

Summary of the project

There is convincing evidence in the literature that chronic neuropathic pain results from abnormal brain processing of pain. Thalamocortical dysrhythmia is one of the main mechanisms resulting in abnormal firing of thalamic neurones in the brain as bursts within the theta frequency range (4-8 Hz). QEEG and SSEPs have been used to visualise the effects of thalamocortical dysrhythmia on the brain cortex in neuropathic pain patients. In chronic neuropathic pain an increase in theta QEEG frequency power (4-8 Hz) is observed with neighbouring areas showing increases in beta (13-25 Hz) QEEG power. Similarly, SSEPs show an increase in amplitude in chronic neuropathic pain in keeping with somatosensory cortical excitation. Spinal cord stimulation (SCS) is offered to patients with medically refractory neuropathic pain of more than 6 months duration and a subjective pain score >5. In the clinical setting the three most frequently used spinal cord stimulator settings are low, high, and burst frequency stimulation. There is debate in the literature on which of these three is the best however it is recognised that after a failed trial of one type of stimulation a second trial of another type of stimulation is effective in another 10-15% patients. After a successful trial of at least 50% pain reduction only 50-70% of true responders maintain sustained benefit after implantation. Therefore, to personalise the treatment for an individual patient there is an argument to trial all three types of stimulation prior to decision making regarding the implant. The ability to predict on an individual level both the therapeutic response to spinal cord stimulation and the best method of stimulation would have significant impact on patient suffering and health economics. Currently there are no objective measures to predict true responders or which method of stimulation is the best for a given patient.

Hypothesis:

An increase in QEEG theta power and augmentation of SSEP amplitudes can be used as biomarkers of chronic neuropathic pain and that theta QEEG power and SSEP amplitudes are reduced by effective spinal cord stimulation using either low, high, or burst stimulation.

Specific aims of the study

To determine if reversal in QEEG theta power and/or SSEP amplitude during the stimulator trial period can predict a sustained response 6 months post implantation

Current progress

This last financial year has been dominated by gaining ethical approval which was achieved on 26th July 2021 from the Research Ethics Committee (IRAS project ID: 283335) and approval from Walton Centre Clinical Governance group for research. Minor amendments were made to the patient information prior to approval. Patient information and consent forms are now completed and ready to be used. September – November 2021 involved a recent review of the current literature which was submitted to Manchester Metropolitan University as a part of a clinical doctorate program for trainee Consultant Clinical Scientists (HSST).

In March 2022 the project was presented to two stake holders – Pain Consultants responsible for recruiting the patients for the project and the neuromodulation nurses involved in changing the stimulator settings. Support was gained from both groups and approval gained to start identifying suitable patients for the project. Challenges identified by the neuromodulation nurses were around room access, a plan has been identified which we aim to trial with the first patient.

The project was also presented to the Neurophysiology department and a small team identified who will assist in data collection.

The project test protocol has been written and the EEG and SSEP recording system (Deymed 3 in 1 EEG/EP system distributed by Neurogen) has been tested on a volunteer. EEG data was successfully exported for data analysis and a sample was analysed for alpha power (resting EEG rhythm seen with the eyes closed). The power maps produced in figure 1 are sufficient for the project which will look for theta power a marker of chronic pain.

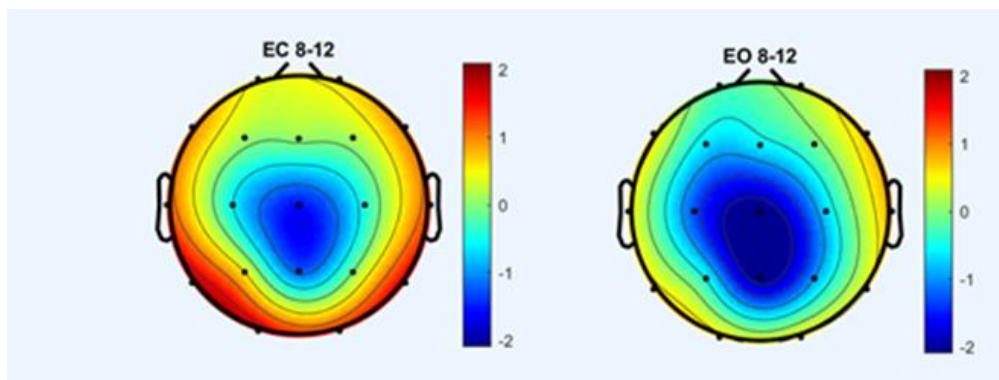


Figure 1: EEG test data for alpha power displayed as a power map

SSEP data export requires an export script to be written which is currently being undertaken by Deymed (system manufacturer) which is scheduled for completion by the end of June 2022. In May 2022, three patients were identified for the project and have been given patient information prior to the consent process. The plan is to start the first few patients in June/July 2022 trailing the project test protocol and trial workflow (figure 2).

