

Impairment of vertical motion detection and downgaze palsy due to rostral midbrain infarction

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Received December 7, 1989 / Received in revised form May 23, 1990 / Accepted August 2, 1990

Summary. We present two cases with acute onset of vertical gaze palsy, mainly consisting of impaired downgaze and apraxia of downward head movements, together with neuropsychological deficits (hypersomnia, impaired attention and disorders of memory and affective control). CT and MRI revealed bilateral post-ischaemic lesions in the dorsomedial thalamus and the mesodiencephalic junction, dorsomedial to the red nucleus, thus being restricted to the territory of the posterior thalamo-subthalamic paramedian artery, which includes the region of the rostral interstitial nucleus of the medial longitudinal fascicle as the main premotor nucleus for the generation of vertical saccades. In our patients, oculographic examination with electro-oculography and magnetic search coil recording showed severe impairment of downward more than upward saccades and only minor deficits of vertical pursuit and the vestibulo-ocular reflex. Visual functions were normal, with one exception: a psychophysical test of motion perception revealed a significant deficit in the detection of vertical movements. This could be due to a central adaptive mechanism which, in order to minimize oscillopsia, might elevate thresholds for vertical motion perception in cases of vertical gaze palsy. As an alternative explanation, lesions within the midbrain tegmentum could have damaged subcortical visual pathways involved in motion perception.

Key words: Vertical motion detection – Downgaze palsy – Eye-head coordination – Paramedian mesodiencephalic syndrome – Midbrain infarction

Introduction

In the literature, the rare syndrome of downgaze paralysis has been described by several authors [1, 10, 22, 28, 29, 33, 35, 38, 42, 48, 54, 56, 58]. From lesion and stimu-

lation studies in monkeys [5, 34] as well as from autopsy findings in humans [1, 6, 28, 50, 55] it may be concluded that the syndrome is due to a bilateral ischaemic lesion of a butterfly-shaped area in the mesodiencephalic junction, dorsomedial to the red nucleus and adjacent to the caudal part of the third ventricle and the aqueduct, also including parts of the medial thalamus, especially the dorsomedial nucleus and the centrum medianum. Infarctions of this location are caused by occlusion of one of the thalamo-perforating arteries, namely the posterior thalamo-subthalamic paramedian artery [55], also called the paramedian thalamic artery [45, 46] or the deep interpeduncular artery [53]. Bilaterality of lesions is due to the fact that the arteries for both sides frequently originate from a common trunk which is a branch of the P1 segment of the posterior cerebral or the basilar artery. Because of the thalamic lesions, this so-called paramedian mesodiencephalic syndrome [8] includes – besides downgaze palsy – neuropsychological deficits, such as hypersomnia (after an initial coma), amnesia and affective disorders [35].

Even if the neuropsychological symptoms are less pronounced, patients with downgaze palsy are often markedly impaired in visuospatial behaviour, more than those with upgaze palsy, possibly because in everyday life, as for example during walking or reading, eye movements into the lower field of gaze are more important than those into the upper field. Furthermore, it has been reported that midbrain infarctions with vertical gaze palsy can be combined with deficits in visual perception [17, 21]. In order to investigate this more specifically, we performed a psychophysical test of motion perception in two patients with downgaze palsy. In addition, to achieve a more precise recording of vertical eye movements than in previous investigations, we applied the magnetic search coil technique.

Case reports

Case 1

A 54-year-old woman suddenly noticed occipital headache, blurred vision, diplopia and an instability of gait, followed by amnesia

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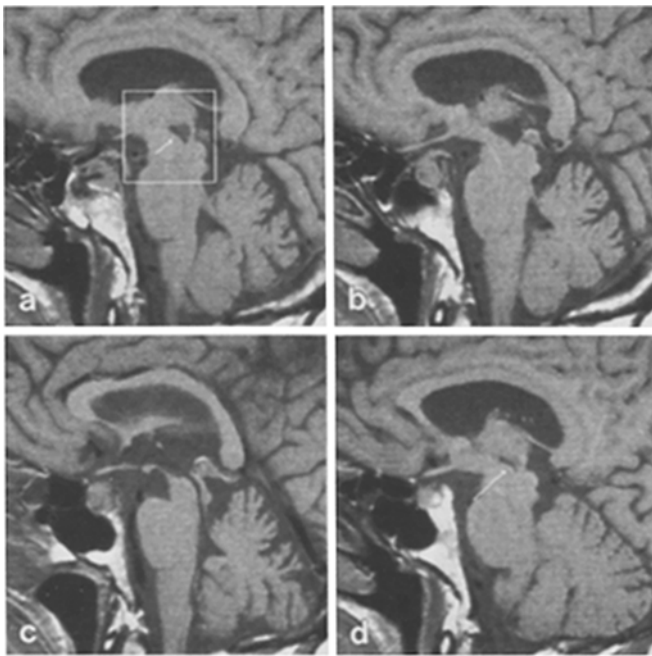


Fig. 1a-d. MRI in case 1, 1.5 T. Two-dimensional spin-echo technique (TR = 600 ms, TE = 15 ms). Slice thickness: 2 mm. Slice positions: **a, b** 6/4 mm right parasagittal; **c** mediosagittal; **d** 6 mm left. Lesions are indicated by *arrows*. The region outlined by the square in **a** is illustrated in Fig. 2a

