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HYPNOSIS WITH SELECTED MOVEMENT DISORDERS

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Abstract

A particular subgroup of movement disorders have aetiologies involving the basal ganglia of the brain, which coordinate signals from the motor cortex and other inputs. Such disorders are classed as resulting from shortages or imbalances of specific neurotransmitters. In recent decades, hypnosis has been used with patients suffering from Parkinson's disease, Tourette's syndrome and the dystonias, either experimentally or to achieve therapeutic changes. In this paper, an attempt to identify common and distinguishing factors is made, centred around the role of the autonomic nervous system, and in particular the role of the relaxation response. The results of previous work with Parkinson's disease, Tourette's syndrome and the dystonias is briefly reviewed, and new work with dystonia and Sydenham's chorea is presented.

Key words: Hypnosis, Parkinson's disease, Tourette's syndrome, dystonia, Sydenham's chorea, treatment

Introduction

A number of movement disorders have aetiologies involving the basal ganglia of the brain. The basal ganglia coordinate afferent signals from the whole cortex and from other parts of the brain. From these ganglia, some efferent signals go direct to the brain stem, and other signals go to the thalamus and thence to the motor cortex to initiate movement.

> [Thus] the behavioral domains of the basal ganglia can be defined to include aspects of preparation for action, the formulation of strategies and responses, and the establishment and selection of emotional responses (Saint-Cyr et al., 1995).

The movement disorders presented here are linked by imbalances of specific neurotransmitters or changes in the motor cortex, or other hypothesized neuronal dysfunction. In recent decades, hypnosis has been used with patients suffering from Parkinson's disease, Gilles de la Tourette's syndrome and the dystonias, either experimentally or to achieve therapeutic changes.

An attempt to identify some common factors is made here, centred on the role of the relaxation response and the effect of hypnosis on the cerebral cortex. Case examples of published work with Parkinson's disease and Tourette's syndrome are cited, and new effects with dystonia and Sydenham's chorea are introduced.

This paper continues a previous study (Medd, 1997) which included speculation on the mechanisms involved in the visible therapeutic effects of hypnosis on cases of torticollis, a subset of dystonia. Contact with a wider range of patients with

movement disorders has since then allowed further observations, and encouraged a search for regularities.

The four selected movement disorders

Parkinson's disease

This commonest movement disorder, which has a mean onset in the fifth decade, is characterized first by tremor, at 3 to 5 beats a second, which is present when the person is at rest and abates with movement. There is also slowness of movement, stiffness of the muscles, loss of emotional expressiveness in the face combined with reduced eye-blink, and a hesitating gait. Cognitive changes also occur, affecting learning and motor routines, and mood, and there may be psychotic experiences possibly ending with dementia.

Important among the changes in the basal ganglia and related areas of the brain which are associated with Parkinson's disease is the loss of pigmented cells in a midbrain nuclear mass, the substantia nigra, leading to a deficiency of the neurotransmitter dopamine. Standard treatment consists of administering L-dopa, the natural precursor of dopamine, in carefully calculated doses. Excess dopamine is associated with schizophrenia. It is normal for Parkinson's disease symptoms to become worse with anxiety or stress.

There is little literature on the use of hypnosis with Parkinson's disease and I will cite just one paper, by Wain and colleagues, published in 1990. The literature references in their paper covering the use of hypnosis with Parkinson's disease numbered only three.

Their synopsis reads as follows:

> Although Parkinsonian tremors typically disappear during sleep and are reduced during relaxation periods, the effects of hypnosis on this type of movement disorder have been generally ignored. We observed a patient's severe Parkinsonian tremor under hypnosis and monitored it with EEG and EMG studies. The patient was taught self-hypnosis and performed it three to four times daily in conjunction with taking medication. The results suggest that daily sessions of self-hypnosis can be a useful therapeutic adjunct in the treatment of Parkinsonian tremors (Wain et al., 1990).

The patient in the Wain study was 76 years old, and had showed the first symptoms of Parkinson's disease when he was 56. By 64, he had started on L-dopa after developing tremor and stiffness. His symptoms became worse, but cognitive abilities showed no decline. Hypnotic induction was achieved using an eye-fixation technique, deepened with progressive relaxation, then imagery using a relaxing scene from his childhood was employed, and finally time distortion, letting a minute seem like an hour of peace. The last method was most effective at reducing all tremors. Since his tremors had got worse after a decade of treatment with varieties of L-dopa, the authors suggest the benefits of a multimodal approach, for example including hypnosis, so that beside the medical help, the patient's sense of self is enhanced 'in a world of drugs and procedures'. In their discussion section, Wain and colleagues examined some earlier neurological hypotheses for the effects of hypnosis. These references were dated no later than 1978.

A recent experiment aiming to produce Parkinson's disease artificially in monkeys by inhibiting dopamine production resulted first in dystonia-type symptoms before the signs of Parkinson's disease were produced (Perlmutter et al., 1997), which is consistent with the view that Parkinson's disease and dystonia are related conditions.

