Lecture N. 8

Thyroid Surgery- Goiter

In the 1960s, with the release of the fifth Italian edition of E. Kaufmann's, prestigious *Trattato di Anatomia Patologica Speciale* (Vallardi), edited by Alfonso Giordano, the renown Milanese pathologist,
I was amazed (puzzled, to say the least) to read *struma* in the feminine gender: for years I had been taught the term was masculine (*lo struma*). Even today, many others (like me) have a predilection for the latter. I satisfied my urge to probe the issue a bit, and found conflicting views. Classic anatomic pathology texts, such as that by A. Pepere - A. Businco, or by G. Lanza, as well as more modern volumes like N. Massaioli's *Chirurgia* (Minerva Medica) and R. Dionigi's effort for Masson Editors, not to mention the Italian edition of Churchill's Medical Dictionary, the term *struma* is used in the masculine. Italian dictionaries offer little help, seeing that *Palazzi's* 2nd Edition classifies it in the masculine, while *Zingarelli* (2002) defines it as a feminine term. Thus, we need to trace the term to its root: in Latin we find *struma-strumae*, hence, a feminine use.
The whole exercise is, we could say, futile. Perhaps both genders can be used. Suffice it to say, however, that the current, above all surgical, use of the term is in the masculine: *lo struma*.
While I was on the subject, I looked into the etymological origins of the word, about which I had already heard (somewhat confusingly) one interpretation that I will cite here, without, however, taking any responsibility. In the Balkans, precisely in Macedonia, the river *Struma* flows. In Turkish, it goes by the name of *Kara-su*, the equivalent of Black River. To the ancient Greeks it was *Στρύμον*, and to the Romans *Sinus Strimonicus*. Well, legend has it that goiters were particularly prevalent along the banks of this river: hence, the term *struma*.

Putting this diversion aside and getting our feet back on the ground, we can say that *struma* is synonymous with *gozzo* (goiter) and, as such, carries the general meaning of enlargement of the thyroid, but also denotes a long enduring benign hyper- or dysplasia of the gland.

A goiter may be parenchymatous (diffuse) or nodular (uni- or multinodular): the former generally does not require surgery, and as such goes beyond the focus of this chapter. Multinodular goiter is the most frequent manifestation: it is estimated that nearly 5% of the world's population is affected with a multinodular goiter, above all in countries of endemic iodine insufficiency.

Indeed, the most common cause of goiters worldwide is iodine deficiency (other causes are the ingestion of goitrogens and thyroid hormone formation defects). In the U.S., where iodine intake far exceeds requirements, prevalence of the condition ranges from 0.4% in Massachusetts to 7.2% in California. In Italy, the prevalence of goiters is high due to the insufficient dietary intake of iodine throughout the country, and

especially in regions such as the Northeast (37-59%), mountainous areas of Piemonte and Liguria (16%), Abruzzo and Molise (47-55%), Campania (19-81%), and still others.

An iodine deficiency is easily corrected: the addition of iodine at the dose of 100-150 μg/die leads to a reduction - tantamount to disappearance - in the prevalence of goiters in the world's younger generations. A number of controlled studies corroborate this assertion. And, indeed, years ago an organization, the *International Council of Iodine Deficiency Disorders* (ICIDD) was established and given the remit of endorsing and coordinating adequate iodine prophylaxis programs. The United Nations and its sister organization UNICEF enlisted the commitment of 130 nations, including Italy, to overcome iodine deficiency and, consequently, goiter. In 1985 the Italian government created the *National Committee for the Prevention of Goiter*, which adopts as its guiding method the one that had thus far proved the easiest and least costly: the addition of iodine to table salt (30 mg of iodine/Kg of salt). Despite the production and sale of iodized table salt now being prescribed by law (see below), the consumption of this *optional*, corrected, difficult to find and poorly publicized comestible has been rather limited over the last 20 years. There was also a fair share of skepticism of many in the general public - including physicians - stemming from erroneously spawn doubts regarding its harmlessness.

The fact is that where iodine deficient diets have been corrected, in Nordic countries and Switzerland, for instance, goiters have for all practical purposes disappeared in younger generations. The same can be said for Italy, where regions such as Alto-Adige and some areas of Tuscany and Sicily have witnessed a sharp drop in prevalence. At the same time, in other nations and in other Italian regions where prophylaxis has not been implemented, goiters still remain a significant health care burden.

Noteworthy is that, with the disappearance or reduced frequency of the condition, other thyroid disorders, including cancer, have been subject to sharp decreases.

The issue of iodine prophylaxis resurfaced in Italy, recently, with the emanation of a bill in 2002 that was finally passed into law by the Italian Parliament in 2005:

Rome - the Council of Ministers of August 30, 2002 approved a bill introducing measures aimed at the prevention of endemic goiter and other iodine-deficiency disorders. The order in essence would guarantee the availability iodine-enriched table salt in retail locations and the sale of un-enriched salt only on specific request. The bill also foresees the issue by the Ministry of Health of a poster to be displayed by retailers informing consumers on the benefits of iodine prophylaxis. Restaurants would also be required to use iodine-enriched table salt and to make it available to customers. The legislative measure would also be extended to the use of iodine-enriched salt as an ingredient in the processing and preservation of food products.

Rome - The House of Representatives definitively passed into law a government-endorsed bill establishing measures to prevent endemic goiter and other iodine-deficiency disorders. Iodine deficiency, of which endemic goiter is the most frequent manifestation, is one of the world's major public health problems, as evinced by the 1994 Annual Report of the World Health Organization (WHO). In Italy no fewer than 5 million people are affected with endemic goiter, most of whom dwell in rural (particularly mountainous and hilly) regions; inhabitants of coastal and urban areas, however, also have a dietary iodine intake which is often lower than requirements. As recommended by the WHO, the simplest and most effective means to achieve iodine prophylaxis in wealthy nations is to substitute normal table salt with iodine-enriched salt. For the sake of continuity, it is also important to complement this measure with adequate consumer-awareness campaigns. Not relying only on appropriate nutrition education actions to alert consumers, health care workers and mass media to iodine prophylaxis and its effectiveness at preventing endemic goiter, the new law introduces

measures to more decisively combat this serious public health burden. Available data show that the consumption of iodine-enriched table salt - currently voluntary and without incentives - is lower than the overall consumption of table salt in general. The law establishes therefore that retail locations of table salt must stock both common and iodine-enriched varieties, and that the sale of the former be made only on the customer's specific request. The law also foresees the design and circulation by the Ministry of Health of a poster to be displayed by retailers informing consumers on the benefits of iodine prophylaxis. Restaurants would also be required to use iodine-enriched table salt and to make it available to customers. The law also includes the possibility to use of iodine-enriched salt as an ingredient in the processing and preservation of food products, and foresees specific labeling for iodine-enriched table salt as well as on products using it as an ingredient. Finally, the Ministry of Health may endorse targeted information campaigns in order to promote thorough public awareness of the disorders caused by iodine deficiency and of the benefits of prophylaxis based on iodine-enriched salt.

