Sweating Disorders

4

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Contents

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4.1 Case Vignette

It all started at the age of 43 years. I noticed increased sweating, interestingly mainly on my trunk. When I had to talk to people, I felt embarrassed about my wet shirt. In my work as a farmer in an alpine area, I did not care too much about it. However, in summer time, it occurred more and more often, that I felt dizzy during hot weather. Over the years sweating increased tremendously and my shirts were always soaked wet. The skin area, that did sweat, however diminished and was lately only an area of about 45 cm of diameter at my chest and back. The winter times in the alps are rough and when it got cold, I felt totally well. In summer time however, dizziness at work and sweating got worse. I went to a hospital and was treated with stellatum blockade. That did in fact stop my sweating but dizziness in the heat exaggerated and I could not work anymore, also at moderate temperatures. So I had to stop my work.

During last summer I had to go to my house and lie down on the kitchen floor. The tiles were pleasantly cool and my dizziness ceased as my body temperature went down. I am worried, because nobody can tell me, what's going on with me.

The patient was seen by a neurologist who noted bilateral pupillotonia and generalised and symmetric hyporeflexia, and a Ross syndrome was diagnosed [8].

4.2 Sweating

Sweating is caused by two different mechanisms: thermoregulation to dissipate heat and emotional sweating. Sweating disorders have to be regarded as severe burden for the patient and eventual medical risk. Excessive sweating is a social problem and mounts into occupational restriction and/or social avoidance behaviour. However, loss of sweating eventually leads to exhaustion, heat stroke or death.

Disorders of sweating (hypo- or hyperhidrosis) can be focal or generalised and appear quite frequently in ANS failure. Evaluation of sudomotor function can provide early diagnosis of small fibre neuropathy [3], particularly in early diabetes mellitus, and is used to provide a measure of cholinergic sympathetic function.

Abnormal sweating results from a wide variety of medications that affect the sympathetic nervous system, the thermoregulatory network or the sweat glands. Furthermore, it may result from sleep-stage disturbances, autonomic nervous system disorders, medullary and spinal cord abnormalities, reduction in serum osmolality or abnormalities of osmoreceptor function, hypercapnia, disorders of hormone secretion (hypothyroidism, postmenopausal syndrome, cortisol) and direct sweat gland stimulation by pressure, heat, trauma or toxins. The key to successful diagnosis of sweating disorders is to differentiate whether the patient suffers hypohidrosis or hyperhidrosis and whether the disorder is focal or generalised.

4.3 Patient's History

In patients, who suffer from sweating disorders, the first approach is to interrogate the subjective disease burden. This will provide the physician with a hint on how extensive clinical investigations and therapeutic interventions need to be.

As mentioned above, the first diagnostic key is whether the dysfunction is focal or generalised. In increased and generalised sweating (i.e. observed on the complete body surface), the patient is suffering hyperhidrosis. The same is true if sweating is absent and generalised. It becomes tricky in increased focal sweating. Does the patient suffer focal hyperhidrosis, or – as in our case vignette – does he suffer compensatory sweating? And in fact the clinical correlate is hypohidrosis in all other regions.

Patients often complain of excessive sweating in warm environment and in emotional or stressful situations (generalised, in certain body regions, or focal), heat intolerance, fatigue and exercise intolerance. Gustatory sweating is a normal phenomenon in most people eating heavily spiced foods and occurs bilaterally focused on scalp and face areas. Unilateral occurrence in contrast is pathologic and may not depend on the type of food. It occurs after parotid surgery and post-traumatic misinnervation of parasympathetic fibres in efferent sympathetic postganglionic neurons innervating sweat glands and blood vessels.

Current medication (Table 4.1) and illnesses (thyroid, gastro-oesophageal reflux, tuberculosis, heart failure, chronic pain, malignancies) have to be interrogated as they may influence sweating. Acute or chronic alcohol and drug intake or withdrawal have a significant effect on sweating.

