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Editorial

ENT SURGEONS SHOULD BE PUBLIC HEALTH SPECIALISTS

ENT is a specialty pregnant with opportunities for addressing public health issues. Acute respiratory infections (ARI), embracing rhinitis, sinusitis, tonsillopharyngitis, adenoid disease, laryngitis and otitis media, has never left the top ten morbidity list of the country. Cancers, including head and neck malignancies, have also inched their way up the top ten causes of mortality, reflecting the growing incidence of non-communicable diseases that accompany industrializing societies. Industrialization also brings with it occupational disorders among which are noise-induced hearing loss as well as allergic and non-allergic rhinosinusitis. The upsurge in accident rates further supports the existence of a transition epidemiologic state upon us and should warn us of more maxillofacial trauma cases within the next years.

We should recognize and use the public health components of these disorders to both prevent and treat them. This approach should be logical enough, considering that current research has demonstrated more and more environmental, behavioral, socio-economic, and occupational risk factors in disease causation. The multifactorial nature of otitis media is well known. Primarily infectious in origin, we now know that familial and racial predispositions provide a fertile soil for the interplay of poor living conditions, inadequate nutrition, passive smoking, allergens and other insults. The contributions of the Epstein-Barr virus, racially determined nasopharyngeal vault size and smoked fish ingestion to the development of nasopharyngeal carcinoma are just as illustrative.

However, we, as specialists, tend to leave patient counseling, family therapy and behavioral modification up to general practitioners. Too often we fail to seize the opportunities presented to us by routine patient consult to advise patients how to complete their treatments, quit smoking, eat sensibly, exercise regularly and avoid health hazards at work. Lack of knowledge, inclination or time may be the culprit here. Those of us who would insist on evidence could ask for proof that addressing preventative aspects of disorders would truly lead to better patient outcomes. And there are many of these. In Africa alone, significant improvements in life expectancy have resulted from increasing primary education[1] . Counseling patients with streptococcal sore throats plus written instructions detailing the need to consume all pills have been shown to be more effective than usual care in promoting patient adherence and treatment outcomes[2] . Similarly, patient rewards and family therapy have been found to be helpful in treating patients with hypertension and schizophrenia.

Admitting a more public health-oriented perspective in our specialty requires both collective and individual effort. As a specialty, we can promote the development and adoption of clinical practice guidelines as tools to improve patient service. Our locally developed guidelines in otitis media, adenotonsillitis and rhinosinusitis can be used to inform decisions made by our patients and policy makers in

accessing health care. We can thus contribute to the rationalization of our public health policies. We can also advocate for the adoption of public health measures to address ENT disorders.

We can add our voice to the clamor against smoking, armed with evidence of its contribution to the development of otitis media, rhinosinusitis and oropharyngeal and laryngeal cancer. We can issue public pronouncements against the indiscriminate use of decongestants-antihistamine combinations as well as antibiotics in managing acute respiratory infections and otitis media, basing our action on the results of several randomized controlled trials and systematic reviews that demonstrate their uselessness[3,4,5].

We can support Department of Health programs and work with private and public schools to screen for hearing disability. We can lobby in Congress for the protection of the rights of and promotion of the welfare of deaf people through legislation. Or add our collective voice to the Clean Air Act, citing the ill effects of pollution on the upper airway. We can help clarify the government's iodination program and point out that neither iodine nor hormone suppression has been found to be reliably effective in shrinking thyroid nodules. We can take the cudgels for millions of laborers who endure noise-saturated, poorly ventilated, chemical-choked factories without the benefit of hearing protection or air filtration devices.

A more public health oriented specialty should also work for the provision of ENT specialists in the major islands of the archipelago. We must develop a master plan for ensuring availability of and access to quality ENT care. Previous experience in other countries have shown that merely increasing the number of doctors do not improve equitable distribution of health care. We must set optimum ENT specialist : population ratio targets. We must nurture, through training consortia, networking and referrals, our provincial training institutions and new diplomates who choose to set up practice in rural settings. We must sustain their efforts to provide more ENT care to those who have less in life by organizing continuing education programs that update their knowledge and skills. Again, by systematically tracking and evaluating the medical evidence for the effectiveness of different ENT interventions and disseminating these in the forms of clinical practice guidelines and reviews, we can rapidly bring the practice of even far-flung specialists into state of the art levels without their having to spend resources in time-consuming literature searches and expensive ENT conventions.

