Christie Noble

The role of EEG in the development of brain-computer interfaces

EEG (electroencephalography) provides us with a window into the complex world of our brains, offering a non-invasive means of monitoring changes in electrical activity. This technique has found diverse uses in both clinical and research practice. Clinically, EEG is the most useful diagnostic procedure for epilepsy, and EEG evaluation is routinely used to aid in diagnosis, to locate the epileptogenic zone, and to characterise seizures for treatment purposes[1]. EEG has a wide variety of other uses, including monitoring patients in status epilepticus[2], monitoring cerebral perfusion during carotid endarterectomy[3] and assessing brain function following traumatic brain injury[4]. EEG has also been used at the extremes of life, in the early assessment of neonates following severe intrapartum asphyxia[5] and as one of several measures used to help determine brain death[6]. EEG changes have also been shown to be a useful indicator of early cognitive impairment and may be useful in detecting the early stages of Alzheimer's disease[7].

An exciting development has been the use of EEG in brain-computer interface (BCI) technology[8]. A BCI provides a direct communication pathway between the brain and an external device, essentially "reading thoughts", with the potential for many applications of varying complexity. Since the BCI output bypasses peripheral nerves and muscles, it provides a means of communication for patients who are effectively 'locked-in' to their own bodies: patients who have severely impaired muscular control due to trauma, including complete spinal cord lesions, or a debilitating disease process, such as amyotrophic lateral sclerosis or brainstem stroke.

There are two ways in which BCIs can be utilised in the rehabilitation of patients who have lost voluntary muscle control. The first is by enabling patients to communicate with their environment directly via brain signals, bypassing the need for neuromuscular output, as discussed above. Through specific patterns of brain activity, such as paying attention to desired stimuli or through motor imagery, a person can use EEG activity to spell out words, to move a cursor on a computer screen, or even to drive a neuroprosthetic limb[8]. The second use is more complex, and is based on the idea that BCIs could be used to guide brain plasticity to restore volitional muscle control[9]. BCIs may promote motor learning by providing feedback in the absence of muscle movement, inducing activity-dependent brain plasticity by demanding close attention to motor tasks. Through popular operant conditioning paradigms, BCIs have also found therapeutic use in several neuropsychiatric conditions, allowing patients to alter their brain activity through neurofeedback[10-12].

Basis of EEG-based BCI technologies

Figure 1[9] illustrates the general principles of a BCI system. The subject must engage in a specific mental task with the resulting brain activity measured by electrodes then translated into an output, such as cursor movement on a computer screen. The subject then receives appropriate feedback on this output, allowing them to alter their brain activity, influencing subsequent output. Part of the definition of a BCI is that it must provide output to the user in a real-time interactive way. Figure 1 also shows that electrical signals from brain activity can be measured from the scalp (EEG activity), at the cortical surface (electrocorticographic, ECoG activity) or within the brain itself (neuronal action potentials). This essay will focus on the use of EEG-based BCIs, which have the advantage of being simple, non-invasive and most accessible for research purposes. Although ECoG and intracortical BCIs have better topographical resolution, concerns over their safety, the risk of tissue reaction and their stability for long-term recording still need to be addressed.

