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neurological diseases.¹ We describe a patient with cerebellar tumour who developed bilateral hypergeusia. The possibility of hypergeusia as a symptom of a posterior fossa lesion is emphasised.

A 73 year old man was admitted to our hospital because of taste and gait disturbances. He was well until two months earlier, when he noted that food was too sweet and salty. His wife cooked the food and did not increase doses of sugar and salt for flavour. The patient related that the food tasted two to three times as sweet and salty as before. His wife tried to season the food with smaller doses of sugar and salt. However, his taste disturbance persisted. One month before admission, unsteady gait developed and slowly progressed.

On examination the patient was alert and oriented. The taste disturbance persisted. Mild dysphagia and poor gag reflex were present. Numbness was present in the fingers and toes of the four limbs. There was mild right hemiparesis. Marked truncal ataxia was present with mild ataxia in the four limbs. Deep tendon reflexes were increased in the right limbs, and plantar reflexes were flexor. There were no clinical signs and symptoms of adrenocortical insufficiency. Taste was examined by a filter-paper disc method that measures threshold of taste,² namely, small filter-paper discs (5 mm in diameter) are impregnated with diluted solutions (five gradations) of the four cardinal taste substances: sugar, salt, tartaric acid and quinine hydrochloride and used for qualitative and semi-quantitative tests. He recognised bilaterally the tastes at the lowest concentrations (8×10^{-4} molar for sugar, $5 \cdot 1 \times 10^{-3}$ molar for salt, $1 \cdot 3 \times 10^{-4}$ molar for tartaric acid and $2 \cdot 5 \times 10^{-6}$ molar for quinine hydrochloride). The results indicated lowered threshold of the four qualities compared with Japanese controls.² One week after the first examination, retesting of taste function showed similar results.

Routine laboratory examinations were normal. Plasma cortisol and ACTH values were also normal. A head CT revealed a mass lesion involving the right brachium pontis and right cerebellar hemisphere with a displacement of the brainstem. Biopsy from the right cerebellar hemisphere showed the tumour to be a glioblastoma multiforme. The taste disturbance lasted until radiation and steroid therapy was started.

Hypergeusia is rare and has been given little attention. Rollin¹ reported one patient with multiple sclerosis who had unilateral hypergeusia and hyperpathia on one side of the body. But the responsible lesion was not clear. In adrenocortical insufficiency, the

taste threshold is lowered.³ Aspirin has been reported to increase the perceptibility of bitter taste.³ Clinical data of our case indicated that adrenocortical insufficiency was not likely and aspirin was not given. All taste fibres are distributed to the nucleus of the solitary tract in the medulla oblongata. The central gustatory pathway from the nucleus ascends without decussation in the homolateral pontine tegmentum.⁴ CT showed a displacement of the brainstem by the cerebellar tumour. We could not attribute the hypergeusia to any other cause except for the posterior fossa lesion. Although the mechanism is not known, hypergeusia may be a manifestation of a posterior fossa lesion.

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Palilalia as a symptom of levodopa induced hyperkinesia in Parkinson's disease

Sir: Souques¹ reported on a particular disturbance of language in a patient with stroke leading to left-sided hemiplegia, which presented as compulsive repetition of semantically adequate answers to the examiners' questions. Neither dysarthria nor aphasia affected the speech of the subject. This symptom, termed palilalia by Souques, was also observed in postencephalitic Parkinsonism, but on the whole it must be considered a

rather rare symptom of this disease (for reviews see refs 2, 3). Postmortem examinations have suggested lesions of the striatum as the anatomical substratum of palilalia.⁴

In the cases reported so far palilalia was either constantly present or varied in degree; it occurred both in spontaneous speech and in replying to questions; the number of repetitions usually ranged between four and eight. Reiterations comprised syllables, words, or sentences. Often the verbal repetitions tended to be uttered with increasing rapidity and decreasing loudness.

Besides sporadic manifestations within postencephalitic Parkinsonism, palilalia has also been observed in pseudo-bulbar palsy, Gilles de la Tourette syndrome, Pick's disease, traumatic lesions of the basal ganglia, idiopathic bilateral cerebral calcinosis or in syndromes of unknown aetiology.⁵⁻⁸ As far as we know, the occurrence of palilalia has hitherto not been documented in idiopathic Parkinsonism; rather palilalia was considered a pathognomonic sign of postencephalitic Parkinsonism.¹⁰ In the following report we describe palilalia in a patient with idiopathic Parkinson's disease, manifesting itself in temporal relationship to peak-dose hyperkinesia.

