Reply from the Author

Dear Sir,

In our original editorial 'Prostacyclin' the effects of prostacyclin infusions on the problems encountered with various extracorporeal circulation conditions such as cardiac bypass, haemodialysis and charcoal haemoperfusion, were briefly discussed. We thank Dr. Weston for drawing the reader's attention in greater detail to his own experience with prostacyclin infusions during haemodialysis and acute renal failure associated with malaria. Unfortunately, in a short review editorial with a limited number of references it is impossible to fully discuss and reference every reported clinical benefit of prostacyclin in the acutely ill patient. We agree entirely that the potential widespread use of prostacyclin to suppress platelet interactions with various artificial surfaces is an important therapeutic advance.

Yours sincerely,
S. Machin

The School of Pathology, The Middlesex Hospital Medical School, Riding House Street, London W1P 7LD, UK

Comment on the Article 'The Influence of Urapidil, a New Antihypertensive Agent, on Cerebral Perfusion Pressure in Dogs With and Without Intracranial Hypertension

by H. Van Aken, Ch. Puchstein, Ch. Anger and P. Lawin

Intensive Care Med (1983) 9:123

Dear Sir,

I very much enjoyed the article "The Influence of Urapidil, a New Anti-hypertensive Agent on Cerebral Perfusion Pressure in Dogs, With and Without Intracranial Hypertension" from Münster. Confronted with a new agent such as Urapidil with such apparently favourable effects on cerebral perfusion pressure, one is immediately interested in the pharmacology of the agent, and indeed we are told initially that it is an alphadrenoceptor blocking drug, and later the nature of the alphareceptor blockade and stimulation are clarified further.

Unfortunately, in the United Kingdom at present there is no available information on this drug. In an international journal, such as Intensive Care Medicine, minor communications difficulties may arise with drug nomenclature. In this particular circumstance, it would have been helpful to have a footnote regarding the availability of this drug in the UK and in other countries, any alternative drug name and perhaps a little more description of closely related drugs. It would be useful to know whether this drug will become available in the UK and the name of the manufacturer. This comment is in no way meant to criticize the excellent paper, merely to facilitate understanding amongst the wide readership of Intensive Care Medicine.

Yours sincerely,
Sheila M. Willatts

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Reply from the Managing Editor

I thank Dr. Willatts for her helpful letter and will bear the comments in mind for future issues of the journal. J. Tinker

Sympathetic Hyperactivity in Tetanus

Sir,

The sympathetic hyperactivity accompanying tetanus remains the main cause of fatality. Kerr [1] described this syndrome as a mixture of alpha and beta hyperactivity and suggested the use of labetalol as the antagonist of choice. This is in agreement with various reports of a good outcome using alpha and beta blockers [2], and fatal complications associated with propranolol [3]. Obviously, the use of propranolol in states of alpha and beta hyperactivity may precipitate cardiovascular failure by reducing cardiac output and further increasing systemic vascular resistance, as could be inferred from the case described by Buchanan (Systolic Blood Pressure (SBP) = 300 mmHg, Diastolic Blood Pressure (DBP) = 220 mmHg, Heart Rate (HR) 120 beats/min).

It is often difficult to classify the sympathetic hyperactivity, because a number of factors may affect the clinical picture: fever, water balance, level of consciousness in a patient ventilated, curarized and exposed to painful and sensorial stimulation. The following case could, however, be classified as one of pure beta hyperactivity successfully treated with a beta selective blocker.

A 29-year-old male heroin addict, presented the typical picture of severe tetanus with respiratory insufficiency and sympathetic hyperactivity. He was treated with mechanical ventilation, acoustic and visual isolation, pancuronium bromide (3 mg hourly), diazepam (100 mg/day), alfaxolone-alfadolone (50 mg hourly) and methadone (10 mg/day) for analgesia and prevention of a withdrawal syndrome. The mean weekly values of certain cardiovascular parameters are shown in Table 1. Comparison with temperature values shows that the decrease in arterial pressure (both systolic and diastolic) registered during the third week was associated with severe hyperpyrexia caused by pulmonary infection.

During the 1st, 2nd, 4th and 5th week, continuous tachycardia, systolic hypertension and profuse sweating associated with diastolic normotension and absence of peripheral vasoconstriction indicated pure beta hyperactivity [4], requiring a beta blocker. Considering the potential hazards of using propranolol, it was administered (1 mg iv) only when the HR was above 140 beats/min and/or SBP was above 180 mmHg. The mean values ± SD of HR (110 ± 6,25), SBP (155 ± 18,8), DBP (91 ± 7,2) and Central Venous Pressure (CVP) (7 ± 3) taken 5 min after the administration of propranolol confirm the known effect of this drug on HR and SBP, and prove its safeness in this case, since SBP never fell below 120 mmHg, HR never below 100 beats/min, and CVP never rose above 12 cm H_2O, DBP showed no significant change.

Table 1. Weekly mean values calculated from twelve daily recordings. HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; T: temperature

Weeks	HR beats/min	SBP mmHg	DBP mmHg	T °C
1	118 ± 19.4	181 ± 14.2	92 ± 7.5	37.8 ± 1.5
2	124 ± 8.3	177 ± 17.9	77 ± 9.5	38.5 ± 2.8
3	128 ± 6.4	150 ± 12.6	76 ± 6.7	39.1 ± 3.3
4	133 ± 13.7	166 ± 7.7	83 ± 6.3	38 ± 1.7
5	129 ± 10.4	166 ± 6.3	90 ± 6.8	37.1 ± 1.2