Such a public health orientation is not without its spillover benefits. Chief among these is public recognition of the intrinsic value of our specialty as an indispensable tool in attaining health for all. If we prove ourselves as specialists constantly in search of improving the quality and relevance of our services through training and research, public relations programs become redundant and turf-protecting unnecessary.

As individual specialists, we must remember that we function within a pluralistic medical system where 70 to 90% of the population heal themselves through traditional means[6]. This means that we must learn to develop and integrate our local and regional health systems within our biomedical

paradigm in order to provide an acceptable and readily available array of ENT services to the remaining third of Filipinos who will decide to consult us. *Akala ko luga lang* [7] graphically demonstrates the clash between indigenous and Western rubrics of the mechanisms and agents of chronic otitis media. This must never be. We should evolve a taxonomy of local terms for ear discharge, search for parallel explanatory models between indigenous health systems and ours, and adapt our immunologic and tubal occlusion concepts into causal theories that our patients will find persuasive and meaningful. Without having to cede their scientific bases, such theories may stand a better chance of engaging the support of our patients' kith and kin and ensuring their adherence to our treatments.

As more and more micro-specialists emerge, the Lamarckian "use it or lose it" possibility becomes imminent. Either we wield the considerable influence that ENT can bring to bear on preventive medicine or lose it to other specialists who have the vision and the energy to serve the real needs of our people. The choice and its consequences, as always, are ours.

Jose Acuin, MD

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Presidential Inaugural Address

MEETING THE CHALLENGE TO CHANGE

Colleagues and friends,

I am not quite sure whether we are one in thinking that the reason why I was luckily elected president of our society is *change*. Six months ago, the distinct honor of being in this position was very remote in my mind. One fateful day, while friends were talking about the necessity for change in our 43-year old society, I was challenged by the idea that I could be an instrument of change.

It is common knowledge that in an organization some members will favor change while others will resist it. That is quite natural for a very active organization such as ours. I believe it is a healthy sign of an organization that is alive, one which desires to grow and move forward. Our membership has grown so rapidly over the last ten years. Access to information has multiplied a thousand fold and the desire for communication seems limitless. And our concerns have likewise multiplied so many times. It is thus inevitable that change would really come our way.

But more than being an agent of change, what gave me greater challenge was the idea of stimulating change and at the same time getting the cooperation of those who resist it. Of being able to harmonize the concerns of those who favor change and those who oppose it. And at the end of the day we can all happily say that what happened was what everyone exactly had in mind. For as one wise man said, "change is just a perception". What constitutes change for one may be just a natural consequence of events for another. Hence, I am challenged to make others see what some think as drastic change in our society as just a natural course of events.

While reviewing the pertinent organizational papers of our society, I noticed that we have never redirected our vision and mission through the years. Perhaps our previous leaders had redirected them in their minds. But nothing was written about them. Nor were they disseminated and made known to the growing membership. Hence, few, if ever, understood those noble visions. As a consequence, only few probably got interested in them. It is thus one of our tasks to write about the future directions of our society, get the opinions, suggestions and comments of the general membership and try to align each member's behavior and beliefs to the commonly agreed upon vision and mission. That may not only clarify our purpose but it may also give us more meaning as an organization. Perhaps such strategy will equip us better to meet the challenges of the next millenium.

Meanwhile, I believe we have to do some organizational realignment. I agree that reorganization brings some negative feelings. A Spanish proverb says, "Cada cura con su locura". In Tagalog, this may be translated, "Bawat bagong pari, may kanya-kanyang pakulo". However, the realignment that I have in mind is one that is arrived at by consensus and in my term we shall always try to achieve things by consensus- a meeting of the minds after due discussion and deliberation. The growing needs and concerns of our members simply call for greater involvement not only among the officers but also from the enthusiastic members as well. Thus, I think it is about time that we empower our members not only in planning and implementing our activities but in making decisions as well. We can thus harness the limitless potentials of our members who are truly committed to the growth and development of our society.

I therefore thank our members who have accepted their responsibilities to be the chairperson and members of the different standing and interim committees and subcommittees. Their enthusiasm further convinces us that we indeed need change, that change is a welcome development and that being an agent of change is quite an honor and a challenge.

Let us therefore work together for the present and the future of our society. Together we will accomplish much.

