Cosmetic Special Topic

Classification and Management of Gynecomastia: Defining the Role of Ultrasound-Assisted Liposuction

Rod J. Rohrich, M.D., Richard Y. Ha, M.D., Jeffrey M. Kenkel, M.D., and William P. Adams, Jr., M.D.

Dallas, Texas

Gynecomastia, or excessive male breast development, has an incidence of 32 to 65 percent in the male population. This condition has important physical and psychological impacts. Advances in elucidating the pathophysiology of gynecomastia have been made, though understanding remains limited. Recommendations for evaluation and workup have varied and are often arbitrary. A diagnostic algorithm is suggested, with emphasis on a comprehensive history, physical examination, and minimizing unnecessary diagnostic testing. Medical management has had limited success; surgical therapy, primarily through excisional techniques, has been the accepted standard. Although effective, excisional techniques subject patients to large, visible scars. Ultrasound-assisted liposuction has recently emerged as a safe and effective method for the treatment of gynecomastia. It is particularly efficient in the removal of the dense, fibrous male breast tissue while offering advantages in minimal external scarring. A new system of classification and graduated treatment is proposed, based on glandular versus fibrous hypertrophy and degree of breast ptosis (skin excess). The authors’ series of 61 patients with gynecomastia from 1987 to 2000 at the University of Texas Southwestern Department of Plastic Surgery demonstrated an overall success rate of 86.9 percent using suction-assisted lipectomy (1987 to 1997) and ultrasound-assisted liposuction (1997 to 2000). The authors have found ultrasound-assisted liposuction to be effective in treating most grades of gynecomastia. Excisional techniques are reserved for severe gynecomastia with significant skin excess after attempted ultrasound-assisted liposuction. (Plast. Reconstr. Surg. 111: 909, 2003.)

Gynecomastia is defined as benign, excessive breast development in male individuals. Initially thought to be a rare condition, with a reported incidence of 8 in 100,000 during World War II, more recent studies have reported an overall incidence of 32 to 36 percent, even as high as 40 percent of men in an autopsy series and 64.6 percent in adolescent boys. The incidence of bilateral involvement also varies in the literature. Bilateral disease occurs in 25 to 75 percent of patients. This wide variation likely stems from an increased appreciation of the psychological, physical, and social impact of the condition. It underscores the lack of a clear and standardized definition within the plastic surgery and medical literature.

The etiology of gynecomastia is multifactorial, but in many cases, an identifiable cause is elusive. Recent investigations have demonstrated pathophysiologic mechanisms to involve either a relative or absolute excess of estrogens, a decrease of circulating androgens, or a defect in androgen receptors. This has been the basis for recent attempts at medical therapy for gynecomastia. However, surgical removal of the hypertrophic breast tissue remains the accepted standard in treatment.

Surgical management, until recently, mostly consisted of excisional techniques. Standard suction-assisted lipectomy had an adjunctive role, as described by Teimourian and Pearlman in 1983. Ultrasound-assisted lipectomy as a primary mode of treatment had been advocated by others. Ultrasound-assisted liposuction, with known mechanical advantages in addressing dense, fibrous lipodystrophy, has recently emerged as the preferred technique for management of gynecomastia.

We discuss the etiology, pathophysiology, di-
agnosis, and our preferences in the treatment of gynecomastia. Based on our experience at the University of Texas Southwestern Medical Center, Department of Plastic and Reconstructive Surgery, over the past 14 years, we propose a new classification system for gynecomastia incorporating the emerging role of ultrasound-assisted liposuction as a safe and effective method for treatment.

ETIOLOGY

Gynecomastia has many identifiable causes, though most cases are idiopathic. Recent studies have shown strong evidence for the estrogen-stimulating effects of breast tissue development and support for an inhibitory androgenic effect. Decreases in the androgen-to-estrogen ratio have also been associated with development of gynecomastia. No clear etiologic classification has been suggested based on hormonal influences solely. Instead, most physicians accept an arbitrary classification based on physiologic, pathologic, pharmacologic, and idiopathic causes (Table I). Idiopathic causes (25 percent) are the most common.6

