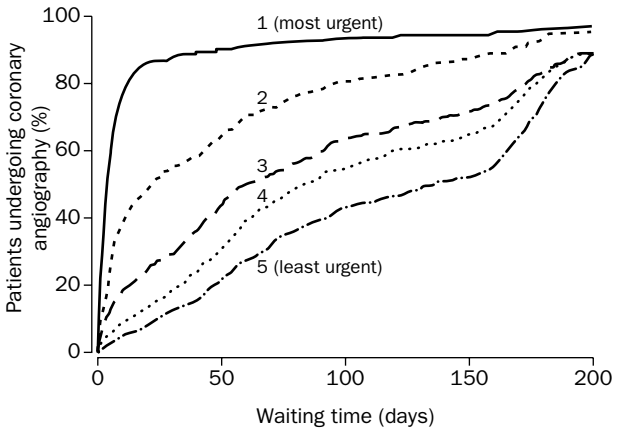

Waiting time to index coronary angiography by urgency category

The strong association between the urgency categories and time spent on the waiting list and clinical outcomes shows, on average, a high degree of clinical order. However, such implicit queueing is imperfect at the level of the individual patient; the interquartile ranges of waiting times overlapped one or two urgency categories, and a perfectly ordered queue would show no association between events while awaiting angiography and urgency category. Further investigation is required to find out how patients, general practitioners, physicians, cardiologists, and hospital managers generate a waiting list ordered by clinical need. However, if the waiting list shows some clinical order in relative terms, the length of wait in absolute terms may pose unacceptable risks. The recommended maximum waiting times for the five urgency categories, according to the Canadian panel were 3 days, 7 days, 14 days, 42 days, and 91 days, for categories 1 to 5, respectively,[4] and a recent US study recommended that no one wait longer than two weeks.[5]

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Imaging hypnotic paralysis: implications for conversion hysteria

Peter W Halligan, Bal S Athwal, David A Oakley, Richard S J Frackowiak

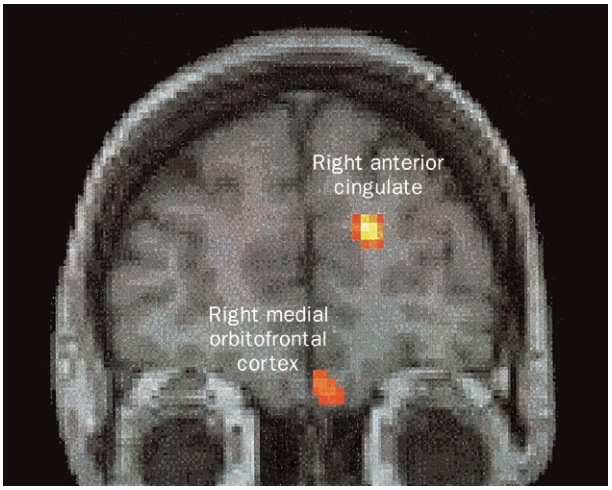
In a single case study with positron emission tomography (PET) functional imaging, hypnotic paralysis activated similar brain areas to those in conversion hysteria, supporting the view that hypnosis and hysteria might share common neurophysiological mechanisms.

Although the clinical criteria for diagnosis of conversion hysteria are well established, controversy continues to surround the neural and psychological mechanisms involved.[1] Since the psychological processes responsible for hysterical paralysis occur via physiological brain activity, functional imaging might reveal some of the neuropsychological mechanisms.

Motor conversion remains the classic presentation of non-organic paralysis; however, hypnotic forms of motor paralysis have been used since the 19th century to mimic the symptomatic behaviour found in hysterical conversion.[2] The two disorders are conceptually linked to the extent that experiments in hypnosis (regarded as a kind of artificial or controlled hysteria) have served as models for the study and treatment of hysterical symptoms. Previous studies suggest that hypnosis selectively involves anterior fronto-limbic inhibitory processes.[3]

In a case study of a patient with longstanding conversion hysteria (leg paralysis) Marshall and colleagues,[1] by positron emission tomography (PET), found that two distinct areas of prefrontal cortex were activated. With their study as a model, we investigated the brain regions activated when lower limb paralysis was hypnotically induced in a healthy man. We postulated that hypnotically produced paralysis would activate similar brain areas to those activated in hysterical paralysis.[4]