Generoso T. Abes, MD

HOW ENTs CAN SURVIVE THE Y2K BUG

The year 2000 starts in only 8 months or so, and with it comes the dreaded Year 2000 or Millenium Bug. Also known as the Y2K Bug, it is the expected malfunction of certain computers on January 1, 2000 and thereon, computers that are dependent on the date for their function and operation. That is to say, if they are not Y2K compliant. The problem involves not only their hardware (the stuff you see when you open a computer's cover: the electronics) but also their software (the instruction codes that tell the computer how to do its job, which is to perform a series of arithmetical operations on data fed to it in the form of numbers) as well.

This whole problem started as a result of programming mainframe computers in the 1950s and 60s when computer memory was very expensive. Today you can buy 32 megabytes of memory for around $40 but in the 1960s, 1 megabyte cost around $1 million ! Input devices then did not use the modern keyboard and mouse but more typically, punched cards. (I still remember my pre-medicine days in the early 1970s when our class cards were punched with holes to encode our names, course, subject and other data relevant to the university.) Since punched cards had space limitations on how much data could be encoded in them, and to save on precious computer memory, programmers in those times shaved off the first 2 digits of the year when encoding this information into the computer. Therefore, the year 1972 was encoded as 72.Everybody including the computer knew that it was 1972. Programmers thought that their programs and computers would be used for only some years, become obsolete, and then be replaced by cheaper and more capable equipment that used the full 4 digits for the year.

They were only partly right. Computer technology has indeed grown by leaps and bounds and many of them are designed to read the year 2000 correctly. But the reality is that many of those old mainframe computers are still around and plugging along as they did 40 years ago. Thus when the year enters 2000, they may think that it is 1900 and get confused, stop working ("crash"), or put out incorrect information. This is a BIG problem for banks, insurance companies, government agencies, national defense, airlines – any organization that uses such computers dependent on the date and year to compute things as the payroll, retirement benefits, loans, airline reservations, even navigation of ships.

The problem is hard to fix because no 2 systems are exactly alike. Current mainframes are complex, custom made hybrid designs built up incrementally over the years. More bad news: their software were written by different programmers who wrote with different styles and flavors, and therefore lines of code are unique, custom-made, and may combine they use of many "languages". No one kept precise records regarding these codes, and the programmers who wrote them are now either dead or unavailable to help fix the system. Still more bad news: the extensive use of "embedded chips" or integrated circuits later inserted into the computer's circuits encoded with hardware information like dates, which help the computer do its job. These chips are not readily accessed by software. Finally, an agency's computer may be Y2K compliant but other network computers connected to it – those that supply it data - may not, and effectively sabotage its computing.

The doomsday sayers paint a bleak picture when year 2000 comes : credit card companies that refuse to honor credit cards that expire in 2000, failure of power and water companies to maintain services, people not getting paid their money from the banks, employees that do not get their retirement benefits because the computer thinks they have not yet been born, commercial airplanes that crash because their navigation systems have, well, crashed, and a medical program that computes a drug dosage for an infant born in 2000 as that for a large 100 year-old man. There are reports in the USA where people have stockpiled canned goods and firearms in the mountains where they can hide out for many months while the rest of civilization experiences anarchy in the confusion and chaos that result from computer malfunction.

Personally, I do not think that any of these it's-the-end-of-the-world predictions will happen. Many agencies are exerting considerable effort to make their computer

systems Y2K compliant, and most of the new computers of today are not affected by the Y2K Bug. Incidentally, Apple MacIntosh computers are, and have always been immune to the Y2K Bug.

For sure, not all of the world will be able to fix their computers on time for the turn of the century. But for us Filipinos, I envision only *some* effects from the Millenium Bug. My programmer friend from MERALCO assures me that they are Y2K compliant already, and banks are asserting the same. There will be some inconveniences for most of us, but none of us can afford to be caught unprepared when January 1, 2000 arrives. For us that regularly use computers, some Internet Web sites offer test programs you can download. These can automatically test your computer's BIOS, installed software and data, and diagnose those areas which are not Y2K compliant. Many of these companies that produced these test programs also offer fixes that automatically correct, for a fee, the software problems and make your computer Y2K compliant.

About a week before the year 2000 arrives, it is also advisable to have updated printed copies of bank, investment and credit card accounts. While bank officials claim that their systems are ready for the turn of the century, having your own copies will help iron out any difficulties more quickly. It is also advisable to obtain a copy of your credit card reports before September 9, 1999, after September 9, 1999 but before December 31, 1999, and after January 1, 2000. Check out these documents for errors. September 9, 1999 (09/09/99) is a problem date because some programmers use "9999" as a signal to reset the system. Also keep printed copies of your different types of life, fire, theft, and pre-need policies with their expiration or maturation dates printed on them.

