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1. Introduction

Sweating is a physiological and vital condition to control thermoregulation of the skin (Rajesh et al., 2003). Hyperhidrosis is defined as an excess of sweating beyond the amount needed to cool down elevated body temperature (Kreyden & Burg, 2000). Primary hyperhidrosis as a disease seems trivial to general public because of its falsely perceived rarity (Eisenach et al., 2005). Furthermore, although not life-threatening (Reisfeld et al., 2002), it is evident that it can lead to severe psychologic, social and occupational dysfunction (Shargall et al., 2008). Nowadays primary hyperhidrosis is being recognized increasingly and its treatment options are gaining widespread attention (Eisenach et al., 2005). Although medical therapies have been the main treatment options for many years, surgical interventions have recently been proven to be an important therapeutic alternative. This shift has corresponded with the evolution of minimally invasive surgical techniques (Grondin, 2008), the main topic of this issue.

2. Classification and causes of hyperhidrosis

Hyperhidrosis can be classified either by pathogenesis in a primary (idiopathic) and a secondary (symptomatic) form or by localisation and extension in a localised and a generalised form (Fig. 1).

![Fig. 1. Classification of hyperhidrosis by localisation and extension](www.intechopen.com)
Secondary hyperhidrosis appears to be more generalised and is triggered by an underlying disease process like infectious, endocrine or neurologic disorders (Eisenach et al., 2005; Kreyden & Burg, 2000; Shargall et al., 2008) (Table 1). Therapy of choice is treating the basic disease.

<table>
<thead>
<tr>
<th>Category</th>
<th>Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Influenza, tuberculosis,</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hyperthyroidism, diabetes, menopause, obesity</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Leukemia, lymphoma</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Spinal cord injury, parkinson’s disease</td>
</tr>
<tr>
<td>Drugs</td>
<td>Corticosteroids, antibiotics, antidepressants</td>
</tr>
<tr>
<td>Psychogenic</td>
<td>Panic disorder, stress, pain</td>
</tr>
</tbody>
</table>

Table 1. Causes of secondary hyperhidrosis

The cause of primary hyperhidrosis, mostly affecting local parts of the body, is still unknown (Duarte & Kux, 2008). There seems to be a genetic predisposition in autosomal dominant fashion with variable penetrance, in 25-50% of cases a positive family history can be detected (Eisenach et al., 2005). The disease tends to begin in early childhood and becomes worse at puberty. It affects males and females equally (Shargall et al, 2008). The exact pathophysiology also remains unknown. There appears to be an overactive response of the eccrine glands to both heat and emotional stimuli, mediated through the sympathetic nervous system (Lee et al., 1999; Shargall et al., 2008) (Fig. 2). Therefore mostly affected areas are where eccrine glands are concentrated like the palms, the axillae, the face and the soles. Nearly half of diseased people suffer from an axillary manifestation (Schlereth et al., 2009). Overall the estimated prevalence of primary hyperhidrosis might be as high as 0.6-1% of the Western population, 2.8% of the US population and even 3% of the Asian population. Asia is also considered as an endemic area (Eisenach et al., 2005).

![Fig. 2. Innervation of the eccrine glands by the sympathetic nervous system](https://www.intechopen.com)
3. Diagnosis and patient evaluation

A substantial part of the diagnosis of hyperhidrosis can be achieved by obtaining a patient's history, followed by a physical examination. Once secondary causes of hyperhidrosis have been ruled out by additional laboratory tests, dermatologists have several techniques to stratify the severity of sweating like gravimetric testing, the Minor starch-iodine test and the ninhydrin test. Quality-of-life assessments may support the categorization of primary hyperhidrosis as a serious medical condition (Eisenach et al., 2005; Solish et al., 2008).

4. Treatment of primary hyperhidrosis

Primary hyperhidrosis is treated symptomatically. There are lots of therapeutic options classified in nonsurgical and surgical treatment. Generally, therapy should be particular to each individual patient and chosen based on disease location, disease severity and expectations for improvement (Gee & Yamauchi, 2008). Dermatologists suggest following a graduated scheme like the guidelines elaborated by the German Society of Dermatology (Wörle et al., 2007).