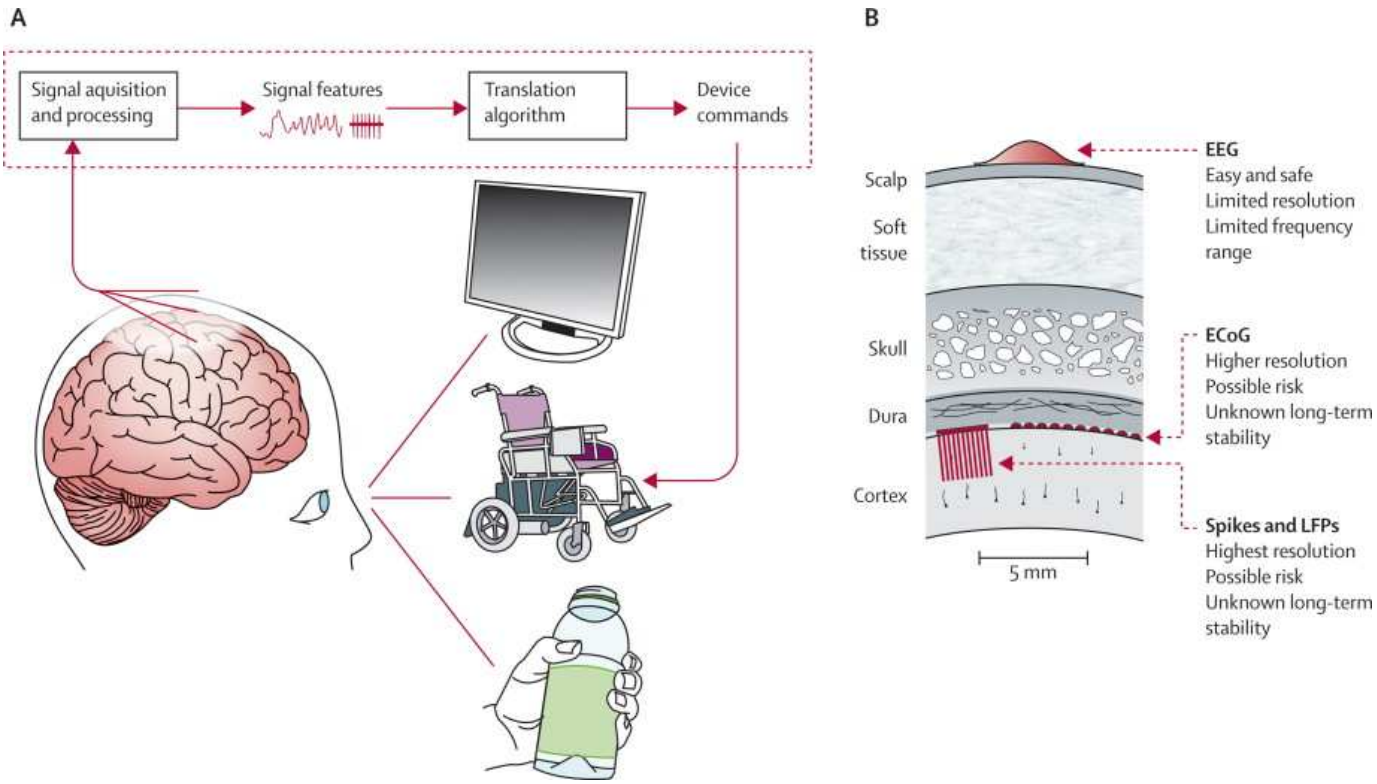

Figure 1 (from Daly *et al*[9]): **A** shows the basic principles of a BCI system. Electrical signals indicating brain activity are recorded from the scalp, then translated into a BCI output that can be used for a variety of functions, such as controlling a computer cursor, wheelchair or neuroprosthetic limb. **B** shows the possible locations of electrodes recording brain activity: on the scalp, on the cortical surface, or within the cortex.

Control of a BCI device

To control a BCI, sustained, stable and controllable brain signals are needed. The brain signals used to drive a BCI can be divided into two groups: the first are generated by stereotyped sensory stimuli, while the second involve spontaneous mental activity[13,14].

1) EEG signals evoked by stereotyped stimuli

The P300 is the most-studied evoked brain signal, and when used in a BCI, allows much faster selection of letters than any other BCI system[10]. The P300 is a component of the event-related potential (ERP), a fluctuation in the EEG that is time-locked to a sensory, motor or cognitive event[15]. The P300 is the largest component of the ERP and can be generated during an 'oddball paradigm', where a subject is asked to attend to a rare stimulus presented within a stream of standard stimuli. The P300 appears as a positive deflection about 300 ms after stimulus presentation and is related to the amount of attention paid to the stimulus. The P300 is part of the process of memory modification, where events only appear to be learned if they are surprising.

The P300 BCI was introduced by Farwell and Donchin in 1988, who created a speller BCI composed of a 6 x 6 matrix containing the 26 letters of the alphabet as well as 10 digits (0-9)[16]. Each row and column is flashed in order. The user must focus on the character that they want to communicate, and count how many times it flashes. The row and column containing the desired character then generate a P300 when they flash, while the other rows and columns do not. Detection of the P300 signals then allows the responses to be matched to one of the rows and one of the columns, so that the desired character can be identified. This allows the user to gradually spell out words, although it requires them to be able to internally spell at reasonably high speeds.

