SHORT REPORT

Visually induced central pain and arm withdrawal after right parietal lobe infarction

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Abstract

A 46 year old man with ischaemic infarction of the right parietal cortex had left hemianaesthesia when his eyes were closed. With eyes open, visual stimuli induced withdrawal of the arm and a burning pain in the numb side of the body. Visually induced central pain is a new clinical finding and may be related to damage of cells with anticipatory inhibitory function in the parietal association cortex.

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Infarcts in the parietal lobe of either hemisphere may be accompanied by a burning or constrictive pain that resembles thalamic pain.¹⁻⁵ This central pain may be induced by various somatosensory stimuli such as touching cold objects, eating hot food, or drinking cold fluids.^{6,7} Exceptionally, a non-somatic stimulus such as intellectual concentration has been reported to cause pseudothalamic pain.^{7,8} The provocation of such pain by visual stimuli has not been reported. It has

been documented only that with a lesion in the parieto-occipital cortex visual stimuli may lead to a contralateral withdrawal reaction.⁹

The subject of this report is a patient with thromboembolic infarction of the parietal cortex subsequent to dissection of the internal carotid artery. He had a unique disturbance of sensation comprising neglect, verbal asomatognosia, and hemianaesthesia for all modalities on the left side of his body, associated with brisk withdrawal and burning pain in the anaesthetised side of his body when the patient saw this side being approached.

Case report

A 46 year old construction engineer experienced pain in the neck on making a sudden jerk of his head to avoid a falling beam. The event was followed by a persistent headache.

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Figure 1 Three dimensional in vivo mapping of lesion in the right hemisphere, reconstructed by manual tracing of contours of the lesion on contiguous MRI slides in the axial plane. (A) View from above; (B) lateral view; (C) the lesion, transferred to a schematic representation of the right hemisphere. It involves the posterior half of the postcentral gyrus (1), supramarginal gyrus (2), angular gyrus (3), inferior parietal gyrus (4), and superior parietal gyrus (5).

Figure 2 Left sided anaesthesia, visually induced burning pain, and withdrawal of the left arm in a 46 year old man with ischaemic infarction of the right parietal cortex. Above (with eyes closed): no reaction when stimulated by pin prick. Complete loss of superficial sensation (and of postural sense and of the ability to move the left arm voluntarily). Below (with eyes open): brisk withdrawal of the left arm as if it had been stung when it is merely approached; at the same time the patient experienced a burning pain in the arm.

Three days later the movements of his left hand became clumsy. He was unable to undress or wash himself properly. On taking a bath he saw a hand that he did not recognise as being his own. Lying in bed, he was again bothered when he saw a supposedly strange arm in his bed. Five days after the initial event, examination showed left hemianopia and left hemianaesthesia, with almost complete loss of all sensory modalities. He had severe postural instability and impaired control of muscle power in the left limbs but no weakness.

CT showed a hypodense area consistent with ischaemic infarction in the posterior part of the postcentral, the supramarginal, and angular gyrus, and in the inferior and superior parietal lobe; MRI confirmed these findings. Some involvement was seen in the white matter that connects the thalamus with the parietal cortex. The thalamus itself was intact. The extent of the infarction over the cortical surface was visualised with three dimensional in vivo mapping (fig 1), based on reconstruction by manual contour tracing of

the area of infarction on contiguous MRI slices in the axial plane.¹⁰ MRI of the neck showed a haematoma in the wall of the internal carotid artery, suggesting subadventitious dissection (not shown).

In the subsequent four weeks the patient improved. The left sided hemianopia regressed completely. His arm, however, gave him a lot of trouble. The patient himself was unaware of his left arm. He had no feeling in the arm and could not use it, but when he saw the arm being approached by someone it would suddenly move sideways as if it had been stung; simultaneously, he experienced a burning pain. The involuntary withdrawal movements of his left arm were so embarrassing that he tied it to his belt.

The patient was transferred to an institution for rehabilitation; he was reassessed after eight months. With eyes closed he had loss of superficial sensation (pain and touch) in the left side of his body, more severely in the arm than in the leg, trunk, and face, the distal parts of the extremities being affected most. No delayed pain reaction occurred. There was also complete loss of postural sense, which resulted in sensory ataxia and pseudo-athetoid movements. Vibration was not perceived. There was lack of awareness of the left half of his body and inability to move his left hand and fingers without visual control. With his eyes open and his gaze directed at his left hand, the patient was able to open and close the hand very slowly. There were no sensory abnormalities on the right side of his body. On seeing that the left part of his body was approached for sensory testing, the patient invariably made a brisk withdrawal movement (fig 2); at the same time he felt a burning pain that was accompanied by grimacing. On moving about, an incidental contact that was not anticipated did not result in pain and withdrawal. When the patient himself approached his left arm with his right hand there was neither pain nor withdrawal.

Discussion

The main residual deficits from infarction of the right parietal cortex in this patient were somatosensory disturbances in the left side of the body.¹¹ There was complete anaesthesia for all forms of stimuli on the affected side when the patient was examined with his eyes closed, but when he could see the approach of a stimulus, involuntary withdrawal and a burning pain were induced. A major difference from classical anaesthesia dolorosa¹ was that the painful reactions in this patient did not occur spontaneously but were evoked exclusively by visual stimuli.

To some extent these findings resemble those reported by Denny-Brown, *et al*⁹ in a patient with an infarct in the right parietal region, described as follows: "when the examiner approached the left hand to grasp it, the hand would move involuntarily as if to avoid the examiner's hand". That patient, however, had incomplete anaesthesia and no pain when visibly approached. We suggest that in our

patient the avoidance reaction and pain sensation induced by visible approach is caused by a lesion of the sensory association area in the posterior parietal cortex.

Neurophysiological studies in monkeys have shown that a minority of neurons in this part of the cortex show anticipatory types of activity on visual stimulation.¹² In these neurons a change in discharge rate occurs whenever a body part is approached, as though actual contact were made. Denny-Brown has suggested that two physiological types of response exist: exploratory and avoiding, which are behavioural antagonists. Frontal lesions release exploratory behaviour, of which instinctive grasping is the prototype. Parieto-occipital lesions release visual avoiding reactions, of which withdrawal is the prototype.¹³ Our hypothesis is that visually induced avoidance and pain in this patient is caused by damage to cells with anticipatory inhibitory activity, presumably located in the sensory association area of the posterior parietal cortex. Diminished output of these inhibitory neurons could lead to release of neuronal activity in the parietal sensory cortex and in the thalamus. Visually induced central pain may be seen as the release of a physiological anticipatory pain reaction, in the same way as visually induced withdrawal in patients with parieto-occipital lesions can be conceived as release of physiological avoidance behaviour.¹⁴ That pain did not occur when the patient saw his left arm being approached by his own right hand might be explained by internal signals cancelling the anticipatory pain induced by visual stimuli that are not self controlled, just as, in physiological circumstances, eye closure on visual threat will be cancelled when someone suddenly approaches his eye with his own hand.

This patient with central pain had an extensive lesion in the parietal cortex result-

ing in complete hemianaesthesia. This is extremely rare in patients with parietal infarction who have no concomitant motor deficits.¹⁵ Perhaps the large extent of the lesion in our patient accounts for the unique feature of visually induced central pain after infarction in the parietal lobe.

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