4.1 Nonsurgical treatment

The initial treatment for primary hyperhidrosis should always be nonsurgical. It includes topical treatments such as aluminium chloride, iontophoresis, oral medications such as anticholinergics and botulinum toxin (Reisfeld & Berliner, 2008). Unfortunately, a lot of cases do not respond sufficiently to these treatment regimes and effects are usually transient (T.S. Lin, 2001). Nonsurgical therapy options are mostly practised by dermatologists, they are listed below (Table 2).

<table>
<thead>
<tr>
<th>Therapy option</th>
<th>Example</th>
<th>Indication</th>
<th>Mechanism/Specifics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psycho-vegetative influence</td>
<td>Autogenic training, hypnosis, psychotherapy</td>
<td>Generalised adjuvant</td>
<td>Psychovegetative damping avoids activation of the reduced threshold of the eccrine glands</td>
</tr>
<tr>
<td>Medications:</td>
<td>Antiperspirants (aluminium chloride hexahydrate)</td>
<td>Axillary</td>
<td>Blocking the lumen of the eccrine duct</td>
</tr>
<tr>
<td>- Topical</td>
<td>Anticholinergics, psychotropics, betablockers</td>
<td>Generalised</td>
<td>Anticholinergic, chemical psychovegetative damping</td>
</tr>
<tr>
<td>- Systemic</td>
<td></td>
<td></td>
<td>Caution: Side effects</td>
</tr>
<tr>
<td>Physical therapy</td>
<td>Iontophoresis</td>
<td>Palmar plantar</td>
<td>Exact mechanism unknown: Ionic current causes a temporary block of the eccrine duct</td>
</tr>
<tr>
<td>Botulinum toxin (BTX)</td>
<td>Local intradermal injection</td>
<td>Axillary palmar plantar</td>
<td>Chemical block: Inhibits the release of acetylcholine at the cholinergic synapse</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Caution: Effect decreases within 6 months</td>
</tr>
</tbody>
</table>

Table 2. Nonsurgical therapy options
4.2 Surgical treatment

Surgery should be reserved to severe hyperhidrosis and should only be contemplated when less invasive nonsurgical options have failed to provide adequate treatment (Naunheim, 2000). It includes local surgical axillary procedures such as excision, curettage or liposuction of the sweat glands and thoracoscopic sympathectomy (Baumgartner, 2008) (Table 3).

<table>
<thead>
<tr>
<th>Therapy option</th>
<th>Procedure</th>
<th>Indication</th>
<th>Specifics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excision of sweat glands (En-bloc-resection of dermis and subcutis)</td>
<td>Radical excision: several techniques with plastic skin suture</td>
<td>Axillary therapy-refractory</td>
<td>Caution: Scarring, contractures</td>
</tr>
</tbody>
</table>
| Subcutaneous Excision of sweat glands (limited resection via tiny incisions) | - Curettage  
- Liposuction | Axillary therapy-refractory | Caution: Hematoma, infection |
| Sympathetic block               | Thoracoscopic sympathectomy: Detailed description follows below | | |

Table 3. Surgical therapy options

4.2.1 Thoracoscopic sympathectomy

The rationale for sympathectomy in the management of primary hyperhidrosis is based on interrupting the transmission of impulses from the sympathetic nervous system to the eccrine sweat glands (Reisfeld et al., 2002). Object of surgery is the sympathetic trunk, a series of ganglia which are located in a line lateral and parallel to the vertebral bodies of the spinal column. The thoracic portion of the sympathetic trunk contains 12 ganglia, where the input is switched over to the effector (Naunheim, 2000). Sweat glands are innervated segmentally, that means a certain ganglion level can be ascribed to a certain localisation of hyperhidrosis (C.C. Lin & Telaranta, 2001). During the surgical procedure on the sympathetic trunk, the ganglia, lying in front of the heads of the ribs and covered by a thin layer of parietal pleura, are readily apparent with the lung retracted caudally (Shargall et al., 2008) (Fig. 3).