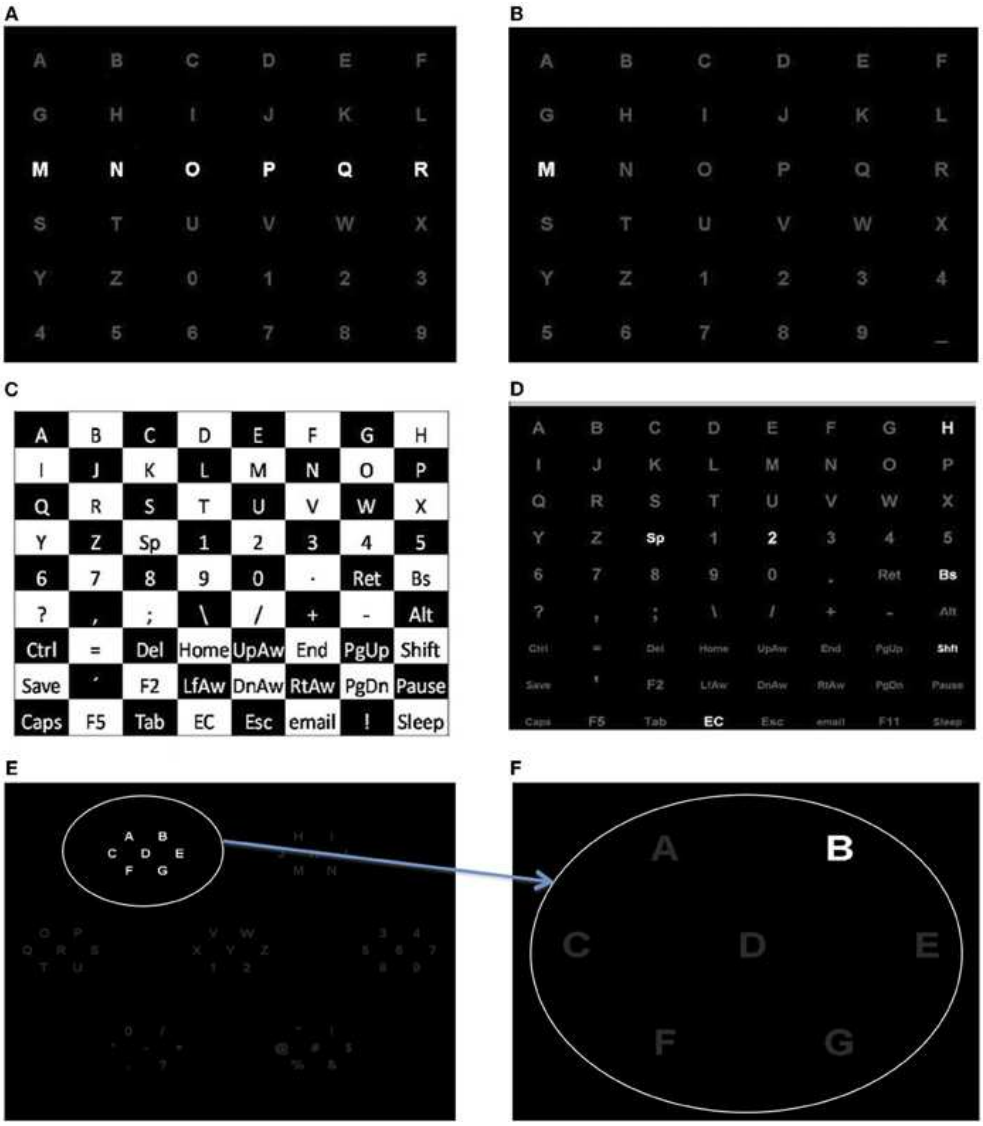

Figure 2 (from Daly *et al*[9]): **A** shows Farwell and Donchin's row/column paradigm, with each individual row and column flashed sequentially. **B** shows a single character paradigm, with each character is flashed. **C** and **D** show Townsend *et al*'s checkerboard paradigm. **E** and **F** show the region based paradigm where a set of characters in level 1 **(E)** are expanded in level 2 for spelling character "B" **(F).** P300 signals are detected when the appropriate group of letters, or individual letter, is flashed, allowing the user to spell out words.