Site	Channel	Impedance	Gain	Filter	Reference
1	EEG	100k	1000	30	Common
2	EEG	100k	1000	30	Common
3	EEG	100k	1000	30	Common
4	EEG	100k	1000	30	Common
5	EEG	100k	1000	30	Common
6	EEG	100k	1000	30	Common
7	EEG	100k	1000	30	Common
8	EEG	100k	1000	30	Common
9	EEG	100k	1000	30	Common
10	EEG	100k	1000	30	Common
11	EEG	100k	1000	30	Common
12	EEG	100k	1000	30	Common
13	EEG	100k	1000	30	Common
14	EEG	100k	1000	30	Common
15	EEG	100k	1000	30	Common
16	EEG	100k	1000	30	Common
17	EEG	100k	1000	30	Common
18	EEG	100k	1000	30	Common
19	EEG	100k	1000	30	Common
20	EEG	100k	1000	30	Common
21	EEG	100k	1000	30	Common
22	EEG	100k	1000	30	Common
23	EEG	100k	1000	30	Common
24	EEG	100k	1000	30	Common
25	EEG	100k	1000	30	Common
26	EEG	100k	1000	30	Common
27	EEG	100k	1000	30	Common
28	EEG	100k	1000	30	Common
29	EEG	100k	1000	30	Common
30	EEG	100k	1000	30	Common
31	EEG	100k	1000	30	Common
32	EEG	100k	1000	30	Common
33	EEG	100k	1000	30	Common
34	EEG	100k	1000	30	Common
35	EEG	100k	1000	30	Common
36	EEG	100k	1000	30	Common
37	EEG	100k	1000	30	Common
38	EEG	100k	1000	30	Common
39	EEG	100k	1000	30	Common
40	EEG	100k	1000	30	Common
41	EEG	100k	1000	30	Common
42	EEG	100k	1000	30	Common
43	EEG	100k	1000	30	Common
44	EEG	100k	1000	30	Common
45	EEG	100k	1000	30	Common
46	EEG	100k	1000	30	Common
47	EEG	100k	1000	30	Common
48	EEG	100k	1000	30	Common
49	EEG	100k	1000	30	Common
50	EEG	100k	1000	30	Common
51	EEG	100k	1000	30	Common
52	EEG	100k	1000	30	Common
53	EEG	100k	1000	30	Common
54	EEG	100k	1000	30	Common
55	EEG	100k	1000	30	Common
56	EEG	100k	1000	30	Common
57	EEG	100k	1000	30	Common
58	EEG	100k	1000	30	Common
59	EEG	100k	1000	30	Common
60	EEG	100k	1000	30	Common
61	EEG	100k	1000	30	Common
62	EEG	100k	1000	30	Common
63	EEG	100k	1000	30	Common
64	EEG	100k	1000	30	Common
65	EEG	100k	1000	30	Common
66	EEG	100k	1000	30	Common
67	EEG	100k	1000	30	Common
68	EEG	100k	1000	30	Common
69	EEG	100k	1000	30	Common
70	EEG	100k	1000	30	Common
71	EEG	100k	1000	30	Common
72	EEG	100k	1000	30	Common
73	EEG	100k	1000	30	Common
74	EEG	100k	1000	30	Common
75	EEG	100k	1000	30	Common
76	EEG	100k	1000	30	Common
77	EEG	100k	1000	30	Common
78	EEG	100k	1000	30	Common
79	EEG	100k	1000	30	Common
80	EEG	100k	1000	30	Common
81	EEG	100k	1000	30	Common
82	EEG	100k	1000	30	Common
83	EEG	100k	1000	30	Common
84	EEG	100k	1000	30	Common
85	EEG	100k	1000	30	Common
86	EEG	100k	1000	30	Common
87	EEG	100k	1000	30	Common
88	EEG	100k	1000	30	Common
89	EEG	100k	1000	30	Common
90	EEG	100k	1000	30	Common
91	EEG	100k	1000	30	Common
92	EEG	100k	1000	30	Common
93	EEG	100k	1000	30	Common
94	EEG	100k	1000	30	Common
95	EEG	100k	1000	30	Common
96	EEG	100k	1000	30	Common
97	EEG	100k	1000	30	Common
98	EEG	100k	1000	30	Common
99	EEG	100k	1000	30	Common
100	EEG	100k	1000	30	Common

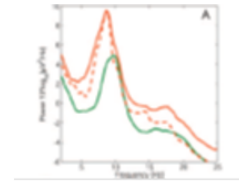
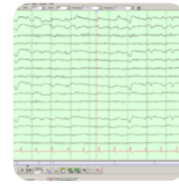
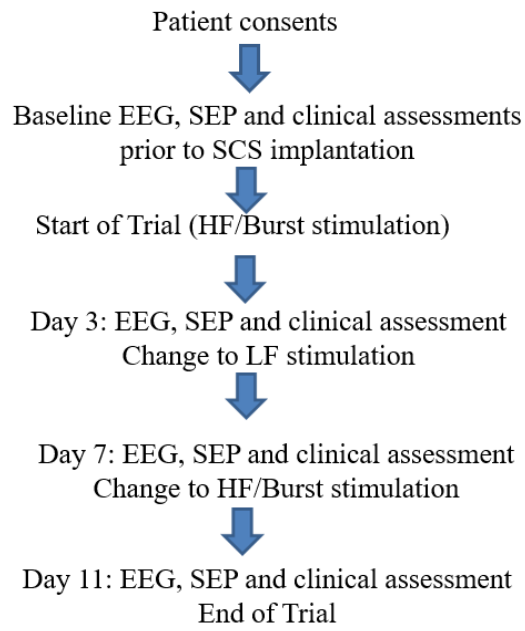
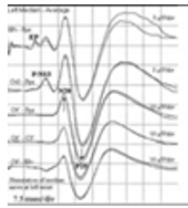


Figure 2: Project workflow