Fig. 3. Anatomy of the sympathetic trunk and applied surgical procedure
4.2.1.1 Surgical approaches and techniques

The first open surgical approaches occurred nearly a century ago (Baumgartner, 2008). These aggressive approaches were associated with significant patient morbidity and a protracted recovery period (Dewey et al., 2006). They often required a moderate to large sized incision in the chest which demanded cutting muscles and separating ribs to expose the sympathetic chain (Naunheim, 2000). Over the past decade, endoscopic sympathectomy, requiring three or two small thoracic incisions, replaced open procedures (Fig. 4). Today magnification and high resolution, attained with videoassisted thoracoscopic surgery, allows a detailed representation of anatomical structures which reduces risk of complications (Zacherl et al., 1999) (Fig. 5). Meanwhile further advances, utilizing microinstrumentation called needlescope surgery or using a uniportal access, enable procedures done on an outpatient basis with minimal risk of surgical trauma and excellent cosmetic results (Dewey et al., 2006; Lee et al., 2000).

Fig. 4. (a) Example for a biportal access. (b) View via videothoracoscope (R=rib)

A short overview of the historical development of sympathetic surgery and its application as to primary hyperhidrosis is listed in the table below (Table 4).

<table>
<thead>
<tr>
<th>Year</th>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>1889, 1896</td>
<td>First open cervical sympathectomies for epilepsy by Alexander and Ionnesco</td>
</tr>
<tr>
<td>1920</td>
<td>First open thoracic sympathectomies for hyperhidrosis by Kotzareff</td>
</tr>
<tr>
<td>1942</td>
<td>First thoracoscopic sympathectomies for different pathologies by Hughes</td>
</tr>
<tr>
<td>1944</td>
<td>Further thoracoscopic sympathectomies for different pathologies by Goetz and Marr</td>
</tr>
<tr>
<td>1954</td>
<td>Further thoracoscopic sympathectomies for different pathologies by Kux and Wittmoser</td>
</tr>
<tr>
<td>1992</td>
<td>First videoassisted thoracoscopic surgery for posttraumatic pain syndrome by Chandler</td>
</tr>
<tr>
<td>1993</td>
<td>Further videoassisted thoracoscopic surgery for hyperhidrosis by Claes and Drott presented at the First International Symposium on Thoracoscopic Sympathectomy, Boras, Sweden</td>
</tr>
</tbody>
</table>

Table 4. Milestones in the history of sympathetic surgery and the therapy of hyperhidrosis
Today much controversy and unanswered questions remain concerning the ideal thoracoscopic sympathetic operation (Baumgartner, 2008). What is the best technique of intervention: Should the sympathetic chain or ganglion be resected (sympathectomy), transected (sympathicotomy) or should only the rami communicantes be divided (selective sympathectomy or ramicotomy)? Sympathectomy represents an aggressive approach, inducing a high rate of compensatory sweating (CS) (Lee et al., 1999). This unrequested side effect, specified below, could be considerably reduced by the nowadays commonly used sympathicotomy (Fig. 6). Another decrease of CS could be ascribed to ramicotomy, a limited technique first described by Wittmoser (Wittmoser, 1992) (Fig. 7). Due to the high rate of recurrence of preoperative symptoms, this approach is actually no longer used (Gossot et al., 2003).

Fig. 5. (a) Sympathicotomy. (b) Ramicotomy

Which instrument should be utilised to dissect the sympathetic trunk: Harmonic scalpel, ultrasonic, laser or thermocoagulation? All of them yield similar results (Inan et al., 2008), but due to electrocautery, care should be exercised to avoid heat damage to the adjoining structures (Krasna, 2008). Another recently upcoming procedure is the clamping method, published by Lin and colleagues in 1998 (C.C. Lin et al., 1998) with the potential advantage of reversibility in those patients unhappy with the outcome (Reisfeld et al., 2002) (Fig. 6). But so far, there have been only a mere handful of such reversals done with the surgical clamping technique and these reported reversals met with varied success (Kwong et al., 2008). Neural cell death is supposed to be responsible, when the clips are not removed relatively soon postoperatively (Dewey et al., 2006).

