

## Some observations upon the determination of capillary filtration coefficient (CFC) in the innervated small intestine of the anaesthetized cat

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Systemic arterial pressure, heart rate, blood flow and capillary filtration coefficient (CFC) were measured in 40-65 g loops of jejunum in the chloralose-anaesthetized cat in a series of 113 experiments, using a plethysmographic technique based upon that of Folkow, Lundgren & Wallentin (1963), modified to preserve autonomic connections as far as possible. CFC is a quantitation of the volume increment caused by the transudation of fluid from the exchange vessels into the perivascular spaces in response to the imposition of controlled increments in venous pressure; it is measured in ml of fluid transuded/(min/mmHg rise in pressure/100 g of tissue). In these experiments, the quantitation was made in terms of the rise in pressure at the veins, and not at the exchange vessels, because of the unproved nature of the assumptions necessary to calculate this value (Folkow *et al.*, 1963; Landis & Pappenheimer, 1963; Richardson, 1973). The determination of CFC depends upon the assumption that the increase in volume resulting from the imposition of a rise in venous pressure is due solely to exudation of fluid, and not to any other factor such as continuing venous dilatation, or arteriolar tone.

A wide variety of drugs was used, including adrenoceptor stimulants and blocking compounds, autacoids and related drugs, and hormones.

In a series of 25 consecutive experiments, control determinations (as the mean of 7-10 CFC determinations over 20-40 min) were analysed in an attempt to reveal any relation between CFC and blood flow or peripheral vascular resistance. CFC, a measure of functional exchange vessel area, determined by precapillary sphincter activity (Pappenheimer & Soto-Rivera, 1948; Mellander, 1960; Folkow *et al.*, 1963) was plotted against blood flow measured in ml/(min/100 g) and vascular resistance, measured in

mmHg/(ml/min/100 g). There was no apparent correlation of CFC with either blood flow or vascular resistance.

In 96 experiments, there was a measurable CFC of 0.015 to 0.030 ml/(min/mmHg/100 g), and low doses of drugs were found to affect the CFC, often by over 50% of the control values, whilst such doses were without marked effect on systemic arterial pressure, heart rate, or blood flow. For example, vasopressin (0.02 U/(kg/min), i.v.) caused a fall in CFC of 75-100% (three experiments), glucagon a rise in CFC of up to 100% (0.25 µg/(kg/min), i.v., five experiments) and pentagastrin a fall in CFC of 40-60% (0.1 µg/(kg/min), i.v., three experiments). In other experiments on the circular muscle fibres of the dog superior mesenteric vein, vasopressin (up to 0.2 U/ml), glucagon (up to 10 µg/ml) and pentagastrin (up to 10 µg/ml) were all without effect.

In 17 out of 113 experiments (15%), under control conditions, the CFC could not be differentiated from zero although blood flow and systemic arterial pressure fell within the range found in the other 96 experiments. This 'zero-CFC' condition could not be reversed by adrenergic sympathetic blockade, or by the administration of doses of drugs which in other experiments caused a rise in CFC of, or in excess of 100% of the control.

These observations point towards the conclusion that capillary filtration coefficient is a cardiovascular quantity independent of blood flow, regional vascular resistance, or indirect effects resulting from direct venous responses.

### References

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