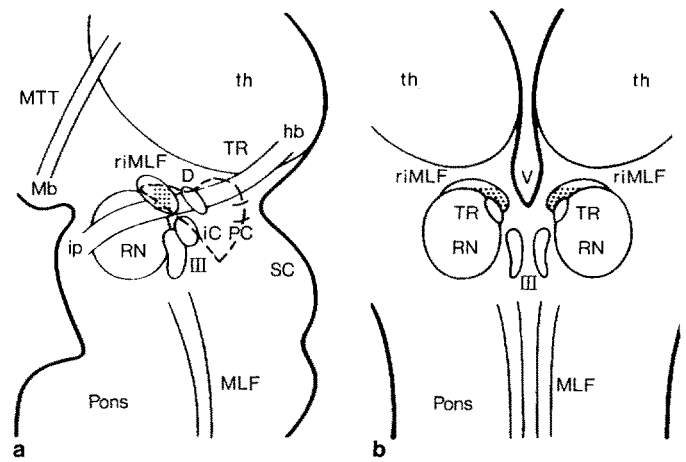


Fig. 2. Anatomical scheme of the mesodiencephalic region in a parasagittal (**a**) and coronal **b** plane. The broken line in **a** is the outline of the right paramedian lesion in case 1, corresponding to Fig. 1a. The *dotted zone* is the most commonly lesioned area in cases of downgaze palsy [48]. *d*, Nucleus of Darkschewitsch; *hb*, habenula; *iC*, interstitial nucleus of Cajal; *ip*, interpeduncular nucleus; *Mb*, mammillary body; *MLF*, medial longitudinal fascicle; *riMLF*, rostral interstitial nucleus of the MLF; *MTT*, mammillo-thalamic tract; *PC*, posterior commissure; *RN*, red nucleus; *SC*, superior colliculus; *th*, thalamus; *TR*, tractus retroflexus; *V*, third ventricle; *III*, oculomotor nuclear complex

for about 36 h. Previously she had always been in good health. The only vascular risk factor was occasional cigarette smoking.

On admission to the hospital about 20 h after onset of symptoms, she was somnolent and only partly oriented, showed a slight left-beating spontaneous nystagmus and dysarthric speech. One day later, she had fully regained consciousness, but still showed deficits of arousal and vigilance, short-term memory and affective control. Neurological examination revealed a complete downgaze palsy and incomplete upgaze palsy, with inability to perform vertical saccades, whereas vertical pursuit and vestibulo-ocular reflex (VOR) were still functioning, though with a reduced gain and amplitude. Furthermore, the patient had impaired convergence, slight divergent strabism and gaze-evoked nystagmus upwards and – to a lesser extent – leftwards. During the first 2 weeks, she was unable to move her head downwards actively, which, in combination with the palsy of downward saccades, prevented her from fixating any target below the horizontal meridian. Passive head motility, however, and head position during sleep were normal. In addition to the oculomotor symptoms the patient showed static and locomotor ataxia, which was more pronounced with the eyes open, because then she experienced vertical oscillopsia during walking and head movements. During the first 10 days, visuospatial orientation was impaired so that she had difficulty in finding directions and localizing objects and persons in extrapersonal space. Pupil reactions, visual acuity and visual fields were normal. The EEG initially showed a mild paroxysmal dysrhythmia. Continuous-wave Doppler sonography of the extracranial vessels and cerebrospinal fluid were normal. CT revealed post-ischaemic lesions of diminished density in both medial thalami, in the posterior limb of the right internal capsule and bilaterally at the mesodiencephalic junction. Four years after the infarction, lesions could be localized exactly by magnetic resonance imaging (MRI, 1.5 T), using 2D-spin-echo and 3D-FISP techniques [43] in sagittal and axial planes. The paramedian sagittal T1-weighted slices showed sickle-shaped lesions of low signal intensity on both sides, a smaller one on the left (Fig. 1d) and a larger one on the right (Fig. 1a), which – as illustrated in Fig. 2 – were located dorsomedial to the red nucleus

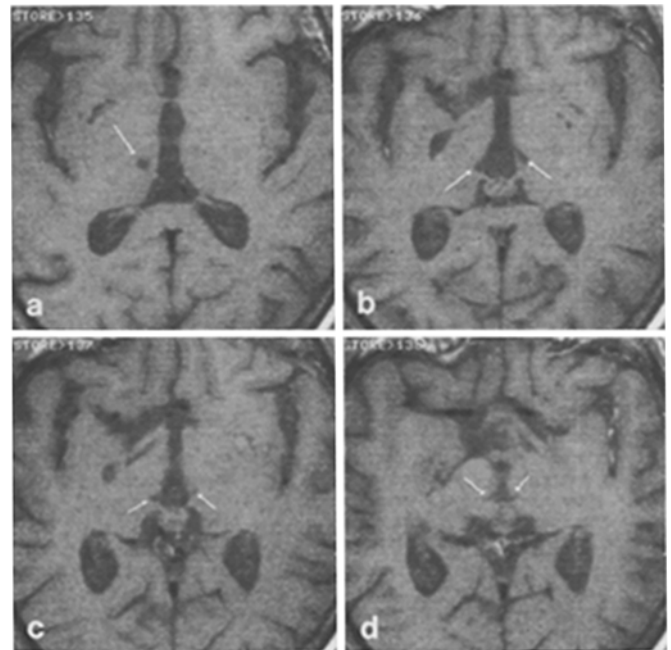


Fig. 3a-d. Spin-echo technique, as in Fig. 1, but in axial plane. Slice positions in craniocaudal sequence: **a** –6 mm; **b** –2 mm; **c** 0; **d** +2 mm

