

NON-LINEARITY IN THE CONTROL OF PERIPHERAL BLOOD FLOW DURING COOLING AND HEATING

E. Howard N. Oakley
Institute of Naval Medicine
Alverstoke, Gosport, Hants PO12 2DL, UK

INTRODUCTION

Long-established studies using plethysmographic and similar techniques have shown that the relationship between skin blood flow and local skin temperature is generally simple and approximately linear over much of its course.¹ More recent methods have enabled measurements of skin blood flow to be made more frequently, and this work was undertaken to investigate the relationship in more detail using ramped thermal inputs.

METHOD

Three male volunteers gave their informed consent to take part in this study, which was approved by the local ethical committee. They rested recumbent in a chamber controlled at $30^{\circ}\text{C} \pm 1.0^{\circ}\text{C}$ dry bulb air temperature, both feet positioned inside a perspex box constructed such that the right foot was covered with a thin plastic bag and immersed above the malleoli in mixed water circulated at approximately 10 l/min from a 2 kW heater-cryostat unit. Blood flow was measured in arbitrary units at a rate of 40 Hz with a Moor Instruments dual-channel laser Doppler rheometer (wavelength 810 nm), with one probe attached to the pad of the great toe, the other to the dorsum of the same toe. At the start of each experiment, the right foot chamber was filled with water at 40°C and left until blood flow had stabilised. This was then cooled linearly to 15°C at a rate of 50°C per hour; the temperature was then held at 15°C for 2 min, and finally rewarmed to 40°C at a rate of 33°C per hour. Temperatures were measured using a thermistor immersed adjacent to the great toe. Blood flow measurements were captured digitally to a computer, filtered off-line using an optimised stack filter, and analyzed on a beat-by-beat basis, according to methods described previously.²

RESULTS

Although blood flow declined as water temperature fell and increased as temperature rose, in all subjects and at both sites, it did so in a highly non-linear manner. Figure 1 shows the mean cooling and warming curves for all three subjects. Cooling was accompanied by little change in blood flow until the temperature had fallen below 25°C , when flow declined exponentially with a half temperature (i.e. the temperature reduction predicted to produce a halving of blood flow) of 11°C . Warming resulted in little increase in mean blood flow until a

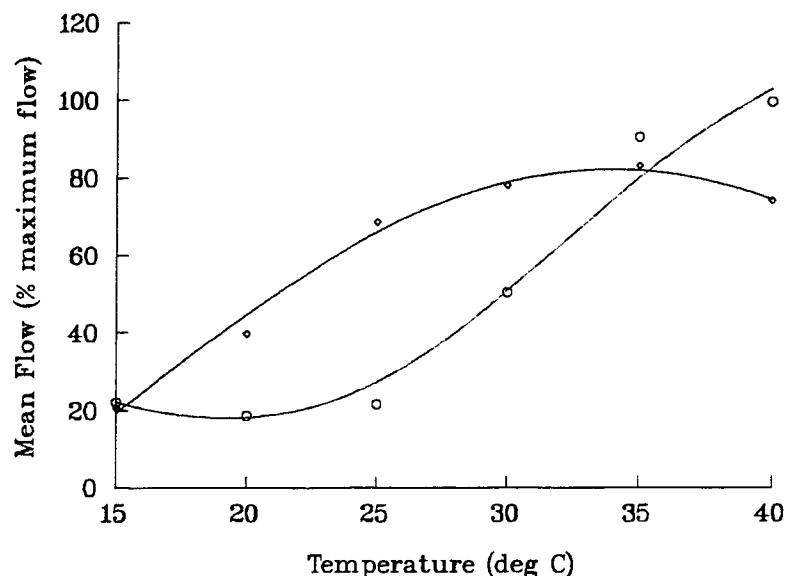


FIGURE 1: Mean data points and weighted least-squares fits of mean blood flow during cooling (◇ symbols and upper line) and warming (○ symbols and lower line) for the three subjects.