In texts we still find a goiter classified as endemic, sporadic or epidemic. To me, this distinction seems somewhat dated: I believe it would be best to say that the condition has a *geographic* distribution, wherever iodine intake is insufficient.

We have said that a goiter is a volumetric enlargement of the thyroid; in its nodular form clinical signs may vary. The classic feature, immortalized in countless works of art and folklore, is the deformation of the neck. Here, to the front, a tumefaction (often voluminous) causes disfigurement, which sometimes spreads uniformly from side-to-side, and sometimes affects only one or the other side. The growth can be rather unsightly: this is much rarer these days, however, since one's personal appearance is more of a priority and, above all, because modern thyroid surgery is much less risky than it once was. There are exceptions, though, judging by the goiters I have extracted in the recent past that weighed from 500 grams to one kilogram.

One likelihood, which in my view should not be overlooked, is the patient presenting to his practitioner and claiming to have had a goiter for many years, but which at a certain point vanished (*spontaneous healing…!*). This is generally the case of an elderly patient complaining of shortness of breath, frequent flushing of the face, and swelling of the jugular veins. Physical examination of the subhyoid region in the front of the neck, however, reveals no tumefaction resembling a goiter: what can be seen, on the other hand, are dilated veins creeping in the anterior thoracic surface (Fig. 3), and Pemberton's maneuver (having a patient elevate his arms) prompts cyanosis of the face and swelling of the jugular veins. Radiological imaging will confirm the diagnosis of an endothoracic (cervico-mediastinal; substernal) goiter. This descent of the mass into the thoracic cavity may occur above all in the elderly, when kyphosis of the cervical-thoracic spinal column presses the cricoid cartilage towards the throat, reducing the confines normally occupied by the gland and thus pushing the goiter in the mediastinum; the entire process is facilitated by the favorable cleavage plane and by intrathoracic pressure. Cervico-mediastinal goiter is generally defined as a thyroid mass with at least two-thirds of its volume located below the thoracic inlet.

Other manifestations include the palpable nodule (usually self-detected by the patient), and that revealed by ultrasound (US) examination, which often turns out to be an "*incidentaloma*".

We have thus far cited the forms of euthyroid goiters, that is growths normal thyroid function. Other overactive (hyperthyroid) forms will be described elsewhere.

Physical examination of the thyroid and its expansion, beyond being visual exploration, is also and above all for purposes of palpation. A normal thyroid is not palpable: when it is, it means that a pathological process is underway. The thyroid is in-line with the larynx, and thus rises with swallowing. This maneuver allows distinguishing between a true tumefaction, like a struma, and other growths unrelated to the thyroid, e.g., lymph nodes and other formations in contiguous tissues.

Two examples from my experience provide good illustration of this. A patient with a large tumefaction in the neck was referred to me for a strumectomy; the mass did not move on swallowing, because the patient had a large lipoma occupying the prethyroid area. Still another case presented a large mediastinal teratoma emerging at the jugular vein and indeed resembling a goiter, but without the trademark movement of thyroid growth.

With goiters known as *plongeant*, when the patient is asked to swallow, the lower portion of the goiter that had invaded the thoracic cavity is raised and the limits of the mass can be defined. As such, any further endothoracic expansion can be ruled out.

The goiter may become complicated, not only because of its size, but also for its expansion and its relation to the patient's age. Growth concentric with the trachea may cause airway compression: textbooks speak often of *tracheomalacia* arising from such circumstances. In my and others' experience, however, this is a truly exceptional event. Of course, a compressed trachea can give rise to dyspnea and, if flaccid, after strumectomy and no longer sustained by the thyroid mass, may collapse and lead to serious respiratory problems.

If the goiter is monolateral (or at least shows greater growth to one side), the trachea and the laryngotracheal axis will gradually be displaced towards the side opposite of the glandular expansion (Fig.s 1 and 2). This event, too, may cause dyspnea. Less frequently, displacement of the esophagus may give rise to dysphagia.

Dysphonia due to compromission of the inferior/recurrent laryngeal nerves, normally monolaterally, is generally the result of an infiltrating neoplastic process. Pressure alone, even from a voluminous goiter, leads to this complication only rarely, since the nerve structure is merely moved and not directly affected, as with cancer. There are, nevertheless, exceptions to this general rule, as can be seen with cervico-mediastinal goiters that become tightly trapped in the upper thoracic inlet.

Two cases drawn from my experience are telling.

The first regards an 82-year-old woman that presented with hoarseness of the voice (EN&T examination revealed hypomobility of the left vocal cord), prevalent dysphagia for liquids (aryepiglottic dysfunction) and an episode interpreted as transient ischemic attack (T.I.A.). Physical examination revealed a relevant tracheal deviation to the right, and by palpation the upper portion of a goiter expanding into the mediastinum is detected. Ultrasound tomography and other radiological tests confirm the diagnosis of a large cervico-

mediastinal goiter with lateral displacement, compression (confirmed by Doppler US) of the left carotid artery and severe dislocation to the right of the laryngotracheal axis. Surgery reveals the degree and extent of mediastinal expansion of the goiter, which fits tightly (*champagne cork*) in the upper thoracic inlet and leads to intensive compressive phenomena in adjacent structures. These compressive symptoms, above all of the nerves, gradually resolve following surgery.

The second case regards a 70-year-old male with a negative medical history, except for a small goiter present for some time. At a certain point the patient suddenly became aphonic. EN&T examination showed paralysis of the right vocal cord. Radiography and US tomography confirm the presence of a goiter situated prevalently to the right with slight tracheal deviation to the left and evidence of some enlarged lymph nodes to the right side. Surgery and perioperative histological cryostat sectioning demonstrate the neoplastic nature of the goiter (infiltrating anaplastic carcinoma with lymph node metastasis in the homolateral recurrent chain). Total thyroidectomy with lymphadenectomy followed by radio- and chemotherapy, however, failed, and the patient died a few months thereafter from brain metastases.

These cases are paradigmatic of the major complications seen with a goiter:

- Cervico-mediastinal migration/expansion;
- Compression phenomena (laryngotracheal, vessels, nerves)
- Neoplastic transformation.

With regard to this latter, we should not overlook the high risk of degeneration of a preexisting goiter - and its relatively frequent neoplastic, often malignant, transformation - in patients over 60 to 70 years of age (as in the case above). Hence, the recommendation for the complete excision of the struma in due time without waiting for age-related complications, of which cancer is undeniably the most dramatic, to set in.