and included the rostral interstitial nucleus of the medial longitudinal fascicle (riMLF), the nucleus of Darkschewitsch (D) and possibly a section of the interstitial nucleus of Cajal (iC). On the axial slices (Figs. 3, 4), lesions were identified in both medial thalami (Figs. 3a, b, 4a, b) and around the caudal part of the third ventricle, which consequently was widened towards both sides (Figs. 3c, d, 4c, d; in parasagittal projection see Fig. 1b). Corresponding to

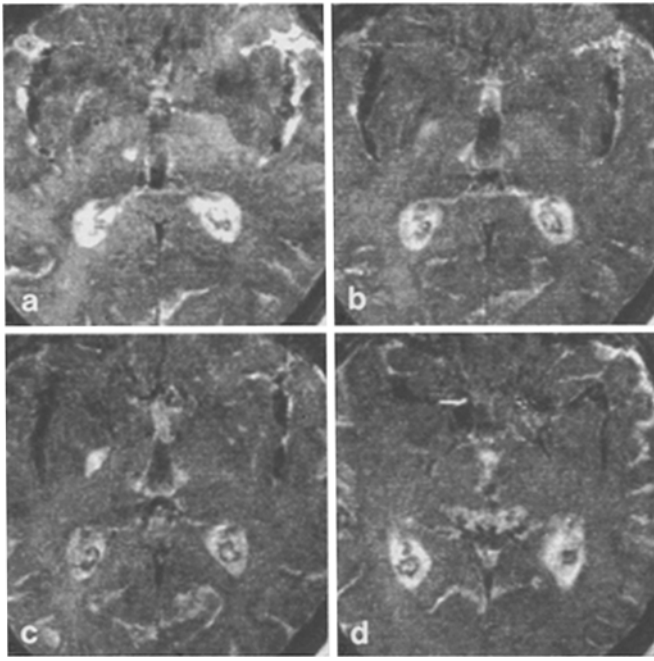


Fig. 4a–d. Three-dimensional FISP technique (TR = 20 ms, TE = 10 ms, Flip angle: 30°). Slice thickness: 2 mm. Slice positions (in axial planes like Fig. 3): **a** –6 mm; **b** –4 mm; **c** –2 mm; **d** +2 mm

the T1-weighted signal decrease (Fig. 3), lesions were outlined as areas of signal increase on T2-weighted 3D-FISP images (Fig. 4).

During the first 10 weeks the patient's condition improved considerably. She developed a strategy to compensate for the persisting lack of downward saccades by performing adequate head movements, accompanied by intervals of eye closure of a few seconds' duration, probably in order to suppress gaze stabilization by fixation and vertical VOR [12, 39, 58]. Finally she regained the ability to perform upward saccades and – after 9 months – also downward saccades of very short amplitude and slow velocity, which always had to be facilitated by blinks [60]. When oscillopsia had disappeared 2 weeks after the ischaemia, ataxia improved gradually, so that the last examination 4 years later revealed only a slight imbalance of gait with rightward deviation, but normal posture. Neuropsychological symptoms like hypersomnia, fluctuating alertness, delay of psychomotor reactions and impaired affective control, which were confirmed by a detailed test examination, persisted for more than 1 year, but had almost disappeared after 4 years.

Case 2

A 47-year-old woman suddenly lost consciousness. When admitted to the hospital, she was comatose and showed non-specific motor responses to painful stimuli. Pupils were equal, reaction to light was weak. Eye position was fixed in divergent strabism, with attenuated oculocephalic reflexes. Babinski's sign was positive on both sides; there was no gross impairment of other neurological functions. General examination, including blood pressure, heart rate and breathing, was normal. Previously, she had been in good health.

Under treatment with steroids and dextran infusions, the patient slowly regained consciousness. On the 5th day, she was partly oriented and able to follow simple verbal commands, although verbal and motor responsiveness were very slow. She showed apathy, fluctuating vigilance, impaired attention and memory. Neurological examination revealed complete vertical gaze palsy, rightward horizontal gaze palsy, dysarthria, dysphagia and mild right-sided hemiparesis. She was not able to stand or to walk, but had no ataxia of the limbs. EEG showed bilateral background slowing in

the delta range. CT revealed large bilateral post-ischaemic lesions in the medial and caudal thalami, resulting in a massive widening of the caudal part of the third ventricle, and a smaller lesion in the right paramedian caudal midbrain. Cerebrospinal fluid and Doppler sonography of extracranial vessels were normal.

The patient's condition improved gradually. When discharged from the hospital 5 months after the onset of symptoms, she was able to walk and to help herself in everyday life, but still had moderate ataxia of posture and gait, with constant rightward deviation, mild dysarthria, almost complete vertical gaze palsy and severe neuropsychological deficits, concerning vigilance, orientation, memory, drive and affective control. Visual acuity and visual fields were normal. EEG had returned to normal alpha rhythm. Ten years afterwards, a detailed neurological examination revealed only minor improvement. Vertical gaze palsy was still complete for saccades and downward pursuit and incomplete for upward pursuit, whereas vertical VOR elicited conjugated slow eye movements without saccades over almost the whole range of vertical eye positions.