The now 68 years old patient suffered from idiopathic Parkinson's disease of a predominantly akinetic-rigid symptomatology since 1974. In the recent years fluctuations in motor performance, manifesting as frequent shifts from akinesia to choreic hyperkinesia and dystonia developed under treatment with levodopa (800-1200 mg per day, administered usually in eight single doses) and bromocriptine (20-30 mg per day). No tremor was present. Neurological examination revealed no other pathological findings. CT showed minimal cortical atrophy.

Our observations were made in a series of three speech examinations performed at 3-months intervals. Each examination lasted for between 40 and 50 minutes and included the recording of a stretch of spontaneous speech and of word- and sentence-repetitions as well as tests of oral diadochokinesis (rapid syllable repetitions) and sustained phonation. The first and third investigation started after intake of 125 mg levodopa, this dose being part of the daily medication schedule. In contrast, the second examination began about one hour after drug intake. Before drug intake the patient presented with mild akinetic symptoms. His speech was characterised by uniform pitch and loudness, accelerated tempo and sometimes imprecise articulation. Often, the patient did not wait until the examiner had finished with his

utterance, but began to speak as soon as he could anticipate the stimulus. After drug intake moderate choreic hyperkinesia, especially of the head, could be observed. In the second examination the legs and the trunk were also included.

Palilalia was observed during the first and the third examination, occurring at a delay of about 30 minutes after drug intake. In the second examination, where drug intake was one hour before the beginning of the speech examination, no palilalic signs were observed. The periods of palilalia in sessions one and three lasted for about 10 minutes and were in both sessions interrupted by the examiner. During these periods, all stimuli presented by the examiner were, without exception, repeated involuntarily several times. In the case of single word repetition tasks (first examination), the whole target word was reiterated four to eight times. In sentence repetition (third examination), the second half of each sentence (five from eight syllables) was repeated two to four times. On occasions, the patient produced spontaneous comments interspersed between repetition tasks. These propositional utterances showed no palilalic disturbance and were uttered only once. In no case did the chain of repetitions of an item span over more than one respiratory cycle. In about one third of the sequences of reiterations a slight decrease of loudness and/or increase of speech tempo could be noted by means of auditory evaluation. This tendency also showed up in measurements of vocal intensity (sound pressure level) and word durations (fig). Further, there was a perceptible decrease in vocal pitch and in articulatory precision within most reiteration chains, which was also corroborated by speech signal analyses (data not presented). Opposite to this general degradation tendency, however, there were instances where the patient achieved a resetting of these parameters within a sequence of repetitions, that is loudness and pitch were raised, articulatory precision improved and/or word duration increased (see fig for an example).

The symptom reported here should at first be differentiated from other types of repetitive verbal behaviour in neurological disorders of language production. It can be distinguished from verbal perseveration, since in all cases the stimulus was repeated correctly in the first instance and involuntary repetitions of stimuli given earlier never occurred. Further, the patient was not echolalic, since he exclusively repeated his own utterances ("auto-echolalia"), and occasional remarks of the examiner between two successive repetition tasks were not

echoed. The reiterations of our patient did not show the characteristic features of acquired ("cortical") stuttering, that is effortful speech or repetitions and prolongations of initial sounds or syllables.¹¹ Finally, differentiation of the repetitive verbal behaviour in our case from aphasic recurrent utterances or schizophrenic verbigeration (verbal stereotypy) goes without saying.

In our patient there was a clear-cut temporal relationship of palilalia to levodopa intake. Palilalia appeared about 30 minutes afterwards, a time interval corresponding to maximum plasma concentration level and delay of action following oral medication with levodopa.^{12,13} Synchronously with palilalia the patient presented hyperkinetic signs, especially of the head, which most probably have to be interpreted as peak-dose dyskinesia.¹⁴ It is known that not only in the acute inflammatory state of encephalitis lethargica, but also within postencephalitic

Parkinsonism hyperkinetic signs such as tic or choreic movements sometimes occurred.¹⁵ But the reports about postencephalitic palilalia give no information about possible hyperkinetic symptoms. A connection between hyperkinesia and palilalia occurs in Gilles de la Tourette syndrome,⁵ and for patients with idiopathic cerebral calcinosis too, a parallel course of palilalia and hyperkinesia was observed.⁶ Thus, it may be conceived that, from the viewpoint of pathophysiology, palilalia is bound to hyperkinesia and not part of Parkinsonism as an akinetic-rigid syndrome. This concept would explain why palilalia is a rare phenomenon within Parkinsonian syndromes.