The participant was a right-handed man aged 25 years preselected for scoring positively on those items of the Harvard group scale of hypnotic susceptibility dealing with ideomotor responses, motor rigidity, and inhibition of movement. An eyes-closed hypnotic induction was done, with relaxation and deepening, involving descent and special place imagery. Suggestions of left-leg paralysis were modelled on those of the previous clinical case study.[1] Hypnotic depth and presence of the paralysis were monitored throughout the procedure. Before induction, neurological examination was entirely normal. After hypnotic induction and hypnotic suggestion for left leg paralysis, the participant could not move his left leg. To compare hypnotic and conversion performance, the same experimental design, statistical analysis, and PET technology were used as in the study by Marshall and colleagues.[1] There was one control (no movement) and four active conditions, each done three times in randomised, counterbalanced order. The four active conditions comprised preparing to move, and attempting to move, the (good) right leg and (bad) left leg in synchrony with a metronome at 0·5 Hz. The prepare conditions involved the participant remaining ready to move immediately upon a signal, which was given only outside the acquisition time on each PET measurement. In the attempt conditions the participant tried to lift the leg. Throughout, both legs were tightly restrained to control for the absence of movement in conditions involving the paralysed leg. PET scanning used the standard oxygen-15 technique with Siemens Ecat (Siemens, Erhlangen, Germany) scanner operating in 3-dimensional mode, with a total of 15 measurements of brain activity. Spatial preprocessing and statistical analysis of images was done



Relative changes in cerebral blood flow associated with attempted movement of the hypnotically paralysed left leg

Brain region	x	y	z	Z-score	p*
Right orbitofrontal (BA 10/11)	4	54	−20	3·77	0·03
Right anterior cingulate (BA 32)	16	52	18	4·01	0·01

Co-ordinates (in standard stereotactic space) refer to maximally activated foci as indicated by the highest Z-score within a cluster of activations: x, distance (mm) to right (+) or left (−) of the midsagittal line; y, distance anterior (+) or posterior (−) to vertical plane through the anterior commissure; z, distance above (+) or below (−) the intercommissural (AC-PC) line. The AC-PC line is the horizontal line between the anterior and posterior commissures. *p-values quoted are after correction for search volumes of orbitofrontal cortex and anterior cingulate estimated from reference standardised brain volume used in SPM99b.

with SPM99b (available at www.fil.ion.ucl.ac.uk/spm). Continuous surface electromyographic (EMG) recordings were taken of both legs throughout scanning to monitor muscle activity.

Moving his (good) right leg activated motor and premotor areas, or both, in the left hemisphere in a similar pattern to that previously reported in studies of healthy individuals. However, when the participant attempted but failed to move the left leg (confirmed by the absence of relevant EMG activity), right orbito-frontal (Brodman area BA10/11) and anterior cingulate (Brodman area BA32) cortex ($p<0·001$, *t*-test) were selectively activated without similar activity in the motor cortex (figure). The interaction shown in the figure is derived from the following comparison of conditions: ([attempt to move left leg−prepare to move left leg]−[attempt to move right leg−prepare to move right leg]). This interaction shows relative regional cerebral blood flow (rCBF) increases when the participant attempts to move the paralysed leg that do not occur when attempting to move the normal right leg. The figure (coronal view) shows activations of the right medial orbitofrontal cortex and anterior cingulate.

Our results and those of Marshall and colleagues[1] are consistent with the hypothesis that hysterical and hypnotic paralysis share common neural systems involving contralateral prefrontal regions. Although the coordinates identified differ slightly,[1] they represent peaks of activity with an overlapping spatial distribution located within the same cytoarchitectural regions. Electrophysiological studies have implicated both areas as part of a cortical region involved in motor inhibition.[5] The rostral parts of the anterior cingulate are intimately connected with adjacent prefrontal, premotor, and orbitofrontal areas and are associated with modulating interactions between motivational processes and motor output. For example, lesions of the anterior cingulate can produce general muscular hypotonia, characteristic of cataplexy elicited by strong emotion.

In our study, the anterior cingulate and orbito-frontal cortex activations probably represent neural activity responsible for inhibiting the participant's voluntary attempt to move his left leg. Alternatively, these activations could represent the management of a mental dissonance produced when the suggestion of paralysis of the left limb conflicts with the explicit instruction to move it. Such an account would equally apply to hysterical people where the activations could reflect the management of a similarly generated internal conflict. While the first interpretation predicts that the recorded activations are specific to hypnotic or hysterical limb paralysis, the second would predict that the pattern of activation might also be seen with the same testing strategy, irrespective of the specific hysterical symptom or its hypnotically produced counterpart. Both interpretations, however, are consistent with the view that for motor paralysis, hypnosis and hysteria share similar mechanisms.

Although these are single-case comparisons, the anatomical proximity of the neural activations suggest that the psychological mechanisms which underlie hypnotic phenomena provide a versatile and testable model for understanding and treating conversion hysteria symptoms.

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Carbon-dioxide portography: an expanding role?

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We describe a new and inexpensive technique of imaging the portal vein in patients with liver disease by use of carbon dioxide.

Portal-vein thrombosis may be the cause of portal hypertension, or may complicate portal hypertension in up to 15% of patients with cirrhosis at transplantation.[1] The presence of portal vein thrombosis has major implications with respect to patient management. Liver transplantation is contraindicated when extensive thrombosis exists, and patients with variceal bleeding are not eligible for radiological shunts. Non-invasive techniques used in the assessment of portal-vein thrombosis include: doppler ultrasound, venous phase contrast enhanced computed