Have 2 or 3 weeks' worth of cash on hand at the end of the year, just in case ATM machines malfunction. For the same reason, keep a 2-weeks' supply of food and other consumables such as necessary medicines in the house. This is not to say that you might withdraw all your money from the banks at the end of the year for fear that the banks might foul up their records and not allow you access to your funds later. If anything at all, do not hoard supplies ! There are limited stocks of everything and if everyone were to hoard, there will definitely be an artificial shortage of goods. We want to spread the word around that panic-hoarding is what we want to avoid in the first place. Keep your supply just enough for your and your family's needs.

Finally, submit medical claims and other requests for benefits from the Social Security System as early as possible. Keep copies of income tax payments and SSS contributions. We do not really know if the government and its agencies are Y2K compliant.

Not many Filipinos are aware of the Y2K Bug and how it may seriously affect their lives, finances and daily routines. Being prepared is perhaps the best thing anyone can do to make the transition to 2000 as smooth and trouble-free as possible.

Bernardo D. Dimacali, M.D.

Original Studies

THE EXTENDED ISLAND TRAPEZIUS MYOCUTANEOUS FLAP: LOCALIZATION OF THE VASCULAR PEDICLE

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Objectives: To identify the location of the transverse cervical artery using fixed, superficial anatomic structures as landmarks and to propose a working guideline in the preservation of the vascular bundle while harvesting of the extended island trapezius myocutaneous flap
Design: Series of cadaver dissections
Setting: Tertiary hospital and a government recognized local funeral parlor.
Materials: 18 adult cadavers providing a total of 36 trapezius muscles with no gross deformity and without history of musculo-skeletal disorders.
Results: The average location of the transverse cervical artery is at the junction of the medial 3/5 and lateral 2/5 along the width of the trapezius muscle.
Conclusion: The transverse cervical artery which provides the dominant vascular supply to the pedicled extended island trapezius myocutaneous flap can be located at the junction of the medial 3/5 and lateral 2/5 along the line drawn between the spine of the 7th cervical vertebra and the acromio-clavicular joint. This location can be utilized in the harvesting of the extended island trapezius myocutaneous flap with minimal post-operative complication on the donor sit.

INTRODUCTION

Reconstructive surgery dates back to the 19th century when defects of the head and neck necessitate reparative procedures for functional and aesthetic reasons. It has, however, underwent an unparalleled rebirth in the late 1970s with the advent of myocutaneous flaps. In the process of its resurgence, the pectoralis major muscle has proven its worth, time and again, to become the "workhorse" of head and neck reconstruction. There are certain reconstruction dilemmas nonetheless that favor the utilization of other myocutaneous flaps which will best serve this purpose.

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In 1977, McCraw et.al.[1] defined the then current concepts and applications of the myocutaneous island flaps based on anatomic and physiologic considerations. Four flaps suitable for head and neck reconstruction are based on latissimus dorsi, pectoralis major, sternomastoid and trapezius muscles, all of which have recognized vascular pedicles. The use of the pectoralis major and the latissimus dorsi flaps have been the subject of several authors' reports in recent literature. The sternomastoid flap has limited use in head and neck surgery because of its location in the operative field. The trapezius myocutaneous flap has been described only recently and its use has not been fully explored.

The trapezius muscle's potential was first described by Mutter[2] in 1842

when he utilized the transfer of a cutaneous shoulder flap for reconstruction of the neck following burn scar contracture, a procedure that was later modified by Zovickian[3] in 1957 for repair and prevention of pharyngeal fistulas using a mastoid-occiput shoulder skin flap. In the following years, attempts had been made to increase the viability of the cutaneous flap and to limit the number of preparatory delay procedures required to ensure survival by elevating the skin in continuity with the underlying trapezius muscle.[4]

The trapezius is triangular, flat and supple muscle of ample size, closely adherent to the overlying skin and richly supplied by al least 4 different feeding vessels. Panje[5] in 1980 described in greater detail the various vascular supplies of the trapezius muscle and its further applications thereby popularizing the trapezius myocutan-eous flap and spearheading a broader acceptance of this technique. In the same year Baek et.al.[6] utilized the caudal placement of the cutaneous segment of the flap to overlay the lower trapezius fibers medial to the scapula. This maneuver increased the effective length of the rotation arc of this vascular pedicle and made use of the more inferior fibers of this muscle.

