tory rhythm was regular and he could not vary it voluntarily, cough, or take a deep breath to command. Tracheal suction generated vigorous coughing. Spontaneous and volitional blinking remained preserved but he could not voluntarily contract the muscles of the upper or lower face; In response to emotional stimuli, however, there were brisk and pronounced contractions of the upper and lower facial muscles that were generally of similar pattern whether he was upset or amused. When this occurred there were concomitant respiratory gasps involving both thoracic muscles and diaphragm on inspiration and abdominal muscles and latissimus dorsi on expiration.

At about five months and just before discharge he had regained some limited volitional control of facial muscles particularly on the right but emotionally induced movements remained much more pronounced. Some voluntary jaw opening and tongue movements had returned, his tracheostomy had been closed, and he was able to take limited nutrition by mouth. Before discharge records of his respiration showed that during quiet breathing his respiratory rate was 21/min with a tidal volume of 0.37 litres: a maximum inspiration from functional residual volume was 0.63 litres, and a maximum expiration from end tidal inspiration was 0.66 litres: involuntary sighs of up to 2.5 litres were recorded. He could hold his breath voluntarily for 10.5 seconds. Carbon dioxide rebreathing yielded a normal ventilatory response and ventilation was maintained in sleep with a normal PaCO_2 . He remained tetraparetic.

This patient shows similar respiratory findings to those recently reported,^{1,2} with loss of voluntary control of respiration but preservation of metabolic control and evidence of activation in response to emotional stimuli. Voluntary control of facial muscles other than blinking was also lost although pronounced activation with emotional stimuli was preserved. Separate non-volitional pathways to the motor nuclei of the facial nerve are known to exist in uncontrolled crying or laughter as seen after stroke,³ whether due to a lesion in the brainstem or due to cortical damage. Such displays of sentiment are activated by the limbic system and are usually under inhibitory cortical control. In this patient the loss of corticobulbar fibres descending through the ventral pons may affect volitional facial and respiratory movement, but emotionally stimulated activity must be mediated independently by fibres descending in the dorsal or lateral pons to terminate on the facial motor nucleus in the caudal third of the ventrolateral tegmentum, and on pontomedullary respiratory motor neurons.

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Screening for cognitive dysfunction in neurodegenerative illness

The current profusion of clinical trials of antidementia compounds adds renewed urgency for accurate patient screening. There are a number of brief assessment instruments for use by the clinician to aid in the determination of dementia or other cognitive dysfunction. All scales may not, however, be equivalent or interchangeable. We have investigated the equivalence of two particularly frequently used scales (mini-mental state examination¹ and the Mattis dementia rating scale²) in three clinically demented populations: a Huntington's disease group (n = 15), an Alzheimer's disease group (n = 13), and a Parkinson's disease group (n = 10). The Mattis scale and mini-mental examination were strongly correlated in the Alzheimer's disease sample (r = 0.78), but not in the Huntington's disease group (r = 0.15) or the Parkinson's disease group (r = 0.15). Further investigation of the subscales in each test yielded a possible explanation for these discrepancies. The tests comprise sets of subscales, each of which assesses function in a different domain of cognitive function. The only common domain covered by both tests is attention and memory. If these are the only domains of interest, then either test will suffice. Both functions are affected in Alzheimer's disease, which may underlie the strong correlation between the two tests in this group. Due to subcortical influences in Huntington's disease and Parkinson's disease, however, frontal lobe dysfunction tends to be a prominent part of the clinical presentation. Only the Mattis dementia rating scale assesses frontal lobe function in its conceptualisation, and initiation and perseveration subscales. In our samples, these two subscales were sensitive to overall dementia severity in the Huntington's disease group and the Parkinson's disease group, but not in the Alzheimer's disease group. No subtest scores on the mini-mental state exam achieved this level of sensitivity to subcortical dementia. Therefore, when integrity of the subcortex and frontal lobe may be of concern, the Mattis dementia rating scale seems to be the more appropriate screening tool to use.

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Role of the pulvinar in ideomotor praxis

The production of learned skilled movements (praxis) is mediated by a modular network of cortical and subcortical structures that may include the thalamus. We report a patient with a left medial occipital, inferior temporal, and pulvinar infarct who showed a bilateral ideomotor limb apraxia. We attribute her apraxia to the pulvinar lesion.