Eye movement recordings

In case 1, eye movements were recorded repeatedly, using conventional EOG with silver-silver chloride electrodes. During the last examination, 4 years after the infarction, we applied the magnetic search coil system for recording of horizontal, vertical and torsional eye movements [11, 51], using 3-foot (0.91 m) magnetic field coils and search coils, consisting of Silastic scleral annuli (Skalar, Delft, Netherlands), which were placed on the patient's left eye. Horizontal and vertical visually guided saccades and smooth pursuit were elicited with a red laser target on a cylindrical screen. The optokinetic stimulus consisted of projected black and white stripes, each subtending 7.5° of visual angle, oriented perpendicular to the direction of stimulation. In case 2, EOG was performed only once, almost 10 years after the infarction. Search coil recording was not possible because of the patient's poor condition.

The results of eye movement recordings are summarized in Table 1. In case 1, downward saccades, whether spontaneous, reflexive or visually guided, were absent in the first three recording sessions. They showed the first signs of recovery after 5 months and reached a maximum velocity of 71°/s and a maximum eccentricity of 17° downgaze after 4 years (search coil recordings, Fig. 5). Upward saccades were present in the first session, but had a slow velocity and a restricted amplitude up to about 10° upgaze. They improved gradually, up to a maximum velocity of 156°/s and a maximum eccentricity of 33° upgaze.

In contrast to vertical saccades, vertical pursuit was less impaired; it reached more eccentric vertical eye positions and had a normal gain when recorded after 4 years with the search coil method. As a probable consequence of the saccadic disorder, vertical optokinetic stimulation at first only led to a tonic eye deviation in the direction of stimulus movement. This was more pronounced with upward stimulation. In the follow-up, vertical optokinetic nystagmus (OKN) partly recovered, slow phases reached a gain of at least 0.3 for downward and 0.15 for upward stimulation (stimulus velocity was 30°/s), and there was a vertical deviation of about 20° (Fig. 5). Vertical VOR, which could be elicited even during the acute phase, led to vertical deviations of both eyes, which later on were unrestrained in upward direction and limited to about 30° of downward gaze, thus representing more eccentric vertical eye positions than could be reached by vertical saccades or pursuit. Quick phases of vertical vestibular nystagmus were missing in the acute phase; 9 months later a few upward beats of low amplitude could be elicited, but no downward beats. Torsional VOR as elicited by passive head movements in the frontal plane led to a tonic counter-roll of the eyes, succeeded by a slow drift back to primary position, but – unlike normal human subjects [11] – without any torsional vestibular saccades. Horizontal and vertical saccades, however, did show a torsional component in the search coil recordings.

In addition, there was a persisting gaze-evoked nystagmus upwards and also slightly leftwards and downwards (Fig. 5). Horizon-

Table 1. Quantitative evaluation of eye movements

Type of eye movements		Case 1 / Time after onset of symptoms						Case 2	
		1 week/ EOG	4 weeks/ EOG	6 weeks/ EOG	5 months/ EOG	8 months/ EOG	4 years/ search coil	10 years/ EOG	
Gaze-evoked nystagmus	Up	+++	+++	++	++	++	++	0	
	Down	0	0	0	0	0	+	0	
	Right	(+)	0	(+)	0	(+)	(+)	0	
	Left	+	(+)	+	0	+	+	0	
Max. vertic. eccen- tricity up/down	Saccadic	10°/0°	25°/0°	30°/0°	30°/5°	32°/10°	33°/17°	8°/3°	
	Pursuit	20°/3°	30°/5°	30°/8°	35°/15°	35°/15°	40°/20°	20°/5°	
	VOR	30°/10°	40°/15°	40°/20°	> 45°/30°	> 45°/30°	> 45°/30°	> 40°/ > 40°	
Maximal saccadic velocity	Up	< 50°/s	< 80°/s	< 120°/s	130°/s	150°/s	156°/s	Slow drift	
	Down	No	No	No	50°/s	50°/s	71°/s	Slow drift	
	Right	400°/s	360°/s		370°/s		400°/s	450°/s	
	Left	370°/s	360°/s		370°/s		420°/s	450°/s	
Gain of smooth pursuit 16°/s	Up	?	?	?			0.92	ca. 0.9	
	Down	?	?	?			0.93	ca. 0.5	
	Right	0.3	0.75	0.90			0.98	1.0	
	Left	0.3	0.88	0.95			0.95	1.0	
OKN-gain 30°/s	stim. dir.:	Up	Dev. 5°	Dev. 15°	Dev. 30°	Dev. 30°	Dev. 35°	0.15	ca. 0.5
		Down	Dev. 3°	0.25	0.28	ca. 0.5	ca. 0.5	0.30	Dev. 30°
		Right	0.43	0.50	0.81	0.97	0.85	0.90	0.76
		Left	0.32	0.58	0.74	0.93	0.67	0.70	1.0

Strength of gaze-evoked nystagmus: 0 (+) + ++ ++++; dev., deviation

tal saccades were normal, apart from a mild hypometria in the beginning. Horizontal pursuit, OKN, fixation suppression of horizontal VOR and gain of leftward VOR (0.44, time constant 12 s; stimulus: stop from 90°/s constant velocity rotation in the dark) were moderately impaired in the acute phase, but recovered later on. In the dark, there was a mild left-beating spontaneous nystagmus (velocity of slow phases 4°/s in the first and 0.5°/s in the last session) and a slight apogeotropic positional nystagmus.