Experimental and anatomic confirmation of the physiologic basis of the trapezius muscle myocutaneous flap quickly followed. Netterville et.al[7] discussed in great detail the three variations of this versatile flap: superiorly based, laterally based and the inferiorly based extended island trapezius muscle myocutaneous flap. He also highlighted areas of particular applicability and the limitations of each type of flap. The utility of this technique in the rehabilitation of a wide range of head and neck defects, including the nose, malar region and orbit has been adequately demonstrated in the literature.

Surgical Anatomy

The trapezius muscle is a broad, flat, triangular muscle covering the area of the back of the neck and shoulders. Its muscle fibers originate in the midline from the occiput and the spinous processes of all the thoracic vertebrae. It inserts on the lateral third of the clavicle , the acromion, and the scapular spine. Motor innervation is via the spinal accessory nerve while sensory innervation is through the upper cervical nerves, C_3 and C_4. The blood supply to the trapezius muscle is variable, but it derives primarily from the transverse cervical artery through its superficial (descending) branch. Branches from the occipital artery, the most consistent among these vessels, along with the posterior intercostal perforating arteries and the dorsal scapular artery provide additional vascular supply. Described in literature, the transverse cervical artery arises from the thyrocervical trunk from the first part of the subclavian artery in most of the cases encountered.

It is the descending branch of the transverse cervical artery that is the major blood supply to the island flap described herein. The descending branch is important in developing the inferiorly based extended island trapezius myocutaneous flap. Colored latex injections of the transverse cervical artery have demonstrated direct musculocutaneous perforators penetrating the anterolateral and inferolateral aspects of the overlying skin.[8]

It is important to mention here that previous studies on the trapezius myocutaneous flap describe a dual vascular supply to the lower half of the trapezius muscle provided by the dorsal scapular artery situated lateral to the

transverse cervical artery, which may be the dominant feeding vessel to the contemplated myocutaneous paddle.[9] It is of critical importance due to its more lateral origin and that it runs a course different and more complex from that of the transverse cervical artery.

Venous drainage is even more variable, but usually is composed of superficial veins in the subdermal plexus and deeper venae commitantes that accompany the perforating arteries. These then drain into the transverse cervical vein, which in most cases accompany the major artery or ocassionally the suprascapular vein inferolaterally.

Pedicled flap survival depends on several factors but what cannot be overemphasized is the preservation of the vascular bundle that will nourish the flap in its new site. Careful planning and harvesting then becomes paramount for successful recon-struction. Studies have described several techniques in terms of vascular pedicle localization for pectoralis major myocutaneous flap but fall short in describing methods specific for the three varieties of the trapezius myocutaneous flap particularly the inferiorly based extended island variant.

It is in this knowledge gap that this study is undertaken. It aims to identify the location of the transverse cervical artery using fixed, superficial anatomic structures as landmarks; and in the process, propose a working guideline in the preservation of this vascular bundle in the harvesting of the inferiorly based extended island trapezius myocutaneous flap

MATERIALS AND METHODS

This is a cross-sectional study involving anatomic dissections performed on 18 fresh and well-preserved cadavers done in a tertiary hospital and a local government recognized funeral parlor. Inclusion criteria included fresh and well-preserved adult cadavers 18 to 60 years of age without gross deformity and without history of musculoskeletal disorders which could possibly alter the anatomy of the back and shoulders.

The anatomic location of the following landmarks was noted: the spine of the7th cervical vertebra, the spine of the 12th thoracic vertebra, the inferior border of the scapula, and the acromio-clavicular joint. The following anatomic parameters were then measured:

1. The *width* or the distance between the acromio-clavicular joint and the spine of the 7th cervical vertebra;
2. The *vascular bundle point* or the distance along the width from the spine of the 7th cervical vertebra to the lateral location of the transverse cervical artery

With the cadaver in the prone position, markers were placed in the above-mentioned landmarks allowing the width to be measured. A vertical skin incision was made from the posterior lower back midway between the medial border of the scapula and the posterior spinal processes from the level of the 12th thoracic vertebra to the level of the clavicle. Medial and lateral horizontal incisions were then made on both the superior and inferior ends of the vertical incision and skin flaps were elevated to expose the lower portion of the trapezius muscle in its entirety.