P300 systems have several advantages in that they are straightforward to use, effective for most users and require little training (since the P300 component results from endogenous attention-based brain function)[15]. More recent P300 BCIs have moved beyond the original row/ column paradigm and can be used for a wide range of different functions. Adaptations in stimulus presentation have improved accuracy, for example the checkerboard paradigm by Townsend *et al*[17], single character flashing or two-stage region-based paradigms developed by Fazel-Rezai and Abhari[18] (see Figure 2). P300 BCIs have also been successfully introduced to home settings to improve the lives of disabled users[19,20]. Sellers *et al*[19] described a 51 year old man with amyotrophic lateral sclerosis who was no longer able to use conventional assistive devices to communicate. He was able to use an EEG-based BCI to run his NIH-funded research laboratory and to send emails to his family and friends, restoring his independence both in social interactions and in work. Therefore, P300-based BCI systems have the potential to significantly improve the quality of life and productivity of severely disabled people.

2) EEG signals created by spontaneous mental activity

Back in 1979, Birbaumer *et al* showed that slow cortical potentials (SCPs) could be brought under voluntary control through operant conditioning[10]. SCPs are positive or negative shifts in the EEG that can last from 300ms to several seconds, generated by depolarisations of large cortical neural assemblies following synchronous firing of individual neurons. Functionally, they constitute a threshold regulation mechanism for local excitatory mobilisation (negative shift) or inhibition (positive shift) of cortical networks. The neurophysiological patterns measured by EEG can be converted into visual or auditory signals, which are continuously fed-back to the subject, allowing them to self-regulate the level of cortical excitability, in a process known as 'neurofeedback'[21].

Changes in brain activity that are made in the desired direction are then rewarded (positively reinforced). This training occurs without the subject being consciously aware of how SCP control is achieved, a voluntary but automatic skill that could be compared to learning to ride a bike. Self-regulation of SCPs through operant conditioning requires extensive training, but has been shown to be useful in basic word processing, for example allowing the subject to move a computer cursor to select letters or words [22].

Sensorimotor rhythms (SMRs) are 8-12 Hz(μ) and 18-26Hz (β) oscillations in EEG signals recorded over primary sensorimotor cortex, with amplitudes altering during sensation, movement and even motor imagery. Subjects can also develop the skill of altering the activity in these different EEG frequency bands through neurofeedback training. Reductions in amplitude occur in preparation for and during movement, as well as during motor imagery, while increases occur in the post-movement period. Due to the somatotopic organisation of somatosensory cortex, imagined movements will activate characteristic areas of cortex (for example, imagining hand movement abolishes SMRs over the 'hand region') which can be reliably located and recorded. Subjects can use motor imagery to control μ and β rhythm amplitudes in the absence of movement or sensation, and can use this control to move a cursor to make selections on a screen, or even to control a simple orthotic device[23]. For example, ALS patients were successfully trained to move a computer cursor to one of two targets by altering their SMRs: low SMR amplitudes (during motor imagery) moved the cursor towards a target at the top of the screen, while high SMR amplitudes (thinking of nothing specific) moved the cursor downwards towards a target at the bottom of the screen[24]. Neurofeedback can be used to aid motor imagery training[25].

Studies have also looked at different, non-motor strategies: one study even found that spatial navigation around a familiar environment or auditory imagery of a familiar tune produced more reliable EEG patterns and BCI control than right and left motor imagery of opening and closing the hand[26]. This may be useful for patients who have been paralysed for many years, or even from birth, in whom motor imagery may be difficult[27].

Using BCIs in the recovery of volitional motor control

In 2000, Pfurtscheller *et al*[28] described the case of a tetraplegic patient, TS, whose only residual muscle activity in his upper limbs following a C4/5 spinal cord lesion was in his left biceps. Their study involved the development of an electronic hand orthosis to support the grasp function of his left hand, as well as extensive EEG training. After a training period of several months, TS learned to control the hand orthosis through motor imagery: imagining movement of both feet (also paralysed) closed the hand, while imagining movement of the paralysed right hand opened it. This control was mainly achieved through TS's ability to voluntarily induce specific beta oscillations on EEG recordings from the area of somatosensory cortex representing the feet. Within five months of training with the EEG-controlled orthosis, TS was able to eat his first apple, using the combination of the residual activity in his left biceps and the grasping function returned to his left hand. Pfurtscheller and his team then went on to develop a similar EEG-based BCI system for TS, also based on motor imagery (foot movement), but which used functional electrical stimulation (FES) as its output[29]. FES could provide increased control of hand grasping by using electrodes to stimulate four different hand muscle groups. The grasp function of the hand was divided into five sequential grasp phases, with each repetition of foot movement imagination (and the resultant beta burst) triggering a transition to the next phase. However, although functional movement was restored to the patient in both these studies, no improvements in voluntary motor function in the paralysed hand were reported[30].

