

formation (though 2 oxime isomers are formed). If oximation is omitted and the Me-TMS derivative is formed, two peaks are seen on GC analysis. The major peak has a retention time (C value=24.3) and mass spectrum identical to 6(9)-oxy-11,15-dihydroxyprosta-7,13-dienoic acid (cyclic ether), as described by Pace-Asciak and Wolfe (1971) and derived from rat stomach homogenates. The second peak (C value=25.7) is probably the open chain form PGF_{1α} during derivitization and gas chromatography and that it is not present in the original extracts. The biosynthetic route from arachidonic acid to 6-oxo-F_{1α} in the uterus is under investigation.

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The potentiation of exogenous noradrenaline by prostaglandins F_{2α} C₂ and D₂ on the canine saphenous vein

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Kadowitz, Sweet & Brody (1971) have shown that prostaglandin F_{2α} (PGF_{2α}) potentiates the response of venous smooth muscle to exogenous noradrenaline (NA). PGD₂ acts similarly to PGF_{2α} on several preparations (Horton & Jones, 1974) whilst PGC₂ has similar actions to PGE₂ (Jones, Kane & Ungar, 1974). Experiments are reported here which compare the potencies of PGF_{2α}, PGD₂ and PGC₂ on a single preparation.

The method used was a modification of that described by Webb-Peploe & Shepherd (1968) and was used to determine the venous response to exogenous NA before and during infusion of the various PGs. Both lateral saphenous veins were cannulated at the ankle and perfused separately, one serving as a control for the other. NA was injected proximal to the pump and once a dose dependent relationship was obtained, the PG infusion was commenced.

PGF_{2α} (1 µg/min) caused a significant potentiation of the response and the calculated potency ratio (R) from the pooled results of three preparations was 6. PGD₂ (1 µg/min) was not as potent (R=3, from six preparations) and PGC₂ (1 µg/min) was the least potent (R=2, from three preparations). Using linear regression analysis, the results during PG infusion (1 µg/min) were significantly different from control

($P < 0.05$). In the two preparations, PGF_{2α} (0.1 µg/min) was found to potentiate the response ($R \approx 2$). However, at 5 µg/min there were considerable baseline changes and no satisfactory tests were performed. At 5 µg/min PGC₂ caused similar baseline changes. Up to 10 µg/min, PGD₂ was apparently no more potent than at 1 µg/min and no baseline shift occurred.

The results with PGF_{2α} are largely in agreement with those of Kadowitz *et al.* (1971) and a similar potency ratio for PGF_{2α} (1 µg/min) has been obtained. We have found PGD₂ to be considerably less potent than PGF_{2α}, whereas it is up to 60 times more potent than PGF_{2α} as a direct vasoconstrictor in the sheep (Jones, 1975). PGC₂ which from our results is only weakly active in potentiating the effects of NA, is in contrast a potent dilator of both resistance and capacitance vessels in the dog (Jones, Kane & Ungar, 1974).

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