Using blunt and sharp dissection, the trapezius muscle was then elevated in its undersurface in a cephalad manner starting from its most inferior attachment at the 12th thoracic vertebra. along the fascia of the latissimus dorsi, lesser and greater rhomboids and levator scapulae muscles, identifying these muscles in the process, and freeing the trapezius from its vertebral attachments at the same time. Further dissection totally mobilized the

trapezius muscle, releasing it in all its inferior and lateral attachments. The descending branch of the transverse cervical artery located in the fascia along the muscle's undersurface was identified and followed superiorly to ascertain the transverse cervical artery. At the level of the pre-measured trapezius width, the vessel's distance from the spine of the 7th cervical vertebra was measured and recorded as the vascular bundle point.

The mean distance of these vascular bundle points from the spine of the 7th cervical vertebra was computed to determine the usual location of the transverse cervical artery..

RESULTS

Table 1. Measurement of trapezius width and vascular bundle point (n=36)

	Age	Sex	Trapezius width (cm)		Vascular bundle pt. (cm)	
			R	L	R	L
1	52	M	20.4	20.4	11.9	11.8
2	46	M	19.5	19.5	11.7	11.6
3	28	M	20.2	20.2	11.9	11.9
4	56	F	18.5	18.5	11.3	11.4
5	38	M	19.0	19.0	11.4	11.4
6	51	F	18.3	18.3	11.1	11.2
7	43	F	18.1	18.1	11.4	11.2
8	46	M	19.5	19.5	11.7	11.8
9	39	M	21.1	21.1	12.0	11.8
10	55	M	22.0	22.0	12.1	12.0
11	59	M	20.2	20.2	11.8	11.9
12	50	F	18.5	18.5	11.5	11.5
13	48	F	18.7	18.7	11.3	11.2
14	27	M	19.3	19.3	11.5	11.4
15	53	F	18.5	18.5	11.1	11.3
16	55	M	20.6	20.6	12.0	12.1
17	50	F	17.2	17.2	10.9	10.7
18	58	F	18.5	18.5	11.3	11.3

A total of 18 cadavers were dissected (10 male; 8 female) providing 36 trapezius muscles for the sample size. The average age is 47 years (Table 1).

The transverse cervical artery is located by the average at the junction of the medial 3/5 and lateral 2/5 along the width of the trapezius muscle. It is in this point that the transverse cervical artery can most often be identified coursing perpendicular to the line drawn from the spine of the 7th cervical vertebra to the acromio-clavicular joint as it runs along the undersurface of the trapezius muscle.

The mean trapezius width was 19.34 cm. The mean distance of the vascular bundle point from the spine of the 7th cervical vertebra was 11.54 cm

The ratio of the mean vascular bundle point to the mean trapezius width was 11.54 / 19.34 or 60%

Thus, the average distance from the spine of the 7th cervical vertebra to the location of the transverse cervical artery is at a point 60% or 3/5 of the line representing the trapezius width.

DISCUSSION

Myocutaneous flap reconstruct-ion has received much popularity in the past 2 decades. The pectoralis, the sternomastoid, and the latissimus dorsi myocutaneous flaps have been used successfully in various head and neck reconstruction procedures. However, when approaching the region of the middle and/or upper face, the distance between the defect and the donor site is longer than the muscle pedicle. There is also the problem in undermining skin especially in cases where radical neck dissection was not performed. The muscle pedicle of these flaps also leaves a bulky deformity in the neck. With these in mind, utilization of the trapezius flap is an excellent alternative.

The trapezius muscle is thin and flat with sparse subcutaneous tissue providing an ample source of thin, supple, hairless skin of uniform thickness. Especially for the inferiorly based extended island flap, the length and thickness of its pedicle allows excellent mobility, can be easily passed through normal neck and leaves minimal bulky cosmetic deformity. It is also an excellent choice in recon-struction for patients undergoing ablative surgery including resection of the spinal accessory nerve. The use of the pectoralis major myocutaneous flap produces the added disability to the shoulder function that is encountered with the loss of both muscles.

In addition, the trapezius flap is more readily tubed making it an excellent candidate for reconstruction of circumferential pharyngeal defects. Finally the inferiorly based flap does not disrupt the anterior aspect of the chest, which would benefit women. The disadvantages of the inferiorly based trapezius flap include the positioning of the patient during harvesting and the resulting shoulder deformity. The most important drawback of this technique is the variable vascular anatomy.

Thus, knowledge of the vascular anatomy of the trapezius muscle, due to its variations, is of crucial importance for the successful utilization of the inferiorly based extended island myocutaneous flap.

Of the four previously men-tioned vessels, the transverse cervical artery is the most critical and variable. It most commonly originates medially from the region of the bifurcation of the thyrocervical trunk. This was noted in 77% of the cases included in this study.

