Vasomotor innervation of the skin of the hand: a contribution to the study of human anatomy

MARIO CAMPERO, RENATO J. VERDUGO AND JOSÉ L. OCHOA

Departments of Neurology and Neurosurgery, Good Samaritan Hospital and Medical Center, Oregon Health Sciences University, Portland, USA

ABSTRACT

The sympathetic vasomotor innervation of the skin of the human hand was studied in 47 subjects who underwent local anaesthetic block of ulnar, median or radial nerves at elbow or wrist levels. Areas of cutaneous anaesthesia were compared with cutaneous territories of paralytic vasodilatation delineated by infrared telethermography. It was found that (1) during ulnar nerve block the area of vasodilatation matched the area of anaesthesia in all 15 cases; (2) median nerve block induced paralytic vasodilatation which, in 14 of 15 cases, matched the area of cutaneous anaesthesia in median territory, but also extended to the unanaesthetised lateral part of the dorsum of the hand; (3) no vasodilatation developed during radial nerve block in 17 of 18 cases, whereas areas of sympathetic sudomotor paralysis matched the area of radial sensory loss in all 5 subjects in whom sweating function was studied. It is concluded that (1) the ulnar nerve supplies vasomotor fibres to its cutaneous sensory territory, no less and no more; (2) the median nerve normally provides supplementary vasomotor innervation to the skin of the radial aspect of the dorsum of the hand; (3) the radial nerve supplies sudomotor innervation for the lateral aspect of the dorsum of the hand, but (4) does not normally contribute vasomotor sympathetic fibres to the skin of the hand.

INTRODUCTION

The sensory, motor and sympathetic innervation territories of human peripheral nerves in limbs are well resolved in textbooks of anatomy and neurology. In terms of cutaneous innervation it is commonly accepted that sensory and sympathetic fibres course together within nerve trunks, ultimately to cover a common province of skin. Legitimate, albeit indirect, evidence in support of this concept derives largely from clinical observations on coincidental territories of sensory loss and sweating loss following nerve injury (Head & Sherren, 1905; Trotter & Davies, 1909; Pollock & Davis, 1933; Highet, 1942) and from the results of local nerve blocks in man disclosing correspondence between areas of cutaneous sensory loss and areas of sweating loss and vasodilatation, reflected by increased temperature (Highet, 1942).

In analysing critically the evidence for the notion that sensory and sympathetic fibres course together in

cutaneous nerves and end in a common territory in the skin, some ambiguities emerge. Indeed, in terms of sudomotor distribution, past observations on the human hand have often been inconsistent. Guttmann (1940) described substantial variability and overlap in the areas of sweating loss after complete lesions of the median, ulnar and radial nerves. In turn, observations on vasomotor innervation have relied on scanty data and relatively coarse criteria (Trotter & Davies, 1909; Highet, 1942).