Table 4.1 Drug-induced hyperhidrosis

Drug class	Common examples	Mechanism
Anticholinesterases	Pyridostigmine	Cholinesterase inhibition
Antidepressants: selective serotonin reuptake inhibitors	Citalopram	Serotonergic effect on the hypothalamus or spinal cord
	Duloxetine	
	Escitalopram	
	Fluoxetine	
	Fluvoxamine	
	Mirtazapine	
	Paroxetine	
	Trazodone	
	Venlafaxine	
Antidepressants: tricyclics	Amitriptyline	Norepinephrine reuptake inhibition with stimulation of peripheral adrenergic receptors
	Desipramine	
	Doxepin	
	Imipramine	

Table 4.1 (continued)

Drug class	Common examples	Mechanism
	Nortriptyline	
	Protriptyline	
Antiglaucoma agents	Physostigmine	Physostigmine = cholinesterase inhibition
	Pilocarpine	Pilocarpine = muscarinic receptor agonism
Bladder stimulants	Bethanechol	Muscarinic receptor agonism
Opioids	Fentanyl	Histamine release
	Hydrocodone	
	Methadone	
	Morphine	
	Oxycodone	
Sialogogues	Cevimeline	Muscarinic receptor agonism
	Pilocarpine	

From Cheshire and Freeman [13]; Cheshire and Fealey [12]

Table 4.2 Questions that may help

Do you sweat in the feet? Are your socks drenched after sports?
Do you sweat in intimate area?
Is there excessive sweating in your armpits?
Did you let things fall down because of sweating in your hands?
Do you feel embarrassed when shaking hands because of sweat sometimes?

Important questions comprise duration of the disorder, diseases and conditions before onset, progression to other body regions, areas of increased or decreased sweating, symmetry, triggers (increased ambient temperature, physical activity, stress), diurnal sweating and comparison to sweating before the onset of disease. Night sweat can be a real distressing symptom with many different reasons. To investigate if the disorder occurs isolated or as part of a generalised autonomic dysfunction, the other autonomic subsystems should be interrogated (s. Chap. 2). The diligent interrogation of a patient suspected of autonomic dysfunction occasionally reveals the phenomenon of gustatory sweating, that is, profuse sweating associated with the ingestion of food. In Table 4.2, we have listed some questions that may help.

For physiology see Sect. 1.2.2, for history taking see Sect. 2.2.2.1.

4.4 Physical Examination

Complete physical and neurological examinations are mandatory, looking for signs of PNP, myelopathy or Horner's syndrome, plexus brachialis lesion, brain stem affection and/or central lesions. To investigate focal sweating dysfunction at

bedside, it is valuable to investigate the patient undressed in a quiet room with comfortable ambient temperature and watch for sweat droplets and areas of skin discoloration. Hairiness and tropical changes of the skin and nails hint to peripheral nerve involvement. Distribution of sweat droplets shall be documented, particularly in regions of increased sweating: the face, dorsal neck, axillae, palms and soles and inguinal region. Measurement of skin temperature in different body regions, lateralisation and distal versus proximal distribution by infrared surface method are recommended. The investigator feels changes in skin moisture and texture by touching. It is notable if there are obvious sweat stains on the clothing. For total absence of sweating, patients should be observed in hot environment, the absence of sweating in isolated areas or restriction of profuse sweating to the upper body, while sweating in the lower body areas is absent (may hint at ANP in diabetes mellitus). Documentation by a standardised sketch or photography may help for follow-up.

4.5 Horner's Syndrome

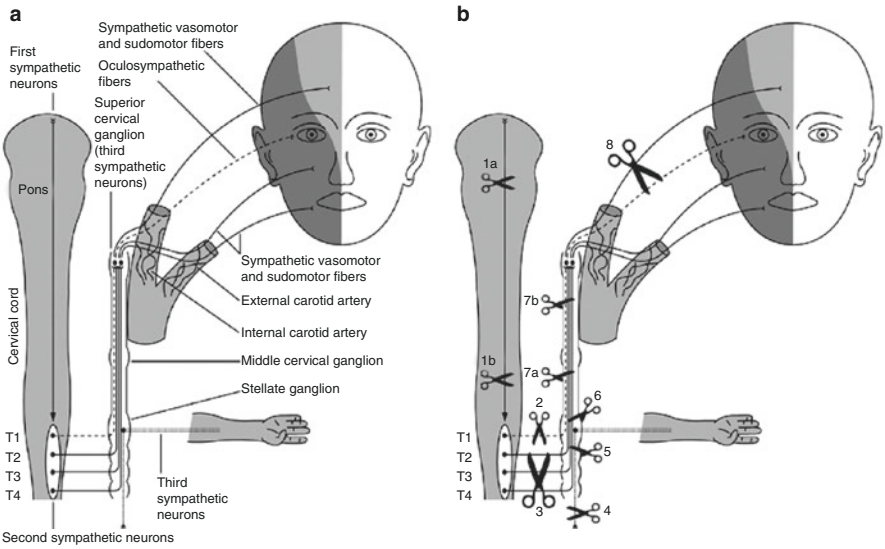
Horner's syndrome (HS) is a central or peripheral sympathetic denervation of the eye. Denervation of the M. tarsalis superior results in ptosis; sympathetic denervation of the inner eye results in parasympathetic predominance causing myosis. HS is best diagnosed in a darkened room. HS is placed in this chapter, because depending on the location of the sympathetic lesion, hypohidrosis occurs to the skin of the forehead/face or ipsilateral arm (Fig. 4.1). Enophthalmus is considered part of the trias. However, enophthalmus per se is only rarely present, being mimicked by ptosis. Therefore a more correct description of HS is:

1. Ptosis
2. Myosis
3. Hypohidrosis

HS is an important hint that – accompanied by other signs – may already help to establish the diagnosis. Clinical evaluation is mandatory along the route of the sympathetic fibres guided by clinical presentation (Fig. 4.1).

HS is also one of the leading signs of *harlequin syndrome*. It is in fact the name of two very different conditions. Here we describe the harlequin syndrome caused by sympathetic lesion; the other condition with this very same name is a skin disorder of congenital ichthyosis.

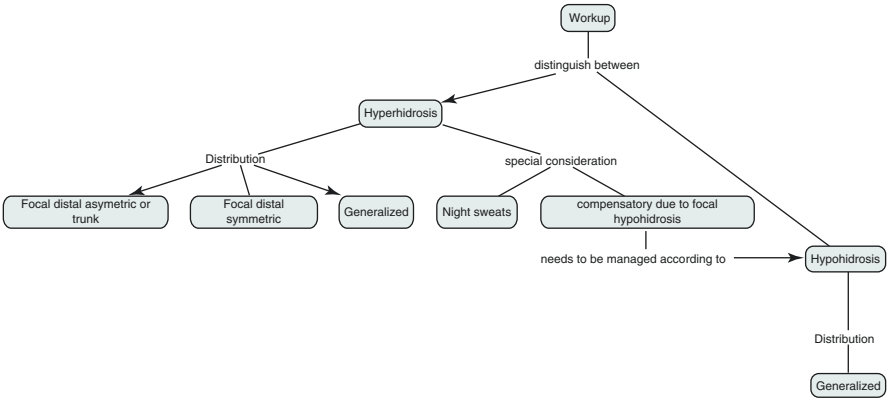
The name is derived from loss of flushing on one side of the face by ipsilateral sympathetic lesion, which prevents sympathetic facial vasodilatation and sweating. The site of the lesion might as well be identified by Fig. 4.1. In rare cases, symptoms of harlequin syndrome are seen in *Adie's syndrome* (tonic pupils with hyporeflexia) or *Ross syndrome* (tonic pupils with hyporeflexia and segmental anhidrosis). The latter two are usually accompanied with a more widespread involvement of the autonomic nervous system.



lesion site	DF	Arm	Horners Syndrome
1	HF	-	central
2	0	=	preganglionic
3	HF	=	0
2+3	HF	=	preganglionic
4	0	-	0
5	HF	-	0
6	HF	-	preganglionic
7	HF	=	preganglionic
8	MFHN	=	postganglionic

Fig. 4.1 Schematic sketch and table helping to diagnose the location of a sympathetic lesion often including Horner's syndrome (Adapted from Wasner et al. [14]); *DF* areas of disturbed facial flushing, *Arm* sympathetic arm innervation, *hf* hemifacial, * medial forehead and nose

4.6 General Considerations for Diagnostic Work-Up



Specific laboratory work-up is necessary only in rare cases and laboratory tests are not widely available [7]. Diagnosis of patients suffering sweating dysfunctions should not depend on the presence of a specialised autonomic laboratory Table 4.1.

4.6.1 Generalised Hyperhidrosis

A carefully taken history should already reveal the generalised character of sweating. In many cases, the patient will tell that "his whole body is soaked wet all the time". It can be time consuming to find out if increased sweating always affects the whole body or only specific areas. What are the exact circumstances and situations when profuse sweating occurs (physical activity, emotional stress, hot environment, night sweat)? How was sweating before the patient noticed the changes, and was there increased focal sweating before? Concomitant diseases, medication and drug abuse have to be evaluated carefully. According to the obtained information, further diagnostic work-up and management have to be planned.