Fig. 6. Clamping method with two clips above and below the third rib
At which level sympathectomy should be performed and what is the correct extent of the procedure? So far, there is no consensus among surgeons, but there seems to be a correlation to postoperative effect and occurrence of side effects. During a long period of time extensive resections from level T2 to T5 were commonly performed (Dewey et al., 2006; T.S. Lin & Fang, 1999; Schmidt et al., 2006). But investigators demonstrated: reducing the extent of sympathectomy leads to a lower incidence of CS (Dumont, 2008). Today surgeons limit the extent of sympathectomy, there is a trend to two-level to the point of single-level surgery. The elected level of intervention is conditioned on the segmentally innervated localisation of hyperhidrosis and is shown in Table 5, based on a review of Krasna in 2008 (Krasna, 2008) (Table 5).

<table>
<thead>
<tr>
<th>Localisation</th>
<th>Level of intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial</td>
<td>T2</td>
</tr>
<tr>
<td>Palmar</td>
<td>T2, T3</td>
</tr>
<tr>
<td>Axillary</td>
<td>T3, T4</td>
</tr>
<tr>
<td>Plantar</td>
<td>&gt;T4 (obsolete)</td>
</tr>
<tr>
<td></td>
<td>L2, L3 (today via endoscopic lumbar extraperitoneal sympathectomy)</td>
</tr>
</tbody>
</table>

Table 5. Localisation of primary hyperhidrosis and related level of intervention

T1 sympathectomy for facial hyperhidrosis is abandoned long time ago due to high risk of Horner’s syndrome. For some time level of choice is T2 ganglia. Although T2 is also seen as centre point of neurologic impulse transmission through the brachial plexus to the hand (Hsia et al. 1999), some investigators suggest preparing lower levels like T3 (Dewey et al. 2006; Kwong et al., 2008; X. Li et al., 2008), because T2 is close to stellate ganglion with a risk of Horner’s Syndrome non-negligible and the fact that higher levels can increase rate of CS (Schmidt et al., 2006). T4 still remains level of choice for axillary hyperhidrosis (Lee et al. 1999; Licht et al. 2005). Isolated plantar hyperhidrosis occurs rarely but rather in combination with palmar or axillary type. As combined manifestation it is often treated by sympathectomy for upper limb hyperhidrosis and leads in 85% of cases to an improvement of plantar symptoms, shown by Reisfeld and colleagues (Reisfeld et al., 2002). Causes of improvement are unknown. Treating plantar hyperhidrosis by preparing lower thoracic levels is no longer practised (Kwong et al., 2008). Today isolated plantar hyperhidrosis is treated at level L2 and L3 by endoscopic lumbar sympathectomy (Loureiro et al., 2008).