It may also arise directly from the suprascapular artery near its origin in the thyrocervical trunk (noted here in 17%) or directly from the first part of the subclavian artery near the base of the tyrocervical trunk (noted in 6% of the cases).

The transverse cervical artery arising from any of these locations has the same clinical presentation. It originates inferomedially in the neck, passes lateral to the phrenic nerve, the brachial plexus, the scalenes, then deep to the omohyoid muscle. Subsequently, it crosses the lateral triangle and enters the trapezius muscle near its midpoint, close to the entrance of the spinal accessory nerve It finally divides into an ascending and descending branches. In some instances, this artery tapers quickly, allowing the descending branch of the dorsal scapular artery to supply the lower trapezius muscle.

Less commonly, the transverse cervical artery originates laterally either from the dorsal scapular artery or directly from the 2nd or 3rd portion of the subclavian artery. It then passes under the middle trunk of the brachial plexus. It continues dorsally, passing under the medial edge of the scapula at the insertion of the levator scapulae superioris muscle. At this point the artery passes laterally either between the levator and the lesser rhomboid muscle or inferior to the lesser rhomboid muscle to enter the deep surface of the trapezius muscle. This descending branch lies along the medial border of the scapula superficial to the greater rhomboid muscle.

The transverse cervical artery or a smaller supplemental branch to the lateral trapezius fibers may send off the dorsal scapular artery at any point along its path. Two points should be noted here. The arc of rotation of the flap whose transverse cervical artery originates in the lateral position may be limited by confinement under the brachial plexus. If the artery also passes under the levator scapula and lesser rhomboid muscles, a microvascular transfer of the flap might be necessary to salvage its use. Otherwise, it may be

possible to transect the lesser rhomboid to free the vascular pedicle, thereby increasing the rotation length.

Identification of the dominant feeding vessel to the inferiorly based extended island trapezius myocutan-eous flap during flap harvesting is paramount to flap survival. Preoperatively, angiography can be utilized to determine the viability of this vascular pedicle but it almost always means additional diagnostic cost as well as subjecting the patient to a tedious procedure. The same can be said about the doppler technique and it is not as graphic as an angiogram.

Localization of the transverse cervical artery, the most common dominant vascular supply to the inferior fibers of the trapezius muscle in 80% of cases, utilizing fixed, superficial anatomic landmarks offers an ideal, economical and more plausible alternative when contemplating an inferiorly based extended island trapezius myocutaneous flap. It is also important to mention that there was no significant difference in the male and female cases in the study as far as the location of the transverse cervical artery is concerned.

Careful identification of these landmarks and accurate measurement gives a two-dimensional picture of the course of the transverse cervical artery thereby guiding the surgeon in designing the flap. Utilizing this technique in the harvesting of the inferioly based trapezius myocutaneous flap provides the advantage of limiting the incision and elevation of the lower portion of the muscle to the size required for reconstruction. It also spares the surgeon the task of mobilizing the entire lower portion of the muscle from all its attachments. In this manner, post-operative complications can be limited.

When the transverse cervical artery is properly identified, the muscle fibers can be divided both medial and lateral to the vessel leaving at least 2 cm cuff of muscle on either side of the artery, which will further debulk the flap's muscular pedicle.

The possibility that the dominant vessel comes from the dorsal scapular artery cannot be overlooked. Thus it is suggested that the segment of the muscular pedicle immediate to the island be of considerable width. Since the distance between the descending branch of the transverse scapular artery and the medial aspect of the scapula, outlining the course of the vascular pedicle as it arises from the dorsal scapular artery, is 4 cm, the pedicle of the inferiorly based flap immediate to the island paddle must be at least 8 cm wide to include the area coursed by both vessels. Only when the dominant vessel is properly identified can we taper the width of this pedicle.

CONCLUSIONS

The transverse cervical artery providing the dominant vascular supply to the inferior portion of the trapezius muscle can be located at the junction of the medial 3/5 and lateral 2/5 along the line drawn between the spine of the 7th cervical vertebra and the acromio-clavicular joint.

By utilizing this technique, it is of importance that the flap be incised and elevated from the trapezius muscle without mobilizing the whole inferior portion of the muscle. The medial extent of the pedicle should be at least 2 cm medial to the location of the transverse cervical artery while the lateral extent should be at least 2 cm lateral to the medial border of the scapula. Careful dissection of the facsia in the trapezius muscle's undersurface will yield the dominant feeding vessel to the flap. Only

then can we taper the pedicle's width to a minimum width of 4cm.