The present work attempted to clarify anatomical uncertainties concerning (1) the occurrence of vasomotor nerve fibres within main nerve trunks supplying the skin of the human hand, (2) their absolute territories of terminal distribution and (3) their distribution relative to somatic sensory nerve fibres. This aim was approached with the advantage of thermography, a noninvasive method that sensitively detects and precisely delineates areas of vasomotor change in the skin. The method was applied to human
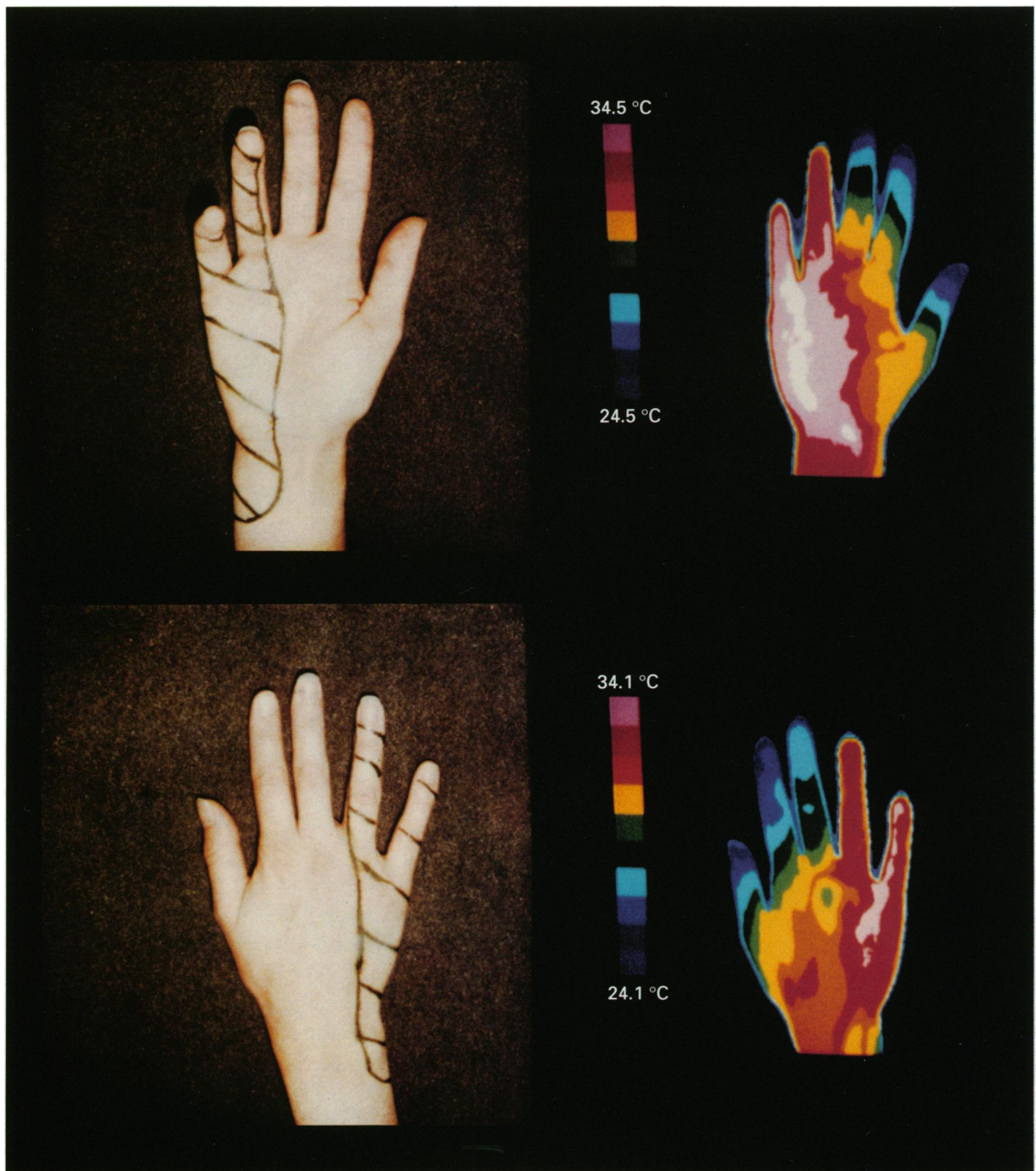
Fig. 1. Local anaesthetic block of right ulnar nerve at wrist level. *Left*: areas of cutaneous anaesthesia during ulnar nerve block in palmar (top) and dorsal aspects (bottom). *Right*: matching areas of cutaneous vasodilatation in palmar (top) and dorsal aspects (bottom) of the hand. Note ulnar claw from motor paralysis. Colour-coded temperature scales on the left of the thermograms for all figures.

subjects who consented to local nerve block aimed at inducing concurrent anaesthesia and paralytic vasodilatation with resulting warming of provinces of skin. Such research strategy could regularly be implemented without difficulty or complication and yielded clear and novel anatomical information.

MATERIALS AND METHODS

Forty-seven subjects, 31 women and 16 men, aged between 27 and 73 years (mean 40.1 years) gave informed consent to local anaesthetic nerve block aimed at the ulnar, median or radial nerves. The procedure was performed in a draught-free room at a constant ambient temperature between 23° and 25 °C. At baseline, telethermograms of the palm and dorsum

Table. *Mean temperature* ($\pm 2s.D.$) *of the skin innervated by the ulnar, radial and median nerves, before and during local anaesthetic nerve block, measured by infrared telethermography*

	Nerve				
	Ulnar (n = 15)		Radial N. (n = 17)	Median N. (n = 15)	
	Palm	Dorsum	Dorsum	Palm	Dorsum
Baseline	30.6±3.1	29.8±2.5	30.5±2.3	32.1±2.4	31.2±2.0
During block	35.2±1.3	34.8±1.6	29.9±2.1	35.6±1.2	34.6±1.2

of both hands were obtained through a Flexitherm Mark V (Westbury, NY, USA). This technique detects infrared thermal emission from the skin with a sensitivity of detection for thermal differences of 0.25 °C. The analogue is then computerised and displayed in a colour code within a temperature range (Pulst & Haller, 1981; Triplett & Ochoa, 1990).

