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Size reduction using Mirror Visual Feedback (MVF) reduces phantom pain

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Following limb amputation patients continue to feel the vivid presence of a phantom limb. A majority of patients also experience pain in the phantom and sometimes (as in our case DS) the pain is severe. Remarkably we find that optically ‘resurrecting’ the phantom with a mirror and using a lens to make the phantom appear to shrink caused the pain to ‘shrink’ as well.

Keywords: Phantom limb; Mirror visual feedback; Neural plasticity; Phantom pain; Somatosensory.

INTRODUCTION
Phantom pain following amputation is notoriously intractable. We have previously shown that pain can be reduced by optical means. The patient looks into a parasagittal mirror to ‘resurrect’ the phantom. Moving the normal hand causes the phantom to feel as though it is moving reducing phantom pain in some cases (Ramachandran & Hirstein, 1996; Ramachandran, Rogers-Ramachandran, & Cobb, 1995). We had also noticed that viewing ones own normal hand through a reducing lens caused it to ‘feel’ smaller as well and caused a curious alienation or the hand, especially if it was wiggled (Ramachandran & Rogers-Ramachandran, 2007). In the fight between vision and proprioception, vision often dominates and vetoes the latter (Rock & Victor, 1964), although not invariably so (see Ernst & Bülthoff, 2004).

We wondered if one could use an optical trick to reduce the size of the phantom limb and whether that would modulate the felt pain.

CASE REPORT
Patient DS is 42 years old. His left brachial plexus was avulsed in a motorcycle accident when he was 19 years old and his (painful) left arm was amputated above the elbow a year later. We examined him 11 years post amputation and again 23 years post amputation. During the first exam he had a vivid, excruciatingly painful, phantom arm which was normal in size and length but could not be moved volitionally even with intense effort. It was as if the actual paralysis of the limb prior to amputation had been carried over into the phantom along with the pain (‘learned paralysis’). We postulated that the continuous mismatch between motor commands and visual feedback from the paralyzed arm (prior to amputation) and indeed from the phantom may be one source of phantom pain. DS was mentally and neurologically completely normal in all other respects except for a Horners syndrome in his left eye caused, presumably, by his avulsion.
We attempted to restore the congruence between the motor commands and the phantom using a mirror (Ramachandran et al., 1995; Ramachandran & Hirstein, 1996; Ramachandran & Rogers-Ramachandran, 1996). The mirror was placed parasagittally on a table in front of the patient and he placed his intact right hand on the right side of the mirror. He then viewed the reflection of the intact hand in the mirror so that it was optically superposed on the felt location of the phantom (a technique we describe as ‘mirror visual feedback’, MVF). He was asked to send commands to both arms to perform symmetrical movements (clenching, unclenching clapping, conducting an orchestra, etc.) to create the visual illusion that his phantom was moving as well. Surprisingly DS reported that this caused the phantom to move as well, partially relieving the painful spasms. Repeated use of the technique actually caused the phantom elbow, wrist and proximal palm to disappear completely – and permanently – along with the associated pain. The hand ‘telescoped’ so it was dangling from the stump and the pain in the digits (and distal palm) remained unaffected. DS often wondered if we could redesign the mirror box to eliminate his fingers as well. The efficacy of MVF in treatment of phantom pain has now been confirmed in a large scale, placebo controlled clinical trial (Chan et al., 2007). It has also been successfully used in the treatment of chronic regional pain syndrome (McCabe et al., 2003) and in rehabilitation from hemiparesis following stroke (Altschuler, Wisdom, Stone, Foster, & Ramachandran, 1999; Dohle et al., 2009; Sathian, Greenspace, & Wolf, 2000; Stevens & Stoykov, 2003).

In the present study we decided to explore whether a similar optical technique could be exploited to ‘shrink’ DS’s phantom. To accomplish this we used the standard MVF, but this time DS viewed the reflection through a minifying lens so that there was a 2-fold shrinkage of the reflection. When DS viewed this reflection he not only saw the hand shrink but FELT it to shrink as well. Presumably this visual capture of the phantom was much more pronounced in him than in normals because of the absence of somatic signals from the (missing) hand failing to veto the visually induced minification.

We then asked him (without prompting) whether he noticed any other change in the phantom. Much to our astonishment – and his – he experienced a striking and immediate reduction in pain as well. The optical shrinkage of the phantom had apparently caused a perceptual shrinkage of the pain! As soon as he shut his eyes (or the lens removed) the pain immediately returned in a matter of seconds.

