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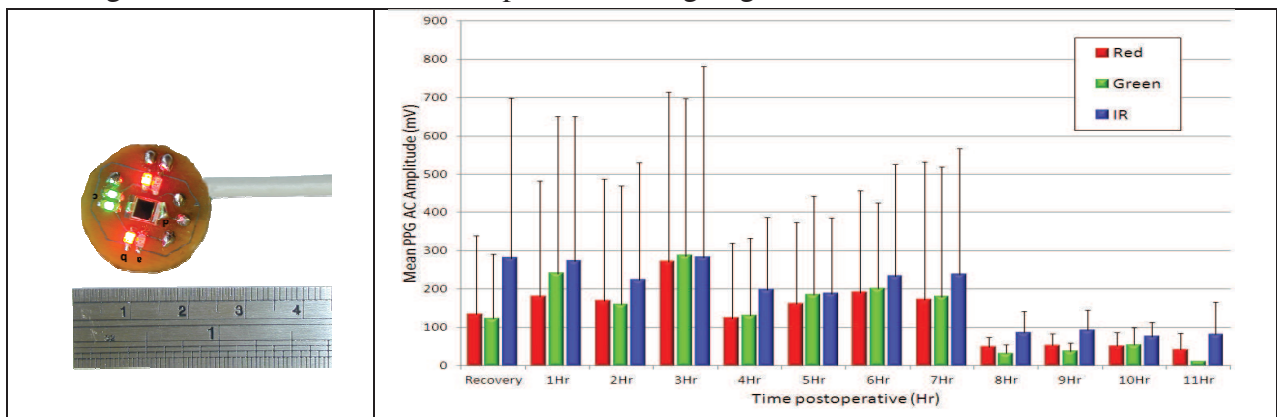
**Title: Pilot investigation of DIEP free flap perfusion utilising a multi-wavelength non-invasive optical sensor.** T Zaman\*<sup>1</sup> SK Pal<sup>2</sup> and PA Kyriacou<sup>1</sup>

<sup>1</sup> City University, London; <sup>2</sup> Broomfield Hospital, Chelmsford, Essex

Deep Inferior Epigastric Perforator (DIEP) free flaps are widely used as a reconstruction option following mastectomy for breast cancer <sup>1</sup>. During such cases partial tissue necrosis can occur due to the insufficient blood supply to the transplanted tissue site. Therefore monitoring of flap perfusion and early detection of flap failure is a prerequisite to flap survival <sup>2</sup>. There is a need to develop a non-invasive, easy to use, reproducible and inexpensive monitoring device to assess flap perfusion postoperatively. A multiwavelength photoplethysmographic (PPG) sensor and battery powered processing system have been developed to investigate PPG signals and estimate free flap blood oxygen saturation continuously and non invasively pre, intra and postoperatively <sup>3</sup>.

The developed reflectance PPG sensor consisted of two infrared, two red and two green ceramic chip surface mount LEDs and a surface mount photodiode, as shown in To evaluate the functionality of the PPG processing system, preliminary clinical investigations were carried out in ten patients undergoing elective breast reconstruction with DIEP Flap. Local research ethics committee approval and patient consent was acquired prior to the study. The sensor was secured onto the exposed skin of the flap using surgical tape. Free flap PPG signals acquisition was conducted in the postoperative period at 15 minute intervals in the first two hours, every 30 minutes for the following four hours and hourly for the next 12 hours. , with a diameter of 20mm in order to be accommodated on the exposed part of the DIEP flaps.

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Figure 1: Reflectance photoplethysmographic flap sensor

Figure 2: The mean amplitude of ac PPGs

An initial observation shows that the amplitude of the PPGs recorded in the postoperative period experience a reduction in amplitude after approximately 7 hours (Figure 2). Such observations need further investigation in order to identify the underlying reasons which cause such change. The utilisation of the green wavelength, with its strong absorption in melanin and haemoglobin and its short depth of penetration, may also provide some blood volumetric and flow activity of the free flap microcirculation<sup>4-5</sup>.

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### **Argon combined with hypothermia protects against hypoxia-ischaemia induced brain damage in rat neonates**

Hailin Zhao\*, Tracy Yushi Cui\*, Daqing Ma

Anaesthetics, Pain Medicine and Intensive Care, Department of Surgery and Cancer, Imperial College London, Chelsea and Westminster Hospital, London

Hypoxic-ischaemic encephalopathy in newborns is a major cause of mortality and disability. Currently, there is no effective treatment apart from therapeutic hypothermia, which provides moderate neuroprotection (1-3). Argon, an abundant and inexpensive noble gas, has recently been demonstrated to be neuroprotective (4). In this study, the effects of argon in combination with hypothermia on neuronal cell death and neuroinflammation induced by hypoxic-ischaemic insult were investigated in a neonatal asphyxia model in rats.

Seven-day-old rats were subjected to unilateral common carotid artery ligation under 1-2% isoflurane anaesthesia (the total anaesthesia time < 10 min) followed by hypoxic (8% oxygen balanced with nitrogen) insult for 90 minutes in a purpose built chamber equipped with a water bath at 37°C. After 1 hour recovery with dame, they were exposed to 70% argon or nitrogen balanced with oxygen at 35°C, or nitrogen balanced with oxygen at 37°C for 2 hrs. Their brains were removed 24 hrs or 4 weeks after gas exposure for immunofluorescence staining and histological analysis respectively.

Argon in combination with hypothermia decreased the fluorescence intensity of cleaved caspase-3 (an apoptotic marker) staining to  $1.11 \pm 0.06$  arbitrary unit from  $1.72 \pm 0.09$  of the injurious controls ( $p < 0.01$ ) in cortex and to  $1.10 \pm 0.04$  from  $1.72 \pm 0.07$  of controls ( $p < 0.01$ ) in hippocampus. NF- $\kappa$ B, an inflammatory marker, was also reduced in cortex ( $1.16 \pm 0.06$  vs  $1.76 \pm 0.09$  of injury controls,  $p < 0.001$ ) and hippocampus ( $1.04 \pm 0.06$  vs  $1.92 \pm 0.31$  of injury controls,  $p < 0.05$ ) with the treatment. Another neuroinflammatory sign astrogliosis induced by hypoxia-ischaemia in hippocampus was significantly decreased in treated group. Argon combined with hypothermia reduced infarction size by 54% ( $p < 0.001$ ) when compared to that of injurious controls. Hypothermia alone also conferred a protection but to much less extent when compared with the combined treatment.