After obtaining a baseline colour thermogram record, 1 of the 3 nerves supplying the hand was blocked locally at wrist or elbow level by injection of 6–10 ml of 2% lidocaine, following a conventional approach as described by Bridenbaugh (1988). At 30 min after injection, development of paralytic vasodilatation was evaluated through further thermography of the palm and dorsum of the hands. Colour photographs of the thermograms were again filed.

Next, the area of cutaneous anaesthesia to pinprick induced by lidocaine was carefully screened through standard neurological testing, and then mapped and photographed. In some subjects the degree of block achieved was also evaluated through measurement of sensory nerve action potentials and through a quantitative somatosensory thermotest, performed both at baseline and after lidocaine injection. The photographed profiles of the areas of lidocaine-induced anaesthesia and of thermograms of the areas of hyperthermia caused by paralytic vasodilatation were then compared visually.

Five of the 47 experimental subjects underwent testing of cutaneous areas of sweating in the hand through standard application of alizarin powder (Guttmann, 1940), at baseline and after anaesthetic block of the superficial radial nerve at the wrist.

RESULTS

Ulnar nerve

Fifteen subjects underwent block of the ulnar nerve: 7 at wrist and 8 at elbow level, 9 in the right arm and 6 in the left. The area of lidocaine-induced anaesthesia was always within the normal anatomical distribution described for the ulnar nerve. It included the hypothenar eminence, the ulnar side of the dorsum of the hand and the entire little finger. The ring finger was split in 14 cases and was totally included in only 1 case.

In all subjects the thermograms obtained during block showed significant hyperthermia in an area which always included the palm and dorsum of the hand and matched the area of pinprick anaesthesia (Fig. 1). In 5 cases, however, splitting of the ring finger, found by mapping the area of pinprick hypoaesthesia, was not shown by thermography, which instead recorded warming of the entire ring finger. Although both the palmar and dorsal aspects of the ulnar cutaneous territory became vasodilated, temperature of the palmar aspect became elevated 0.3–1.3 °C higher (mean 0.4 °C) than the temperature of the ulnar dorsum of the hand (Table). In 2 subjects whose ulnar nerve was blocked at elbow level there was no warming up of the ulnar dorsum of the hand although the dorsum of the little finger showed significant increase in temperature in both subjects.

Radial nerve

Nine subjects underwent anaesthetic block of the superficial radial nerve at the wrist and 8 underwent block of the radial nerve trunk at elbow level, 10 on the right and 7 in the left arm. In all cases the resulting area of pinprick anaesthesia again followed the anatomical textbook distribution for the normal radial nerve. This area included the lateral part of the dorsum of the hand and, to a variable extent, the dorsum of the proximal phalanx of the thumb and index finger. There was no difference in the area of lidocaine-induced anaesthesia when the block was performed at the wrist as opposed to the elbow. Not surprisingly, block of the radial nerve at elbow level resulted in significant weakness of extensor muscles of the fingers and wrist.
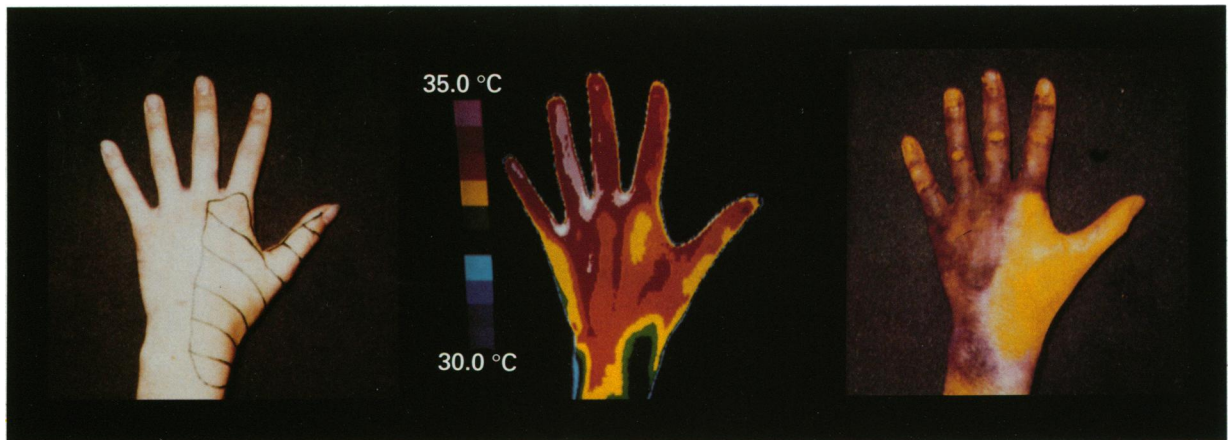
Fig. 2. Local anaesthetic block of left superficial radial nerve at wrist level. *Left*: area of cutaneous anaesthesia in radial nerve territory. *Centre*: absence of discernible changes in thermal emission profile of the radial dorsum of the hand. *Right*: selective absence of sweating in radial nerve territory, as revealed by an alizarin test.

