TMS IS AN EFFECTIVE THERAPY OF APHASIA (NO)
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Both TMS and TDCS (transcranial direct current stimulation) have after-effects on cortical excitability that outlast the period of stimulation. They are reduced or abolished by drugs that interfere with function of NMDA receptors and hence may involve the initial stages of synaptic potentiation or depression. The protocols also have been demonstrated to interact with normal processes of learning in motor and language systems, with some protocols improving performance and others worsening it. Clearly these methods may be promising candidates to improve recovery from brain damage, and in particular to improve the response to language therapy in aphasic patients. There are two questions that have to be answered before we can conclude that they are effective therapies: first has improvement been conclusively demonstrated and second if so, is it clinically significant and robust enough to apply widely in general practice?

Reviewing the literature, it may appear that there is good evidence that TMS/TDCS treatment can improve language function after stroke. However, the majority of the studies are case reports in small numbers of patients, almost all of which find a significant improvement in chronic aphasics. Although encouraging, case reports on the effects of a new therapy are well known to have a highly skewed publication bias in favour of positive results, so that we have little idea of how many times the same protocols have proved ineffective. In addition, there is variation in which protocol was applied in any one particular report making the power of the combined observations weaker than it would be with a standard approach. The conclusion is that there is far too little evidence available to conclude that rTMS/TDCS is having any therapeutic benefit in aphasia.

Nevertheless, there are many positive indicators that the methods might be useful. However, the number of potential treatment approaches is large and this will make validating rTMS/TDCS as therapies very difficult in practice. For example, should therapy concentrate on suppressing excitability on the right of the brain or enhance excitability on the left of the brain? The answer may well depend on the pre-stroke organisation in each individual, the size and location of the stroke and the time after stroke that the therapy is applied. It may be possible to obtain some insight into the best therapy from functional imaging studies, but this is not going to be a practical approach for widespread therapeutic uptake, even if scientifically interesting. An effective therapy would need a cheap alternative indicator of best stimulation site. A second question concerns when to apply the stimulation. Some centres apply TDCS at the same time as language therapy with the idea that stimulation will improve the response to therapy, effectively "targeting" the TDCS to the synaptic connections most involved in relearning. In other cases, similar improvements have been found when rTMS has been given while patients are at complete rest. Which approach is most effective?

Answers to these questions will be found, but the problem is that large trials are needed to justify a new clinical therapy. The worry is that the treatment of aphasia is so complex and patient-oriented that such work may never be completed.