Similar results were obtained in case 2. Vertical saccades were not possible, apart from a slow drift of the eyes. Vertical pursuit was less impaired; its performance was better upwards than downwards (maximal eccentricity 20° upwards and 5° downwards). The normal range of vertical eye positions, however, could be reached by vertical VOR. Horizontal eye movements were normal, apart from a slightly reduced gain of OKN, pursuit and VOR (0.63) with rightward stimulation. There was no spontaneous nystagmus and no gaze-evoked nystagmus in this case.

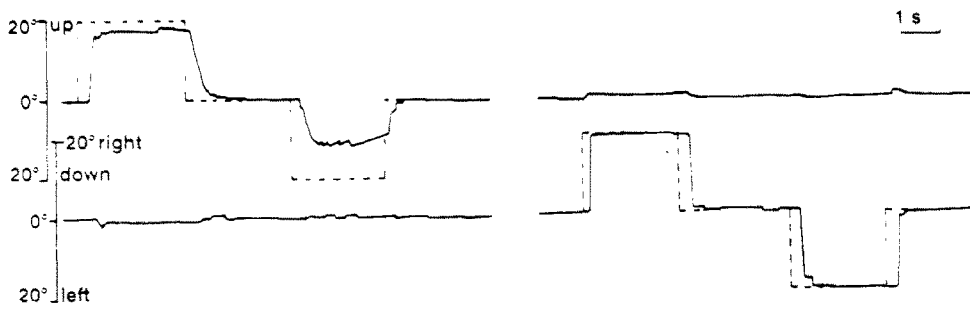
Psychophysical testing of motion detection

Methods. To test motion detection, a bright bar of 60' (= minutes of arc of visual angle) length and 1' width was moved on an *x/y*-monitor (Tektronix 608 with green P 31). For each trial, stimulus duration was 150 ms, which is short enough to prevent pursuit eye movements [59]. The bar always appeared at the fixation point and moved smoothly either to the left or to the right. We kept the presentation time constant and varied the amplitude of motion between 10' and 50'. Accordingly, the speed of motion varied between 1.1 and 5.6°/s. The monitor could be rotated by 90° to test vertical motion detection. The sequence of presentations was pseudo-random; the relative probabilities were balanced. Room illumination was in the photopic range (250 asb). The patients sat at an observation distance of 3.2 m from the monitor and had to indicate the direction of the motion in a two-alternative forced choice

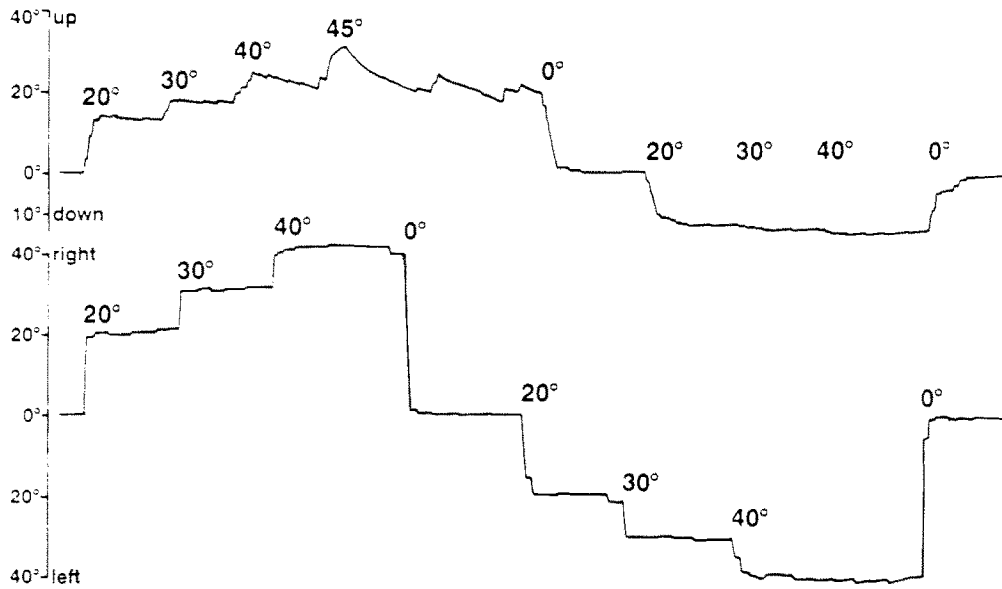
task by pressing one of two push-buttons. The left-hand button signalled leftward (or downward motion), the right-hand button rightward (or upward) motion. The responses were monitored using a digital computer that also produced the analogue voltages controlling position and movement of the stimulus on the monitor. Thresholds were defined by the level of 84% correct responses on a curve fitted according to probit analysis [19]. In order to decrease the influence of artefacts due to fluctuating concentration and motivation, we verified the results by measuring the same condition twice, whenever the patient's state was appropriate.