4.6.2 Generalised Hypohidrosis

This is a rare and sometimes life-threatening condition, because of hyperthermia or heat-related illness, as body core temperature homeostasis cannot be maintained. Most patients remain unaware of hypohidrosis and may report on heat intolerance with dizziness, vertigo, dyspnoea and even fainting. It has to be differentiated if the condition increased gradually, maybe as a severe form of progressive hypohidrosis beginning in the extremities, or if it had sudden onset. In the latter case, drugs may play a causal role including antimuscarinic anticholinergic agents, carbonic anhydrase inhibitors and tricyclic antidepressants [12]. These drugs are quite often found to cause sudomotor side effects in elderly patients. Other neurological side effects such as overactive bladder, neuropathic pain, irritable bowel syndrome, reactive airway disease, Parkinson's disease, dizziness, depression, nausea and headache may contribute to heat-associated illness in this population (for a detailed description of the route of action of different drugs, see Cheshire and Fealey [12]). Further factors include decline in sweating responses with ageing and fluid restriction. As acetylcholine is the principal neurocrine mediator, anhidrosis is one of the clinical hallmarks by which acute anticholinergic toxicity may be recognised. The symptom of dry-mouth often accompanies the less apparent symptom of hypohidrosis. In any case, drug screening may be valuable including a drug history, eventually also drug abuse.

4.6.3 Focal Distal Symmetric Hyperhidrosis

This disorder may be easily interrogated from patients. In many cases, focal distal symmetric hyperhidrosis occurs in the course of some other neurological disorders. Chronic alcoholic patients often present with hyperhidrosis in the feet and

sometimes palms [10]. Some of these patients will report on soaked socks. If no other cause for a secondary hyperhidrosis is known, diagnostic management will proceed as delineated in primary hyperhidrosis. Distal hyperhidrosis as generalised hyperhidrosis may cause severe social embarrassment leading to avoidance behaviour. Secondary social and psychological consequences of the disease increase disease burden. Even more, especially plantar hyperhidrosis is eventually causing bromhidrosis (foul-smelling sweat), infection and secondary skin lesions.

4.6.4 Focal Distal Asymmetric or Trunk Hyperhidrosis

This particular form of sweat disorder is often the presentation of a partial hypo- or anhidrosis with compensatory sweating in the preserved body areas. For example, compensatory sweating may be observed in the upper body or trunk and facial region, due to reduced sweating in extremities. However, patients will much more likely notice the sweating than the hypohidrotic areas. In these cases, the distribution and degree of sweat disturbance have to be evaluated carefully and documented for follow-up investigations. To screen for a secondary sweating disorder, other neurological signs are providing valuable hints.

4.6.5 Secondary Hyperhidrosis

Spinal cord injury causing autonomic dysreflexia frequently induces secondary hyperhidrosis, which can occur years after the injury. A number of drugs induce hyperhidrosis (see Table 4.1). Biochemical agents including chemical warfare and pesticides are a rare but not neglectable cause. In addition, other conditions, e.g. hypoglycaemia, anxiety and menopause, induce hyperhidrosis.

Paroxysmal sweating: Neurologic diseases including diencephalic epilepsy, pontine ischaemia and carcinoid syndrome lead to episodes of sweating. Endocrinologic causes for paroxysmal sweating are thyrotoxicosis, which might well respond to beta-blocker and diabetes mellitus. In pheochromocytoma, excess catecholamines lead to recurrent sweating episodes.

4.6.6 Night Sweats

Night sweats are symptoms commonly linked to menopause, tuberculosis and lymphoma. However, night sweats are often reported by persons without these conditions. Other important differential diagnoses include human immunodeficiency, infectious diseases, endocarditis, gastro-oesophageal reflux disease (GORD), sleep-associated breathing disorders (obstructive and central sleep apnoea syndromes and nocturnal hypercapnia in neuromuscular patients with respiratory failure, as observed by one of the authors), hyperthyroidism, hypoglycaemia, later stages of PD, MSA, depression and anxiety disorders (C: medication induced!). A more complete list is presented in Table 4.3. Alcohol use, particularly alcohol dependency and

Table 4.3 Causes of night sweats

Malignancy	Lymphoma
	Leukaemia
	Other neoplasms
Infections	Human immunodeficiency virus
	Tuberculosis
	Mycobacterium avium complex
	Infectious mononucleosis
	Fungal infections (histoplasmosis, coccidioidomycosis)
	Lung abscess 2
	Endocarditis
	Other infections
Endocrine	Ovarian failure
	Hyperthyroidism
	Diabetes mellitus (nocturnal hypoglycaemia)
	Endocrine tumours (pheochromocytoma, carcinoid tumour)
	Orchiectomy
Rheumatologic	Takayasu's arteritis
	Temporal arteritis
	Others
Obstructive sleep apnoea	
Gastro-oesophageal reflux disease	
Chronic fatigue syndrome	
Granulomatous disease	
Chronic eosinophilic pneumonia	
Lymph node hyperplasia	
Diabetes insipidus	
Prinzmetal's angina	
Anxiety	
Depression	
Pregnancy	
Drugs	
Antipyretics (salicylates, acetaminophen)	
Antihypertensives	
Phenothiazines	
Substances of abuse	Alcohol, heroin
Over-bundling	
Autonomic overactivity	