Physiologic gynecomastia can be further subdivided into neonatal, pubertal, and elderly periods. Circulating maternal estrogens transferred by the placental-fetal circulation are thought to contribute to excessive development of breast tissue in neonates. Because this is usually a self-limited process lasting weeks to months, treatment is rarely indicated. Adolescent boys (up to 65 percent) often exhibit varying degrees of gynecomastia, which usually resolves over several months to years.5 The degree of breast enlargement can be so minor as to escape recognition unless clinically palpated. Relative excess of plasma estradiol compared with testosterone is implicated in the pathogenesis of pubertal gynecomastia.12,13 Older men (beginning at age 65) will often develop breast enlargement as plasma testosterone levels decline and peripheral conversion of testosterone to estrogen (peripheral aromatization) occurs, thus effectively increasing the plasma estrogen-to-androgen ratio.6

Pathologic gynecomastia occurs as a result of various metabolic disorders (alcoholic cirrhosis, refeeding after a starvation state, feminizing adrenal tumors), endocrine disorders (hyperthyroidism, adrenal cortical hyperplasia, hypothyroidism), acquired hypogonadal states (orchitis, testicular trauma, granulomatous disease, renal failure, alcoholism, myotonic dystrophy), congenital hypogonadal states (Klinefelter syndrome, congenital anorchia, androgen resistance), and increased estrogen states (bronchogenic carcinoma, true hermaphroditism, testicular tumors).6,14–16 The specific pathophysiologies are beyond the scope of this discussion.

Pharmacologic gynecomastia occurs by several mechanisms, including increased direct estrogenic activity, increased secretion of estrogen, decreased testosterone synthesis, and decreased androgen sensitivity. There are also many drugs with poorly understood mecha-

<table>
<thead>
<tr>
<th>TABLE I Causes of Gynecomastia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Developmental/physiologic</strong></td>
</tr>
<tr>
<td>Neonatal</td>
</tr>
<tr>
<td>Pubertal</td>
</tr>
<tr>
<td>Aging</td>
</tr>
<tr>
<td>Drug-induced (see Table II)</td>
</tr>
<tr>
<td>Hypogonadism (decreased androgen synthesis or increased androgen resistance)</td>
</tr>
<tr>
<td>Primary</td>
</tr>
<tr>
<td>Acquired (trauma, infection, torsion, radiation exposure, mumps, chemotherapy)</td>
</tr>
<tr>
<td>Congenital</td>
</tr>
<tr>
<td>Secondary</td>
</tr>
<tr>
<td>Hypogonadotropic hypogonadism</td>
</tr>
<tr>
<td>Kallmann syndrome</td>
</tr>
<tr>
<td>Pituitary failure (infarction, infection, neoplasm)</td>
</tr>
<tr>
<td>Tumors (increased estrogen production)</td>
</tr>
<tr>
<td>Steroid-producing (adrenal, testis)</td>
</tr>
<tr>
<td>Human chorionic gonadotropin-producing (testis and others)</td>
</tr>
<tr>
<td>Aromatase-producing (testis)</td>
</tr>
<tr>
<td>Bronchogenic carcinoma</td>
</tr>
<tr>
<td>Systemic</td>
</tr>
<tr>
<td>Thyrotoxicosis (altered testosterone/estrogen binding)</td>
</tr>
<tr>
<td>Renal failure (acquired testicular failure)</td>
</tr>
<tr>
<td>Cirrhosis (increased substrate for peripheral aromatization)</td>
</tr>
<tr>
<td>Adrenal (adrenocorticotropic hormone deficiency or congenital adrenal hyperplasia)</td>
</tr>
<tr>
<td>Congenital disorders</td>
</tr>
<tr>
<td>Klinefelter syndrome</td>
</tr>
<tr>
<td>Enzyme defects of testosterone synthesis (may be late onset)</td>
</tr>
<tr>
<td>Vanishing testis syndrome (anorchia)</td>
</tr>
<tr>
<td>Androgen resistance syndromes</td>
</tr>
<tr>
<td>True hermaphroditism and related conditions</td>
</tr>
<tr>
<td>Increased peripheral tissue aromatase</td>
</tr>
<tr>
<td>Familial</td>
</tr>
<tr>
<td>Miscellaneous</td>
</tr>
<tr>
<td>HIV</td>
</tr>
<tr>
<td>Chest wall trauma</td>
</tr>
<tr>
<td>Psychological stress</td>
</tr>
<tr>
<td>Spinal cord injury</td>
</tr>
<tr>
<td>Malnutrition/refeeding (increased substrate for peripheral aromatization)</td>
</tr>
<tr>
<td>Herpes zoster infection</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Alcoholism</td>
</tr>
<tr>
<td>Myotonic dystrophy</td>
</tr>
</tbody>
</table>

isms that are associated with gynecomastia\textsuperscript{6,17,18} (Table II).