Although, judging by the area and depth of anaesthesia induced by lidocaine, a satisfactory block of the superficial radial or the parent radial nerve trunk was achieved in all 17 cases, hyperthermia of the area of anaesthesia was observed in only 1 case, while in 16 there was no significant change in thermal emission profile (Fig. 2). Furthermore, in the 1 case in whom hyperthermia was observed, the increase in temperature was mild, not exceeding 1 °C, and involved only a small fraction of the area of hypoaesthesia. In 3 cases the substantial degree of block achieved was also documented by electrophysiological study, which showed loss of the sensory nerve action potential in the distal superficial radial nerve. In those 3 cases, a quantitative somatosensory thermotest revealed loss of thermal-specific and thermal-pain sensations in the territory of lidocaine-induced anaesthesia for stimuli between 0° and 50 °C.

These 3 and 2 additional subjects underwent evaluation of sweat function, and showed unimpaired even sweating of the palmar and dorsal aspects of the hand at baseline. In contrast, during anaesthetic block of the superficial radial nerve, absence of sweating was recorded by an alizarin test in the area of lidocaine-induced hypoaesthesia on the radial aspect of the dorsum of the hand in all 5 cases (Fig. 2).

Median nerve

Fifteen subjects underwent block of the median nerve, 8 at wrist and 7 at elbow level. In all, the resulting area of pinprick anaesthesia involved the thenar eminence and the palmar aspect of the thumb and the index and middle fingers. Pinprick anaesthesia also developed to a variable extent in the ring finger and dorsum of the thumb, index and middle fingers. In none of the

subjects did the area of anaesthesia include the radial aspect of the dorsum of the hand or the dorsum of the proximal phalanx of the 2nd finger or thumb. There was no difference in the area of anaesthesia when comparing effects of block at wrist and elbow.

Postlidocaine thermography showed significant warming of the area of lidocaine-induced anaesthesia in all 15 subjects. Unexpectedly, in addition to vasodilatation in the area of anaesthesia, significant warming of the lateral part of the dorsum of the hand, an area obviously not anaesthetised, was observed in 14 of the 15 cases (Fig. 3). While the radial part of the dorsum warmed significantly in comparison with the medial part, it did so by 1.2–5 °C (mean 3.52 ± 1.6 °C) less than the median cutaneous territory.

DISCUSSION

Relationships between cutaneous anaesthesia and vasodilatation during nerve block

As given, local anaesthetic block abolished cutaneous sensation in characteristic domains described in classical textbooks for the ulnar, superficial radial and median nerves. Unsurprising variants encountered consisted of anaesthesia of the entire ring finger during ulnar nerve block in 1 case and variable extents of anaesthesia of the dorsum of the thumb, index and middle fingers during median nerve block in most cases.

