by mild muscle contraction in the index finger".[1] According to *Anatomy for Acupuncture* by Dorsher and Cummings (http://www.anatomy.tv/atv2/mainwindow.aspx?ref=0&titleid=26&svrid=0&App=acupuncture), sensation from the skin of the first dorsal web space is conveyed by the terminal radial nerve, and the muscles most often needled at this point are the first dorsal interosseous and adductor pollicis brevis, both of which are innervated by the ulnar nerve (myotome C8-T1). Deep needling closer to the first metacarpal bone may penetrate flexor pollicis brevis, which is innervated by the ulnar and median nerves (myotome C8-T1), but this is not usually achieved with needling to 1.25 cm depth at LI4. If the needle were to have been directed ventrally to the second metacarpal bone (in the direction of SI3, on the ulnar border of the hand at the level of the palmar crease), the needle might have reached the first lumbrical, which is also innervated by the median nerve.[2] In this case, flexion of the metacarpophalangeal joint with extension of the interphalangeal joints of the second finger would have been elicited with EA.

When describing the movement of the index finger or forefinger when performing EA at LI4, it should have been mentioned whether there was abduction of the index finger, adduction of the thumb, or both, reflecting stimulation of the first dorsal interosseous, the adductor pollicis brevis, or both. If flexion of the metacarpophalangeal and carpometacarpal joints of the thumb was elicited, the needle would likely have been placed in the flexor pollicis brevis. Flexion of the metacarpophalangeal joint with extension at the interphalangeal joints of the second finger would indicate needling of the first lumbrical. Stating the direction of insertion or, even better, identification of movement during EA, would have identified which muscle(s) the needle had reached and which nerves were being stimulated.[2]

In the description of the experimental EA protocol, several aspects require elucidation.

It is stated that a "continuous rectangular waveform (pulse width 30 ms) at a frequency of 4 Hz"[1] was applied. The polarity of the current applied should have been stated as this has implications for the experimental protocol. When using a polar current, nerve fibre stimulation, even if less intense than at the negative pole, may also be elicited at the positive pole (figure 1). In the experimental protocol the electrode was placed onto the surface of the leg, which was not under anaesthesia, so cutaneous afferent stimulation could have elicited brain activity, thus affecting the outcome of the experience. It should have been

Interscalene brachial plexus blockade does not guarantee complete C8-T1 root block

Dear editor,

After carefully reading the recent paper by Gu *et al*,[1] a few questions and concerns arose.

In the Background it is mentioned that "LI4 is supplied by the median nerve".[1] No reference is given to support this statement. Also, it is stated that determination of electroacupuncture (EA) intensity "was generally accompanied

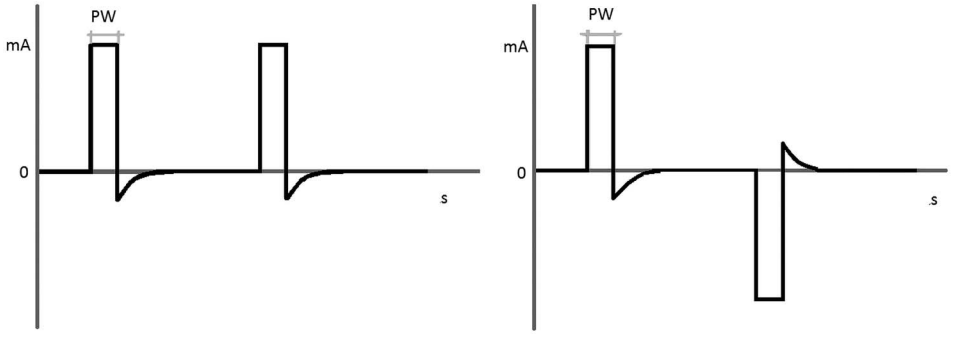

Figure 1 Examples of polar (left panel) and apolar (right panel) currents, also called monophasic or biphasic, respectively. Polar currents pose a greater risk of acid-base reactions leading to tissue damage, and electroacupuncture devices using polar currents should be used with greater care. Apolar currents may depolarise nerve fibres at both poles, as the negative deflection is counter-balanced by an equal, positive deflection. Intensity of current (mA) and time(s) are shown on the X and Y axes, respectively. PW, pulse width (μs).

stated whether any sensation occurred in the leg during the experiment. Alternatively, placing the electrode on the same arm (which was under brachial plexus blockade) would have avoided any stimulation at the positive pole.

Moreover, a pulse width of 30 ms (milliseconds) is not common with EA devices, which usually operate in the range of microseconds (μs), usually up to 500 μs;[3] 30 ms equals 30 000 μs—was this a typographical error?