Histologically, Bannayan and Hajdu\textsuperscript{19} identified the following three patterns with varying degrees of stromal and ductal proliferation: florid, intermediate, and fibrous types. The florid pattern shows increased numbers of budding ducts in a highly cellular fibroblastic stroma. Fibrous type has extensive stromal fibrosis with minimal ductal proliferation. Intermediate type demonstrates overlapping of the fibrous and florid histologic patterns. Most observers agree that these patterns represent a transition related to duration of gynecomastia and its symptoms, with florid type seen usually in gynecomastia of less than 4 months’ duration and fibrous type seen in gynecomastia lasting longer than 1 year.\textsuperscript{19}

**ASSOCIATED RISKS OF MALE BREAST CANCER**

One percent of all breast cancer occurs in men. Patients with Klinefelter syndrome, however, have up to a 60 times greater risk of developing breast cancer, and the incidence is approximately 1:400 to 1:1000.\textsuperscript{20} Many studies reviewing male subjects with gynecomastia have demonstrated no increased risk for breast cancer as compared with the normal male population.\textsuperscript{6} Thus, in all non-Klinefelter patients, it is reasonable to treat gynecomastia surgically with both liposuction and excisional techniques without adding considerable morbidity of delayed detection of male breast cancer.

In those patients in whom hypogonadism is suspected, Klinefelter syndrome must be ruled out because this will ultimately impact treatment. Because of the oncologic risk in these patients, excisional techniques are preferred in this scenario.

**DIAGNOSIS**

Clinical evaluation of patients with gynecomastia is paramount. However, further workup is rarely indicated. History elicited should include age, duration and onset of breast enlargement, symptoms of pain or tenderness, medications and recreational drug use, and psychological and social effects. A review of systems should involve signs/symptoms of hepatic disease, hyperthyroidism or hypothyroidism, recent weight gain or loss, adrenal disease, alcoholism, renal failure, and malignancies.

Physical examination of the breasts should involve assessment for glandular or fat predominance (by the pinch test), degree of glandular ptosis, skin excess, nodules/masses, and nipple abnormalities or discharge. Glandular or parenchymal tissue is characterized by rubbery breast tissue that is mobile and extends from a subareolar, centric position. Suspicious nodules or masses may have abnormal firmness, overlying skin ulceration, eccentric location, or abnormal nipple discharge. The normal male breast is typically flat with some fullness around the nipple-areola complex. The nipple-areola complex is normally 2 to 4 cm in diameter (average, 2.8 cm)\textsuperscript{21,22} and located over the fourth intercostal space. Nipple to sternum notch distance is, on average, 20 cm.\textsuperscript{23} A muscular male chest may exhibit superior fullness with a transition to a flat inferior chest near the inframammary fold. Completion of the physical examination should in particular assess for testicular enlargement/atrophy and asymmetry, thyromegaly, hepatomegaly, pulmonary abnormalities, and abdominal masses.

Additional diagnostic testing should be individualized to address abnormalities identified in the history or physical examination. Individualized workups have also been suggested depending on age at the time of development of

**TABLE II**

**Drug-Induced Gynecomastia**

<table>
<thead>
<tr>
<th>I. Drug classes associated with gynecomastia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estrogens</td>
</tr>
<tr>
<td>Gonadotropins</td>
</tr>
<tr>
<td>Androgens (aromatizable)</td>
</tr>
<tr>
<td>Anti-androgens (cyproterone, flutamide)</td>
</tr>
<tr>
<td>Cancer chemotherapy agents (especially alkylating agents)</td>
</tr>
<tr>
<td>Calcium channel blockers (verapamil, nifedipine, diltiazem)</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme–inhibitors (captopril, enalapril)</td>
</tr>
<tr>
<td>Anti-hypertensives (methyldopa, reserpine)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II. Individual drugs commonly associated with gynecomastia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cimetidine</td>
</tr>
<tr>
<td>Spironolactone</td>
</tr>
<tr>
<td>Ketoconazole</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III. Miscellaneous drugs related to gynecomastia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
</tr>
<tr>
<td>Auranoxin</td>
</tr>
<tr>
<td>Clomiphene</td>
</tr>
<tr>
<td>Etretinate</td>
</tr>
<tr>
<td>Metronidazole</td>
</tr>
<tr>
<td>Omeprazole</td>
</tr>
<tr>
<td>Penicillamine</td>
</tr>
<tr>
<td>Sulindac</td>
</tr>
<tr>
<td>Theophylline</td>
</tr>
</tbody>
</table>