Modified from Viera et al. [15]

acute withdrawal, may cause night sweats. Overall, the prevalence estimates ranged from 10% among older primary care patients to 60% among women on an obstetric inpatient unit [9]. However, some individuals may be less tolerant of either sweat or its cooling effect or anxious about symptoms, like night sweats, that might indicate illness [9].

Management After exclusion of all treatable disorders as causative factors, patients should be assured of the benign character of the disorder, and possible behavioural and environmental measures can be discussed that may help to relief their bothersome symptoms. Alpha-adrenergic blockers may reduce night sweats in patients taking serotonin reuptake inhibitors.

4.6.7 Differentiating Hyperhidrosis from Hypohidrosis with Compensatory Sweating

It may be tricky to differentiate compensatory hyperhidrosis from primary focal hyperhidrosis. Compensatory hyperhidrosis – as mentioned above – occurs in the skin areas, which are less affected in those patients who have severe hypohidrosis in a wide area of their bodies, which might be unnoticed by the patient. There are a number of techniques to investigate the function of the sweat glands (QSART, TRST) or epiphenomena of sweating (e.g. SSR); however, if those are not available, it might be helpful to wrap the patient into a white blanket in an environment of increased temperature, eventually having the patient drink hot tea. Areas which do not sweat will leave no stains on the blanket.

4.6.8 Sweating Disorders Associated with Other Diseases

Sweating disturbances are common (64%) and distressing symptoms in PD that are related mainly to autonomic dysfunction, off periods and dyskinesias [16]. Hyperhidrosis may occur in association with wearing-off phenomena. Compensatory hyperhidrosis may be observed in the upper body, due to reduced sweating in extremities (unlike MSA). PD is characterised by a length-dependent involvement of postganglionic sudomotor fibres, whereas MSA is characterised by widespread, early and preganglionic autonomic failure. In diabetic ANS, length-dependent hypohidrosis may be observed, thus beginning in the soles and palms. Also gustatory sweating may occur in PD patients.

4.7 Laboratory Investigations

Laboratory tests can assess central and peripheral sudomotor function, as the thermoregulatory sweat test (TST, [1]), or postganglionic function alone, the quantitative axon reflex test (QSART, [4]), the sympathetic skin response test (SSRT), the

quantitative direct and indirect axon reflex test (QDIRT, [2]) and the dynamic sweat test (DST, [5]) [6].

TST The test is performed in a temperature- and humidity-controlled chamber (45–50 °C). The whole body is covered with an indicator dye and the changes in colour due to sweat production are documented. Asymmetric patterns due to focal anhidrosis or stocking and glove distributions in length-dependent neuropathy may be observed.

QSART This test measures postganglionic axon reflex-mediated sweat production in a small restricted area of the skin. A multicompartmental sweat capsule is used to stimulate sweat glands by iontophoresis of acetylcholine. A first, direct sweat response in the area of iontophoresis is discriminated from the indirect, axon reflex-mediated response in the surrounding area (e.g. see [3] for details). See Fig. 1.4 for a schematic sketch on how this test works.

SIT The test evaluates postganglionic sympathetic cholinergic sudomotor function by measuring the direct and axon reflex-mediated sweat response. Sweat glands are stimulated by iontophoresis of acetylcholine, pilocarpine or methacholine, followed by application of a thin layer of mouldable material on the skin. Sweat droplets displace the silicone material during polymerisation resulting in permanent impressions that can be quantified by various methods. A number of droplets, size and distribution are reported.

QDIRT and DST These methods quantify sudomotor function with spatial and temporal resolution. They combine the stimulation of sweat glands and sudomotor axons by iontophoresis of a cholinergic agonist into the skin with the colour change of an indicator as sweat pours out. Each sweat droplet results in a colour spot which increases in perimeter and number over time. The evolving pattern of spots is recorded by high-resolution digital photography or video and evaluated with automated image analysis software.