Although these somatic anaesthetic nerve blocks abolished sensation in all subjects, cutaneous hyperthermia was not a universal development, but when it occurred it did so systematically. Indeed, for ulnar nerve block, cutaneous anaesthesia matched the area of vasodilatation with trivial exceptions, whereas for
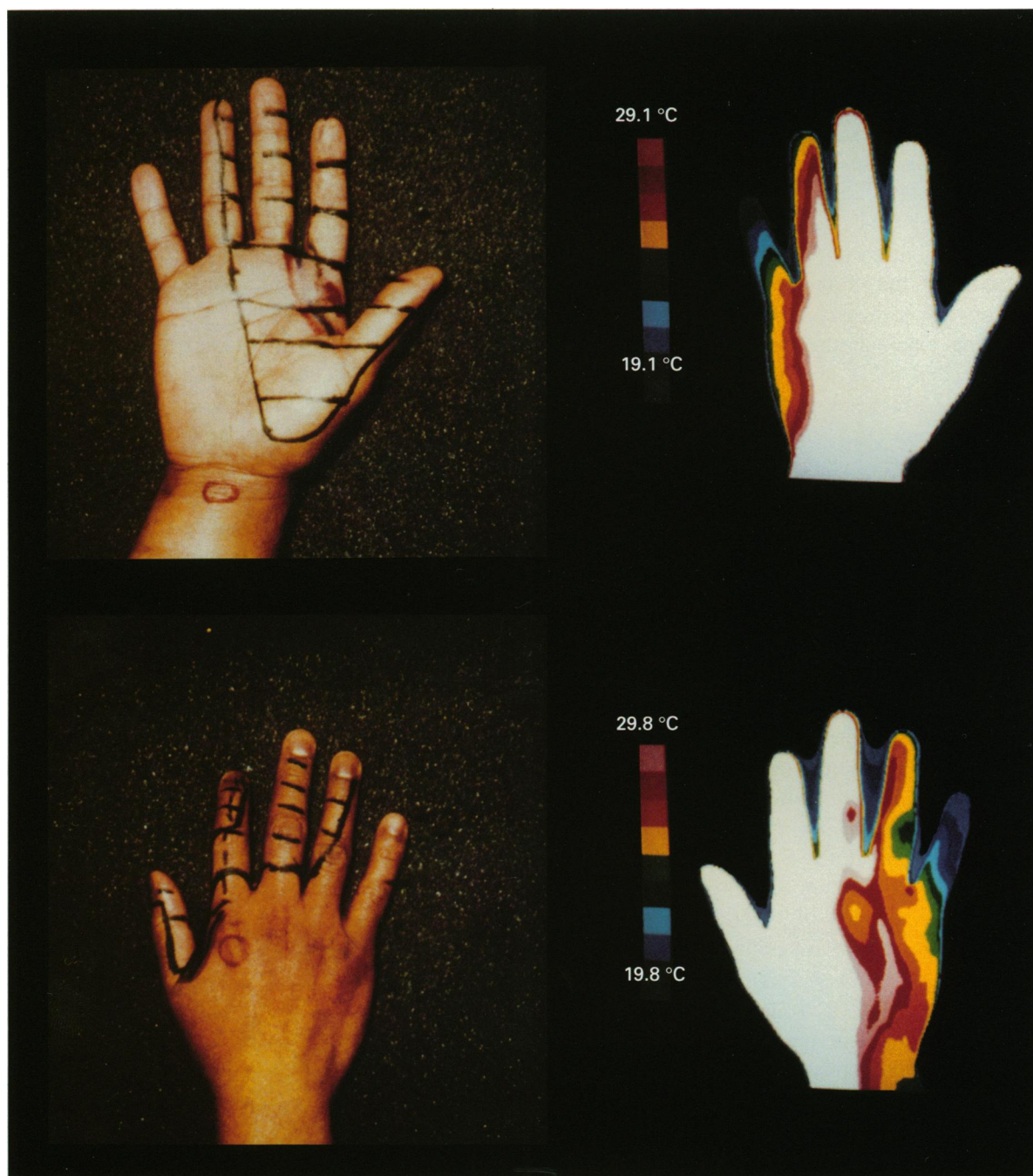
Fig. 3. Local anaesthetic block of left median nerve at wrist level. *Left*: area of cutaneous anaesthesia follows the normal anatomical territory of the median nerve in palmar (top) and dorsal (bottom) aspects. *Right*: paralytic vasodilatation induced by nerve block matches the area of sensory deficit (top) but also includes the radial nerve territory on the dorsum of the hand (bottom).

median nerve block the area of hyperthermia extended onto the dorsum of the hand. For radial nerve block the textbook area of cutaneous anaesthesia was unaccompanied by hyperthermia except in 1 out of 17 subjects.

The absence of cutaneous vasomotor change following radial nerve block has not been appreciated previously. Trotter & Davis (1909), in their study on innervation of human skin, described transient in-

crease in temperature, changes in colour and 'variations in the reactions to stimulation of the circulatory conditions of the part' following experimental division of their own cutaneous nerves. The distribution of these vasomotor changes was in correspondence 'with the outline of the hypoaesthesias rather than with that of the anaesthesias'. They described an exception only for section of their great auricular nerve, after which the distribution of paralytic vasodilatation was limited

to an area smaller than the area of anaesthesia. If Trotter & Davis had sacrificed their superficial radial nerves they would undoubtedly have found the presently described exception.