Two further complications are relatively frequent: intraparenchymal bleeding and secondary thyrotoxicosis (*Basedow's* goiter, or better yet, toxic multinodular goiter - Plummer's disease). The first may arise more frequently in cystic goiters (colloid-cystic goiter), thus leading to what is defined as *encystic hemorrhage*. In these cases the goiter grows rapidly, ultimately causing increasingly serious dyspnea in the patient and requiring emergency care, such as tracheal intubation, tracheotomy and surgical intervention. Two luck cases in my experience regard two patients awaiting surgery. The patient experiences sudden neck tension, pain and a sense of suffocation: it takes little to correctly interpret these signs, and the patient is swiftly delivered to the operating room, intubated and operated. The goiter looks unusual, resembling more a large hematoma. This event may arise even in small cysts, and has been known to occur in presumably normal thyroid glands.

The multinodular toxic goiter will be discussed in the chapter on thyrotoxicosis.

The diagnosis of a goiter is made on basis of patient history, physical examination and a few instrumental examinations. These generally follow a well prescribed protocols.

The first and most important of these is US tomography. This examination yields useful information about the goiter's size, its single or multiple nature of nodes, the structure of its formation(s) (cystic, solid, mixed, calcified), vascularization, growth features, and possible compressive phenomena.
Simultaneously, thyroid function is measured via screening for thyroid hormones (FT3, FT4, thyroglobulin) and TSH. In case of hyperfunction, detected principally by a decrease in TSH, scintigraphy (where hyper-intake areas are "warm" and hypo- or no intake areas are "cold") is indicated. Standard radiological examination (antero-posterior and lateral) adequately reveals details about compressive phenomena, namely tracheal (displacement of the trachea) and about possible mediastinal migration (Fig.s 1 and 2). Only in difficult-to-interpret cases are CT scan (Fig. 1) or MRI helpful. Care must be taken in reading these findings, since they often yield data that tend to overestimate the extension and complexity of the struma, especially with mediastinal expansion, and thereby insinuate operative difficulties that ultimately are not borne out by the facts.
Still other tests are useful for differential diagnosis. These include: thyroid antibodies (revealing an autoimmune component) and calcitonin (to differentiate from medullary carcinoma). In case of suspected malignancy, e.g., for a solitary nodule that is solid (US tomography) and cold (scintigraphy), cytological sampling by fine-needle aspiration (FNA) is recommended. For previous thyroid surgery patients, the second opinion of an EN&T specialist for examination of the vocal cords is advisable.

On the subject of surgery of the goiter, endocrinologists and surgeons diverge on indications. The former hold that *"surgery plays a rather secondary role in the treatment of goiter"*, to be resorted to only in the presence of relevant goiter-induced consequences (size, compressive phenomena, endothoracic development, etc.). This far, surgeons clearly agree.

These latter, nevertheless, often at odds with endocrinologists, propose other indications for thyroid goiter surgery based on their observations of goiters developing at a relatively young age and which were treated exclusively by medical rather than surgical means. Such sequelae, which often may be severe, were already mentioned in the paragraph on complications. Again, the most serious are undoubtedly malignant neoplastic transformation in patients over 60 to 70 years of age and cervico-mediastinal migration, two events that, also because of advanced age, jeopardize surgical intervention.
Only when a nodular goiter is small in size, generally less than 1 centimeter in diameter, do I, as others (and not only surgeons) opt for a more conservative approach. The goiter should, nonetheless, be monitored regularly by US, and surgical treatment undertaken at the first signs of growth.

Before delving into surgical details, a brief review of the nomenclature is requisite.

- Enucleation or partial lobe resection;
- Lobectomy (right or left);

- Isthmusectomy;
- Lobectomy with isthmusectomy;
- Subtotal thyroidectomy (STT) = residual gland equal to normal parenchymal volume;
- Near Total Thyroidectomy (NTT) = residual gland < 10 grams;
- Total Thyroidectomy (TT).

Surgical therapy for goiter is, in essence, the strumectomy, the removal of the goiter. While this may seem to be a claim to be taken for granted, we will see below why it is not.

Our increased understanding of the physiopathology and natural history of disorders affecting endocrine glands has lead to the concept that surgery must be *functional*, i.e., it must resolve the disease but must leave intact as much parenchyme as possible. Because a euthyroid goiter - the subject of our discussion - is a benign disease, a general principal of surgery must be applied beyond the consideration mentioned above, namely, that the desired result must be achieved with the least possible risk of mortality and morbidity.

This is why our School, like many others, prefers as first line treatment for goiter a partial thyroidectomy and never total in principle as advocated by endocrinology groups and carried out by some surgery centers. The reasons for this choice, TT, are founded on three suppositions:

1. The whole thyroid gland, not only the areas affected by the goiter, is diseased;
2. As such, a high degree of relapse after only STT or NTT is foreseeable;
3. Hormone substitute treatment is easier.

These arguments are not confirmed by our clinical findings, however, in consideration of the above-mentioned concepts. Out of nearly 7,000 thyroid surgeries performed by us, benign euthyroid goiters constituted the majority of cases: in 1996 we published the results of our experience, reviewing goiter patients treated and followed up for time spans ranging from 10 to 20 years (Ann.Ital.Chir., LXVII, 3: 341-345, 1996). The same findings readily reflect outcomes achieved since then, and confirm the validity of conservative goiter therapy. The notions underlying this success will be discussed below.

Insulin Growth Factor (IGF) is known, together with other growth factors such as Epidermal Growth Factor (EGF) and various immunoglobulins and cytokines, to act as a causative agent underpinning the dysplastic development of a goiter. The IGF content in nodular tissue and in apparently healthy tissue in multinodular goiters varies greatly, showing much higher values in the former, thereby suggesting the possible presence of cell clones with different dysplastic potential. In essence, such evidence would disprove the notion that the entire gland, including its extranodular portions, is diseased. The low number of relapsing goiters after NTT or STT seen in our and others' cohorts readily substantiates this claim. In this regard, it is best to bear in mind what is meant from a surgical standpoint by relapsing goiter, and that is a resumption of disease warranting re-operation. The residual thyroid gland, which in our experience generally consisted of a posterior glandular wall whose thickness varied depending on whether an STT or an NTT had been performed, presents a cut anterior surface; subjected to the regenerative process that the gland is undergoing (typical of all parenchymal organs, e.g., the liver), the wall develops projecting pseudonodular growths,

which, on US may be mistaken for relapsing nodules by the inexpert radiologist. This phenomenon can frequently alarm patients, as well as their attending physicians. In other cases, the nodules detected by US, because of their structure and vascularization, will permit the experienced radiologist to correctly identify them as relapsing disease. These are in the vast majority of cases small, and respond adequately to the treatment options mentioned above for nodules under 1 cm. in diameter (i.e., observation and hormone therapy, if necessary). In our (and others') experience only a very slight percentage of relapsing nodules ever reached a volume and extension that required re-operation (4.9% after years of follow up).

