskov, H. (1969). Vibratory perception during ischaemia in uraemic patients with mild carbohydrate intolerance. *Journal of Neurology, Neurosurgery, and Psychiatry*, **32**, 519–524.
- Gregersen, G., and Pilgaard, S. (1971). The effect of ischaemia on vibration sense in hypo- or hypercalcemia and on demyelinated nerves. *Acta Neurologica Scandinavica*, **47**, 71–79.
- Poole, E. W. (1956a). Ischaemic and post-ischaemic paraesthesiae: normal responses in the upper limb with special reference to the effect of age. *Journal of Neurology, Neurosurgery, and Psychiatry*, **19**, 148–154.
- Poole, E. W. (1956b). Ischaemic and post-ischaemic paraesthesiae in polyneuritis. *Journal of Neurology, Neurosurgery and Psychiatry*, **19**, 281–288.
- Sarett, H. P. (1943). A direct method for the determination of N¹ methyl derivatives of nicotinic acid in urine. *Journal of Biological Chemistry*, **140**, 159–164.
- Seneviratne, K. N., and Peiris, O. A. (1968). The effect of ischaemia on the excitability of sensory nerves in diabetes mellitus. *Journal of Neurology, Neurosurgery, and Psychiatry*, **31**, 348–353.
- Seneviratne, K. N., and Peiris, O. A. (1970). Peripheral nerve function in chronic liver disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, **33**, 609–614.
- Seneviratne, K. N., Sananayake, N., and Nimalasuriya, A. (1973). Post-ischaemic paraesthesiae in diabetes mellitus. *Journal of Neurology, Neurosurgery, and Psychiatry*, **36**, 288–295.
- Shah, D. R., Singh, S. V., and Jain, I. L. (1971). Neurological manifestations in pellagra. *Journal of Association of Physicians of India*, **19**, 443–446.
- Shahani, B., and Russell, W. R. (1969). Motor neurone disease. An abnormality of nerve metabolism. *Journal of Neurology, Neurosurgery, and Psychiatry*, **32**, 1–5.
- Spies, T. D., Chinn, A. B., and McLester, J. B. (1937). Severe endemic pellagra: a clinical study of fifty cases with special emphasis on therapy. *Journal of the American Medical Association*, **108**, 853–857.