Woollard & Phillips (1923–3) reported changes in colour and temperature of the skin during anaesthetic block of different nerves in their own upper limbs. They concluded that vasomotor fibres follow the distribution of sensory fibres for the ulnar and median nerves, although they did not describe the presence or absence of temperature change in the dorsum of the hand during block of the median nerve. They did block the radial nerve in 1 experiment after which 'Anaesthesia corresponding to their distribution was obtained and within this area the skin became smooth and slightly flushed', but there was no detectable rise in temperature. Nevertheless, the authors did not discuss whether or not this finding reflected dichotomy of vasomotor and sensory innervation for the radial dorsum of the hand.

Significance of lidocaine-induced vasodilatation following local nerve block

In measuring skin temperature, largely a function of cutaneous circulation under autonomic nervous control (Cannon & Rosenblueth, 1949), infrared tele-thermography provides an analogue of vasomotor sympathetic function (Pulst & Haller, 1981; Ochoa & Yarnitsky, 1990; Lindblom & Ochoa, 1992). In the absence of other causes of vasodilatation, such as neurogenic antidromic vasodilatation (Lembeck, 1983; Cline et al. 1989), it may be assumed that the hyperthermia that develops during local block of nerve impulse conduction is a result of vasodilatation due to loss of vasoconstrictor, C fibre-mediated, neural sympathetic outflow destined for the skin.

For the radial nerve it might be argued that absence of vasodilatation in response to effective local anaesthesia to pinprick may have been due to selective block of A fibres without functional impairment of vasomotor sympathetic C fibres.

Documentation that C sympathetic fibres in the superficial radial nerve were indeed blocked is provided by the observation that, in spite of the absence of paralytic vasodilatation, temporary block of sweating clearly occurred in the area of lidocaine-induced anaesthesia on the radial aspect of the dorsum of the hand. Additionally, in all cases in which a quantitative somatosensory thermotest was performed, there was significant increase in threshold for warm sensation relative to baseline. It is known that warm sensation is mediated at peripheral nerve level

by unmyelinated C fibres (Kenshalo, 1976; Hallin et al. 1981; Yarnitsky & Ochoa, 1990, 1993; Verdugo & Ochoa, 1992). It is therefore clear that the lack of development of hyperthermia in the radial aspect of the dorsum of the hand after block of the median nerve, means that this part of the dorsum of the hand is a supplementary and exclusive median nerve target of vasoconstrictor innervation.

The radial nerve enigma

Although it seems clear that the vasomotor innervation of the radial dorsum of the hand is provided by the median nerve, the radial nerve does contain sympathetic efferent fibres to the skin. Block of the radial nerve resulted in unquestionable sweating loss in the radial dorsum of the hand in our volunteers. Earlier, Guttmann (1940) had described variable areas of anhidrosis on the dorsum of the hand after complete lesions of the radial nerve. In turn, many of Wallin's (1981) classic experiments on microneurographic recording and activation of sympathetic efferent fibres were performed on superficial radial nerves and did document sudomotor effector responses, as tested through skin resistance. Remarkably, the absence of vasomotor fibres in the human superficial radial nerve was not described in those classic studies because circumstantially they were targetted on sudomotor function.

Bini et al. (1980a, b) did search differentially for vasomotor and sudomotor outflow in cutaneous nerves, using standard microneurographic criteria (Vallbo et al. 1979; Delius et al. 1972; Hagbarth et al. 1972). They did not find vasomotor activity within the superficial radial or the posterior antebrachial nerve in human volunteers and concluded that 'in the hairy skin on the dorsal side of the forearm and hand reflex thermoregulation is to a large extent executed via sudomotor fibres.' Thus Bini and coworkers did not encounter information to alert them to the fact that the skin of that body segment does have vasoconstrictor control, but that it is supplied through the median nerve.

Role of sympathetic fibres in vasodilatation of the skin

Parasympathetic vasodilator fibres have been demonstrated in human facial nerves and are responsible for the flushing of the face through a trigeminal-parasympathetic reflex (Drummond, 1992; Drummond & Lance, 1992). However, sympathetic vasodilator systems have not yet been definitively es-

tablished. Blumberg & Wallin (1987) reported increase in bloodflow of the skin during and following painful intraneural microstimulation of the superficial peroneal nerve in normal volunteers. The authors proposed that the increase in capillary flow was a reflex phenomenon mediated through sympathetic vasodilator fibres. However, direct evidence for the existence of this type of sympathetic nerve fibres in humans is not yet available. In the present study, somatic anaesthetic block of the median and ulnar nerve caused significant hyperthermia of the pertinent skin territory. Thus, if sympathetic vasodilator fibres indeed exist, their effect was overridden by vasoconstriction paralysis. With the radial nerve block, the thermal emission profile either did not change or became mildly hypothermic (0.6 °C). Whether this minor vasoconstrictor effect was due to block of vasodilator fibres contained in the radial nerve or was a somatosympathetic reflex response exerted through vasoconstrictor fibres in the median nerve could not be determined.

