Effects of primary thoracic blast injury on the response to haemorrhage in the anaesthetised rat

E. Kirkman, M.A. Sawdon, M. Ohnishi, G.J. Cooper* and P. Watkins*

*Department of Biological Sciences, University of Durham, Durham DH1 3LE and *Biomedical Sciences, DERA, Porton Down, Salisbury SP4 0JQ, UK*

Primary blast injury to the thorax results in bradycardia, hypotension and apnoea (Guy *et al.* 1998). The bradycardia and apnoea are due to a vagal reflex (Ohnishi *et al.* 1998). Haemorrhage induces a biphasic chronotropic response: initial tachycardia followed by a vagally mediated bradycardia (Little *et al.* 1989). This study aimed to investigate the interaction between the responses to blast and severe haemorrhage.

Male Wistar rats (231–265 g body weight) were used in three groups of eight rats. Surgical anaesthesia was induced and maintained with isoflourane (3·0–3·5% in O_2–N_2O, $F_{I,O_2} = 0{\cdot}5$). Following surgery anaesthesia was maintained with alphadolone/alphaxalone (19–21 mg kg^{-1} h^{-1} I.V.). Heart period (HP) was measured from the electrocardiogram and mean arterial blood pressure (MBP) via the tail artery. Body temperature was maintained at 38·0 °C. At the end of the study the animals were killed with an overdose of anaesthetic.

Group I were subjected to sham blast while groups II and III received a blast wave focused on the ventral thorax (Guy *et al.* 1998). Ten minutes after sham or real blast groups I and II received a haemorrhage of 40% total estimated blood volume (BV; 6·06 mg kg^{-1}; Little *et al.* 1989) at 2% BV min^{-1}, while group III did not. Sham blast (group I) produced no significant cardiovascular response. Blast (group II) induced a significant bradycardia (HP increasing by 194 ± 10 ms from 145 ± 6 ms; mean ± S.E.M., $P < 0{\cdot}05$, ANOVA) and hypotension (fall in MBP of 53 ± 5 mmHg from 101 ± 4 mmHg). Ten minutes after blast HP had returned to 168 ± 8 ms while MBP was 87 ± 7 mmHg. The response in group III was not significantly different from that in group II. In group I haemorrhage produced the expected biphasic pattern of response with HP initially decreasing by 12 ± 4 ms after the loss of 7% BV while MBP was maintained, followed by a significant increase in HP by a maximum of 53 ± 6 ms above pre-haemorrhage levels and a fall in MBP to 33 ± 2 mmHg after the loss of 33% BV. In group II the tachycardia was absent: HP increased by 37 ± 6 ms above pre-haemorrhage levels after the loss of 7% BV and continued to rise by a maximum of 51 ± 6 ms after the loss of 24% BV while MBP fell throughout haemorrhage. In group III there was no significant change in HP and a gradual rise in MBP between 10 and 30 min after blast.

These results indicate that the response to thoracic blast abolishes the first and augments the second, bradycardic, phase of the response to haemorrhage.

Guy, R.J., Kirkman, E., Watkins, P.E. & Cooper, G.J. (1998) *J. Trauma* **45**, 983–987.

Little, R.A., Marshall, H.W. & Kirkman, E. (1989). *Q. J. Exp. Physiol.* **74**, 825–833.

Ohnishi, M., Kirkman, E. & Watkins, P. (1998). *Br. J. Pharmacol.* **123**, P82.