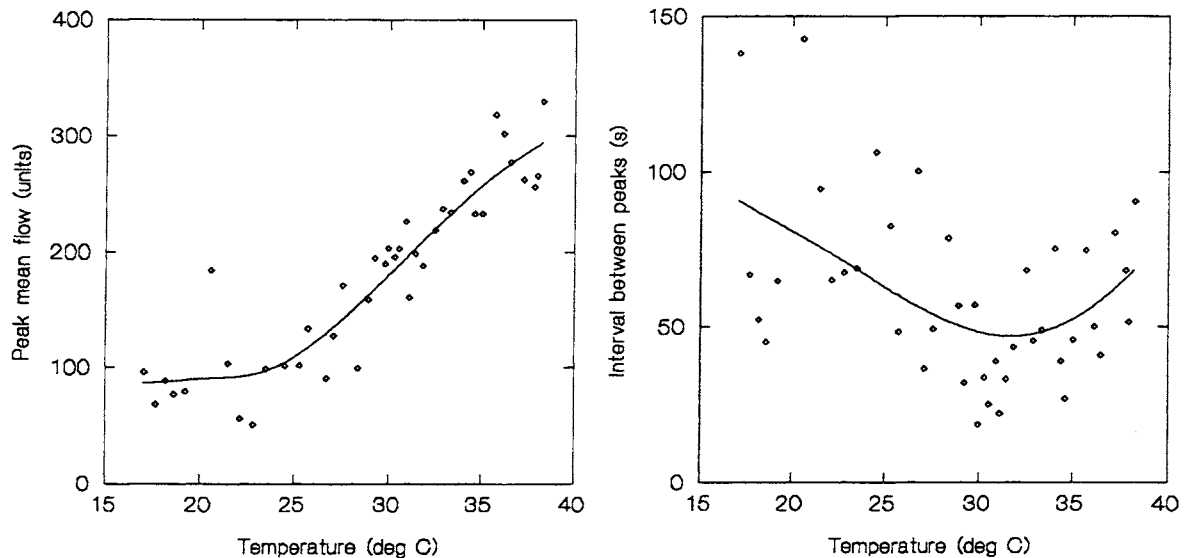


FIGURE 2: Peak amplitude and period of vasomotion during rewarming for one subject, with weighted least-square fitted curves.

temperature of 25°C had been reached, when an exponential relationship of opposite curvature was apparent. The half temperature of this curve was computed to be 9°C. The two curves described a hysteresis that amounted to a difference between heating and cooling flows of up to 100% (at 25°C) of the mean flow. Furthermore, the pattern of flow during almost all of the warming (up to 35°C) showed pronounced periodic vasoconstrictive episodes resembling a slow vasomotion. Figure 2 shows the relationship between the amplitude of peak vasodilatation with temperature, and that between the period of 'vasomotion' with temperature. The peak of the dilation phase of the 'vasomotion' increased, and the duration of the constriction phase reduced, with increasing temperature, until steady flow was re-established at 35°C. On return to 40°C, blood flow was higher than that originally recorded at 40°C.

CONCLUSIONS

Although the difference in rate of cooling and warming, and differences between water temperatures and those of deeper tissues, may have contributed some of the non-linearity observed, it is unlikely that they can account for more than a small proportion at most. For instance, this would require the difference between surface-subcutaneous tissue temperature gradients during cooling and warming to amount to 10°C for a given water temperature. It is also of significance that the half-temperature for the more rapid process of cooling was computed to be longer than that for the slower process of warming, although interestingly both are close to corresponding to the theoretical Q_{10} of 2.0. The phenomenon of periodic vasoconstriction would also be hard to explain in this way, given that the lowest water temperature was in excess of accepted thresholds for cold-induced vasodilatation,³ and the fact that this pattern of flow persisted until the temperature had reached 35°C, some 27 min after the temperature had risen above 20°C. Time-averaging techniques of measuring blood flow such as plethysmography obscure dynamic changes in skin blood flow that were apparent particularly during rewarming. These results not only show that the control of peripheral blood flow with respect to local temperature is more complex than that conventionally portrayed,^{1,3} but also suggest that models that use or compute such blood flow may need to take account of this more complex relationship.

REFERENCES

1. Allwood M.J. and Burry H.S. 1954, The effect of local temperature on blood flow in the human foot, *J. Physiol.* 124, 345-357.
2. Oakley E.H.N. 1993, Techniques for filtering noise from laser Doppler rheometer data, Proceedings of First Asian Congress for Microcirculation, Osaka, Japan, September 1993.
3. Keatinge W.R. and Harman M.C. 1980, *Local mechanisms controlling blood vessels* (Academic Press, London).