

## Changes in blood pressure, cardiac output and rate, and the response to lower body negative pressure (LBNP) in trypanosome-infected rabbits

D.B. YATES†  
(introduced by A.T. BIRMINGHAM)

Department of Physiology & Pharmacology, The Medical School, Queens Medical Centre, Clifton Boulevard, Nottingham NG7 2UH

Goodwin (1971) demonstrated that trypanosome infections damage the microcirculation and Boreham (1968) showed increases in blood kinin levels. Measurements of the effects of trypanosome infection on heart rate, blood pressure and cardiac output are now reported.

New Zealand White rabbits were infected with *T. brucei*. On the 12th, 19th and 26th day after infection groups of animals were anaesthetised intravenously with 1% chloralose in 25% urethane (5 ml/kg). Blood pressure was measured via a left brachial arterial cannula and heart rate was derived from this via a Devices instantaneous rate meter. Cardiac output was estimated by thermal dilution, by injection of saline into the right atrium and measurement of the blood temperature change in the aortic arch using a thermistor cannula inserted via the right carotid artery. Total peripheral resistance (T.P.R.) was de-

rived by dividing mean blood pressure (mm Hg) by cardiac output ( $\text{ml kg}^{-1} \text{min}^{-1}$ ).

Table 1 shows that, as the infection progressed, there was an increase in cardiac output accompanied by hypotension, a fall in T.P.R. and bradycardia. Twelve-day infected animals were hypotensive but there was no increase in cardiac output.

To test the effectiveness of the sympathetic compensatory reflexes the technique of lower body negative pressure (L.B.N.P.) was used (Yates & Fentem, 1974). In order to grade the cardiovascular stress, three suction levels were used (–25, –50 and –70 mm Hg) on control and on 12-, 19- and 26-day infected rabbits. The compensatory response of most of the infected rabbits was not significantly different from that of uninfected rabbits. However, blood pressure compensation during exposure to L.B.N.P. was sluggish in 12-day infected animals and virtually non-existent in 26-day infected animals at –70 mm Hg; tachycardia and vasoconstriction were not significantly different from control responses.

### References

- BOREHAM, P.F.L. (1968). Immune reactions and kinin formation in chronic trypanosomiasis. *Br. J. Pharmac.*, **32**, 493–504.
- GOODWIN, L.G. (1971). Pathological effects of *Trypanosoma brucei* on small blood vessels in rabbit ear chambers. *Trans. R. Soc. Trop. Med. Hyg.*, **65**, 82–88.
- YATES, J.M. & FENTEM, P.H. (1975). The effects of lower body negative pressure on the cardiovascular system of the anaesthetised rabbit. *Cardiovascular Research*, **9**, 190–200.

† Present address: Research Department, The Boots Co. Ltd., Nottingham, NG2 3AA.

**Table 1** Table showing the mean blood pressure (BP), cardiac output (CO), heart rate (HR) and total peripheral resistance (TPR) of normal rabbits and of rabbits infected with *T. (T.) brucei* at 12, 19 and 26 days of infection

	BP (mm Hg)	CO ( $\text{ml kg}^{-1} \text{min}^{-1}$ )	HR (bts/min)	TRP (CO/BP) (arbitrary units)
Control ( <i>n</i> = 15)	113.0 ± 2.9	205.4 ± 10.3	321 ± 5.3	0.58 ± 0.04
12-day infected ( <i>n</i> = 4)	81.1 ± 9.8***	188.2 ± 20.3	294.3 ± 17.5	0.39 ± 0.03***
19-day infected ( <i>n</i> = 14)	84.6 ± 3.6***	256.6 ± 14.3**	268.0 ± 10.4	0.34 ± 0.02***
26-day infected ( <i>n</i> = 5)	70.3 ± 8.0***	339.3 ± 34.4***	269.7 ± 13.6***	0.21 ± 0.0***

\*\* *P* < 0.01; \*\*\* *P* < 0.001.

Data were compared using Student's *t*-test.