4.2.1.2 Complications and side effects

Complications in hyperhidrosis surgery are rare and exceptional. Some can be avoided by experience or by technical improvement of the surgeon, others are unforeseeable. However any complications are less acceptable than for other sorts of thoracic operations because sympathectomy is a functional surgery for young patients in good health (Dumont, 2008). In 2004 Ojimba and Cameron did a Medline search using the term thoracoscopic sympathectomy and analysed all publications for reported complications (Ojimba & Cameron, 2004) (Table 6): No death has ever been reported in any published series but there are anecdotal reports of nine deaths following thoracoscopic sympathectomy. Five patients died from excessive haemorrhage, three due to incidents of narcosis and one death remained unexplained. Nevertheless, mortality associated with thoracoscopic sympathectomy is a rare condition compared to the high number of surgical procedures. The most common complication is pneumothorax. Up to 75% of patients have some residual
gas or air in the thorax at the end of procedure, mostly resolving spontaneously. A temporary tube drainage is only required in 0.4-2.3% of cases, usually either after direct trauma to the lung at the time of trocar insertion, after dissolving adhesions or after rupture of a bulla as a consequence of anaesthesia, if high inflation pressures are used. Apart from the deaths mentioned above, reports of serious intraoperative bleeding are rare. Bleeding usually arises from disrupted intercostal veins or bleeding at the site of trocar insertion. The highest rate of significant bleeding with an incidence of 5.3% was reported by Gossot and colleagues. They also described one laceration of the subclavian artery demanding an immediate thoracotomy (Gossot et al., 2001). Horner’s Syndrome is the mostly feared complication. Occurred by irritation or damage to the stellate ganglion T1, it causes miosis, ptosis and enophthalmus on the same side of the face. Symptoms are often transient and decrease within weeks or months, but can also persist (Kaya et al., 2003). Since introduction of the videothoracoscope, which allows a better view, rate of postoperative Horner’s Syndrome could be significantly reduced (Zacherl et al., 1999). However it is mentioned in almost all series. Gossot and colleagues found three main causes: damage by a direct or indirect current diffusion using diathermy, by excessive traction on the nerve during dissection or misdetermination of the ribs by the surgeon (Gossot et al., 2001). Pain in the form of intercostal neuralgia with dysesthesia at the site of trocar insertion is rarely documented but more frequent than generally recognized. Many centres perform short-stay surgery that may lead to underestimation of pain results. In most series pain resolves within months, but Walles and colleagues could detect a persistence for years (Walles et al., 2008). Further unfrequent complications are wound infection, pneumonia, chylothorax arising from laceration of an accessory thoracic duct (Gossot, 1996), rhinitis caused by increased parasympathetic stimulation of nasal mucosa (Herbst et al., 1994) and cardiopulmonary modification. The latter is recently paid particular attention: In a case report in 2009 O’Connor and colleagues presented a patient with postoperative asystole. After successful resuscitation, permanent bradycardia required a pacemaker treatment (O’Connor et al., 2009). Surveys including pre-, peri- and postoperative measurements of cardiopulmonary function presented: the decreased activity of sympathetic nervous system after sympathectomy is comparable to the effect of a beta-blocker. It reduces heart rate and worsens pulmonary function. But the clinical importance of these findings was not significant (Vigil et al., 2005). However patients suffering from vasovagale syncope or high performance athletes should be advised of possible bradycardia and also asthmatics should be informed about potential deterioration of obstructive lung disease.

<table>
<thead>
<tr>
<th>More frequently reported complications</th>
<th>Less frequently reported complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
<td>Wound infection</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Horner’s Syndrome</td>
<td>Chylothorax</td>
</tr>
<tr>
<td>Pain and Dysesthesia</td>
<td>Rhinitis</td>
</tr>
<tr>
<td></td>
<td>Cardiopulmonary modification</td>
</tr>
</tbody>
</table>

Table 6. Overview of possible complications caused by thoracoscopic sympathectomy

Side effects are almost constant and unavoidable. They occur in nearly all series of surgery and therefore they are main topic of numerous articles (Dumont, 2008). Compensatory sweating (CS) represents the most common side effect. It is defined as a postoperative increased sweating in body regions unaffected by sympathectomy (Lyra et al., 2008) (Fig. 7). The exact mechanism remains poorly understood. It is speculated that a
greater amount of sweating elsewhere in the body compensates for the lack of sweating in the treated body area in order to maintain sweating balance of the whole body in a thermoregulatory way (Licht & Pilegaard, 2004). In 2008 Lyra and colleagues tried to study the exact pathogenesis and assumed that sympathetic block causes a dysfunction of control loop with lack of negative feedback to the hypothalamus resulting in CS (Lyra et al., 2008).

