It is now recognised that there are several distinct approaches to treatment decision making that doctors can use with their patients—the paternalistic, the shared, and the informed (or consumerist) approach. Each has different implications for the roles of doctors and patients in communicating information and for the type, amount, and flow of information between the two.[4] Moreover, some approaches are more amenable than others to incorporating patients' voices and eliciting patients' agendas.

Doctors who adopt a paternalistic approach, for example, are unlikely to have much interest in discussing patient concerns expressed "in the voice of the life world."[1] They are more likely to want short descriptions of physical symptoms that they can transform into diagnostic categories. In the "pure type" of this approach doctors can then make a treatment decision that they think is in their patients' best interest without having to explore each patient's values and concerns.

In the informed approach patients are accorded a more active role in both defining the problem for which they want help and in determining appropriate treatment. In the pure type of this approach the doctor's role is limited to providing relevant research information about treatment options and their benefits and risks so that the patient can make an informed decision.

Only in the shared approach do doctors commit themselves to an interactive relationship with patients in developing a treatment recommendation that is consistent with patient values and preferences.[5] To enable this to happen, the doctor needs to create an open atmosphere in which patients can communicate all their agenda items. In this approach information exchange helps the doctor understand the patient and ensures that the patient is informed of treatment options and their risks and benefits. It also allows patients to assess whether they feel they can build a relationship of trust with their doctor.

Actual behaviour, of course, rarely corresponds to ideal types, and most doctor-patient encounters combine elements from different models.[6] Moreover, the approach adopted at the beginning of an encounter may change as the doctor gains a better sense of whether the patient has a good understanding of the available treatments.

To develop effective interventions to promote better communication, it is useful to explore specific communication patterns within the broader context of the type of decision making process within which communication is embedded. For example, there may be a mismatch between the decision making approach that the doctor wants to use and patients' desire to voice their own agenda in their own words. Understanding the reasons why communication problems occur can help researchers develop interventions designed specifically to address potentially different types of communication issues.[7 8]

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1 Mishler EG. *The discourse of medicine, dialectics of medical interviews*. Norwood, New Jersey: Ablex Publishing Corporation, 1984.
2 Britten N, Stevenson FA, Barry CA, Barber N, Bradley CP. Misunderstandings in general practice prescribing decisions: qualitative study. *BMJ* 2000;320:484-8.
3 Barry C, Bradley C, Britten N, Stevenson F, Barber N. Patients' unvoiced agendas in general practice consultations: qualitative study. *BMJ* 2000;320:1246-50.
4 Charles C, Gafni A, Whelan T. Shared decision-making in the medical encounter: what does it mean? (or it takes at least two to tango) *Soc Sci Med* 1997;44:681-92.
5 Charles C, Gafni A, Whelan T. Decision-making in the physician-patient encounter: revisiting the shared treatment decision-making model. *Soc Sci Med* 1999;49:651-61.
6 Charles C, Gafni A, Whelan T. What do we mean by partnership in making decisions about treatment? *BMJ* 1999;319:780-2.
7 Gafni A, Charles C, Whelan T. The physician-patient encounter: the physician as a perfect agent for the patient versus the informed treatment decision-making model. *Soc Sci Med* 1998;47:347-54.
8 Whelan T, Gafni A, Charles C, Levine M. Lesson learned from the decision board: a unique and evolving decision aid. *Health Expectations* (in press).

Treating hyperhidrosis

Surgery and botulinum toxin are treatments of choice in severe cases

Physiological sweating from cutaneous eccrine glands maintains normothermia and skin hydration. Properly hydrated palmar skin contributes to the effectiveness of normal grip and permits tasks such as turning the pages of the *BMJ*. Hyperhidrosis is unphysiological and excessive sweating, which squanders water and electrolytes without compensatory cooling from the latent heat or enthalpy of evaporation. It affects about 1% of the United Kingdom population. What help can be offered to patients with this disabling condition?