The use of BCIs to help encourage and guide neural plasticity, promoting the restoration of motor function, particularly as part of stroke rehabilitation, is a relatively new research direction[31]. Motor recovery after stroke is associated with structural and functional changes, including increased axonal sprouting in the region around the infarct and increased synaptogenesis[9]. By providing a feedback

signal in the absence of normal volitional movement, EEG-based BCIs can support the process of cortical reorganisation[9]. The use of a BCI requires intense focus of concentration on the intended motor task, one of the critical motor learning principles needed for acquisition of motor skills and motor learning[32].

A 2011 study[33] used transcranial magnetic stimulation to assess whether sensorimotor rhythm-based BCI training could induce persistent functional changes in motor cortex. They found that training healthy, naïve subjects to move a cursor using motor imagery resulted in a significant increase in motor cortical excitability. Subjects who developed a motor imagery strategy based on hand grasping developed an enhancement of hand muscle representation in the cortex, specifically the hand muscle engaged by the imagined motor action – opponens pollicis. In addition, the motor imagery training appeared to induce more widespread changes in brain network topology, with a global efficiency decrease observed, suggesting more optimal local information processing. This supports the idea that using motor imagery strategies to control a BCI can lead to plastic changes in the brain, a principle that may be useful in stroke rehabilitation.

More recent studies have worked on this theory and provide evidence that BCI devices can promote the return of volitional motor control. For example, Daly *et al* described the case of a 43-year-old woman who had suffered a stroke 10 months previously[32]. She had both gait and upper limb motor deficits, including in her right hand, where she had partial volitional movement but was unable to produce isolated movements of any of her fingers. One of the important factors in motor learning is to be able to practise the desired movement in as normal a manner as possible. If abnormal movements are repeated, cortical reorganisation may lead to these movements becoming progressively more abnormal, impeding a return to normal function. Since many stroke survivors are unable to move in a way that approximates normal movement, FES can be used to assist movement (as described in one of the studies above), allowing practice of the desired coordinated movement. Using EEG recordings from the lesioned hemisphere, Daly *et al* used a BCI to trigger FES for movement practice in this woman (individual extension of the fingers of the right hand). The BCI+FES system involved trials that involved both imagined and attempted finger movement and relaxation. After only nine training sessions, the woman demonstrated recovery of volitional isolated index finger extension (from no movement before training, to an extension of 26 degrees). This is an area that needs further investigation, but there is clearly promise in the ability of an EEG-based BCI to promote the restoration of independent volitional movement through an impact on neuronal plasticity.

EEG-based BCIs in therapeutic neurofeedback training

As discussed above, 'neurofeedback' training is a useful application of BCI technology in which subjects learn to gain self-control over their EEG patterns. Activity in different EEG frequency bands can be decreased or increased, while training of SCPs addresses the regulation of cortical excitability[21]. Training to self-regulate brain waves in this way has also been reported to have therapeutic effects in a variety of psychiatric and neurological conditions, including schizophrenia[12], obsessive compulsive disorder[34], epilepsy and ADHD[35]. Interestingly, only SCPs originating from central and frontal areas of the brain appear to be susceptible to operant control, while SCPs originating from posterior parietal or occipital sources cannot be voluntarily regulated[10]. Neurofeedback has found particular use in patients with drug-refractory epilepsy, who are able to reduce the frequency of seizures through self-regulation of SCPs[11].

Several studies have found encouraging results in relation to the use of EEG neurofeedback in improving attention and behavioural control in children with attention deficit hyperactivity disorder (ADHD), although this treatment has yet to be widely accepted into clinical practice[35]. Abnormal patterns of cortical activation are seen in power spectral analysis and event-related cortical potentials in children with ADHD[36]. Neurofeedback training can be run as a computer game and can therefore be effectively adapted for use in these children. In a randomised controlled trial run by Gevensleben *et al*

in 2009[37], 102 children with ADHD were trained to decrease activity in the theta band of their EEG (4-8Hz), and to increase activity in the beta band (13-20Hz), decreasing the theta/beta ratio. (ADHD has been associated with increased slow wave activity (theta) and reduced alpha/ beta activity). They found that the children who were able to reduce theta activity during training showed improvements on an ADHD behavioural rating scale. Training children to self-regulate SCPs has also been implicated as a potential treatment for ADHD[38]. SCP shifts in an electrically negative direction reflect the depolarisation of large cortical neural assemblies, reducing their excitation threshold during states of cognitive preparation. Studies have shown that negative SCP shifts are reduced in children with ADHD, and that learned self-control of SCPs, with enhancement of negative SCPs, is accompanied by a reduction in impulsivity errors[39].

