pooled values of individual jitter were compared between study and control groups. SFEMG examination in radiculopathy patients did not show a definite jitter abnormality in any individual patient. Three patients had borderline increases of mean MCD, but these were within the normal limit values of Trontelj et al.[4] According to Stålberg, the severity of jitter and blocking abnormality gives information about the rate of denervation–reinnervation process.[2] In cases of fast progression, such as amyotrophic lateral sclerosis, jitter is increased and blocking is commonly seen.[2] Our results show that this process is slow in radiculopathies, as supported by the observation that the patients with lesser symptom duration demonstrate significantly increased individual jitter.

Unlike other axonal disorders, jitter increase was not prominent in radiculopathy. This may be explained by the sole involvement of the posterior primary ramus in radiculopathy, where myotomal overlap makes localization inaccurate.[5] Forty-five percent of our patients had only paraspinal abnormalities. However, our patients with solely anterior primary ramus involvement did not show a significant increased abnormality in individual jitter. The method employed may also play a role, because, in myasthenia gravis stimulation SFEMG yielded fewer abnormalities than the voluntary activation method.[1] On the other hand, the older age of our affected subjects compared with controls does not explain the increased jitter, because jitter does not increase with age in stimulation SFEMG.[3]

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THE CALCIUM CHANNEL BLOCKER VERAPAMIL IN HYPOKALEMIC PERIODIC PARALYSIS

Hypokalemic periodic paralysis (HypoPP) is an autosomal-dominant disorder belonging to the ''muscle ion channel diseases.''[3] The channel involved in HypoPP is supposed to be a calcium channel, as point mutations in the gene for the dihydropyridine (DHP) receptor CACNL1A3 localized on chromosome 1q31-32.[2] However, the relation between genetic defect and clinical features is unclear and therapy is empirical. The reported positive effect of the Ca-channel antagonist, verapamil, in one patient[1] has prompted us to investigate the effect of verapamil in patients with HypoPP.

Nine patients from two unrelated Dutch families with HypoPP were tested (see Table 1). All patients were using acetazolamide and potassium chloride before the study. Patients 1–5 stopped their acetazolamide intake 3 weeks before the study. Patients 6–9 refused to discontinue their acetazolamide use. During the study period, no other medication was used except potassium chloride, the intake of which was recorded. Verapamil (240 mg daily) and placebo (lactose) were given in randomized order for 3 weeks each using a crossover design. The patients were seen in the first and last week of each treatment period. At each visit, blood pressure, possible side-effects, ECG, and muscle strength of 13 muscle groups, measured by a handheld dynamometer, were assessed. Blood samples were taken at each visit to measure serum electrolytes and creatine kinase activity. The patients recorded daily fluctuations and potassium use. A partial attack of muscle weakness was defined as a decline in muscle strength of one or two limbs interfering with normal daily activities, and a total attack defined as tetraparalysis.

The assessment of effect of therapy was based on a comparison of muscle strength measurements (sum of left and right, primary outcome measure), number of paralytic attacks, and potassium use (secondary outcome measure), during 3 weeks on placebo and 3 weeks on verapamil.

The Wilcoxon matched-pair signed-rank test was used for statistical analysis (significance $P < 0.05$).

Muscle strength measurements were not significantly different between the treatment and the placebo period when the results of all the different muscle groups were combined. Only one of the patients (no. 6) mentioned a subjective increase in muscle strength and feelings of well-being during the verapamil period. Although this was supported by objective measurements, it was outweighed by contrasting findings in other patients.

During the study period only partial attacks were reported. The cumulative number of partial attacks in the verapamil period was 13 compared with 23 in the placebo period (not significant) and could be attributed to patient nos. 1 and 2 (see Table 1).

Serum potassium and CPK values were similar in the treatment and placebo periods. ECG abnormalities and side-effects were not found. There was no difference in potassium intake between the two periods.

In conclusion, this small trial was unable to document a beneficial effect of verapamil in HypoPP. However 2 patients showed considerable subjective and objective benefit. We suggest that verapamil may be tried in patients with HypoPP that is resistant to conventional treatment.

Table 1. Characteristics of the patients and results.

Patient no.	Patient characteristics						Potassium intake (mmol)		Attacks of weakness	
				Normal attack frequency						
	mutation	Age	Gender	Partial	Total	Medication	V	P	V	P
1	Arg528His	20	M	1/wk	1/yr	Potassium	—	150	3	9
2	Arg528His	45	M	1/wk	—	Potassium	650	650	5	8
3	Arg528His	42	F	—	—	Potassium	1200	1200	*	*
4	Arg528His	44	F	—	—	Potassium	—	—	*	*
5	Arg1239His	35	F	1/2 mo	1/3 yr	Potassium	273	256	0	1
6	Arg1239His	42	F	—	—	Potassium acetazolamide	1688	1715	0	1
7	Arg1239His	39	M	—	4/yr	Potassium acetazolamide	999	838	1	0
8	Arg1239His	38	M	1/d	1/yr	Potassium acetazolamide	2058	1826	1	3
9	Arg1239His	28	F	—	1/yr	Potassium acetazolamide	184	176	3	1

*F = female; M = male; d = day; wk = week; yr = year. Potassium intake was based on complaints, varying from one to three times daily and before going to bed to once a week or month. Acetazolamide was used 1–3 × 125 mg daily. *Daily complaints of fluctuating muscle strength. Duration of partial attacks 4–12 h, of total attack 10–20 h.*

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