gynecomastia (Table III). These considerations are important, but arbitrary use of “screening” tests or laboratory data is not recommended. Rarely is an extensive workup indicated, and often it will not affect treatment. Certain clinical findings, however, should prompt further evaluation. In the scenario of a young patient (especially prepubertal) with a negative history and bilateral gynecomastia, a testicular ultrasound in conjunction with a careful testicular examination has been found to be cost-effective. The incidence of a functional endocrine tumor in this group is significant. Physical examination findings of feminization, including small testicular size (< 3 cm in length or 8 cc in volume), lack of male hair distribution, or a eunuchoid body habitus, suggest a possible feminizing tumor. Endocrine testing in this situation (serum testosterone, luteinizing hormone, estradiol, and the sulfate salt of dehydroepiandrosterone) is appropriate. If feminizing characteristics are found in association with a marfanoid body habitus, a karyotype and endocrine referral is indicated to rule out Klinefelter syndrome.

A patient should consider proceeding with surgical management once diagnosis of gynecomastia is established of nonphysiologic causes or of duration greater than approximately 12 months, because hypertrophic breast tissue beyond this stage usually becomes irreversibly fibrotic. An algorithm for the diagnosis and directed treatment of gynecomastia is suggested (Fig. 1 and Table IV).

### TABLE III

**Historical Guidelines for the Clinical Evaluation of Gynecomastia**

<table>
<thead>
<tr>
<th>Adult, male</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Testicular failure (viral orchitis, trauma)</td>
<td>1. LH/FSH</td>
</tr>
<tr>
<td>2. Ectopic gonadotropin production</td>
<td>2. Testosterone (total and free)</td>
</tr>
<tr>
<td>3. Feminizing adrenal carcinoma</td>
<td>3. Estradiol</td>
</tr>
<tr>
<td>4. Drug use</td>
<td>4. Karyotype</td>
</tr>
<tr>
<td>5. Chronic liver disease/cirrhosis</td>
<td>5. Scans or exploration as indicated</td>
</tr>
<tr>
<td>6. Renal failure</td>
<td>+/− androgen receptor analysis</td>
</tr>
<tr>
<td>7. Thyrotoxicosis</td>
<td>+/− androgen biosynthetic intermediates</td>
</tr>
<tr>
<td>8. Idiopathic</td>
<td>Tests</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pubertal, male (with ambiguous genitalia)</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Androgen biosynthetic defects</td>
<td>1. LH/FSH</td>
</tr>
<tr>
<td>2. Partial androgen resistance</td>
<td>2. Testosterone (total and free)</td>
</tr>
<tr>
<td>3. True hermaphroditism</td>
<td>3. Estradiol</td>
</tr>
<tr>
<td>5. Scans or exploration as indicated</td>
<td>+/− androgen receptor analysis</td>
</tr>
<tr>
<td>6. If estradiol is elevated, adrenal computed tomographic scan</td>
<td>+/− androgen biosynthetic intermediates</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pubertal, male (with normal external genitalia)</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Physiologic</td>
<td>1. LH/FSH</td>
</tr>
<tr>
<td>2. Klinefelter syndrome</td>
<td>2. Testosterone (total and free)</td>
</tr>
<tr>
<td>3. Congenital anorchism</td>
<td>3. Estradiol</td>
</tr>
<tr>
<td>5. Feminizing carcinoma (Leydig cell tumor, adrenal carcinoma)</td>
<td>5. If testosterone low and LH/FSH elevated, karyotype</td>
</tr>
<tr>
<td>6. If estradiol is elevated, adrenal computed tomographic scan</td>
<td>Tests</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prepubertal, male</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider:</td>
<td>1. Serum estradiol</td>
</tr>
<tr>
<td>1. Premature thelarche</td>
<td>2. Computed tomographic scan abdomen/adrenal</td>
</tr>
<tr>
<td>2. Exogenous hormones or drug use</td>
<td>3. LH/FSH (elevations may be only at night)</td>
</tr>
<tr>
<td>3. Premature puberty</td>
<td>4. Bone age</td>
</tr>
<tr>
<td>4. Feminizing adrenal carcinoma</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prepubertal, female</th>
<th>Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider:</td>
<td>1. Serum estradiol level</td>
</tr>
<tr>
<td>1. Premature thelarche</td>
<td>2. Computed tomographic scan abdomen/adrenal</td>
</tr>
<tr>
<td>2. Exogenous hormones or drug use</td>
<td>3. LH/FSH (elevations may be only at night)</td>
</tr>
<tr>
<td>3. Premature puberty</td>
<td>4. Bone age</td>
</tr>
<tr>
<td>4. Feminizing adrenal carcinoma</td>
<td></td>
</tr>
</tbody>
</table>