The published rates of CS vary widely from 1.2-90% (Dumont, 2008): On the one hand evaluating of CS is subjective and varies according to the patient (Leão et al. 2003). On the other hand most authors do not quantify a severity code. Some investigators only report on patients who have severe CS. They believe that almost all patients develop mild CS after sympathectomy (Ueyama et al., 2004). But also climate plays a decisive role (Lyra et al., 2008). High rates of CS are mostly found in studies of countries with warmer temperatures and humid weather (X. Li et al., 2008). Surgical technique also seems to influence the risk of CS: The lower the level of division and the smaller the extent of sympathectomy, the lower the incidence of CS (Dewey et al., 2006). Treatment options for severe CS are limited: Some investigators try local injection of botulinum toxin in areas where CS is the most severe (Bechara et al., 2006), others use the clamping method with moderate success. At the end of the nineties, Telaranta successfully performed reconstruction with nerve graft by open thoracotomy for a patient suffering from severe CS (Telaranta, 1998). But it is an individual case and a complex procedure. It should be able to avoid CS, because severity does not change over time in 70% of cases (Dumont, 2008). In Taiwan patients suffering from serious CS have already formed a support group based on an internet discussion forum to request the government to take this problem seriously (Hsu & Y.C. Li, 2005). Therefore surgeons are searching for preoperative measurements to determinate postsympathectomy CS. In 2008 Miller and colleagues developed a new technique of a temporary thoracoscopic sympathetic block of the nerve with a local anesthetic that can hopefully predict severity of postoperative CS (Miller & Force, 2008).

Gustatory sweating (GS) is defined as facial sweating when eating certain foods particularly spicy or acidic food (Licht & Pilegaard, 2006) (Fig. 8). This phenomenon has no real
explanation, the pathophysiology may be quite complex (Licht et al., 2005). GS is less commonly reported than CS. The reported incidence of GS varies considerably from 0-38% (Dumont, 2008). Except one study published by Licht and Pilegaard in 2006, which analyses the relation between extent of sympathectomy, primary localisation of hyperhidrosis and the incidence of GS (Licht & Pilegaard, 2006), there are only a few investigators dealing with this issue. This occurrence is probably not estimated as very troublesome, both by surgeons and by patients. Furthermore triggers can be easily avoided by patients. Thus treatment options for GS including topical or systemic medications and the injection of botulinumtoxine are rarely performed (Eckardt & Kuettner, 2003).

Fig. 8. Patient suffering from gustatory sweating

Due to unforeseeable and unacceptable complications and unavoidable side effects, careful patient selection is important for surgery. Patients should be fully informed before they decide on surgical treatment (Dumont, 2008).

4.2.1.3 Postoperative results and patient satisfaction

Literature suggests: Endoscopic thoracic sympathectomy is a safe and effective therapeutic strategy in patients suffering from severe primary hyperhidrosis with excellent results and high rates of patient satisfaction (Henteleff & Kalavrouziotis, 2008). Postoperative results seem to depend more on severity and primary localisation of hyperhidrosis than on surgical technique: Best results can be achieved in patients with severe palmar hyperhidrosis (Baumgartner & Konecný, 2007). Patients with isolated axillary hyperhidrosis do not benefit sufficiently from sympathectomy (Gossot et al., 2003; Herbst et al., 1994). One possible explanation for the lower success rate may be that there is a combination of eccrine and apocrine sweat glands in the axilla. The eccrine sweat glands are innervated by sympathetic fibres, but the apocrine glands respond primarily to epinephrine. They are not blocked by sympathectomy and continue to function (Licht et al., 2005; Reisfeld et al., 2002). Therefore local surgical axillary procedures should be recommended as first-line therapy. As already mentioned, isolated plantar hyperhidrosis should be treated by endoscopic lumbar extraperitoneal sympathectomy. Individual reports with positive experiences already exist (Wörle et al., 2007). Also patients with facial hyperhidrosis or blushing do not universally and overwhelmingly benefit by sympathectomy, a case-by-case evaluation is required (Baumgartner, 2008). But patients with severe hyperhidrosis presenting...
for surgery mostly suffer from combined site hyperhidrosis (Eisenach et al., 2005). Reisfeld requests to establish indication for surgical therapy carefully: Thoracoscopic sympathectomy should only be performed in patients with severe palmar hyperhidrosis, other localisations should only be treated that way if combined with palmar site (Reisfeld et al., 2002). Short-term studies on sympathectomy can be detected frequently, they continuously present great outcome depending on primary localisation. But unsatisfactory immediate results can occasionally be detected (de Campos et al., 2003). Causes for persistent postoperative sweating are inadequate knowledge and orientation of the surgeon or unrecognised variances of anatomic structures (D.H. Kim et al., 2005; Reisfeld et al., 2002; Yoon et al., 1999) including Kuntz nerve, a communicating sympathetic ramus crossing the second rib (Chung et al., 2002) (Fig. 9). Therefore some authors recommend extension of the sympathectomy line to about three or five centimetres lateral to the sympathetic chain by coagulating the surface of the corresponding rib, a method first described by Linder and colleagues in 1994 (Linder et al., 1994). Adequacy of sympathectomy is also tried to be detected by perioperative use of monitoring device like measuring skin surface temperature or plethysmographic blood flow (Lee et al., 1999; Yoon et al., 1999).