It should be stressed that our patient presented palilalia only in repetition tasks and not in spontaneous speech as the postencephalitic Parkinsonian patients usually did. In contrast to our observation, LaPointe and Horner⁷ found an even lower incidence of reiterations in repetition tasks than in spontaneous speech, reading, or picture description. It is generally assumed that palilalia does not affect nonpropositional speech, such as recitations, counting, or naming the letters of the alphabet.^{16,17} Our results point at a differential effect with respect to a further dimension, namely the modality dimension. The special nature of the modality of verbatim repetition has been demonstrated in several studies of normal (for example McLeod and Posner¹⁸) and abnormal (for example Friedrich *et al.*¹⁹) speech production. These investigations have provided evidence for a direct link between the auditory percept of a word and its motor representation, a loop which seems to have a certain autonomy in that it is not subject to interference from competing tasks. In the case presented here it was obviously this particular auditory-articulatory link which, once activated in the repetition task, was set into palilalic resonances.

In accord with previous reports (for example Critchley²), our patient presented a tendency for acceleration, articulatory degradation, and decreasing pitch and loudness during the course of reiterating an utterance, that is the repetitions were not realised as phonetically identical copies. Such changes may, at least partially, be ascribed to the fact that the patient was gradually running out of breath during the repetition sequences, that is to constraints of motor execution. But sometimes the patient improved again both in articulatory accuracy and in vocal parameters, yet without being able to interrupt the reiterative loop. This observation has not been reported before, probably

Figure Changes in word duration (top) and peak intensity (bottom) in palilalic reiterations of five test words of different lengths.

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since most communications were not based upon detailed phonetic characterisation of changes along repetitions. An exception is a case report of assumed simple schizophrenia,²⁰ documenting that there was no consistent decrease in either word duration and intensity along repetition trains (see also LaPointe and Horner⁷). Presumably, our finding of an improvement in articulatory and vocal performance along repetition trains does not reflect voluntary control, since this would not conform with the patient's inability to interrupt the repetition loop. A more plausible explanation would rather ascribe the observed resetting of phonetic parameters to spontaneously occurring shifts of psychomotor drive ("energising").

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Ophthalmoplegic migraine with bilateral involvement

Sir: Ophthalmoplegic migraine is characterised by attacks of unilateral headache and transient dysfunction of one or more ocular motor nerves. To our knowledge, bilateral involvement in a single attack has not previously been described.

A 37 year old Caucasian woman had experienced migraine attacks approximately

twice monthly since aged 12 years. Usually 2 hours scintillating photopsia was replaced by left frontal headache, with vomiting, diarrhoea and occasional tingling of hands and feet. Her mother experienced frequent similar episodes. The patient suffered a typical attack. At its onset she took her usual therapy of two capsules, each containing 1 mg ergotamine tartrate, 5 mg prochlorperazine, 5 mg chlorthalidone, 250 mg paracetamol, 250 mg aspirin and 8 mg codeine. The following day she awoke with left ptosis and diplopia.

Visual acuity was 6/6 in each eye, visual fields were full and the optic fundi were normal. There was moderate left ptosis. Both saccadic and pursuit eye movements were abnormal. Gaze to left, right and downwards was limited to about 10° (fig). Upgaze was impossible, and convergence severely limited. Abduction was slightly greater than adduction on right lateral gaze, although eyes were otherwise conjugate. No improvement occurred with oculoccephalic testing. Both pupils were 5 mm diameter and unresponsive to light and accommodation. The remainder of a full neurological and general medical examination was normal.

Blood count, ESR, automated biochemistry, treponemal serology, red cell transketolase and chest radiograph were normal. Computed tomography of brain with and without contrast enhancement was unremarkable, as was bilateral carotid and left vertebral angiography. Cerebrospinal fluid contained no cells and oligoclonal banding was absent, although protein was 0.69 g/l (0.15-0.40) and IgG: albumin ratio 0.29 (0-0.10). Nerve conduction studies and visual evoked potentials were normal.

One week later, ptosis and headache had largely resolved, pupil reactions to light and convergence had returned, and range of eye movements had increased. Ocular movements, however, had become more dysconjugate with adduction being 5° greater than abduction on left and right lateral gaze, and depression of the left eye being 5° less than the right on downgaze.

By 10 weeks the ophthalmoplegia had resolved. Pizotifen was then administered and resulted in reduced headache frequency. There has been no recurrence of ophthalmoplegia after five years.

In 1882 Saundby¹ described a young woman with recurrent migraine and ophthalmoplegia. Eight years later Charcot² labelled a similar case as "migraine ophtalmoplegique". Many early cases, however, were subsequently discovered to have structural intracranial pathology, prompting development of diagnostic criteria. Those of