Level of median-to-radial sympathetic vasoconstrictor cross-over

The fact that hyperthermia develops in the dorsum of the hand after block of the median nerve at wrist level indicates that the median vasomotor fibres destined for the radial dorsum of the hand exit from the nerve distal to the wrist. The fact that the supplementary vasoconstrictor territory of the median nerve covers radial nerve territory implies that it is the latter nerve that distributes those fibres ultimately.

Early studies had remarked that the vasomotor nerves to the arteries supplying the skin, corresponded 'roughly with the distribution of nerves to muscles and skin' (Kramer & Todd 1914). They stated 'the portion of the dorsal carpal arch associated with the little and ring fingers was supplied by the dorsal cutaneous branch of the ulnar nerve but the major portion of the arch...received its supply from the superficial ramus of the radial nerve.' Kramer & Todd also stated that 'the more distal arteries are supplied by sympathetic fibres which have travelled to their distribution along special nerve-trunks and not along main vessels' but they did not describe the innervation of these distal arteries. In an anatomical study in cadavers, Coates (1931–32) described 'The radial artery was found in 3 cases to receive a branch from the median nerve in the region of the wrist. No branches from the radial nerve were found to supply the artery in any part of the forearm.' Later, Pick (1958) stated that 'the distal part of the radial artery

was supplied by one filament from the superficial branch of the radial and, unusually enough, by eight individual twigs from the lateral antebrachial cutaneous' which Pick proposed might also originate from the radial nerve. Nevertheless, Pick did not describe the innervation of more distal branches even in his more recent treatise on the autonomic nervous system (1970).

Thus, as supported by the observation of paralytic vasodilatation of the radial cutaneous territory of the hand induced by local anaesthetic median nerve block, and by the absence of vasomotor effects on blocking the radial nerve, previous anatomical evidence is in harmony with the concept that the sympathetic vasodilator cutaneous innervation of the radial dorsum of the hand derives from the median nerve below the wrist.

Clinical implications

The present results entail several points of clinical interest. (1) The commonplace assumption that cutaneous sensory and vasomotor distribution of peripheral nerves as a rule coincide is in error, at least for the human hand. (2) Vasoconstriction or vasodilatation in the dorsum of the hand following injury to the median nerve need not mean that an additional nerve – the radial – has been injured. (3) Radial nerve blocks that do not vasodilate the dorsum of the hand need not be failed blocks. (4) Thermography is of no use in evaluating sympathetic phenomena following injury to the radial nerve. However, the method is useful in evaluating antidromic sensory vasomotor phenomena following injury to any kind of somatic nerve. (5) Evaluation of median nerve function must include testing of the dorsum of the hand.

REFERENCES

BINI G, HAGBARTH KE, HYNNINEN P WALLIN BG (1980 a) Thermoregulatory and rhythm-generating mechanisms governing the sudomotor and vasoconstrictive outflow in human cutaneous nerves. *Journal of Physiology* **306**, 537–552.

BINI G, HAGBARTH KE, HYNNINEN P, WALLIN BG (1980 b) Regional similarities in thermoregulatory vaso- and sudomotor tone. *Journal of Physiology* **306**, 553–565.

BLUMBERG H, WALLIN BG (1987) Direct evidence of neurally mediated vasodilatation in hairy skin of the human foot. *Journal of Physiology* **382**, 105–121.

BRIDENBAUGH LD (1988) The upper extremity: somatic blockade. In *Neural Blockade in Clinical Anesthesia and Management of*

Pain (ed. M. J. Cousins & P. O. Bridenbaugh) 2nd edn, pp. 387–416. Philadelphia: J. B. Lippincott.

CANNON WB, ROSENBLUETH A (1949) *The Supersensitivity of Denervated Structures: a Law of Denervation,* New York: Macmillan.

CLINE MA, OCHOA JL, TOREBJÖRK HE (1989) Chronic hyperalgesia and skin warming caused by sensitized C nociceptors. *Brain* **112**, 621–647.

COATES AE (1931–32) Observations of the distribution of the arterial branches of the peripheral nerves. *Journal of Anatomy* **66**, 499–507.