Hyperhidrosis commonly affects the palms of the hands, the soles of the feet, or the armpits, but in a small number of patients it occurs over the whole body surface. In most patients palmar and/or axillary hyperhidrosis is the major problem, and it is freedom from sweating in the hands or armpits that they seek. In some patients hyperhidrosis affects only the hands or armpits or soles of the feet. Patients with palmar hyperhidrosis have a slippy grip and a cold wet handshake, and their sweat drips into computer keyboards, wets paper, and smudges ink. Exuberant axillary and plantar hyperhidrosis stains and damages clothing and shoes. Eccrine sweat is initially odourless, but patients are embarrassed and inconvenienced by having sodden clothing and damp hands.

Conventional medical therapy with anticholinergic drugs or topical aluminium chloride hexahydrate is inconvenient, unpleasant, and temporary. Patients usually stop using anticholinergic drugs because of a dry mouth, and aluminium chloride hexahydrate often

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causes skin soreness and irritation. Antiperspirants can provide useful palliation in patients with moderate hyperhidrosis, but in severe cases they are ineffective. Most patients with severe localised hyperhidrosis need to consider either surgery or injections of botulinum toxin.

Eccrine sweat glands are innervated by the sympathetic nervous system. Uniquely, the secretomotor neurotransmitter is acetyl choline not noradrenaline.[1] The sympathetic nerves to the arm arise from spinal cord segments T2-T6 and leave the spinal canal in the corresponding ventral rami to synapse or pass through the 2nd to 6th thoracic sympathetic ganglia. All postganglionic sympathetic fibres to the hand, forearm, and arm except the axilla run with the somatic nerves of the brachial plexus (root origins C5-T1). Consequently, division of the sympathetic trunk between the first and second thoracic ganglia will interrupt all the sympathetic innervation to the arm, comprising preganglionic and post-ganglionic fibres.[2]

A study of 54 patients treated by thoracoscopic division of the sympathetic trunk with scissors on the 2nd rib and followed up for up to five years showed an initial cure rate of 100% for palmar hyperhidrosis[3] and no cases of Horner's syndrome, which can occur with electrocoagulation or diathermy division of the intact sympathetic trunk.[4] Hyperhidrosis may recur nine to 12 months later from regrowth of preganglionic nerves. Reinnervation can be impeded by inversion of the cephalad and electrocoagulation of the caudad sympathetic trunk after severance.

Thoracoscopic excision or destruction by electrocoagulation of the T2 and T3 sympathetic ganglia is difficult. Unless both ganglia are ablated axillary sweat glands can still be innervated by sympathetic fibres in the 2nd and 3rd intercostal nerves. It is probably for this reason that axillary hyperhidrosis is often not cured by sympathectomy.

Pedal sympathetic denervation requires removal of the 2nd lumbar sympathetic ganglion. In men ejaculatory impotence after bilateral surgery is almost certain. Some surgeons have reserved the operation for women, but this is questionable since genitosexual innervation is analogous in men and women.[5]

For palmar hyperhidrosis alone the treatment of choice is thoracoscopic sympathetic trunkotomy. Patients with both palmar and axillary hyperhidrosis can be treated by thoracoscopic excision of the T2 and T3 sympathetic ganglia. Those with only axillary or plantar hyperhidrosis are best served by topical botulinum toxin injection.

For axillary hyperhidrosis 240 units of botulinum A toxin-haemagglutinin complex (Dysport) per armpit is injected in 12 0.1 ml aliquots subdermally covering the hyperhidrotic area. Pedal hyperhidrosis is more tedious and uncomfortable to treat because of the large surface area and sensitivity of the feet: 24 to 36 subdermal injections of 0.1 ml are required to cover the entire plantar surface of foot and toes.

