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Stimulation induced biopotential amplifier saturation due to common mode voltage

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Introduction

Functional Electrical Stimulation (FES) is frequently used in the rehabilitation of Spinal Cord Injury. For research purposes it is often necessary to record Electromyographs (EMG) during stimulation to assess the efficacy of FES treatments, however stimulation artefact can corrupt or distort the EMG signal. Three main causes of stimulation artefact are known: voltage gradient induced potential difference between recording electrodes due to stimulation current flowing through the tissue [1]; residual charge on the electrode-skin interface at the end of a stimulation pulse [2], and; amplifier saturation due to common mode (CM) voltages exceeding the amplifier's CM voltage range [3]. In practice, it can be difficult to ascertain which of the causes are responsible for stimulation artefact interference if the capability of the amplifier is not known. Here we present a method to simulate excessive CM voltage as a means to characterise the sensitivity of a biopotential amplifier to CM voltages.

Methods

A Bipolar Junction Transistor (BJT) configuration (fig. 1a) was used to realise a differential signal relative to the EMG reference (\sim 2 mV_{p-p}, 100 Hz) as a proxy for an EMG signal to the amplifier (Digitimer, D360, gain: 100x, bandwidth: 10-500 Hz). A Digitimer, DS8R stimulator was used to generate a train of 10 stimulation pulses (80 mA constant-current, biphasic, 1 ms pulse width, 30 Hz) that were coupled into the amplifier input through a large resistor used to simulate skin and electrode impedance. The stimulation pulse was sufficient to generate a CM voltage of approximately 15 V relative to the EMG reference electrode. The amplifier output was captured using a CED Power 1401 data acquisition system and visualised with CED Signal software (Version 6), sampling frequency 2 kHz.

Results & Discussion

The amplifier is desensitised for at least 30-50 ms after each stimulation pulse (fig. 1b). The effect was present with both monophasic and biphasic stimulation. At stimulation current intensities below 80 mA, the effect was worse until a CM voltage of around 11 V was reached when the effect stabilised; most likely due to ESD protection built into the preamplifier. Connecting parallel back to back clamping diodes between the amplifier inputs and the amplifier reference have a beneficial effect by limiting the CM voltage to approximately 0.7 V thus reducing the impact and the recovery time of the amplifier to around 20 ms. In a clinical setting consideration should be given to the positioning of the stimulating and recording electrodes so as to minimise the CM voltage.

Conclusion

The circuit presented provides a means to characterise a biopential amplifier in response to stimulation induced CM voltage. Such knowledge allows an amplifier's suitability (amplifier recovery time vs stimulation frequency) for an intended application to be determined.

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References

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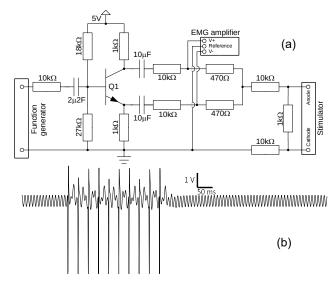


Fig 1: (a) Circuit used to realise an EMG signal proxy and to couple the stimulation pulse to the amplifier input. (b) Amplifier output captured in response to a 10-pulse stimulation train.