The third point under discussion regards hormone therapy. Advocates of TT outright hold that TSH-suppressive therapy must follow NTT or STT on the conviction that it protects against recurrence. This is objectionable for a number of reasons, the first of which is the finding that relapse may occur in patients with normal or even suppressed TSH levels, also because (as already mentioned) TSH may not be the only goitrogenic factor. Here again, our experience corroborates this possibility, since relapsing disease in our series of operated patients occurred regardless of whether they were medically treated or not.

As we will see below, conservative surgical procedures, like those carried out by us, are modulated according to the goiter's extension. As a result, a nearly normal amount of parenchyma may be maintained, as may a relatively exiguous residual gland (after the most advanced NTT). Postoperatively, therefore, patients will present conditions of either euthyroidism or hypothyroidism, and hormone therapy will have a substitutive function as is seen in TT. The administration of levothyroxine must consequently be tailored to the residual functional needs. Telling of this is the fairly commonplace observation of a progressive recovery of glandular function to an extent that allows gradually reducing or even suspending hormone replacement.
Total thyroidectomy necessitates *lifetime* administration of levothyroxine: *athyreosis*, the absence of thyroid function, is a condition with a serious prognosis, both *quoad valetudinem* and *quoad vitam*. The necessary high doses are not always free of adverse effects, above all in the elderly; not all countries maintain stocks of the drug; the patient disdains his "slavery" to the pill; health care priorities often overlook the lifelong surveillance and hormone monitoring required in TT patients; levothyroxine replacement does not protect against other thyroid (e.g., calcitonin) functions.

In essence, these are the reasons why we and many others do not acknowledge thyroidectomy as first-line treatment for benign diseases of the thyroid gland, beginning with a goiter.

What follows is a description of the surgical technique used by us for the treatment of a goiter.
General anesthesia with tracheal intubation; patient is placed in supine position with the head -neck stretched; incision at the base of the neck (Kocher's collar) 1 cm. above the jugular, along a crease in the neck ("*Venus' collar*", if present), that unites the anterior margins of the sternocleidomastoid muscles (this long, ample access is strongly advised in order to achieve a optimum view of the operating field and to avoid stretching of the margins of the skin, a trauma which adversely impacts a successful cosmetic result); incision of the platysma muscle; transverse section of the prethyroid, sternohyoid and sterno-thyroid muscles

(advisable for complete control of the gland and contiguous formations); inspection and palpation of the gland (US probe?) and of adjacent extrathyroid structures; assessment of mediastinal extension (Fig.s 4 and 5a); identification, mobilization and excision (whole, with the rest of the gland) of the pyramidal lobe (Fig 5b), if present; preparation of the superior margins of the gland; isolation and interruption of the superior thyroid arteries, paying close attention to the superior laryngeal nerve; identification of the inferior thyroid artery, which is never interrupted (bearing in mind parathyroid vascularization), but if necessary passed underneath with a rubber loop; this maneuver facilitates identification of the inferior/recurrent nerve (Fig. 6B) and localization of the inferior parathyroid; with TT the recurrent nerve is also passed under by a soft loop that expedites its localization and completes visual control of the nerve up to its laryngeal end; isolation of the parathyroid glands at their delicate antero-superior connections with the thyroid capsule, carefully preserving the vascular inferior-lateral peduncles; preparation of the gland's inferior margins; gradual extraction of the mediastinal extension, if present, by delicate and progressive digital and/or bidigital maneuvers, avoiding at all cost traction with transfixed strings (only rarely was it necessary to resort to sternotomy). At this point glandular demolition is performed: STT, NTT (Fig.s 6a, 6c) or TT, according to indications resulting from US tomography, preoperative exploration and histological examination of cryostat sections. Bipolar and/or harmonic instrumentation may prove useful, except in lateral areas of the gland (i.e., due to risk of recurrent laryngeal nerve and/or parathyroid gland damage). The operation is completed with tubular suction drainage, suture of the prethyroid muscles, of the platysma muscle (using absorbable sutures) and the skin. The surgical team moves away from the operating table only after removal of the intubation and the resumption of spontaneous respiration. At 24 hours after the operation it is normally possible to remove the drainage tube (which, obviously, was left in place if necessary) and to begin removing cutaneous sutures (which is fully accomplished at 48 hours after surgery). Discharge usually comes about on the 3rd postoperative day. On or around the 20th postoperative day, levels of FT3, FT4 and TSH are monitored to ascertain the need for hormone replacement therapy, if necessary.

The major possible complications resulting from surgery for uni- or multinodular goiter basically regard bleeding and disorders of parathyroid function or of the laryngeal nerves.

The hemorrhagic episode most feared by surgeons occurs in the hours immediately following the operation, usually as the result of causes (namely, cough, vomiting, psychomotorial excitement) that lead to sudden hypertension of neck vessels and possible dehiscence of hemostatic seals. If bleeding is massive, suction drainage is ineffective at removing blood from the residual thyroid cavity, which thus soon fills, leading to compression of the trachea and suffocation. Such a condition requires immediate emergency re-operation to achieve hemostasis. Events of this kind were seen only very rarely by us.

The main disturbance of parathyroid gland function entails a significant drop in serum calcium levels (hypocalcemia); patients who are hypocalcemic may present limb paresthesias (tingling) and, if serious, tetany ("*midwife's hand*", etc.). Two observational maneuvers allow detection of the disturbance, even if latent. The Chvostek sign is elicited by lightly tapping the facial nerve in the preauricular area and observing for contractions in the orbicular of the mouth and the buccinator muscles; the Trousseau sign by induction of

tetany through compression of the upper arm manually or by means of upon inflation of a blood pressure cuff. This complication may be transient and may disappear a few days following surgery; it may, however, be permanent and require treatment (calcium, vitamin D2 replacement). In our experience episodes of this kind were observed in 0.3% of patients, a rate in keeping with other reports. According to some authors (e.g., Chirurg., 74 (5):437-43, 2003), this complication develops more frequently after TT.

Laryngeal nerve defects must be distinguished into those affecting the superior and those the inferior recurrent nerve. The first, which finds little mention in the literature and was an exceptional event in our experience, is revealed by changes in the voice. The latter is more serious, entailing paralysis of one or both vocal cords. Monolateral paralysis may be accompanied by respiratory disorders (biphasic stridor, "cornage" and/or activation of complementary respiratory muscles and hollowing of the respiratory areas, "tirage") and dysphonia; with bilateral paralysis occlusion of the glottis compels emergency tracheotomy. Monolateral recurrent laryngeal nerve defects may be temporary, but may also be permanent: in our experience, this complication totaled 1.1% of cases, reflecting findings in the literature. With this event, too, the highest incidence rates reported in the literature are seen following TT.

These complications of thyroid surgery very often depend on particular technical difficulties entailed with surgical intervention, which in turn are usually related to disorders that make the recognition, isolation - and hence, preservation - of anatomic structures difficult: examples of potential events include possible dislocations of large goiters, inflammatory and neoplastic processes, as well as and scarring phenomena following previous surgery. Frequently, it is the operating surgeon's poor familiarity (resulting from an incomplete learning curve) with the neck's intricate anatomy that accounts for such events. Our group, expert in thyroid surgery, saw a significant number of patients already treated in other centers where such intervention was episodically performed; and indeed, these patients presented parathyroid and recurrent laryngeal nerve complications that exceeded average rates reported in the literature, namely 4.7 and 8.9%, respectively.