A clinical case series was recently published on the efficacy of quantitative EEG-guided neurofeedback treatment in a group of patients suffering from schizophrenia refractory to antipsychotic medication[12]. With a prevalence of 1 in 100, schizophrenia can be a devastating illness because of its early onset and the destructive nature of its symptoms for the patient and their family. Of 48 patients who completed an individualised neurofeedback treatment protocol (completing an average of about 60 sessions), 47 showed clinical improvement in their symptoms (using the Positive and Negative Symptom Scale, PANSS). This study provides the first evidence that neurofeedback is an effective treatment in schizophrenia, and suggests that this is an area that may need further research. The same researchers have also published a case series on obsessive compulsive disorder, demonstrating that neurofeedback training appears to have similar efficacy, and that the improvement in symptoms is often long-lasting[34].

Conclusions

The electroencephalogram has found diverse functions within the field of brain-computer interfaces, transforming the lives of many severely disabled patients. It has provided a means of communication with the outside world, as well as promoting rehabilitation in paralysed or even 'locked in' patients, and has also found therapeutic use in several neuropsychiatric disorders. Looking to the future, the impact of these simple, non-invasive BCI devices will be dependent on further improvement in the ease and convenience of their day-to-day use, but further technological advances are likely to greatly improve their abilities for communication and control. In particular, further research into their effects on cortical plasticity will reveal the role that this technology may play in promoting the return of independent, voluntary movement.

References

1) Noachtar S, Remi J (2009) The role of EEG in epilepsy: a critical review. Epilepsy Behav 15:22-33
2) Bleck TP (2012) Status epilepticus and the use of continuous EEG monitoring in the intensive care unit. Continuum (Minneapolis Minn) 18:560-78
3) Florence G, Guerit JM, Gueguen B (2004) Electroencephalography (EEG) and somatosensory evoked potentials (SEP) to prevent cerebral ischaemia in the operating room. Neurophysiol Clin 34:17-32
4) Arciniegas DB (2011) Clinical electrophysiological assessments and mild traumatic brain injury: state-of-the-science and implications for clinical practice. Int J Psychophysiol 82:41-52
5) Lukaskova J, Tomsikova Z, Kokstein Z (2008) Cerebral function monitoring in neonates with perinatal asphyxia – preliminary results. Neuro Endocrinol Lett 29:522-8
6) Sediri H, Bourriez JL, Derambure P (2007) Role of EEG in the diagnosis of brain death. Rev Neurol (Paris) 163:248-53
7) Onishi J, Suzuki Y *et al* (2005) Predictive model for assessing cognitive impairment by quantitative electroencephalography. Cogn Behav Neurol 18:179-84
8) Wolpaw JR, Birbaumer N *et al* (2002) Brain-computer interfaces for communication and control. Clin Neurophysiol 113:767-91
9) Daly JJ, Wolpaw JR (2008) Brain-computer interfaces in neurological rehabilitation. Lancet Neurol 7:1032-43
10) Birbaumer N, Cohen LG (2007) Brain-computer interfaces: communication and restoration of movement in paralysis. J Physiol 579:621-36
11) Kotchoubey B, Strehl U *et al* (2001) Modification of slow cortical potentials in patients with refractory epilepsy: a controlled outcome study. Epilepsia 42:406-16
12) Surmeli T, Ertem A *et al* (2012) Schizophrenia and the efficacy of qEEG-guided neurofeedback treatment: a clinical case series. Clin EEG Neurosci 43:133-44
13) Cabrera AF, Dremstrup K (2008) Auditory and spatial navigation imagery in brain-computer interface using optimised wavelets. J Neurosci Methods 174:135-46
14) Curran EA, Stokes MJ (2003) Learning to control brain activity: a review of the production and control of EEG components for driving brain-computer interface (BCI) systems. Brain Cogn 51:326-36
15) Fazel-Rezal R, Allison BZ *et al* (2012) P300 brain computer interface: current challenges and emerging trends. Front Neuroeng. 5:14
16) Farwell LA, Donchin E (1988) Talking off the top of your head: toward a mental prosthesis utilising event-related brain potentials. Electroencephalogr Clin Neurophysiol 70:510-23
17) Townsend G, LaPallo BK *et al* (2010) A novel P300-based brain-computer interface stimulus presentation paradigm: moving beyond rows and columns. Clin Neurophysiol 121:1109-20
18) Fazel-Rezai R, Gavett S *et al* (2011) A comparison among several P300 brain-computer interface speller paradigms. Clin EEG Neurosci 42:209-13
19) Sellers EW, Vaughan TM, Wolpaw JR (2010) A brain-computer interface for long-term independent home use. Amyotroph Lateral Scler 11:449-55
20) Kleih SC, Kaufmann T *et al* (2011) Out of the frying pan into the fire – the P300-based BCI faces real-world challenges. Prog Brain Res 194:27-46
21) Heinrich H, Gevensleben H, Strehl U (2007) Annotation: neurofeedback – train your brain to train behaviour. J Child Psychol Psychiatry 48:3-16
22) Kubler A, Neumann N (2001) Brain-computer communication: self-regulation of slow cortical potentials for verbal communication. Arch Phys Med Rehabil 82:1533-9
23) Machado S, Araujo F *et al* (2010) EEG-based brain-computer interfaces: an overview of basic concepts and clinical applications in neurorehabilitation. Rev Neurosci 21:451-68
24) Kubler A, Nijboer F *et al* (2005) Patients with ALS can use sensorimotor rhythms to operate a brain-computer interface. Neurology 64:1775-7
25) Hwang HJ, Kwon K, Im CH (2009) Neurofeedback-based motor imagery training for brain-computer interface (BCI). J Neurosci Methods 179:150-6