MANAGEMENT OF GYNECOMASTIA

Nonsurgical management has involved correction of underlying causes, medications to regulate hormonal imbalances, and irradiation. Medical therapy with testosterone, antiestrogens (clomiphene, tamoxifen), and danazol has had only limited success.6,25

Gynecomastia of long duration is unlikely to regress spontaneously and will often progress to irreversible dense fibrosis and hyalinization despite medical therapy. Withdrawal of an offending drug, correction of a systemic disorder, or the natural course of physiologic gynecomastia often results in spontaneous regression of gynecomastia; however, correction of an underlying disorder may not be effective in treating gynecomastia, especially if the duration of disease is lengthy and fibrosis of breast tissue has already occurred.26 Thus, surgery remains the accepted standard for management. The evolution of surgical techniques for gynecomastia can be traced in the literature:

Fig. 1. Algorithm for evaluation and treatment of gynecomastia.
TABLE IV
Classification and Management of Gynecomastia

<table>
<thead>
<tr>
<th>Classification</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I - Minimal hypertrophy (&lt; 250 g of breast tissue) without ptosis</td>
<td></td>
</tr>
<tr>
<td>I A. Primarily glandular*</td>
<td>Ultrasound-assisted liposuction or suction-assisted liposuction</td>
</tr>
<tr>
<td>I B. Primarily fibrous*</td>
<td>Ultrasound-assisted liposuction</td>
</tr>
<tr>
<td>Grade II - Moderate hypertrophy (250–500 g of breast tissue) without ptosis</td>
<td></td>
</tr>
<tr>
<td>II A. Primarily glandular*</td>
<td>Ultrasound-assisted liposuction or suction-assisted liposcopy</td>
</tr>
<tr>
<td>II B. Primarily fibrous*</td>
<td>Ultrasound-assisted liposuction</td>
</tr>
<tr>
<td>Grade III - Severe hypertrophy (&gt; 500 g of breast tissue) with grade I ptosis</td>
<td></td>
</tr>
<tr>
<td>Glandular or fibrous*</td>
<td>Ultrasound-assisted liposuction with or without staged excision†</td>
</tr>
<tr>
<td>Grade IV - Severe hypertrophy with grade II or III ptosis</td>
<td></td>
</tr>
<tr>
<td>Glandular or fibrous*</td>
<td>Ultrasound-assisted liposuction with or without staged excision†</td>
</tr>
</tbody>
</table>

* Fatty and glandular tissue is determined by a pinch test medially, laterally, and beneath the nipple-areola complex.  
† Delayed excision of remaining ptotic breast skin and/or breast parenchyma is performed 6 to 9 months after ultrasound-assisted liposuction to allow maximal skin retraction.

1. Semicircular intra-areolar incision — resection of cone-shaped mass of breast tissue centrally with peripheral taper; reaproximation of fat beneath areola
2. Inverted omega intra-areolar incision — improved exposure to undersurface of areola
3. Horizontal transareolar/intra-areolar incision — improved exposure, easier dissection and hemostasis
4. Superior periareolar incision with skin excision — for moderate to large gynecomastia; excess skin resected and nipple transposed superiorly
5. Nipple transposition on single dermal pedicle with oblique incision — nipple transposed medially and superiorly
6. Nipple transposition on vertical bipedicle with horizontal incision
7. Excision of redundant skin/breast tissue with free nipple graft — excision located as ellipse around nipple
8. Radial intra-areolar incision
9. Excision of redundant skin/breast tissue, nipple grafting with inframammary fold scar — en bloc excision of breast tissue/skin with free nipple grafting; inframammary fold scar not in continuity with new nipple position
10. Transaxillary approach — 2-inch incision in axilla
11. Dermal pedicle
12. Concentric circle technique — half-circle serves as dermal pedicle, remaining half resected with breast tissue
13. Single superior dermal pedicle
14. Concentric circle technique modification — no dermal pedicle; apple core around nipple/areola, vascular supply from chest wall
15. Suction-assisted lipectomy with excision — used in periphery to smooth contour
16. Suction-assisted lipectomy — used to evacuate both adipose tissue and dense parenchymal tissue in the breast, citing use of smaller 2.4-mm cannula as critical component for effective evacuation of the dense parenchyma
17. Suction-assisted lipectomy — useful in selected patients with predominantly fatty breasts and well-located nipple/areola
18. Suction-assisted lipectomy
19. Suction-assisted lipectomy — axillary approach for suction liposcopy
20. Eccentric skin resection and pursestring closure
21. Endoscopic transaxillary approach
22. Circumareolar skin and suction-assisted liposuction — technique described for patients with severe gynecomastia.
23. Ultrasound-assisted liposuction — technique reviewed, successful use in all three grades of gynecomastia
24. Pinwheel technique
25. Ultrasound-assisted liposuction — excellent results using ultrasound-assisted liposuction for most types of gynecomastia