In the early 1990s, on the basis of the experience we were achieving with videothoracic and laparoscopic surgery, we devised a minimally invasive approach for thyroid surgery (Ann. Ital. Chir., LXVII, 4, 1996). We soon abandoned the procedure, however, as we were unconvinced about its safety and clinical effectiveness. Today, innovative technologies and dedicated instrumentation allow skilled surgical groups to perform such minimally invasive thyroid (and parathyroid) intervention in selected cases.

We have already mentioned the use, almost routinely with the same criteria, of preoperative fine-needle aspiration (FNA) and perioperative examination of cryostat histological samples in the case of suspected neoplasia (i.e., nodules that are isolated, single or few, or are solid on US tomography). Obviously, such scrutiny of all nodules in a multinodular goiter is not possible, and only postoperative analysis will be able to define histomorphological characteristics. At times, this examination (delayed with respect to surgical intervention) may reveal the presence of neoplasia arising among the many, often numerous, nodular formations making up the goiter; the lesion is generally well-differentiated, papillary in structure, localized

and with a size ranging from 10 to 15 mm. Such a finding constitutes a so-called "occult tumor", which will be the subject of a forthcoming lecture on thyroid cancer. In these instances, we usually consider surgery sufficient, as long as an STT was performed, with the patient subsequently undergoing scrupulous follow-up (especially by US) for the detection of possible neoplastic progression. Under the conditions mentioned above, an event of this kind never occurred in our series.

In conclusion, we can summarize by saying that a goiter is still a frequent disease for which surgical therapy is often indicated on the condition that:

- it shows signs, even benign, of growth;
- it causes compressive syndromes of adjacent delicate structures;
- it expands into the mediastinum, thereby compromising important intrathoracic structures;
- it is at-risk of neoplastic transformation;
- it presents cosmetic problems.

Surgical treatment of a nodular (uni- or multi-) goiter is thyroidectomy in one of its three modalities: subtotal (STT), near total (NTT) or total (TT). The risks and major complications of these surgical procedures include: postoperative bleeding, permanent hypoparathyroidism, permanent recurrent laryngeal nerve injury. These risks are minimized by the deliberate intraoperative visualization of anatomic structures (by means of ample surgical access) and by the experience of the surgeon.

Iconography
(Taken from the series of patients operated on c/o the Surgical Clinic, University of Genoa Medical School)

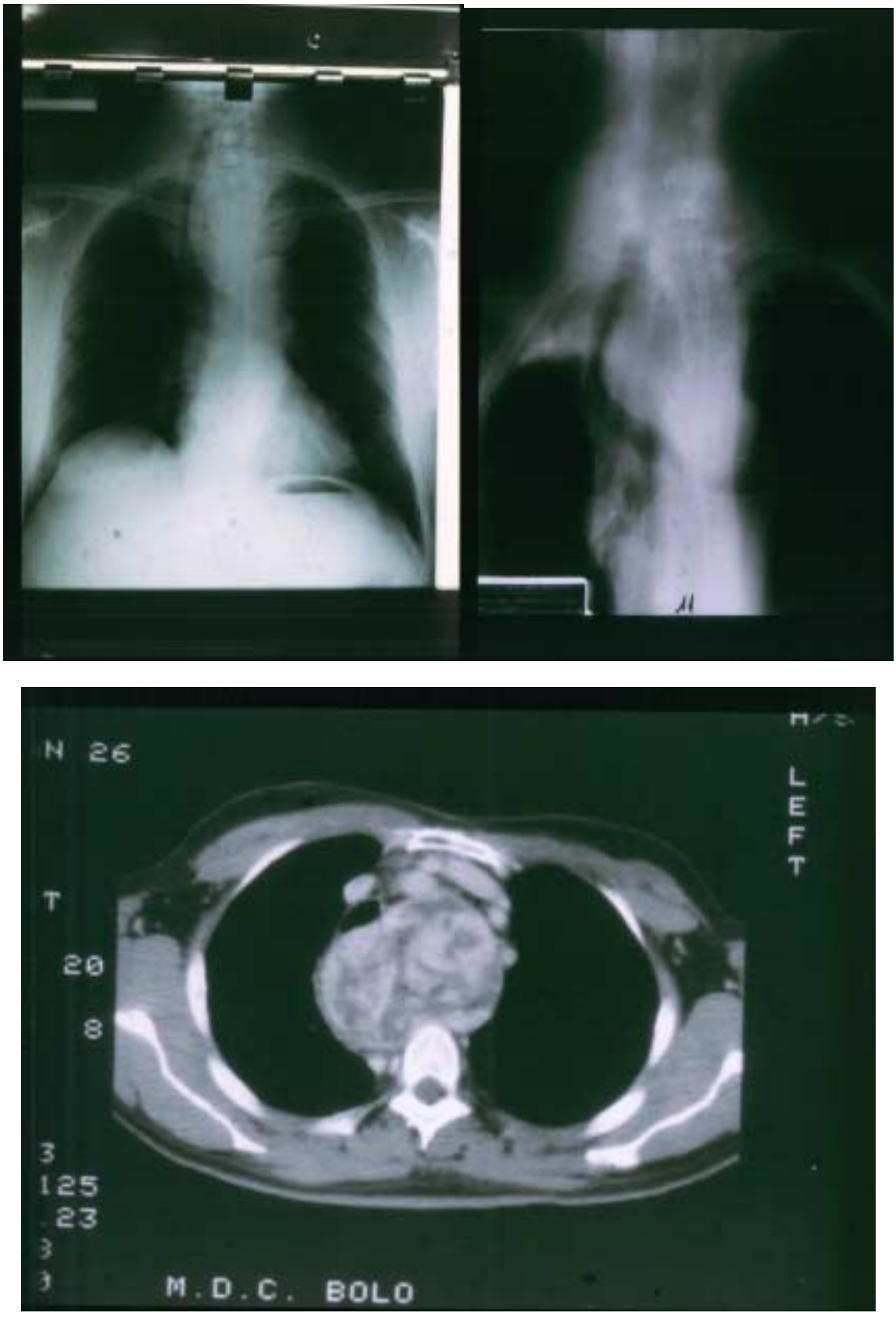


Fig. 1: Deviation to the right and compression of the trachea down to the bifurcation of a large intrathoracic cervico-mediastinal (substernal) goiter.