Fig. 9. Anatomy of Kuntz nerve (INK= intrathoracic nerve of Kuntz) and rami communicantes (arrowheads)

Long-term outcomes of more than 10 years are rarely reported (Zacherl et al., 1998). Investigations show that unfortunately results of sympathectomy deteriorate with time (T.S. Lin & Fang, 1999; Walles et al., 2008). This recurrent postoperative sweating may be due to local nerve regeneration but has not yet been proven (Lee et al., 1999).
Today some surgeons offer redo-operations in cause of persistent or recurrent postoperative sweating, also called re-sympathectomies. Usually these procedures can be reperformed by videothoracoscopy, severe pleural adhesions requiring thoracotomy are rarely documented (D.H. Kim et al., 2005; T.S. Lin, 2001). However there is a lack of long-term results too. Lots of investigators use patient satisfaction as a common parameter to describe overall effectiveness of sympathectomy. Some studies reveal that patient dissatisfaction is primarily associated with persistence or recurrence of preoperative symptoms and to a lesser extent with incidence of side effects (Kwong et al., 2005; Walles et al., 2008). But assessment of surgical result using the conventional method patient satisfaction is imprecise and inaccurate (Leão et al., 2003). Main problem in requesting patient satisfaction, mostly based on patients self-report in questionnaires postoperatively, is subjectivity (Shargall et al., 2008). In some series several quality-of-life measures for assessment of improvements in daily life after treatment of hyperhidrosis are already used to get a more objective point of view (de Campos et al., 2003; Tetteh et al., 2009) (Table 7). In combination with quantitative measurements, which are often not practicable in the clinical setting, a precise evaluation of the effectiveness of sympathectomy would be possible (Cetindag et al., 2008).

<table>
<thead>
<tr>
<th>General tools</th>
<th>Specific hyperhidrosis tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Illness Intrusive Rating Scale (IIRS)</td>
<td>- Hyperhidrosis Impact Questionnaire (HHIQ)</td>
</tr>
<tr>
<td>- Medical Outcomes Trust Short Form</td>
<td>- Hyperhidrosis Disease Severity Scale (HDSS)</td>
</tr>
<tr>
<td>12 or 36 (SF-12 or SF-36)</td>
<td></td>
</tr>
<tr>
<td>- State-Trait Anxiety Inventory (STAI)</td>
<td></td>
</tr>
<tr>
<td>- Symptom Distress Scale (SDS)</td>
<td></td>
</tr>
<tr>
<td>- Dermatology Life Quality Index (DLQI)</td>
<td></td>
</tr>
</tbody>
</table>

Table 7. Several tools used for Quality-of-life assessment

5. Conclusion

In literature database hundreds of citations can be identified concerning treatment of primary upper limb hyperhidrosis by thoracic sympathectomy and nearly all investigators suggest that patients can significantly benefit from this procedure. But fundamental limitations arise: the great majority of currently available studies are retrospective single-centre series. The heterogeneity of study population, the inconsistent definition and terminology of the word sympathectomy, the variety of surgical techniques with the optimal procedure remaining elusive and the lack of uniform measures at both the exposure and outcome levels make comparison and generalisability of these series quite impossible. In future, in addition to standardization, both long-term studies with large numbers of patients and multicentre randomised controlled trials are mandatory to clearly define the role of sympathectomy in treatment of primary hyperhidrosis (Henteleff & Kalavrouziotis, 2008).

6. References


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