Since DS was a highly reliable and observant patient we had no reason to doubt the veracity of his reports of pain reduction. But as ‘control’ we had him look at the mirror through a magnifying (instead of minifying) lens. This time, despite his minor concern that the pain might increase (temporarily) he noted that there was no change in pain whatever, even though the phantom looked 2-fold bigger. This renders it highly unlikely that the reported reduction was simply a response bias or even confabulatory in origin; for if that were the case why would he not experience increased pain with an amplifying mirror despite his expectation (and trepidation)?

We ran two experimental sessions separated by a 2-h lunch break. In each session there were 18 trials; 12 with the reducing lens and 6 with the magnifying lens. Each trial lasted about 20 s followed by removal of the lens and a 30-s intertrial interval. The minifying and magnifying lenses were randomly interleaved. At the end of session 2 an additional experiment was carried out in which a 4× minifying lens was used (four trials) instead of 2×.

Pain ratings (on a scale of 1–10) were obtained before and after each trial. On a majority of trials the pain level was at 8, but in two trials it was reported to be an ‘excruciating 14 or 15’ (even though it was explained to him that this was not part of the scale) Upon using the mirror, the pain fell to 2 and this was remarkably constant across trials. A few seconds after the lens was removed the pain went back to the level (8) that it was before the lens was removed. On three trials the pain dropped from 8 to zero. Pain dropped to 4 on the two occasions when it was 15 at the start (Figure 1).

On some trials DS noted that the lens caused the phantom hand to not only shrink but fade away completely except for a small region below the dorsum of the index finger; and there was no pain (zero) in the faded regions.

When using the magnifying lens, there was no change in pain on any trial except one (on a single trial it increased to 9 from 8). Conversely, when a 4× minifying lens was used there was an even further reduction to about 1, although we did not measure this systematically. A ‘dose response curve’ may be useful in future studies. Pain levels climbed back to original levels (8) a few minutes after the last trial of session two. It remains to be
seen whether repeated use of the lens (as with standard MVF) would lead to more lasting reduction from pain.

These striking results—suggesting powerful inhibition of somatic pain using optical shrinkage—complement our earlier findings of pain reduction using MVF. They should, nonetheless, be regarded as preliminary until they are replicated in other patients. It is, however, highly unlikely that the pain modulation with optical minification we observed in DS was a result of suggestibility (or confabulation) given the observation that the magnifying lens had no effect even though he fully expected it to have one. Secondly he reported that he had started using Neurontin during periods of pain and that the drug was less effective than optical reduction, although its effect was more long-lasting. Thirdly, on some trials, he reported an actual disappearance or fading of the phantom hand itself except for a small part on the dorsal hand skin below the index finger; and the pain disappeared selectively in the faded parts alone. There would be no reason for such a patchy reduction; it would be a strange confabulation.

DISCUSSION

These observations on the modulation of pain using visual feedback—whether with mirrors or lenses—have broad implications for understanding central pain mechanisms as well as therapeutic applications for treating chronic pain (Ramachandran & Altschuler, in press). As we have noted in previous publications visual feedback can strongly modulate pain sensations. We now take these observations further by showing the optically induced shrinkage of the phantom can shrink the corresponding pain as well. It remains to be seen whether repeated use of the lens-mirror arrangement would cause a more permanent reduction.

Apart from our own work, there is now a wealth of new experimental evidence suggesting that the various ‘specialized’ modules are highly malleable in response to changes in sensory inputs and that they interact to a significantly greater extent with each other than previously assumed (Lacey & Sathian, 2008). Here, we have now shown that optical shrinkage can shrink phantom pain.

The broader theoretical implications are equally important. Many artificial intelligence researchers and neurologists have long believed that the brain consists of a number of quasi-autonomous specialized modules (e.g., for vision of touch or pain) that are hardwired. Each module is thought to compute and make explicit some aspect of incoming sensory information that is then relayed to the next module in the hierarchy. This ‘serial hierarchical bucket-brigade’ model needs to be radically revised with a view in which different brain regions are in a constant dynamic equilibrium with each other and with the environment. Disease—in this view—is caused not by ‘punching out’ a module for ever but by functional shifts in equilibrium that can, in theory, be restored by simple procedures such as MVF or lenses.