Vernier acuity was measured as a control in two sessions for both horizontal and vertical orientation of the stimulus, using a similar experimental procedure. Stimuli consisted of two bar-shaped segments, each 60' long, with a Vernier break in between that amounted to 10'' (= second of arc) at minimum and 80'' at maximum (cf. inset of Fig. 6d). In a two-alternative forced choice task, the patients had to indicate by pressing a push-button whether the lower (or right) segment was offset to the right or left, i.e. up or down.

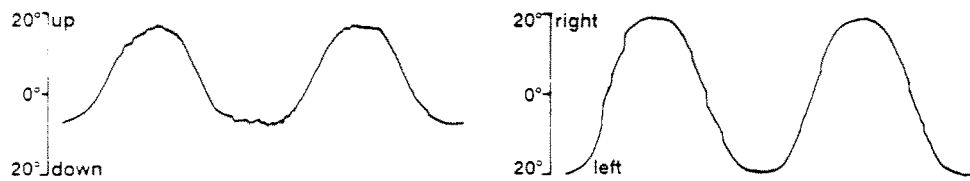
Results. Thresholds for motion detection, as calculated from the percentages of correct responses to the different amplitudes of movement, are plotted in Fig. 6. When first tested, case 1 could detect motion in horizontal directions, but not in vertical directions, as indicated by the arrows in Fig. 6b, c. Healthy observers do not show such a difference between stimulus orientations [30]. At the following four test sessions, up to 5 months after the onset of symptoms, thresholds for vertical motion detection were significantly higher than those for horizontal motion detection ($z = -2.8$; $P < 0.01$, Wilcoxon test). Thresholds for all directions of motion were higher than in healthy control subjects, who showed thresholds of around 150''. Performance improved with time, especially for the vertical directions. The difference between vertical and horizontal stimulus orientations had disappeared in the last two



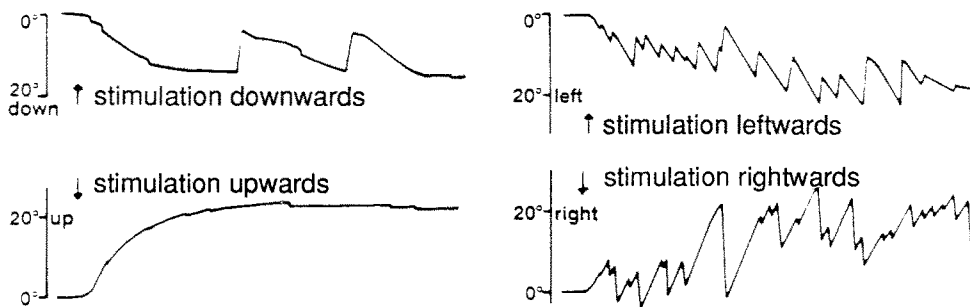
a



b



c



d

Fig. 5. Original traces of vertical and horizontal eye position recorded with the search coil method (coil in the left eye) in case 1, 4 years after the ischaemia. **a** Saccades (20°): ---, laser target; **b** eccentric eye positions; **c** pursuit (sinusoidal target motion: 0.2 Hz, amplitude 40°); **d** OKN (stimulus velocity 30°/s), *left*: vertical, *right*: horizontal