Dystonia or the dystonias

Sporadic hypnosis work over the past three decades has reported intermittent therapeutic benefit with dystonia patients. Most successful work has concerned torticollis, or spasms of muscles in the neck. Torticollis accounts for about 25% of all idiopathic dystonia (Butler and Duffey, 1996). Such a focal dystonia is usually treated by injections of *botulinum* toxin into the offending muscles to prevent stimulation. A small number of patients are treated with drugs, usually anticholinergics. Symptoms become worse under stress.

The case I give here is of Angus, presented previously in 1997, and referred to me in my capacity as a dystonia counsellor.

Angus is a man in early middle age, who had suffered from spasmodic torticollis for 15 years. It caused permanent pain or discomfort in his neck and shoulder, one muscle fairly often went into spasm so that it could sometimes be seen to be twitching several times a second, and his head was slightly pulled over to the right. One of the first signs had been a noticeable tremor of the face which became a permanent feature of the torticollis and had quickly attracted a nickname at work. For reasons of unsteadiness in work-tasks in the construction industry, and through embarrassment which eventuated as a social phobia, he had been unemployed for 15 years and was treated with a variety of medications for anxiety, poor sleep and depression. All stresses made his condition worse. He rarely left the house, and then only by car to uninhabited places or to safe relatives.

I first saw him late in 1995 and we met irregularly over a protracted period into 1998. His wife, who came to all counselling sessions, finally attended alone. In the latter months, having made personal progress, he could not bear to return to the scene of painful memories at the clinic. Much work of a cognitive nature was done regarding perceptions and beliefs within the extended family and its past history, with relaxation practice, role-play and assertiveness training. Three hypnosis sessions over the first year were spent focusing on general relaxation and specifically bringing ease to the offending muscles in the neck. A good degree of comfort was obtained by this brief treatment. Use of antidepressants ceased in the first months (Medd, 1997).

Angus' attendance at counselling sessions fell away, which entailed his wife's working on her role in family dynamics. His modest improvement remained, and after one year he discontinued taking his nightly diazepam for good. In mid-1997 he experimentally stopped receiving injections of *botulinum* toxin for torticollis, which over the next 30 weeks became a permanent change.

He returned to counselling for four months in August 1997, during which time, at his own request, hypnosis was used again three times, again to link general relaxation with confidence building and help for the troubled muscles. One session was taped for him to add extra practice at home. Since early 1998, he has been quite free of tremor, spasms, torticollis and pain. Only at times of unusual stress such as an accident is there a slight discomfort in the old site of trouble. His wife estimates he is more than 95% better than he has been for many years. He can now make simple shopping trips, buy his own petrol at the filling station and manage limited mingling among strangers at public occasions.

Wain and colleagues (1990) in their paper on Parkinson's disease claimed that movement disorders showed an elimination of symptoms during deep rest or sleep. However, many idiopathic dystonia patients' partners report that the movements continue during sleep and the patients themselves report that they are aware of movements continuing during the night. It may be hypothesized that REM sleep, so close to wakefulness but marked by physical immobility, serves as a preparation for

the return of movements by activating relevant areas of the cortex. Consciousness itself, however, does not automatically correlate with involuntary movements since the hypnotized dystonia patient is both conscious and still.

An experiment with somatosensory evoked potentials (Reilly et al., 1992) found that, if a stimulus is applied to the median nerve of the hand of someone with dystonia, the evoked motor cortical potential has an unnaturally high amplitude compared with that of control subjects. It was concluded that cells in the supplementary motor cortex are unduly sensitized through endlessly repeated stimulation by signals from the basal ganglia. Parkinson's disease patients, unsurprisingly, show a below-average cortical evoked potential. Some dystonic movements – especially in drug-induced dyskinesias, which I observe respond equally well to hypnosis – closely resemble the signs of the next two types of disorder.

Gilles de la Tourette's syndrome

De la Tourette identified this disorder as having an organic origin. Up to the 1970s, it was rarely recognized. The disorder may be conceived of as a syndrome of multiple tics, usually beginning in children between the ages of five and 10 years. It is marked by twitches and jerks of the face and head, with the addition of sniffing, snorting and grimacing, and frequently the uttering of obscenities. In about one-third of cases, there is a familial clustering. Up to 40% of Tourette's syndrome children also show signs of obsessive compulsive disorder. A hereditary factor has been proposed, suggesting obsessive compulsive disorder as an alternative phenotype for a putative Tourette's syndrome gene (Singer and Walkup, 1991). Chlorpromazine and haloperidol have been used to reduce symptoms. The syndrome can be lifelong.

In the 1980s, several papers on Tourette's syndrome in young people were published in the American hypnosis literature, three involving hypnosis (Culbertson et al., 1987; Young and Montano, 1988), and one using 'intensive training' in stress-management relaxation methods (Michultka et al., 1989). Neurologically, some people conceive Tourette's syndrome in terms of a failure of normal inhibitory mechanisms following arousal irregularities whereas others implicate the reticular formation, or reticular activating system, and unnaturally prolonged neuronal firing, which in turn leads to over-arousal of the cerebral cortex.

