MgSO<sub>4</sub>, 1.2 mm; NaHCO<sub>3</sub>, 30 mm; NaH<sub>2</sub>PO<sub>4</sub>, 1 mm; glucose, 11.1 mm; heparin 1500 iu/litre) initially passed retrogradely through this cannula to perfuse the coronary circulation. Cannulae were also inserted into the inferior vena cava and common pulmonary artery. The superior venae cavae were ligated. The heart was then excised from the animal and connected to the perfusion apparatus.

The coronary circulation was perfused with physiological saline (composition above) containing NaNO<sub>2</sub> (1  $\mu$ g/ml), gassed with 95% O<sub>2</sub>/5% CO<sub>2</sub>, delivered to the heart at 37°C and at pressure of approximately 110 mm Hg. The perfusion line incorporated a bubble trap and a PALL Ultipore blood transfusion filter (SQ40) proximal to the heart to prevent coronary emboli.

A second perfusion circuit involved delivery of normal saline to the right atrium via the inferior vena caval cannula. This saline was ejected by the right ventricle, through the pulmonary artery against a resistance provided by an adjustable constriction in the arterial cannula. Pulmonary arterial pressure was measured using a blood pressure transducer (Bell & Howell 4-422) and the resistance adjusted to give a mean pressure of 15-20 mm Hg. Physiological elastance and compliance of the pulmonary arterial circuit was mimicked by the connection of a 1 ml air-

filled chamber to a side arm on the arterial cannula.

The preparation thus permits evaluation of rightside cardiac performance. Coronary flow was measured by recording the decrease in weight of the fluid reservoir supplying the aortic cannula. The base to apex surface electrocardiogram was recorded by including one nichrome wire electrode in the ligature on the interior vena cava which secured its cannula in position. A second nichrome wire electrode was sutured to the apex of the left ventricle. The fluid in the coronary perfusion reservoir was connected to earth.

The preparation has been used to examine the effects of high (8 mm) and low (0.5 mm) potassium concentrations in, and removal of calcium from the coronary perfusion fluid. A videotape showing all these experiments was demonstrated.

### References

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## An analysis of the response of the perfused vas deferens of the rat to field stimulation

## A.R. JACKSON & D.R. TOMLINSON

Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Nottingham NG7 2UH

A method for the perfusion of tubular organs using a flow-variable apparatus has been described (Jackson, Short & Tomlinson, 1978). This method permits constrictor responses of tubular organs to be generated with concomitant and measurable reductions in perfusate flow and hence very small changes in transmural pressure. When compared with the conventional method of perfusion at constant flow whilst recording changes in perfusion pressure (Fastier & Smirk, 1947), the flow-sensitive apparatus is advantageous in that very brisk constrictor responses are not damped by the compliance of the organ and apparatus and potentially damaging transmural pressures are not generated.

Vasa deferentia from 200 to 250 g rats were perfused with Krebs' solution, previously gassed with 95%  $O_2/5\%$   $CO_2$ , delivered to the tissue at 37°C from a Mariotte bottle at a pressure head of 80 cm  $H_2O$ . The vas was immersed in Krebs' solution (gassed as above) between parallel platinum wire electrodes at 37°C in an organ bath. The flow of perfusate through the system was measured as described by Jackson, Short & Tomlinson (1978).

The response of the vas to field stimulation for periods of 10 s or longer at frequencies of 1 Hz or above (200 µs pulses at 150 V) was biphasic comprising a very rapid and brief occlusion ('phase 1') followed by a slower sustained constriction ('phase 2'). The latter achieved complete occlusion at frequencies greater than 4 Hz.

Both phases of the response were completely absent in the presence of tetrodotoxin  $(1 \times 10^{-6} \text{ m})$  though increasing the pulse width to 5 ms achieved direct electrical activation of the smooth muscle of the vas.

Phase 1 of the response could be elicited without the slower Phase 2 by very brief pulse trains; indeed, in many vasa, a single pulse generated a brief but complete occlusion. Repeated generation of Phase 1 in isolation (every 5 s for 5 min) produced a marked but incomplete tachyphlaxis. When this was followed immediately by stimulation for 30 s, to generate the full biphasic response, phase 2 was also found to have been substantially reduced in magnitude. Thus stimulus parameters which generate only phase 1 also activate and fatigue the mechanisms responsible for phase 2.