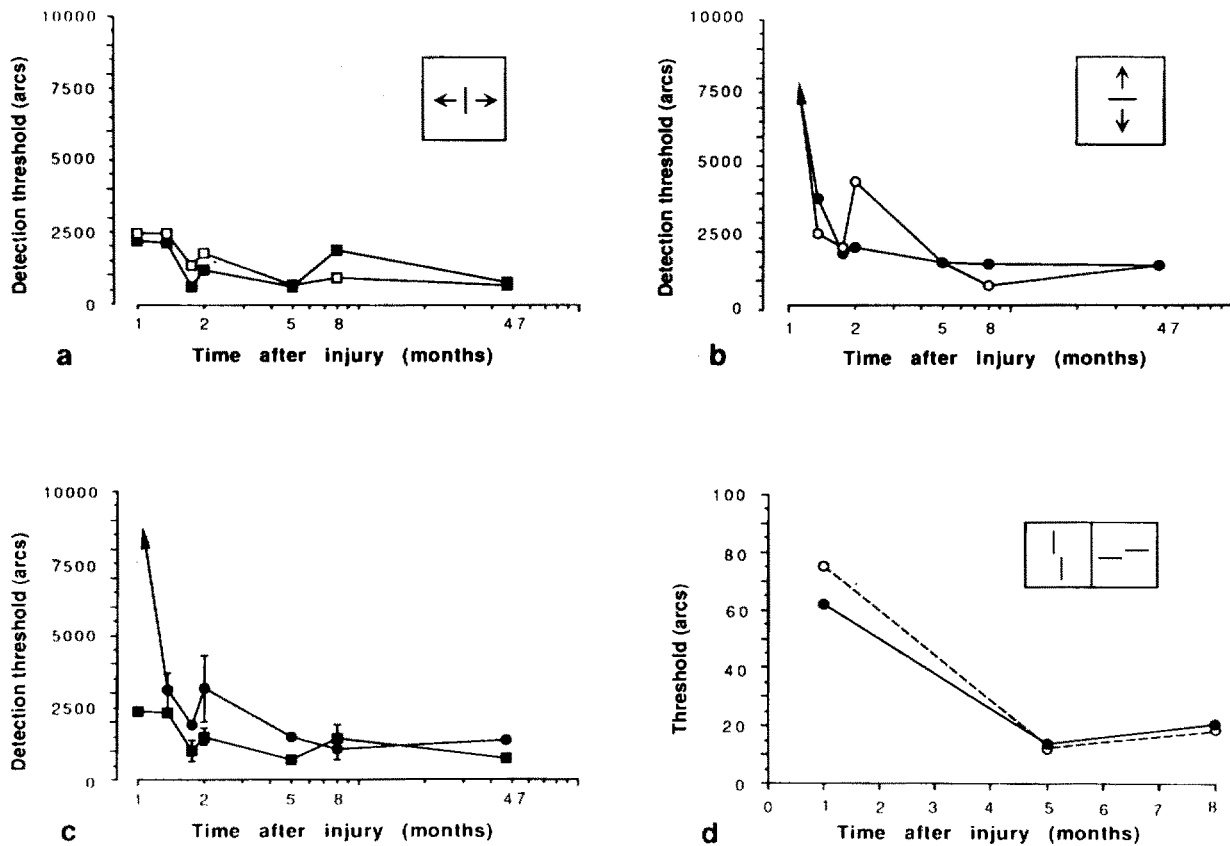


Fig. 6a–d. Thresholds for the identification of motion direction in case 1, calculated using probit analysis and the criterion of 84% correct responses as the threshold. The average thresholds for horizontal and vertical motion are plotted in **c**. As a control, thresholds for the detection of a stationary vernier displacement are shown in **d**. **a** Leftward (■) and rightward (□) motion; **b** downward (●) and upward (○) motion; **c** horizontal (■, a) and vertical (●, b); **d** vernier horizontal (●) and vernier vertical (○)

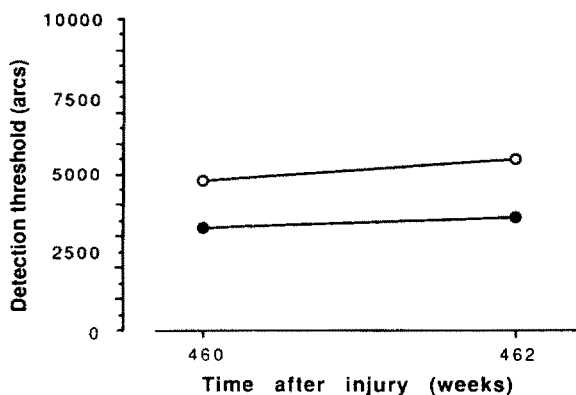


Fig. 7. Thresholds for the correct identification of motion direction in case 2 (●, horizontal; ○, vertical)

sessions, 8 months and 4 years after the ischaemia. Vernier thresholds were measured as a control (Fig. 6d). Initially, they were clearly increased for both stimulus orientations, but without a significant orientation bias.

The second patient, who was tested only twice, almost 10 years after the onset of symptoms, also showed a clear difference be-

tween the thresholds for vertical versus horizontal motion detection (Fig. 7).

Discussion

Both cases showed the typical clinical signs (combination of down-gaze palsy and neuropsychological disorders) of the so-called paramedian mesodiencephalic syndrome that was described in the Introduction. The distribution of the neuroradiologically confirmed lesions is in accordance with the territory of the posterior thalamo-subthalamic paramedian artery. The extent of thalamic infarctions was quite variable in our cases as it was in the cases reported in the literature. Therefore neuropsychological symptoms can range from minor disturbances to the full clinical syndrome of “thalamic dementia” or “akinetic mutism” [13, 14, 21, 35, 38, 55].

Vertical gaze palsy, predominantly involving down-gaze, is a rather constant symptom in most of the reported cases, as well as in our patients. By recording eye movements not only with the usual EOG, but also with the more sensitive search coil technique, we were able to confirm the clinical observations of four previous reports [39, 52, 54, 58] that downward saccades were impossible in the acute phase and – more than upward saccades – showed a persistent deficit (reduced amplitude and velocity) later on, in contrast to vertical pursuit and vertical VOR, which either remained intact or recovered rapidly. The saccadic deficit is the result of a bilateral lesion of the riMLF, which is considered to be the main premotor

nucleus for the generation of vertical saccades [4, 5]. It belongs to the vascular territory of the posterior thalamo-subthalamic paramedian artery [6, 53, 55] and is located dorsomedial to the anterior pole of the red nucleus, rostral to the interstitial nucleus of Cajal and lateral to the nucleus of Darkschewitsch (Fig. 2). In monkeys it contains the medium lead (= short latency) burst neurons for vertical and torsional saccades [25, 31]. Since no topographical segregation has been found between neurons for upward and downward saccades [27], the dissociation of downgaze and upgaze palsy could rather be due to lesions of the efferent fibres of riMLF neurons, leaving the riMLF at different sites, because only those for upward saccades are assumed to decussate in the posterior commissure. This would be in accordance with the localization of lesions found in humans and monkeys with upgaze and downgaze palsy [6, 34, 44, 48, 50]. Furthermore, our finding of an absence of torsional saccades is consistent with recent experimental data, which indicate that even unilateral riMLF lesions in monkeys cause deficits of ipsilateral torsional saccades [25]. As vertical VOR and vertical pursuit have never been persistently deficient in cases of riMLF lesions, it can be concluded that their neural pathways do not include the riMLF, but more probably the interstitial nucleus of Cajal [32, 50].