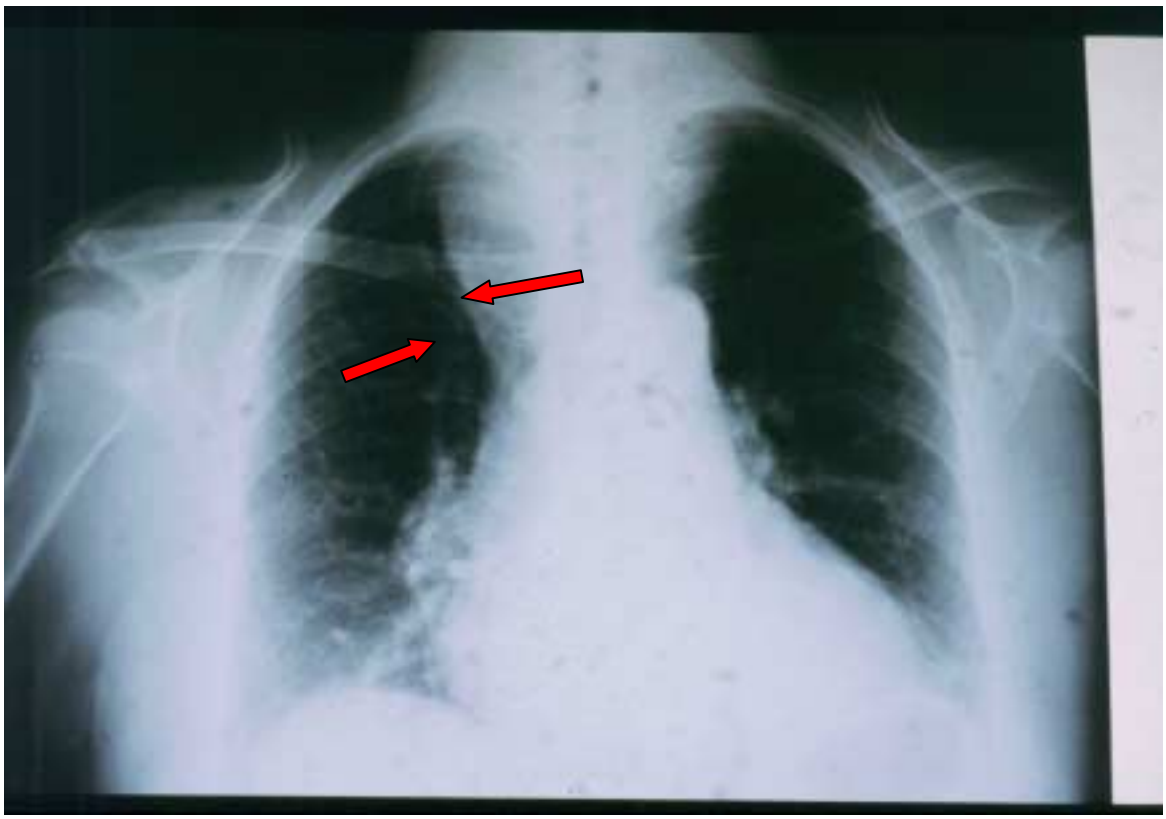


Fig. 2: Serious tracheal (↓)displacement and compression caused by a large goiter showing intrathoracic cervico-mediastinal (substernal) migration.

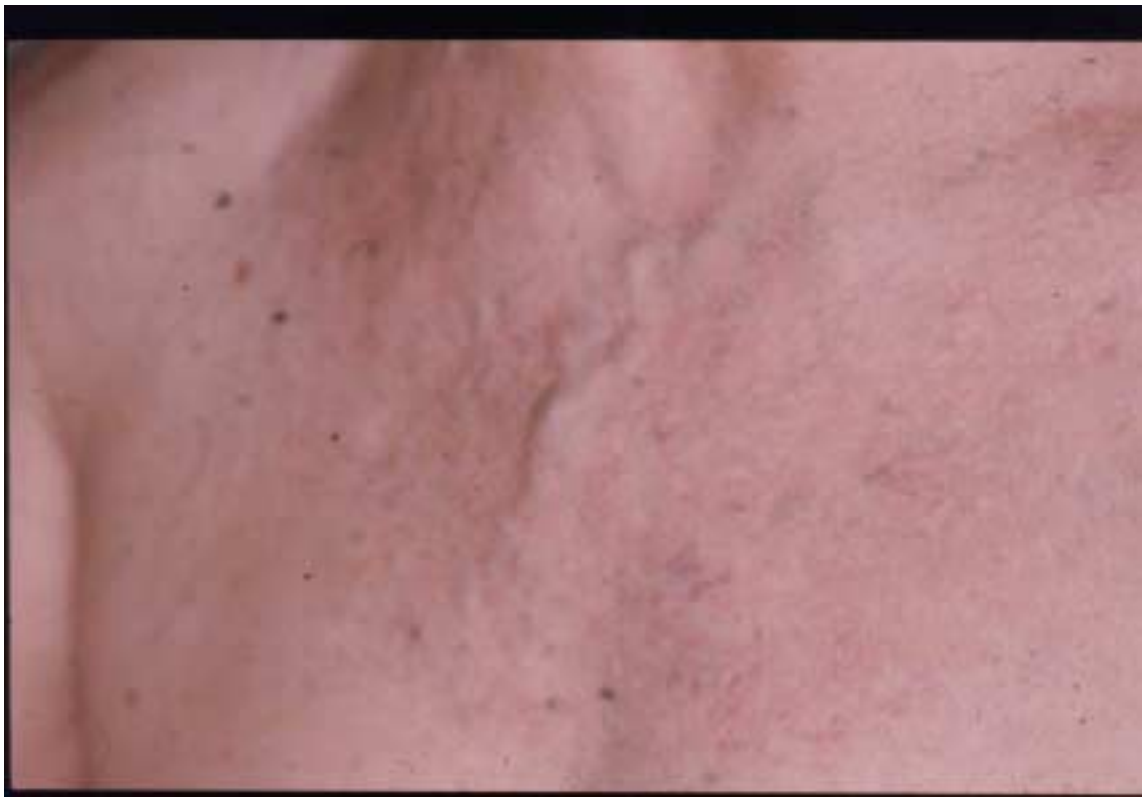


Fig. 3: Collateral subcutaneous venous circles arising from a mediastinal compressive syndrome caused by cervico-mediastinal (substernal) goiter.