As mentioned, suction-assisted liposcopy has been used to treat gynecomastia primarily or adjunctively with some success; limitations were encountered with severe cases or in breasts with primarily fibrous tissues. Ultrasound-assisted liposuction has extended the role of lipoplasty in the management of gynecomastia. Zocchi first described the technique of using ultrasonic energy transmitted by means of excited piezoelectric crystals placed on terminal ends of suction cannulas to emulsify fat while preserving adjacent nervous, vas-
cicular, and connective tissue elements. Emulsi-

fication is effected through cavitation of fat
cells in tumesced fields.

Rohrich et al.\textsuperscript{50–53} have further refined the
ultrasound-assisted liposuction technique, in-

corporating a three-stage process of subcutane-

ous infiltration of a wetting solution, followed by
ultrasound-assisted liposuction and com-

pleted by evacuation and final contouring by
suction-assisted lipectomy. Endpoints for ul-

trasound-assisted liposuction (time and loss of re-

sistance) differ from those for standard suc-

tion-assisted lipectomy (pinch and contour)

but now provide parameters for safe and effi-
cient lipectomy.

Ultrasound-assisted liposuction has several
advantages over suction-assisted lipectomy in
the treatment of gynecomastia. Several studies
have confirmed the selective emulsification of
fat leaving higher density structures, such as
fibroconnective tissue, relatively undamaged.
This allows for more efficient fat removal in
areas that have higher densities of fibroconnec-
tive tissue, such as the male breast. At higher
energy settings, ultrasound-assisted liposuction
is effective in removal of the denser, fibrotic
parenchymal tissue that suction-assisted lipe-
tomy is inefficient in removing. Also, ul-

trasound-assisted liposuction performed in the
appropriate subdermal plane affects the der-

mis, allowing for skin retraction in the postop-
erative healing period. Further advantages of
ultrasound-assisted liposuction include the re-
duced physical demand for large-volume lipo-
suction of the breast, allowing the surgeon im-
proved attention to precise contouring.\textsuperscript{6,23,54,55}

Previous clinical classification systems, as
suggested by Simon et al.\textsuperscript{33} and Cohen et al.,\textsuperscript{6}
offered excellent guidelines for graduated
management of gynecomastia based on de-
grees of lipodystrophy and skin excess. Most
recommended techniques were excisional with
suction-assisted lipectomy offered as an adjunc-
tive measure for final contouring. We offer a
new classification system for gynecomastia and
the preferred treatment at each grade, incor-
porating the role of ultrasound-assisted lipo-
suction as a superior method for safe and ef-

fective removal of male breast tissue, based on
our experience with 61 patients evaluated and
treated for gynecomastia from 1987 to 2000
(Table IV).

Our classification of gynecomastia is based
on the amount and character of breast hyper-
trophy and the degree of ptosis. Preoperative
and postoperative examples are shown to dem-

onstrate the efficacy of ultrasound-assisted lipo-
suction. Grade I patients (Fig. 2) have minimal
hypertrophy (< 250 g of breast tissue), and
grade II patients (Figs. 3 and 4) have moderate
hypertrophy (between 250 and 500 g of breast
tissue). These grades can be further subdivided
into IA and IIA for primarily fatty breast tissue
and IB and IIB for primarily fibrous breast
tissue. Grade III and IV patients (Figs. 5 and 6)
have severe hypertrophy (> 500 g of breast
tissue) but are distinct because of a greater
degree of ptosis. Grade III gynecomastia pa-

patients exhibit grade 1 ptosis, and grade IV gy-

necomastia patients show grade 2 or 3 ptosis.