Another sign was the inability to carry out downward head movements in case 1 during the first 2 weeks after the ischaemia. In combination with the paralysis of downward saccades, it prevented the patient from visual exploration of the complete lower hemifield, thus causing a gross impairment of visuospatial orientation. According to the literature [47, 52], this symptom has so far been observed in only four cases, all with paramedian thalamo-subthalamic infarctions. As in our case, the deficit was temporary, at most for 6 months, and referred only to active downward head movements. There was no paresis, so it can be called an apraxia. The exact neural pathway and the neurophysiological mechanism for vertical head movements and vertical eye-head coordination is not yet clear. Various simultaneous phenomena of vertical or torsional eye and head movements have been reported as the result of electrical stimulation or lesions of the subthalamic region, the midbrain tegmentum and especially the iC in cats [24, 26, 41] and monkeys [7, 16, 17, 44]. Single unit recordings in the mesencephalic reticular formation near the iC of awake monkeys revealed both "burst tonic neurons" responding to vertical eye movements and "irregular tonic neurons" responding to vertical visual tracking, possibly correlated also to head movements [32]. Furthermore, a group of iC neurons project to the cervical spinal cord [9, 61]. These findings as well as evidence from other studies in the iC of cats [20] have led to the conclusion that the anatomical region of the iC could be an integrative centre for the control of vertical gaze (eye position in space) involving coordinated eye and head movements. This is supported by clinical findings, not only in our case 1, where the iC was at least partly lesioned (Fig. 2), but also in patients with supranuclear palsy, where bilateral degeneration of the iC is assumed to be responsible for the

characteristic upturned head posture [15, 40], which is also found after bilateral iC lesions in cats [20].

Besides vertical gaze palsy, our patients showed an increase of at least one afferent visual threshold, namely that involving vertical motion detection, as was confirmed by means of a psychophysical test. The explanation of this perceptual deficit is difficult. It can only partly be attributed to a lack of arousal and directed visual attention or inaccurate fixation, since horizontal motion detection and Vernier acuity for both vertical and horizontal orientations were less affected and recovered rapidly. During the test sessions visual fixation (straight ahead) and head position were stable so that the signal-to-noise ratio was not reduced by oscillopsia. Furthermore, the deficit can hardly be explained on the basis of the visual afference, since in the main visual pathways, including the lateral geniculate nucleus, the occipital cortex and the superior colliculus, no pathology was evident, according to CT and MRI.

On the other hand, impaired vertical motion detection could be a consequence of the vertical gaze palsy, mediated by a central adaptive mechanism that elevates thresholds for movement detection in order to reduce oscillopsia, which is due either to efference copy of the intended vertical saccades or to a deficient vertical VOR. Such a threshold elevation could have led to the disappearance of the initially observed vertical oscillopsia during walking in one of our subjects. A similar mechanism was proposed by Brandt and Dieterich [3, 18] for patients with extraocular eye muscle paresis, who showed elevated thresholds for the detection of slow horizontal object motion and did not experience oscillopsia during head movements, in spite of a considerable retinal slip. Their results, however, are not entirely comparable to ours, as they investigated not the discrimination of directions but the detection of only very slow movements, using rather long exposure times, so that the initiation of pursuit eye movements may have played a role. This was avoided by short-term exposure (150 ms) in our experiments. Also their conclusion that the deficits of motion detection were independent of the direction of object motion in each case cannot be generalized because they applied only stimuli moving horizontally, that means within the meridian of the oculomotor deficits.

Only a few authors have mentioned disorders of visual or visuospatial perception in cases of paramedian thalamo-subthalamic infarction, but these deficits lack further identification [2, 21]. The single more detailed analysis was given by Denny-Brown and Fischer [17]. In monkeys with experimental lesions of the midbrain tegmentum and disorders of vertical eye and head movements, they found an impaired ability to shift visual attention away from the point of fixation, most prominent in the vertical dimension and for moving stimuli, as a cause of a marked constriction of the effective visual field and a reduction of visually directed behaviour. For explanation, they assumed a lesion of subcortical visual pathways within the mesencephalic tegmentum involved in motion perception and visuospatial behaviour. In support of this hypothesis, there is anatomical evidence in monkeys that the midbrain tegmental nuclei, especially

the riMLF, receive indirect cortical input from the frontal eye fields [31] and the inferior parietal lobule (= IPL) via the superior colliculus [23, 36, 49, 57] and from the middle temporal visual area (= area MT) via the inferior pulvinar complex [37]. Both areas are considered to be the most important cortical areas for the mediation of motion perception (area MT) and visuospatial orientation (IPL).

Acknowledgements. We thank Mrs. Weller (Department of Neurophthalmology), Mrs. G. Schönwälder and Dr. M. Fetter (Department of Neurology) for their help during the recording sessions and Mrs. M. Wagner for typing the manuscript. The work was supported by the Deutsche Forschungsgemeinschaft (SFB 307, A2; Fa 119/5-1).

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