Culbertson (1989) describes one case study and provides a good history of the hypnosis interventions involved. The patient was a 16-year-old adolescent male with Tourette's syndrome. He was taught Jacobson's progressive relaxation; a finger-tip temperature biofeedback procedure was used to encourage warmth as a sign of relaxation; Spiegel's eye-roll and hand-levitation were used to teach self-hypnosis; and imagery of a peaceful scene was used in such a way that the peaceful quality was transferred to his face and mouth. Six sessions, accompanied by frequent self-hypnosis practice at home, produced excellent results. Follow up was good, and the patient later joined the USA Air Force without the Tourette's syndrome being of concern.

Sydenham's chorea

Chorea may arise from the effects of drugs, infections, oral contraceptives, pregnancy and degenerative diseases. Treatment varies according to aetiology. The case presented here is of Sydenham's chorea, a form closely linked to childhood rheumatic fever, and now less common through treatment with penicillin. Up to the Second World War, as St Vitus' Dance, its prevalence reflected poor living conditions.

Sydenham's chorea is characterized by 'rapid, irregular, aimless involuntary movements of the muscles of the limbs, face and trunk' and may be associated with muscular weakness (Carter, 1989).

The movements are frequently confined to one side of the body, and are thought to involve the basal ganglia of the opposite side. A hypersensitivity to dopamine is probably also involved, as an after-effect of the infection. There appear to be no literature references to the use of hypnosis with Sydenham's chorea.

My client is female, retired, and began experiencing symptoms after rheumatic fever at the age of six. Soon there were marked muscular spasms on the right side of her face and neck, passing down into her right arm and leg. Schooldays were a misery because of teasing from other children, although her symptoms subsided somewhat in adult life.

As a result of continued spasms in the neck, my client now has a dystonic muscle which led to a referral through the local dystonia society to the *botulinum* toxin clinic where I work. She sought my help for depression and considerable social anxiety resulting from shakes of the head, shrugs of the shoulders and some tremor. She also had permanent discomfort from lower back problems. The neurologist had told her that there was no cure for her chorea, and drugs to reduce the spasms made her feel suicidally depressed.

She enquired about hypnotherapy and during the first hypnosis session the spasms stopped before induction was completed. At irregularly spaced sessions, hypnosis was used, and then taught to her as a self-hypnosis routine for relaxation and ego-strengthening. She worked hard at her self-hypnosis practice. After the fourth hypnosis session, the tremors began to subside sometimes when she was outdoors, but the spasms continued to be very bad in the evenings when she was tired. They were worst when she lay down to sleep, but eventually subsided after half an hour. By the tenth hypnosis session, she estimated that the spasms had reduced by about 50%. She had now had her first evening at home without spasms, and social life was much improved. Progress with the chorea has not been uniform. Other counselling work is now of equal priority after 24 meetings in nine months, but recently she reports that most evenings are now spasm-free and her social life is almost fully restored.

Conclusions

In these four movement disorders, disturbances to neurotransmitters in the basal ganglia, or irregularities in related neuronal function, have been variously identified or hypothesized. With relaxation there is the visible and experienced deactivation of the sympathetic nervous system (Benson, 1989). It is agreed by patients and researchers (Jankovic and Fahn, 1980) that stress makes symptoms worse in all of the four disorders outlined above. Conversely, peacefulness and relaxation make the symptoms reduce (Wain et al., 1990). However, in nearly every reported case, relaxation methods alone do not arrest or greatly reduce symptoms enough to be called a therapeutic advantage. The addition of hypnosis seems to be effective in achieving such a gain.

With hypnosis there are neurological effects, both observed and subjectively experienced, with all four diseases. Different mechanisms to explain the effect of hypnosis in Parkinson's disease, the dystonias and Tourette's syndrome have been proposed by several authors. There is also oversensitivity or hyperreactivity of the motor cortex for dystonia, Tourette's syndrome and Sydenham's chorea, and undersensitivity in Parkinson's disease. Instead of proposing different hypotheses for the effects of relaxation or hypnosis with each of these conditions, the principle of Occam's razor would

suggest that we try to keep the number of hypotheses to a minimum, initially by seeking common factors. First, it would seem that one common factor in the phenomena of all four conditions is the influence of the autonomic nervous system. Sympathetic arousal worsens symptoms; parasympathetic arousal ameliorates them. The hypothesized significant cause here may be the influence of adrenaline. Second, a single hypothesis regarding the effects of hypnosis with all four disorders might be that it merely amplifies the effects of relaxation.

A less simple alternative to the second hypothesis would be that hypnosis inhibits motor cortical activity in three conditions, dystonia, Tourette's syndrome and Sydenham's chorea, but reduces tremor in Parkinson's disease by another mechanism, such as by inhibiting output to the brain stem. Viewed psychologically, the permanent therapeutic advantages made with all four disorders by sustained use of hypnosis may perhaps be conceptualized most simply in terms of conditioned responses. Overall, though, it seems clear that a more physiological approach to research is required, to identify the mechanisms that lie behind the hypnotically achieved reductions in involuntary movements with these four neurological conditions.

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