



**ROBERT GORDON
UNIVERSITY•ABERDEEN**

OpenAIR@RGU

The Open Access Institutional Repository at Robert Gordon University

<http://openair.rgu.ac.uk>

This is an author produced version of a paper published in

Proceedings of the Nutrition Society (ISSN 0029-6651, eISSN 1475-2719)

This version may not include final proof corrections and does not include published layout or pagination.

Citation Details

Citation for the version of the work held in 'OpenAIR@RGU':

BROOM, J., BRACKENRIDGE, I. E., SIMPSON, E., MILLER, J. D. B. and MORRISON, I., 1986. The effects of post-operative metabolic support on lipolytic rates in patients undergoing elective abdominal surgery. Available from *OpenAIR@RGU*. [online]. Available from: <http://openair.rgu.ac.uk>

Citation for the publisher's version:

BROOM, J., BRACKENRIDGE, I. E., SIMPSON, E., MILLER, J. D. B. and MORRISON, I., 1986. The effects of post-operative metabolic support on lipolytic rates in patients undergoing elective abdominal surgery. *Proceedings of the Nutrition Society*, 45 (1), 20A.

Copyright

Items in 'OpenAIR@RGU', Robert Gordon University Open Access Institutional Repository, are protected by copyright and intellectual property law. If you believe that any material held in 'OpenAIR@RGU' infringes copyright, please contact openair-help@rgu.ac.uk with details. The item will be removed from the repository while the claim is investigated.

The effects of postoperative metabolic support on lipolytic rates in patients undergoing elective abdominal surgery. By J. BROOM¹, I. E. BRACKENRIDGE², E. SIMPSON¹, J. D. B. MILLER¹ and I. MORISON³, ¹*Surgical Metabolic Unit, Department of Surgery;* ²*Department of Therapeutics and Clinical Pharmacology* and ³*Department of Pharmacy, Aberdeen Royal Infirmary, Foresterhill, Aberdeen AB9 2ZD*

The metabolic response to trauma is associated with mobilization of energy substrates including fat or fat-derived substrates. This lipolytic response is inhibited by the administration of dextrose-containing solutions (Blackburn *et al.* 1973; Swaminatham *et al.* 1980). Recently, *in vivo* lipolysis rates have been determined by extrapolation from the measurement of glycerol turnover rates *in vivo* using stepwise glycerol infusions (Carpentier *et al.* 1979; Broom *et al.* 1985).

Glycerol turnover rates were determined in two groups of patients (*n*4) undergoing elective abdominal surgery. Group 1 received 2 litres dextrose (25 g/l) – saline (9 g sodium chloride/l) postoperatively, whilst group 2 received the same volume of intravenous fluid but containing 1 litre isotonic amino acids (Perifusin) and no dextrose. Glycerol turnover rates were determined preoperatively and 24 and 72 h postoperation; plasma glucose and insulin concentrations were determined throughout.

In group 1, in all cases except one, the glycerol turnover was increased at 24 h but had fallen below preoperation values by 72 h. In group 2 the 24 h glycerol turnover was again increased but at 72 h had further increased over the preoperation values. The 72 h glycerol turnover rates in groups 1 and 2 were statistically significantly different ($P < 0.05$), each patient being used as his own control. Plasma glucose concentration increased from fasting levels of 5.1 (SD 0.5) and 5.3 (SD 0.2) mmol/l in groups 1 and 2 respectively to 10.6 (SD 2.0) and 6.7 (SD 0.5) 24 h postoperation; at 72 h the levels had fallen in the Perifusin group (group 2) to fasting concentrations (5.4 (SD 0.5)) but remained elevated in the dextrose group (group 1) (7.8 (SD 0.5)). Plasma insulin concentrations were higher when plasma glucose concentrations were increased.

Thus, during a 3 d period of study, there were obvious differences in lipolytic rates between the two groups, with group 2 apparently switching to more of a fat-based fuel economy and lower circulating concentrations of glucose and insulin. This *in vivo* kinetic analysis of fat metabolism substantiates the claims that non-dextrose-containing regimens support endogenous fat breakdown post-operatively.

This work was supported by a grant from Merck Pharmaceuticals.

- Blackburn, G. L., Flatt, J. P., Clowes, G. H. A. & O'Donnell, T. E. (1973). *American Journal of Surgery* **125**, 447–454.
- Broom, J., Scalfi, L., Pearson, D. W. M., Khir, A. S., Miller, J. D. B., Ross, I. S. & James, W. P. T. (1985). *Clinical Nutrition Supplement* (In the Press).
- Carpentier, Y. A., Askanzi, J., Elwyn D. H., Jeevanandam, M., Gump, F. E., Hayman, A. I., Burr, R. & Kinney, J. (1979). *Journal of Trauma* **19**, 649–654.
- Swaminatham, R., Hill, G. H., Bradley, J. A. & Morgan, D. B. (1980). *Postgraduate Medical Journal* **56**, 652.