These benefits should not be denied to patients, including those facing the special challenges of epilepsy, on the basis of tenuous speculations unsupported by any credible evidence.

Yours faithfully
Dr. Roger Chalmers

Competing interest statement

Roger Chalmers is a full-time locum general practitioner and has derived more than 99% of his income from UK National Health Service clinical work over the past 9 years. He became a teacher of Transcendental Meditation in 1975, and has lectured widely on research and medical applications of this and related techniques over the past 28 years. From 1982 to 1996 he was directly involved with institutions publicly advocating TM, holding a variety of non-salaried academic positions and co-editing volumes 2—4 of collected research papers on TM.[1] From 1987 to 1991, he worked in full-time private medical practice utilizing the complementary system known as Maharishi's Vedic Approach to Health, which includes TM, alongside modern medicine.

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Hypnosis and meditation: Similar experiential changes and shared brain mechanisms

Newberg and Iversen have recently proposed in Medical Hypothesis a neural model underlying meditative states [1]. Here, we draw attention to the excellent correspondence between hypnosis and meditation both in terms of their experiential qualities and their underlying brain mechanisms.

Hypnosis has been characterized by changes along several dimensions of subjective experience [2,3] including mental relaxation and absorption. Mental relaxation refers to a fluid flow of thoughts; while mental absorption involves a shift from externally directed and active (effortful) forms of attention to internally directed and passive attention. Those changes lead to a reduction in temporal and spatial orientation. Hypnosis is further characterized by an altered sense of self-monitoring and self-agency manifested experientially by reduced resistance to alternative experiences and by feelings of automaticity associated with thoughts or actions. While mental relaxation and absorption are generally considered essential to achieving meditative states, the broadening of experiential space and the altered sense of self may be more specific to certain forms of meditation [4].

Neuroimaging studies of hypnosis have identified many of the same cerebral responses posited in the model of meditation proposed by Newberg and Iversen. In both meditation and hypnosis, attention drives the prefrontal and cingulate cortices which interact with other structures including nuclei of the thalamus and brainstem as well as parietal cortices, resulting in states of decreased vigilance and increased attention. Furthermore, hypnosis studies have demonstrated distinctive associations between certain brain networks and mental relaxation and absorption [5,6]. Specifically, hypnotic relaxation involves brain areas known to regulate arousal and vigilance [6,7] while mental absorption involves a brain network underlying attention mechanisms [7,8]. Additional increases in occipital rCBF during guided meditation [9] and hypnosis [5] may reflect a decrease in vigilance and in cross-modality suppression, associated with decreases in the cortical release of norepinephrine [5,10], and leading to a facilitation of experiential changes [3].

Meditative techniques form a dichotomy roughly akin to the extremes of the allegorical spotlight of attention. Concentrative techniques involve sustained focal attention (e.g. on the breath) whereas receptive techniques involve unfocused sustained attention (e.g. mindfulness meditation) [4]. Further, meditative techniques may be self guided or externally guided via an instructor or recording. Similarly, hypnosis can be self induced or induced by a hypnotist. Considering the striking similarities in their experiential and brain correlates, meditation and hypnosis appear to be closely related phenomena and hypnosis may be conceived as a western form of guided meditation.

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Acidosis might make breast cancer cells more susceptible to metastasize by shifting CXCL12 monomer–dimer equilibrium to monomeric state

Sir,

Tissue hypoxia occurs when there is an inadequate supply of O_2 that compromises normal biological processes in the cell [1,2]. This stressful microenvironment is a hallmark of solid tumors, meaning that O_2 delivery to the respiring cancer cells is reduced or abolished. Most tumors larger than 1 mm^3 in volume contain regions of hypoxia as a result of the disordered blood vessel structure. The lactate is produced when oxidative phosphorylation is switched to glycolysis to maintain energy production and eventually this causes an acidic microenvironment to occur in tumor cells [1]. This acidosis is likely to be more pronounced in hypoxic areas of the tumor, with experimental tumors having a mean pH of