The intensity of the EA used in the treatment protocol was stated as '5–12 mA'.[1] From our own personal experience, this seems to be too high. We usually use EA up to 3 mA, and, if the needle tip is correctly inserted in the vicinity of the intended muscle(s)' motor point(s), currents as low as 1 mA may be sufficient to elicit strong muscle contraction. A 4 Hz pulse with 30 000 μs of pulse width at an intensity of 5–12 mA would most likely have stimulated skin, fascia and muscle nociceptor fibres, making it very painful and difficult to withstand for 10 min in each EA session.

It is also stated that "one week before the experiment, the intensity of EA was tested for each patient" and "the same individual intensity as that established was used during all three treatments for each participant".[1] Intensity of stimulation depends on how close the needle tip or shaft is to the sensory or motor nerves stimulated by EA, and it is unreasonable to expect that the needle will lay at the same exact point during each needling session. Therefore the intensity of stimulation should have been adjusted based on muscle contraction or sensation for each individual patient and separately for each of the three sessions of EA, making sure that adequate stimulation intensity was achieved every time. Most importantly, the method for establishing the intensity of stimulation poses a serious problem. While under brachial plexus block, sensation (from skin, fascia, and muscle afferents) is abolished. Thus, the only way nerve stimulation by EA at the site of insertion can be proven is by visible or palpable muscle contraction, and this observation was not stated in the experimental protocol. Consequently, the procedure to determine intensity of stimulation does not assure us that proper stimulation of local nerve fibres was achieved in every patient, especially under brachial plexus blockade. Thus, lack of elicitation of specific cerebral activity by EA while under brachial plexus blockade may have been due to inadequate peripheral intensity of EA rather than blockade of nerve transmission from the insertion site by the brachial plexus block.

The chosen method of brachial plexus blockade also poses a serious problem for the experimental protocol. The interscalene blockade does not assure complete anaesthesia or motor paralysis of the entire upper limb, as roots C8 and T1 may be spared by the procedure[4] in as many as 15–20% of cases (http://www.nysora.com/techniques/nerve-stimulator-and-surface-based-ra-techniques/upper-extremitya/3346-interscalene-brachial-plexus-block.html). While the skin of the dorsal aspect of the first web space usually corresponds to the C6 dermatome, the muscles usually needled at LI4 are innervated mostly by the C8 and T1 spinal nerve roots. As for the methodology used to control for any central effects of lidocaine (intramuscular injection into the deltoid), this poses a risk of myotoxicity,[5] and perhaps a safer technique (eg, interfascial plane infiltration) should have been considered.

In summary, the conclusion by the authors that brachial plexus nerve block abolishes activation of specific brain regions by EA at LI4 must be reconsidered in light of all of these concerns.

Gustavo Pereira,[1] Fabio Athayde,[2] António Paulo Martins da Encarnação[3]

[1]Department of Anesthesiology, Hospital de Braga, Braga, Portugal
[2]TechAccus, Curitiba, Paraná, Brazil
[3]Braga, Portugal

Correspondence to Dr António Paulo Martins da Encarnação, Rua do Carvalhal, 25, Dume, Braga 4700-051, Portugal; antoniopauloencarnacao@yahoo.co.uk

Acknowledgements We would like to thank Rafael Vercelino for pointing out an important anatomical detail regarding LI4, as referred to in Chapple.[2]

Contributors All authors participated in the writing and discussed the final text.

Competing interests None declared.

Provenance and peer review Not commissioned; internally peer reviewed.

To cite Pereira G, Athayde F, Martins da Encarnação AP. *Acupunct Med* 2016;**34**:156–157.

Accepted 24 October 2015
Published Online First 18 November 2015

▶ http://dx.doi.org/10.1136/acupmed-2015-010901
▶ http://dx.doi.org/10.1136/acupmed-2015-010901corr1
▶ http://dx.doi.org/10.1136/acupmed-2015-011031

Acupunct Med 2016;**34**:156–157.
doi:10.1136/acupmed-2015-010996

REFERENCES

1 Gu W, Jiang W, He J, *et al*. Blockade of the brachial plexus abolishes activation of specific brain regions by electroacupuncture at LI4: a functional MRI study. *Acupunct Med* 2015;33:457–64.
2 Chapple W. Proposed catalog of the neuroanatomy and the stratified anatomy for the 361 acupuncture points of 14 channels. *J Acupunct Meridian Stud* 2013;6:270–4.
3 Mayor DF. *Electroacupuncture: a practical manual and resource*. Elsevier, 2007:39–40.
4 Barash PG, ed. *Clinical anesthesia*. 7th edn. Philadelphia: Lippincott Williams & Wilkins, 2013:1448.
5 Zink W, Graf BM. Local anesthetic myotoxicity. *Reg Anesth Pain Med* 2004;29:333–40.

Interscalene brachial plexus blockade does not guarantee complete C8-T1 root block

Gustavo Pereira, Fabio Athayde and António Paulo Martins da Encarnação

Acupunct Med 2016 34: 156-157 originally published online November 18, 2015
doi: 10.1136/acupmed-2015-010996

Notes