

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 stereotaxic 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 (Brodmann area BA10/11) and anterior cingulate (Brodmann 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 colleagues¹ 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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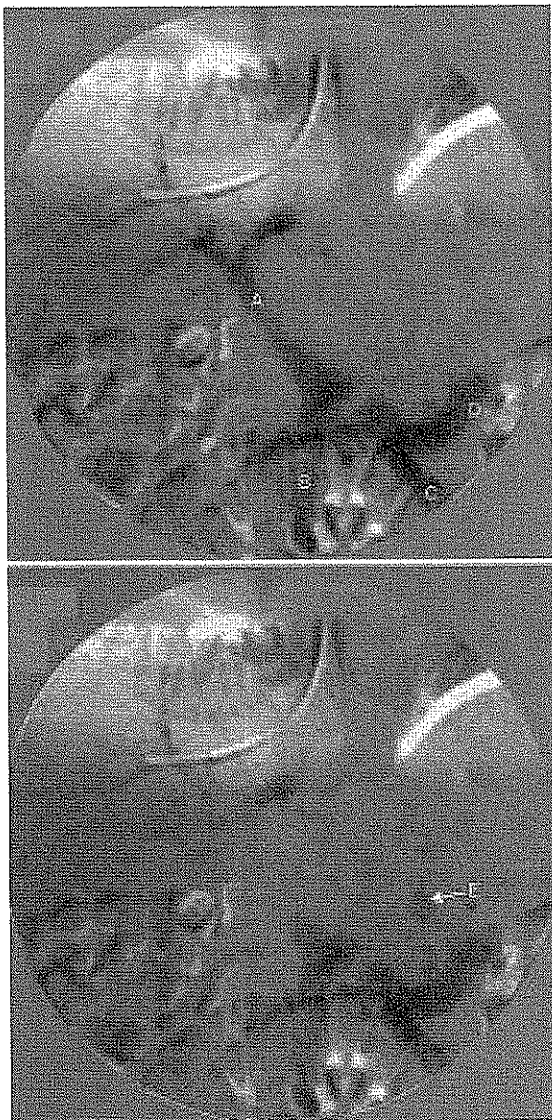
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Carbon-dioxide portography: an expanding role?

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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



Portal vein patency confirmed on carbon-dioxide portography

A, portal vein; B, superior mesenteric vein; C, inferior mesenteric vein; D, splenic vein; E, varices.

tomography, and magnetic resonance angiography. However, these imaging modalities are not always definitive, and under these circumstances direct angiographic methods are still required, such as venous phase splanchnic arteriography or transhepatic or transsplenic portography.

Hepatic venous catheterisation is the method of choice to assess portal pressure in patients with cirrhosis.² During this procedure, contrast medium can be injected to obtain a retrograde portogram. Unfortunately, the use of iodine contrast medium rarely achieves adequate visualisation of the portal vein. Furthermore, adverse reactions to contrast material, including idiosyncratic reactions and contrast-induced nephropathy continue to occur in a few patients. Carbon dioxide has been used as an alternative to iodinated contrast material since the 1950s.³ Its advantages are numerous,⁴ and many of these are associated with the low viscosity (1/400) of carbon dioxide, allowing the use of finer (3F) catheters. The rapid clearing of extravasated carbon dioxide results in much less parenchymal staining, which is a drawback with iodine contrast medium. Carbon dioxide has

a proven safety record⁵ and is inexpensive, at 1/1000 the cost of iodine contrast medium.

Until recently, carbon dioxide angiography had focused on imaging arterial systems. We did a prospective assessment of carbon-dioxide-wedged hepatic venography to investigate portal vein thrombosis. We examined 11 patients undergoing liver transplant work-up, in whom portal-vein thrombosis had been diagnosed by at least one other imaging modality. A control group of 30 patients was also examined. These patients were also undergoing liver transplant work-up, or a transjugular intrahepatic portosystemic shunt (TIPS) procedure. A (7F) balloon occlusion catheter (Cordis Europa, Roden, Netherlands) was placed into a hepatic vein, via a common femoral or internal jugular vein approach, and wedged in position and the balloon inflated. In suspended respiration a rapid hand injection of 50 mL medical grade carbon dioxide was given. The carbon dioxide then refluxed retrogradely through the liver sinusoids to fill the portal-venous system (figure). Image capture was done in a standard fluoroscopy suite (Siemens, München, Germany), with a rapid frame rate of 6 frames per s.

Of 11 patients with equivocal imaging of the portal vein, six were confirmed to have portal-vein thrombosis by carbon dioxide portography. Five (45%) patients were found to have unequivocally patent portal veins. All five have since undergone successful liver transplantation. In the control group the portal vein was clearly patent in all patients. Only minor complications were seen. Transient non-specific chest discomfort occurred in four patients who had no other clinical signs and normal electrocardiograms and chest radiographs. These complications settled conservatively within 15 min. One patient had a suspected liver capsule leakage shown on carbon-dioxide portography, but no rupture was seen with conventional contrast medium injection, and there was no clinical sequelae. No further intervention was necessary.

We conclude that carbon-dioxide portography may be useful in examination of portal circulation. This technique has now become established as our definitive method to prove portal vein patency, when other methods give conflicting results. Carbon-dioxide portography is also used in real time during TIPS procedures to guide the puncture of the portal vein. Our experience so far suggests that this technique is safe, well tolerated, and has a major impact on patient management. Carbon-dioxide portography may be done at the time of transjugular liver-biopsy, and hepatic wedge pressure may also be undertaken, providing fundamental histological, haemodynamic, and anatomical information for the clinician. In the era of single visit, day case medicine, we have termed this series of investigations "the one-stop liver shop".

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