Botulinum toxin binds to presynaptic nerve membranes and then inhibits release of acetyl choline by disrupting the Ca^{2+} dependent K^{+} evoked release mechanism.[6] A small before and after study of patients with disabling hyperhidrosis refractory to drug treatment showed that botulinum toxin completely abolished sweating in the injected areas after a delay in onset of two to three days.[7] The effect lasts for up to 11 months.[8] Second and third treatments have been of undiminished effectiveness, but antibody formation may reduce long term therapeutic potency. Attempts to use botulinum toxin to treat palmar hyperhidrosis were complicated by paresis of the intrinsic muscles of the hand.[9] The risk of hand dysfunction is a high price to pay when thoracoscopic sympathetic trunkotomy is a safer alternative. Local toxin diffusion in the axilla or foot is, however, unlikely to produce symptomatic muscle weakness, and we have observed no adverse effects in over 20 patients we have treated.

Thoracoscopic sympathetic trunkotomy and ganglionectomy are safe procedures and operative complications are rare. Deaths and permanent neurological damage from anoxia have occurred when surgeons have unwisely performed immediate consecutive bilateral thoracoscopy rather than two separate operations.[10] The eye signs of Horner's syndrome are produced by deliberate or inadvertent destruction of the first thoracic sympathetic ganglion. This complication is unnecessary and avoidable. Intercostal neuralgia can occur if the sympathetic ganglia are destroyed by electrocoagulation rather than excision.

The incidence of compensatory hyperhidrosis is proportional to the surface area rendered anhidrotic. Compensatory sweating is uncommon after unilateral sympathetic trunkotomy but usual after bilateral T2, T3 ganglionectomy.[3] Its occurrence after axillary botulinum toxin injection in a patient with bilateral sympathetic trunkotomy but incomplete ganglionectomy regressed as axillary sweating returned. Post-sympathectomy gustatory sweating has been reported in 28% of patients.[11]

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1 Schotzinger R, Yin X, Landis S. Target determination of neutrotransmitter phenotype in sympathetic neurons. *J Neurobiol* 1994;25:620-39.
2 Haxton HA. The sympathetic nerve supply of the upper limb in relation to sympathectomy. *Ann R Coll Surg Eng* 1954;14:247-66.
3 Fox AD, Hands L, Collin J. The results of thoracoscopic sympathetic trunk transection for palmar hyperhidrosis and sympathetic ganglionectomy for axillary hyperhidrosis. *Eur J Vasc Endovasc Surg* 1999;17:283-9.
4 Gordon A, Collin J. Thoracoscopic sympathectomy. *Eur J Vasc Surg* 1994;8:247-8.
5 Berard EJ. The sexuality of spinal cord injured women: physiology and pathophysiology. A review. *Paraplegia* 1989;27:99-112.
6 Ashton AC, Dolly JO. Characterization of the inhibitory action of botulinum neurotoxin type A on the release of several transmitters from rat cerebrocortical synaptosomes. *J Neurochem* 1988;50:1808-16.
7 Naumann M, Hofmann U, Bergmann I, Hamm H, Toyka KV, Reiners K. Focal hyperhidrosis: effective treatment with intracutaneous botulinum toxin *Arch Dermatol* 1998;134:301-4.
8 Bushara KO, Park DM, Jones JC, Schutta HS. Botulinum toxin—a possible new treatment for axillary hyperhidrosis. *Clin Exp Dermatol* 1996;21:276-8.
9 Shelley WB, Talanin NY, Shelley ED. Botulinum toxin therapy for palmar hyperhidrosis *J Am Acad Dermatol* 1998;38:227-9.
10 Cameron AEP. Complications of endoscopic sympathectomy. *Eur J Surg* 1998;164(suppl):33-5.
11 Rex LO, Drott C, Claes G, Gothberg G, Dalman P. The Boras experience of endoscopic thoracic sympathicotomy for palmar, axillary, facial hyperhidrosis and facial blushing. *Eur J Surg* 1998;164(suppl):23-6. 6.