26) Curran E, Sykacek P *et al* (2004) Cognitive tasks for driving a brain-computer interfacing system: a pilot study. IEEE Trans Neural Rehabil Eng 12:48-54
27) Cabrera AF, Farina D Dremstrup K (2010) Comparison of feature selection and classification methods for a brain-computer interface driven by non-motor imagery. Med Biol Eng Compu 48:123-32
28) Pfurtscheller G, Guger C *et al* (2000) Brain oscillations control hand orthosis in a tetraplegic. Neurosci Lett 292:211-4
29) Pfurtscheller G, Muller GR *et al* (2003) 'Thought'-control of functional electrical stimulation to restore hand grasp in a patient with tetraplegia. Neurosci Lett 351:33-6
30) Birbaumer N, Murguialday AR, Cohen L (2008) Brain-computer interface in paralysis. Curr Opin Neurol 21:634-8
31) Grosse-Wentrup M, Mattia D, Oweiss K (2011) Using brain-computer interfaces to induce neural plasticity and restore function. J Neural Eng 8:25004
32) Daly JJ, Cheng R *et al* (2009) Feasibility of a new application of non-invasive brain computer interface (BCI): a case study of training for recovery of volitional motor control after stroke. J Neurol Phys Ther 33:203-11
33) Pichiorri F, De Vico Fallani F *et al* (2011) Sensorimotor rhythm-based brain-computer interface training: the impact on motor cortical responsiveness. J Neural Eng 8:25020
34) Surmeli T, Ertem A (2011) Obsessive compulsive disorder and the efficacy of qEEG-guided neurofeedback treatment: a case series. Clin EEG Neurosci 42:195-201
35) Monastra VJ, Lynn S *et al* (2005) Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. Appl Psychophysiol Biofeedback 30:95-114
36) Monastra VJ (2008) Quantitative electroencephalography and attention-deficit/hyperactivity disorder: implications for clinical practice. Curr Psychiatry Rep 10:432-8
37) Gevensleben H, Holl B *et al* (2009) Distinct EEG effects related to neurofeedback training in children with ADHD: a randomised controlled trial. Int J Psychophysiol 74:149-57
38) Strehl U, Leins U *et al* (2006) Self-regulation of slow cortical potentials: a new treatment for children with attention-deficit/hyperactivity disorder. Pediatrics 118:e1530-40
39) Heinrich H, Gevensleben H *et al* (2004) Training of slow cortical potentials in attention-deficit/hyperactivity disorder: evidence for positive behavioural and neurophysiological effects. Biol Psychiatry 55:772-5