DELIUS W, HAGBARTH K-E, HONGELL A, WALLIN BG (19872) Manoevers affecting sympathetic outflow in human skin nerves. *Acta Physiologica Scandinavica* **84**, 177–186.

DRUMMOND, PD (1992) The mechanism of facial sweating and cutaneous vascular responses to painful stimulation of the eye. *Brain* **115**, 1417–1428.

DRUMMOND PD, LANCE JW (1992) Pathological sweating and flushing accompanying the trigeminal lacrimal reflex in patients with cluster headaches and in patients with a confirmed site of cervical sympathetic deficit. *Brain* **115**, 1429–1445.

GUTTMANN L (1940) Topographic studies of disturbances of sweat secretion after complete lesions of peripheral nerves. *Journal of Neurology and Psychiatry* **3**, 197–210.

HAGBARTH K-E, HALLIN RG, HONGELL A, TOREBJÖRK HE, WALLIN BG (1972) General characteristics of sympathetic activity in human skin nerves. *Acta Physiologica Scandinavica* **84**, 164–176.

HEAD H, SHERREN J (1905) The consequences of injury to the peripheral nerves in man. *Brain* **28**, 1–224.

HALLIN RG, TOREBJÖRK HE, WIESENFELD Z (1981) Nociceptors and warm receptors innervated by C fibers in human hand. *Journal of Neurology, Neurosurgery and Psychiatry* **44**, 313–319.

HIGHET B (1942) Procaine nerve block in the investigation of peripheral nerve injuries. *Journal of Neurology, Neurosurgery of Psychiatry* **5**, 101–116.

KENSHALO DR (1976) Correlations of temperature sensitivity in man and monkey, a first approximation. In *Sensory Functions of the Skin in Primates, with Special Reference to Man* (ed. Y. Zotterman), Wenner-Gren International Symposium, vol. 27, pp. 305–330. Oxford: Pergamon.

KRAMER JG, TODD TW (1914) The distribution of nerves to the arteries of the arm. *Anatomical Record* **8**, 243–255.

LINDBLOM U, OCHOA JL (1992) Somatosensory function and dysfunction. In *Disease of the Nervous System,* vol. II, 2nd edn (ed. A. K. Asbury, G. M. McKhann & W. I. McDonald), pp. 213–228. Philadelphia: Ardmore Medical.

OCHOA J. YARNITSKY D (1990) Triple cold ('CCC') painful syndrome. *Pain* (Supplement) **5**, S278.

OCHOA J, YARNITSKY D (1993) The triple cold ('CCC') syndrome: cold hyperalgesia, cold hypoesthesia and cold skin in peripheral nerve disease. *Brain,* in press.

PICK J (1958) The innervation of the arteries in the upper limb of man. *Anatomical Record* **130**, 103–123.

PICK J (1970) The sympathetic nerves of the upper extremity, with special reference to the nerve supply of the arteries. In *The Autonomic Nervous System. Morphological, Comparative, Clinical and Surgical Aspects,* pp. 341–349. Philadelphia: J. B. Lippincott.

POLLOCK LJ, DAVIS L (1933) *Peripheral Nerve Injuries.* New York: Paul B. Hoeber.

PULST ST-M, HALLER P (1981) Thermographic assessment of impaired sympathetic function in peripheral nerves. *Journal of Neurology* **226**, 35–42.

TRIPLETT B, OCHOA JL (1990) Contemporary techniques in assessing peripheral nervous system function. *American Journal of EEG Technology* **30**, 29–44.

TROTTER W, DAVIES HM (1909) Experimental studies in the innervation of the skin. *Journal of Physiology* **38**, 134–246.

VALLBO AB, HAGBARTH K-E, TOREBJÖRK HE, WALLIN BG (1979) Somatosensory proprioceptive and sympathetic activity in human peripheral nerves. *Physiological Reviews* **59**, 919–957.

VERDUGO R, OCHOA JL (1992) Quantitative somatosensory thermotest. A key method for functional evaluation of small calibre afferent channels. *Brain* **115**, 893–913.

WALLIN BG (1981) New aspects of sympathetic function in man. In *Clinical Neurophysiology* (ed. E. Stålberg & R. R. Young). London: Butterworth.

WOOLLARD HH, PHILLIPS R (1932–33) The distribution of the sympathetic fibers in the extremities. *Journal of Anatomy* **67**, 18–27.

YARNITSKY D, OCHOA JL (1990) Release of cold-induced burning pain by block of cold-specific afferent input. *Brain* **113**, 893–902.