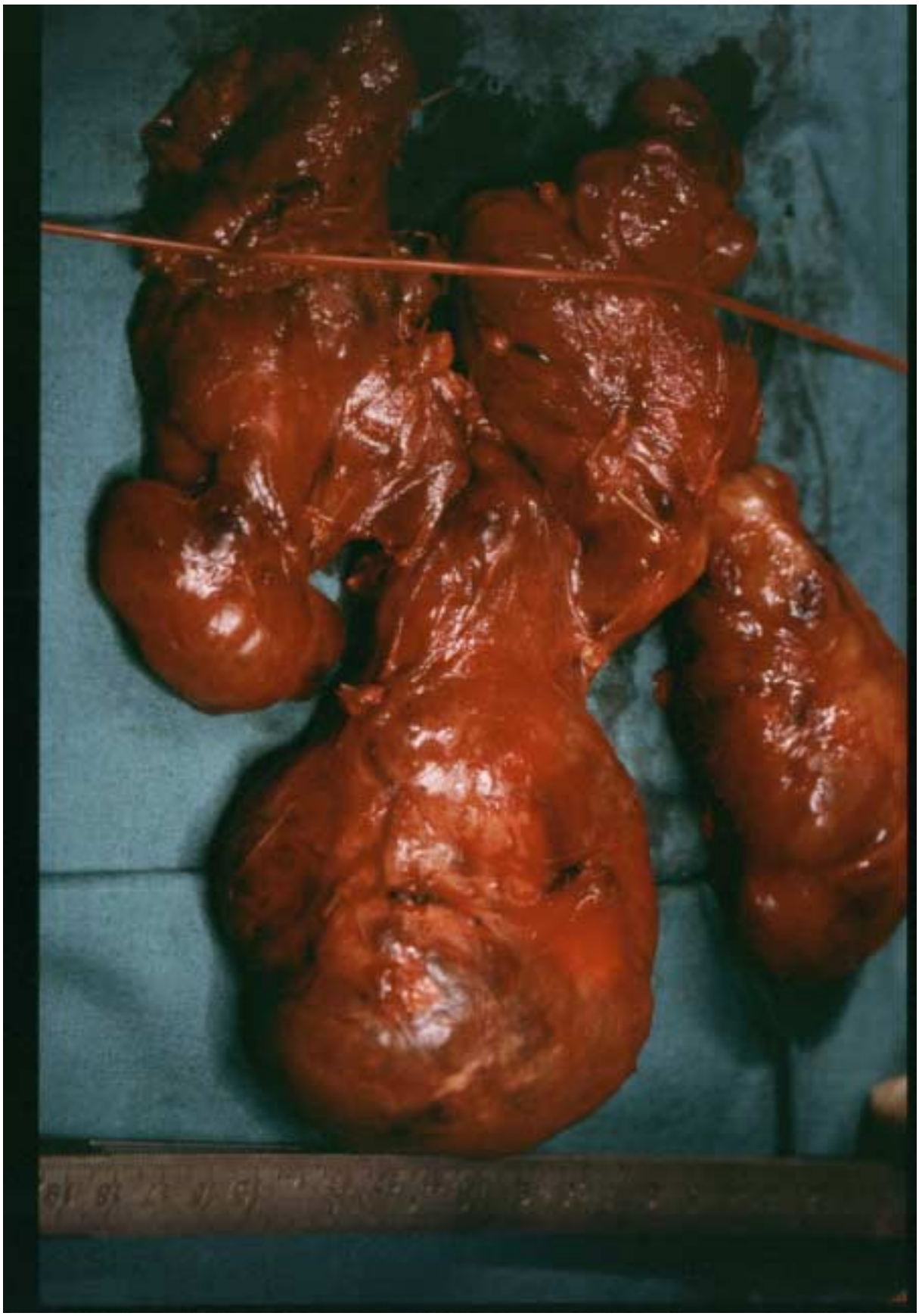
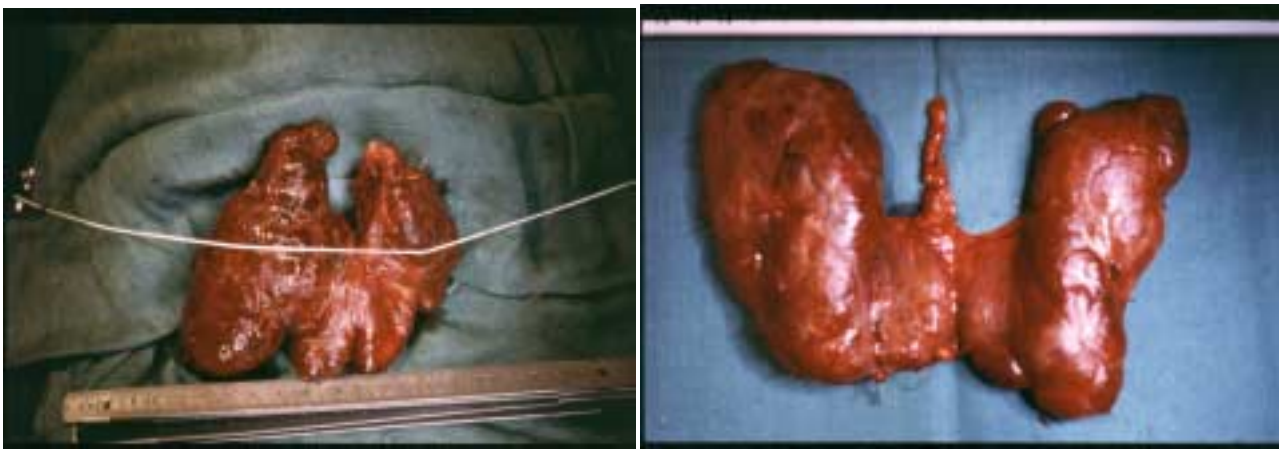
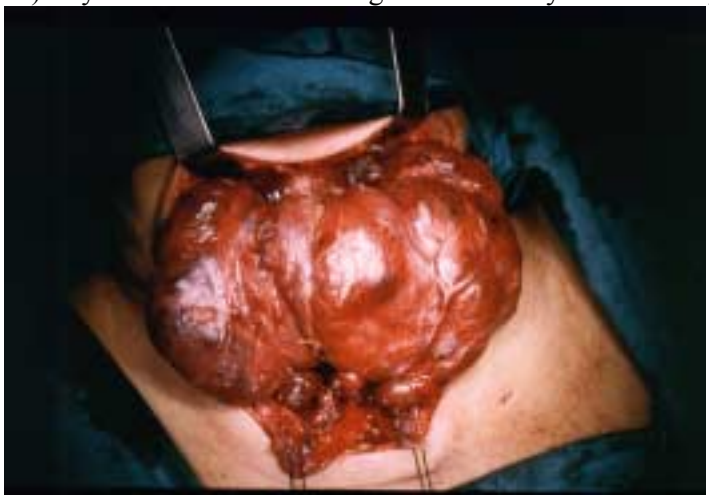


Fig. 4: Large cervico-mediastinal (substernal) goiter. Only the portion overlying the red rubber loop was in a suprajugular cervical position.