Suction-assisted lipectomy can be used in
grade IA and grade IIA gynecomastia with suc-
cess. However, it is a suboptimal treatment for
more severe gynecomastia (grade III and IV)
and for any gynecomastia with primarily fi-

brous tissue.

Ultrasound-assisted liposuction is effective in
all grades of gynecomastia. Usually, no further
treatment is needed in grade I or II gyneco-

mastia and, often, single ultrasound-assisted lipo-
suction treatment is all that is necessary for
grades III and IV, especially in those with mild
ptosis and good skin quality. If removal of re-
dundant skin and/or resistant lipodystrophy is
still required after ultrasound-assisted liposuc-
tion, a staged excision is delayed for 6 to 9
months to allow for maximal skin retraction
and healing, thus potentially allowing down-
staging of the magnitude of the excisional

 technique (and therefore minimizing

scarring).

**Ultrasound-Assisted Liposuction Technique**

After thorough evaluation of a patient, in-
cluding a detailed history and physical exami-
nation, markings are made to outline bound-
aries of treatment. The inframammary fold
should be marked preoperatively with the pa-

ient sitting or standing. Adherent zones in the
upper outer quadrants, as the breast retreats
into axillary tissue, should be suctioned with
caution (Fig. 7). Patients receive general anes-
thesia and are positioned supine with arms
abducted. Incisions (3 to 4 mm) are made with
a no. 11 scalpel in the lateral inframammary
folds bilaterally for optimal access to the dense
breast parenchyma. This route also provides
superior access to obliterate the inframammary
fold and to suction the medial chest.\textsuperscript{11}

Subcutaneous infiltration of wetting solution

Vol. 111, No. 2 / GYNECOMASTIA MANAGEMENT 915
in the intermediate fat layer is performed using a standard infiltration pump and a 3.0-mm cannula. A “superwet” technique, with a 1:1 ratio of infiltrate to estimated aspirate, is used with a solution containing 1 liter of lactated Ringer solution mixed with 1 ampule of 1:1000 epinephrine and 30 cc of 1% Xylocaine. Uniform infiltration can be challenging, and it is important to systematically infiltrate all treatment areas in multiple layers.

Ultrasound-assisted liposuction is performed with an ultrasound generator [Lysonix (Carpinteria, Calif.) or Mentor (Santa Barbara, Calif.)], a 5-mm blunt-tip titanium cannula or a 4-mm golf-tee cannula, and a standard surgical aspirator for evacuation. The Mentor generator is set at an energy level between 70 to 90 percent, with 90 percent being especially effective in the denser subareolar areas. Lysonix generators should have energy levels set between 5 and 7. Volumes of aspirate and time of application are...
recorded. Stroke technique involves constant, deliberate passes of the cannula through the intermediate fat layer in a radial fashion from the lateral inframammary fold incisions. Additional passes are concentrated in the unique subareolar region where maximal fibroconnective density occurs. A bimanual technique is used with the nondomi-
nant hand guiding the ultrasound-assisted liposuction cannula through the subdermal layer. Liposuction of the subdermal layer is not a traditional level of treatment in ultrasound-assisted liposuction, but it is particularly important in addressing the dense fibrous tissue of gynecomastia and allowing for maximal skin retraction. The periphery is treated for feathering and contouring. Disruption of the inframammary fold is essential in achieving a more gradual transition of the breast to the abdomen, which is characteristic in men (as opposed to the distinct demarcation of the inframammary fold in women). The adherent zone should be suctioned minimally, if at all (only for contouring), and extreme caution should be exercised.

Suction-assisted lipectomy is performed in standard fashion using a 3.7-mm cannula for final contouring and evacuation of emulsified fat. Evacuation should occur from deep fat layer to the intermediate fat layer. Great care is
taken to avoid overcontouring of the area overlapping the upper lateral pectoralis major muscle.

In the situation of small, residual, dense subareolar tissue, it is reasonable to excise this after ultrasound-assisted liposuction through a periareolar incision. Our preference is not to address skin excess at the initial operation. We recommend allowing for maximal skin retraction after ultrasound-assisted liposuction before staged skin excision, if required.

FIG. 5. Preoperative (left) and postoperative (right) patient photographs: grade III gynecomastia.
After evacuation is complete, incisions are closed with 5-0 fast absorbing plain gut. The chest wall is dressed with a double layer Topifoam, and a compressive vest is to be worn by the patient for 4 weeks continuously followed by 4 weeks at nighttime only.