SSRT This neurophysiologic measure of electrodermal activity provides a surrogate marker of sympathetic cholinergic sudomotor function. An arousal stimulus (electric, acoustic, deep breath) induces a change in skin potential, which is recorded from the palms and soles of the feet most often. SSRs are reported as present or absent. For amplitude and latency, normative values have been published.

4.8 Management

4.8.1 Nonpharmacological

Avoid spicy foods, coffee, tea, nicotine and alcohol.

Supporting measures are diverse relaxation techniques (e.g. progressive muscle relaxation).

4.8.2 Treatment of Hypohidrosis

If generalised hypohidrosis is of clinical relevance and drug induced, a reduction of the inducing drug should be considered. Deficient sweating may further be managed by avoiding situations of heat stress and cooling the skin with externally applied water.

4.8.3 Treatment of Hyperhidrosis

Treatment of first choice is topical applications of aluminium salts. Those are already added in many antiperspirant agents; however, a higher dose is necessary in hyperhidrosis (15–25%) several times a day. Side effects include skin irritation and dysaesthesias. Patients should take care not to get in contact with dark clothing directly after application due to discoloration of their clothes. In Frey syndrome, e.g. after surgery, topical application of 0.5% glycopyrrolate is recommended.

Tap water iontophoresis is very effective in palmar and plantar hyperhidrosis. Standard settings are continuous direct current or eventually pulsed direct current, which might be slightly less effective [11]. Patients with implanted pacemakers and pregnant women should abstain from this therapy. Side effects include skin irritations including erythema, blistering and local burning sensations.

If the previous measures were not able to relieve symptoms, botulinus toxin injection may be considered. The application is intradermal. In many patients, a good effect is seen up to 7 months. When applied in palmar hyperhidrosis, paresis of small hand muscles is often observed.

Sympathectomy of the sympathetic ganglia Th2/Th3 is used foremost in palmar hyperhidrosis. However, compensatory hyperhidrosis might complicate this therapy in the long run. Surgery-related complications include haemothorax, pneumothorax, injury to the thoracic duct and the phrenic nerve and Horner's syndrome. See Text Box 4.1.

Generalised hyperhidrosis is treated with anticholinergic drugs (e.g. methanthelinium bromide (2 × 50 mg/day)). Only few data exists on administration. Side effects of anticholinergic drugs might limit the therapy (urinary retention, constipation, memory impairment, drymouth, reduced accommodation). Alternative treatment options are antidepressants (especially tricyclic antidepressants like amitriptyline, but also paroxetine), beta-blockers or calcium channel antagonists (e.g. diltiazem).

Text Box 4.1. Endoscopic Transthoracic Sympathectomy (ETS)

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Indication. The classic indication is primary focal axillar and/or palmar hyperhidrosis (HH). Facial blushing, Raynaud's disease, angina pectoris, reflex sympathetic dystrophy and plantar HH are considered as further possible indications. However, the latter is not yet sufficiently documented in literature.

Surgical procedure. ETS is a video-assisted endoscopic keyhole procedure under general anaesthesia. The endoscope is inserted via a single skin incision in the armpit. Through the working channel, a cautery probe is inserted to place two to three thermo-lesions on the sympathetic trunk between ganglia 2, 3 and 4. We also coagulate aberrant, mostly invisible connections up to 3 cm laterally as prevention against recurrences.

Even though the complication rate is fairly low, we operate one side and do the procedure on the other side 6 weeks, thereafter. Instead of thermo-lesions, also cutting is described in literature, or clamping, that can be reversed in case of adverse effects.

Risks and complications. The most common unwanted effect is compensatory sweating on the torso, whereas surgical complications such as infection and bleeding or symptoms from residual pneumothorax are extremely rare.

Results. At the Department of Neurosurgery, Medical University of Graz, we reviewed a total of 44 patients over a period of 60 months. The mean success rate by increased quality of life was 92% (96% for palmar HH and 85% for axillary HH). The recurrence rate was 14%, whereas only two patients required a reoperation. There was no serious complication. Fifty-five percent of the patients reported signs of compensatory HH; however, 18% denoted it onerous, and only one patient (2%) would not have the same procedure again. Compensatory hyperhidrosis was habitually noted temporarily or was accepted in a lesser stage by most of the patients.

Take Home Messages

Sweating disorders:

- Need special considerations.
- Anatomic representation plays a huge role (focal, generalized; hypo-, hyperhidrosis).
- Sweating disorders based on systemic diseases have to be ruled out.
- Non-pharmacological and pharmacological management is available.

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