The patient was a 76 year old, right handed woman who had a left posterior cerebral artery embolic infarct. We followed up the patient from five to the end of 17 months after the stroke during which time her examination did not change. On examination she had a right homonymous hemianopia and mild increase of reflexes on the right with normal strength and sensation. She was fully orientated except to year. She produced fluent anomic speech with preserved auditory comprehension and repetition, had amnesia, colour anomia, acalculia, a lexical agaphia, and read by a letter by letter strategy. Her figure copying was apraxic. Oral praxis was normal. She showed an ideomotor limb apraxia bilaterally.

Magnetic resonance imaging of the brain with horizontal, coronal, and sagittal slices was performed at five months after the stroke (figure). The stroke involved the left medial occipital lobe, inferoposterior temporal lobe, and the pulvinar nucleus of the thalamus.

We tested the patient with several sections of the Florida apraxia battery. She was able to recognise all tools (for example, hammer, scissors) used in testing.

She was given the name of each of 20 tools (transitive gestures) and 10 intransitive gestures (meaningful gestures that do not involve tool use—for example, salute) and asked to demonstrate the appropriate gesture. She was asked to use her left hand to perform all requested gestures and, subsequently, to use her right hand. Error types¹ included content errors (the correct movement but for the wrong tool), temporal errors, spatial errors (errors in the movement, relation of the hand to the tool, or the

MRI of the brain performed five months after the patient's stroke. The infarct involves the left medial occipital lobe, inferoposterior temporal lobe, and the pulvinar nucleus of the thalamus.

tool to the object), unrecognisable errors, and no response errors.

When using her left hand she was correct on nine of 10 (90%) intransitive gestures making one unrelated content error and she was correct on three of 20 (15%) transitive gestures making 19 spatial, three temporal, and two related content errors. When using her right hand she was correct on eight of 10 (80%) intransitive gestures making two spatial and one temporal error and she was correct on four of 20 (20%) transitive gestures making 22 spatial and four temporal errors.

The gesture to command task was also given to 12 normal men and women in their late 60s. They performed 10 of 10 (100%) intransitive gestures and 17 to 20 of 20 transitive gestures correctly (85% to 100%, mean 18.9, 94.5%). Normal subjects made 11 temporal errors and two spatial errors.

When given the actual tool to use, she was correct on eight of 20 (40%) trials when using her left hand and nine of 20 (45%) trials when using her right hand.

She was given the name of the tools and intransitive gestures from the gesture to command subtest. For each item she was presented with two gestures performed by the examiner, one the correct gesture and one an incorrect gesture (spatial error), and she was asked to choose the gesture that best fitted the item. Each set of gestures was performed twice. She was correct on 25 of 30 (82.5%) trials. Five age, education, and sex matched normal controls were correct on 28 to 30 of 30 (93% to 100%, mean 29.2 (97%)) trials.

The patient was shown 50 two and three step non-meaningful gestures involving the hand and arm on videotape. Each gesture was shown twice and she was asked to imitate the gesture. She was unable to imitate any of the 50 gestures correctly.

She was given a rotary pursuit task to assess her motor skill learning and did not show any motor skill learning with either hand.

She showed an ideomotor limb apraxia as the result of a left posterior cerebral artery infarct. The praxis system is a network that anatomically includes the parietal lobes, the frontal lobes, the white matter connections between them (both intrahemispheric and interhemispheric (corpus callosum)), and possibly the basal ganglia.² The neural structures subserving the praxis network are fed by the branches of the middle and anterior cerebral arteries whereas apraxia is not typically reported after posterior cerebral artery infarcts.³

Her apraxia was characterised by impaired production of meaningful and non-meaningful gestures with relative preservation of gesture recognition. Her apraxia improved but was still present when using tools. Her apraxia was more severe for transitive than for intransitive gestures. She also had absent motor learning on a rotary pursuit task as has been found in other apraxic patients.⁴

Damage to the occipital or temporal lobes has not been implicated in limb ideomotor apraxia. Apraxia secondary to dominant thalamic injury has been reported although not often investigated. Most of these cases have been thalamic hemorrhages that may have produced apraxia secondary to compression of non-thalamic structures. Graff-Radford *et al*⁵ described three patients with thalamic infarcts in the territory of the

tuberothalamic artery who showed limb apraxia.