Our experience has demonstrated that staged excision can be accomplished through a periareolar approach in most cases. An inferior 180-degree incision through the areolar margin is used to gain access to any residual subareolar tissue. Excision through extensive breast mound incisions has rarely been required.

FIG. 6. Preoperative (left) and postoperative (right) patient photographs: grade IV gynecomastia.

RESULTS AND COMPLICATIONS

Courtiss\(^9\) reviewed 159 patients who underwent surgical management for gynecomastia, with 101 patients (192 breasts) treated with excisional techniques. He reported a high complication rate with excisional techniques, including overresection (18.7 percent), unattractive scarring (18.7 percent), hematoma (16.1 percent), seroma (9.4 percent), and underresection (21.9 percent).

A series of 60 patients treated by ultrasound-assisted liposuction was reported by Gingrass and Shermak.\(^{48}\) No major complications, hematomas, or episodes of skin necrosis have
been reported over a 4-year follow-up. Results were reported as “uniformly good to excellent.”

Our experience at the University of Texas Southwestern Department of Plastic Surgery consists of 61 patients treated with liposuction (suction-assisted lipectomy and ultrasound-assisted liposuction) from 1987 to 2000. We have used ultrasound-assisted liposuction exclusively since 1997 for all grades (I to IV) of gynecomastia. No additional treatment was required in 86.9 percent of patients (53 of 61) treated with either ultrasound-assisted liposuction or suction-assisted lipectomy (Table V). Eight patients, four with grade III disease and four with grade IV gynecomastia, required staged excision of remaining breast tissue and skin (6 to 9 months after ultrasound-assisted liposuction) to optimize results.

**DISCUSSION**

Gynecomastia is a benign condition that affects as many as 65 percent of male individuals. Advances have been made in elucidating its causes and pathophysiologic mechanisms. However, most cases remain idiopathic. Medical therapies have largely been ineffective. Historically, surgical management of gynecomastia has involved primarily excisional techniques with suction-assisted lipectomy used adjunctively for contouring. Most excisional techniques are effective in achieving reduction of glandular and fibrotic breast tissue. However, these techniques can subject the patient to large, visible scars on the chest wall. More recently, ultrasound-assisted liposuction has emerged as the preferred surgical technique for most cases of gynecomastia, providing the advantages of minimal scarring and efficient removal of both glandular and fibrotic breast tissue. The precise effects of ultrasound-assisted liposuction on skin contraction are not yet known, although our experience has shown a considerable amount of retraction, particularly in patients with grade III and IV gynecomastia, depending on the patients age and skin elasticity. The degree of ptosis or skin excess has not altered our treatment algorithm with regard to ultrasound-assisted liposuction. We recommend that ultrasound-assisted liposuction be used first to allow for maximal skin retraction. If residual skin excess or breast tissue exists 6 to 9 months later, we recommend considering excisional techniques at that time. Our experience has demonstrated that more than 85 percent of patients treated with ultrasound-assisted liposuction did not require extensive skin incisions for removal of the remaining tissue.

A new classification system is suggested adopting ultrasound-assisted liposuction as a safe and effective treatment for most grades of gynecomastia. Specific treatment is directed by grade, with the goal of achieving a normal-appearing male chest while minimizing residual deformity and avoiding excessive scars.

**TABLE V**

<p>| University of Texas Southwestern Department of Plastic Surgery Gynecomastia Series (1987-2000) |
|---------------------------------------------------------------|---------------------------------|</p>
<table>
<thead>
<tr>
<th>Grade</th>
<th>Patients (%)</th>
<th>UAL/SAL Success Rate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>18 (29.5)</td>
<td>100.0 (18/18)</td>
</tr>
<tr>
<td>II</td>
<td>24 (39.3)</td>
<td>100.0 (24/24)</td>
</tr>
<tr>
<td>III</td>
<td>12 (19.7)</td>
<td>66.7 (8/12)</td>
</tr>
<tr>
<td>IV</td>
<td>7 (11.5)</td>
<td>42.9 (3/7)</td>
</tr>
<tr>
<td>Total</td>
<td>61 (100.0)</td>
<td>86.9 (53/61)</td>
</tr>
</tbody>
</table>

*Success rate = percentage of patients requiring only initial UAL/SAL with no further surgery. UAL, ultrasound-assisted liposuction; SAL, suction-assisted lipoplasty.

**ACKNOWLEDGMENT**

REFERENCES

1. Webster, G. V. Gynecomastia in the Navy. The Military Surgeon November 1944.

46. Botta, S. A. Alternatives for the surgical correction of