Experiments were performed in which different portions of the vas were perfused and stimulated. Phase 1 of the response was generated by all portions but Phase 2 was absent from the quarter of the vas at the prostatic end.

Phase 1 of the response was more resistant than

phase 2 to guanethidine, but at  $1 \times 10^{-4}$  M both were blocked.

It is therefore suggested that both phases of the response are mediated by noradrenergic nerves. The preparation and results referred to above will be demonstrated.

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# The use of gamma cameras to measure the disposition and fate of iodide in the rabbit

J.G. HARDY<sup>1</sup>, MICHELE LEESON<sup>1</sup> & C.G. WILSON<sup>2</sup> (introduced by T. BENNETT)

<sup>1</sup>Department of Medical Physics and <sup>2</sup>Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Clifton Boulevard, Nottingham NG7 2UH

The use of gamma cameras to follow the distribution of  $\gamma$ -emitting radiopharmaceuticals as a diagnostic aid in patient management has been well documented

(Craddock & MacIntrye, 1977). We have now used the gamma camera to follow the distribution of [131]-sodium iodide, since we wish to use this nuclide as a marker of novel drug formulations.

Seven rabbits received, by intravenous administration, 0.2 ml of sodium iodide solution (100 picomoles) containing 100  $\mu$ Ci of <sup>131</sup>I in isotonic saline. For the ensuing 200 h anterior images of the rabbit were recorded at intervals using a Searle LFOV gamma camera linked to a Varian V76 data processor. Regions of interest were defined over the neck, stomach, urinary bladder and thigh muscle; and the radioactivity within these regions was quantified. The data obtained were corrected for radioactive decay and for background activity. The results are shown in Table 1. Whole body elimination of iodide was

Table 1 Whole body elimination of <sup>131</sup>I-sodium iodide and distribution to bladder, stomach and neck with time. Means ( ± s.e. mean) derived from seven rabbits

% Administered dose remaining with time (h) Region of interest				
Time (h)	Whole body	Neck	Stomach	Bladder
0	$100 \pm 0.00$	$0.00 \pm 0.00$	$0.00 \pm 0.00$	$0.00 \pm 0.00$
2	$102.54 \pm 2.22$	$1.08 \pm 0.29$	$2.48 \pm 0.53$	$1.77 \pm 0.68$
5	$93.35 \pm 4.33$	0.93 ± 0.26	2.52 ± 0.74	7.02 ± 1.77
10	80.93 ± 3.35	1.71 ± 0.51	3.10 ± 0.84	10.16 ± 3.47
15	$60.71 \pm 5.90$	$2.53 \pm 0.60$	$3.14 \pm 0.72$	$4.53 \pm 1.28$
20	$50.21 \pm 7.16$	$2.80 \pm 0.78$	$2.59 \pm 0.81$	$2.42 \pm 0.76$
25	$41.44 \pm 8.67$	$3.04 \pm 0.54$	$2.10 \pm 0.60$	$1.62 \pm 0.83$
30	$34.87 \pm 7.54$	$3.03 \pm 0.52$	$1.82 \pm 0.91$	$3.38 \pm 0.49$
40	$29.15 \pm 6.91$	$3.42 \pm 0.68$	$0.82 \pm 0.34$	$3.28 \pm 1.20$
50	$22.50 \pm 6.92$	$3.71 \pm 0.73$	$0.66 \pm 0.35$	$1.49 \pm 0.64$
60	17.86 $\pm$ 6.46	$4.06 \pm 0.78$	$0.24 \pm 0.14$	1.14 ± 0.76
80	$14.14 \pm 5.66$	$4.09 \pm 0.72$	$0.32 \pm 0.31$	$0.47 \pm 0.35$
100	$12.20 \pm 4.85$	$4.23 \pm 0.88$	$0.10 \pm 0.10$	$0.03 \pm 0.03$
150	$9.21 \pm 3.41$	$3.86 \pm 0.69$	$0.09 \pm 0.09$	$0.05 \pm 0.05$
200	$7.10 \pm 2.40$	$3.59 \pm 0.67$	$0.15 \pm 0.13$	$0.03 \pm 0.02$