The pulvinar has connections with the inferior parietal cortex and lateral prefrontal cortex, cortical structures important for praxis, thus making it a likely candidate for contributing to gesture processing. Based on its connections, we suggest that the pulvinar may participate in the selection of the movement representation, the translation of the representation into spatiotemporal coordinates, and the mediation of feedback for gesture monitoring.

The thalamus has been posited to function as a gated relay to filter out irrelevant information and may selectively engage relevant cortical neurons thereby creating transient cortical networks (Nadeau and Crosson, personal communication). Thalamic function is dependent on its thalamocortical connections for input and output and is regulated by the frontal lobe-inferior thalamic peduncle-nucleus reticularis-centromedian system. Therefore apraxia could arise from damage to the pulvinar as the result of its connections with the parietal and frontal cortex as shown by our patient or from damage to the frontal lobe-inferior thalamic peduncle-nucleus reticularis-centromedian system as shown by the patients with tuberothalamic infarcts reported by Graff-Radford *et al*.⁵

Further cases of thalamic lesions will help define the role of the thalamus in the praxis network.

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Anomia for proper names after left thalamic infarct

Proper names constitute the category of words most exposed to the "tip of the tongue" phenomenon.¹ Older people are more susceptible to this difficulty, which is their most common spontaneous complaint regarding memory performance.² A pronounced impairment in naming faces is often noted in Alzheimer's disease,³ but has only been reported rarely as an isolated

deficit after focal cerebral lesions.⁴⁻⁹ We describe a patient who presented an impairment in retrieving proper names as the only linguistic deficit after left thalamic stroke.

The patient was a 59 year old ambidextrous man, working as a senior executive. He had a history of migraine, hypertension, and hypercholesterolaemia. One morning his secretary noticed that he was abnormally drowsy and slow and had moderate speech difficulties. These abnormalities disappeared within 24 hours. When the patient consulted a few days later, he only had vague recollections of this initial day. He spontaneously complained, however, of severe difficulties in retrieving the names of familiar people. When asked to enumerate recent French prime ministers from memory, he could provide accurate descriptions of their physical aspect, city of origin, and political tendency, but could not find their names. He was unable to find the names of two close members of his own family. There was no motor, sensory, oculomotor, or visual defect, and no aphasia, apraxia, or spatial neglect.

Cerebral MRI (figure) showed a left thalamic infarct in the territory of the anterior polar artery, affecting the ventral anterior and ventral lateral nuclei, the mamillo-thalamic tract, and possibly part of the dorsomedial nucleus. PET with ¹⁵O labelled H₂O showed a diffuse and moderate hypometabolism of the left hemisphere.

The patient scored at ceiling level in all subtests of the Boston diagnostic aphasia examination. On the revised Wechsler memory scale, he had a visual index of 106, but a verbal index of 79 (1.2 SD below the mean).

To assess the patient's ability to name people, he was presented with a set of 45 pictures of famous faces and asked to name them. He could name only six of these, performing significantly worse than five control subjects matched for age and educational level ($t(4) = 3.36$, $p < 0.03$). He identified most targets, however, as evidenced by accurate biographical comments. Moreover, in 78% of the trials on which he declared having identified the target face without being able to retrieve the name, the patient could produce the correct name as soon as he was provided with its initial syllable. Two observations further support the hypothesis of an impaired name retrieval with intact face perception and recognition. Firstly, the patient scored in the normal range in Benton's facial recognition test. Secondly, as noted before, his word finding deficit was also apparent in non-visual tasks such as spontaneous speech and naming on definition.

Although the patient had major difficulties retrieving proper names for output, comprehension was apparently normal. On verbal presentation of their name, he provided a precise verbal characterisation of 14 of 15 famous people, of which he could name only three when confronted with a picture of their face.

To evaluate the selectivity of the naming deficit, the patient was asked to name 80 line drawings of objects belonging to 10 semantic categories, as well as 12 patches of colour, and 20 body parts pointed out on the experimenter's body. His performance was flawless except for two items, which he could readily name as soon as he was provided with the first syllable. To establish whether all kinds of proper names were