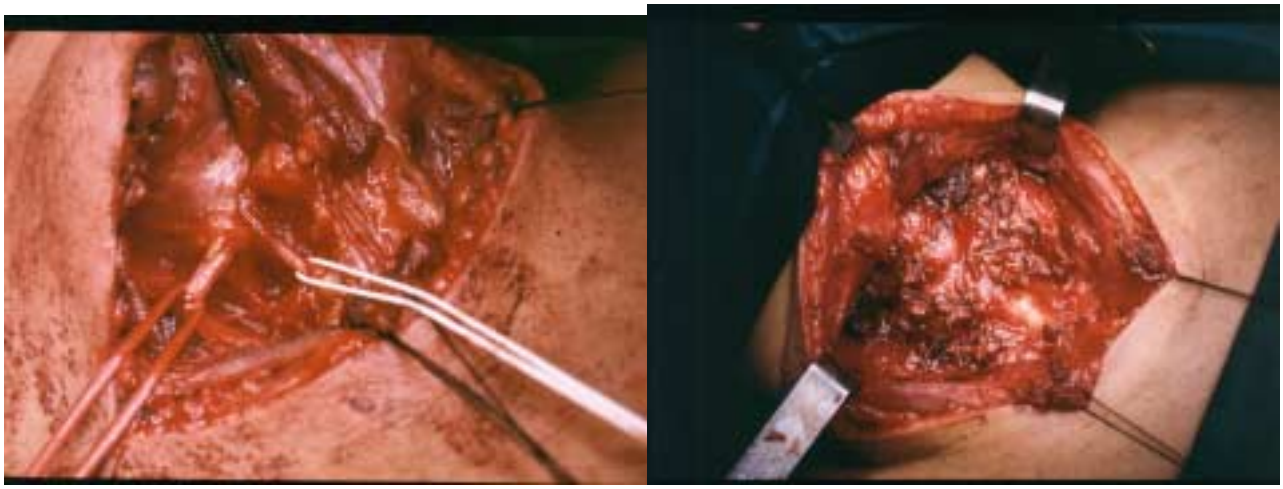


a b

Fig. 5: a) cervico-mediastinal (substernal) goiter: the suprajugular cervical portion lies above the white rubber loop. b) Thyroid with multinodular goiter treated by near total thyroidectomy (NTT).

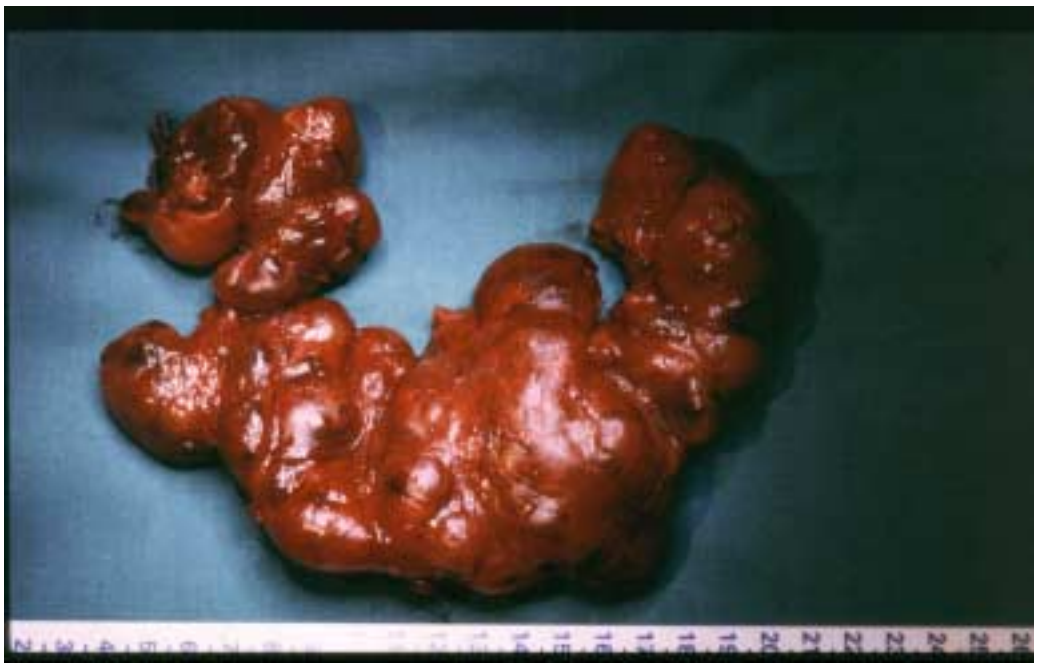


a

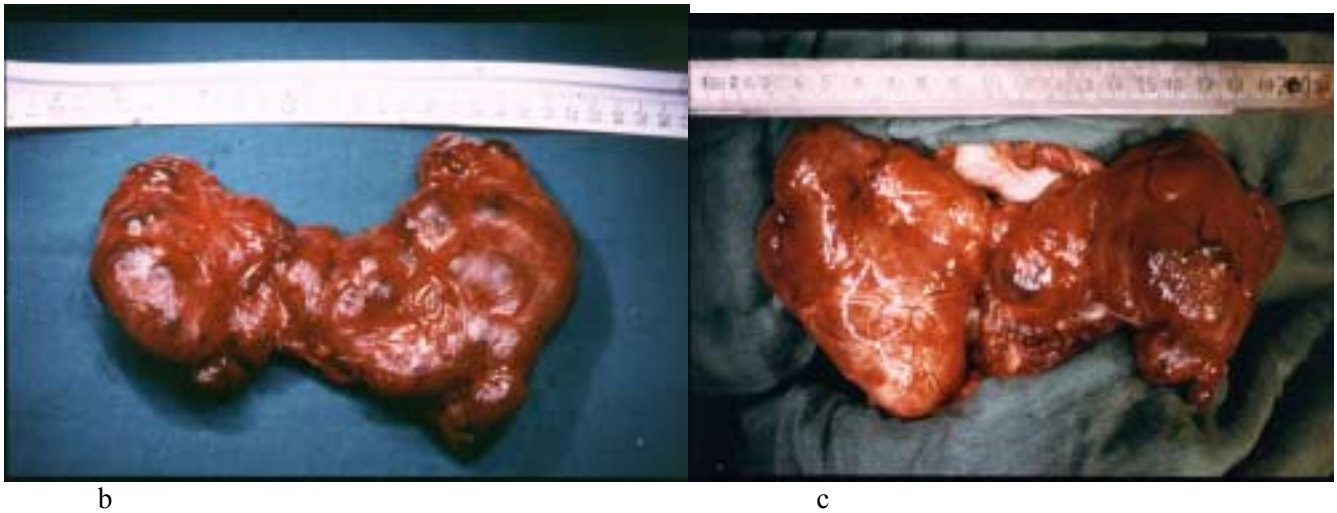


b c

Fig. 6: a) Near total thyroidectomy (NTT) for diffuse bilateral multinodular goiter. b) Identification and preservation of the right inferior recurrent laryngeal nerve (white rubber); the red rubber lies under the inferior thyroid artery that emerges posteriorly to the common carotid artery; c) residual parenchyma (posterior wall) following NTT.



a



b c

Fig. 7: a), b), c) Different features of diffuse bilateral multinodular goiter.

Selection of publications by the Surgical Clinic on the topic

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