Being a pilot study, it is recommended that further investi-gations should be conducted with a larger sample size to establish a norm representative of the population. Correlation may also be evaluated between the measurement of the vascular pedicle with variables such as age, sex, height and weight. Having established these, deviations from the average measurements may need further attention and scrutiny as indications of certain abnormalities among patients.

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Original Studies

COMBINED SUPERO-INFERIOR RELEASE FOR CERVICAL TRACHEAL RESECTION

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The medical and surgical management of patients with airway obstruction is a difficult and challenging problem that stems, in part, from the complexity of both the etiology of the disorder and the medical history of the patients with airway obstruction. This can be classified either as an upper or a lower airway obstruction, but, the main root to this dilemma is still securing a good airway. Tracheal stenosis remains one of the most challenging problems facing an otolaryngologist. Although this is not a common clinical entity, it still presents a significant problem despite recent endoscopic advances. It is therefor the objective of this study to find a new technique of preventing tracheal restenosis and to avoid further complications through early decannulation.

INTRODUCTION

Tracheal stenosis remains one of the most challenging problems facing the otolaryngologist. Recurrent stenosis and disruption of the anastomosis are serious and all too frequent complications of its repair. suture. The most common cause of tracheal stenosis is trauma from an endotracheal or tracheotomy tube placed for prolonged ventilation[3]. The reported incidence following tracheotomy ranges from 0.6 to 21%, while following endotracheal intubation the incidence ranges from 6 to 21%[1]. The most appropriate treatment for circumferential tracheal stenosis, greater than 1cm in vertical diameter and with loss of cartilaginous support is still tracheal resection and end-to-end anastomosis[8]. But, excessive tension on the line of anastomosis would lead to recurrent stenosis and disruption of the sutures. Releasing techniques were therefore introduced to obtain a tension-free approximation of the proximal and distal tracheal stumps.

This paper describes our experience with tracheal resection performed in 2 patients and illustrates
the surgical maneuvers we used to facilitate resection and reanastomosis of the stenotic lesions without undue tension.

CASE HISTORIES

Case No.1:

C.C., a 33-year-old male, was intubated 8 months prior to admission while being managed for complications arising from a gunshot wound on his right thigh. After prolonged intubation, he underwent tracheotomy. He complains of persistent cough productive of whitish to greenish sputum and dyspnea.

On physical examination, a tracheotomy tube was in place and indirect laryngoscopy revealed good mobility of the vocal cords. There was no pooling of saliva at the pyriform sinuses and granulation

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tissue was noted above the tracheotomy site. The rest of the ENT examination was unremarkable.

A laryngeal tomogram revealed tracheal stenosis at level C5-C6 (*fig.1*). CT scan revealed tracheal stenosis involving the segment above the stoma about 2-3cm from the subglottic area, with no evidence of stenosis at the area of the cuff.

Case No. 2:

B.B., a 50-year-old male, ws admitted because of stridor. The patient went into cardiopulmonary arrest three months prior to admission and was intubated for 2 days. He was discharged improved after 4 days. After a month, he developed wheezing and dyspnea on exertion that later progressed to dyspnea at rest accompanied by stridor. Indirect laryngoscopy revealed good mobility of vocal cords. Berci-ward examination revealed tracheal stenosis of approximately 50-70%. A laryngeal tomogram failed to show any tracheal narrowing (*fig.*2) while the CT scan revealed tracheal narrowing of the subglottic area measuring about 9 mm long with around 30% narrowing in its right posterior portion. He underwent emergency tracheotomy 2 days prior to his operation.

Surgical Technique:

Endotracheal anesthesia was used in both cases via the tracheotomy site. The neck was then maximally extended to bring as much of the trachea up from the mediastinum into the cervical area. A horizontal skin incision was made at the level of the tracheal stoma and was carried laterally to the anterior border of the sternocleidomastoid muscle and superiorly to the level of the thyrohyoid membrane to gain access to the suprahyoid area. Inferiorly, the flap was created up to the level of the clavicle.

A vertical midline incision was made through the anterior tracheal wall above and below the stoma until normal-sized lumen was found. The stenotic segment was then resected (tracheal rings 1-4 for the first case, tracheal rings 1-5 for the second case). Care was taken to keep the dissection on the surface of the tracheal rings to avoid injury to the recurrent laryngeal nerves. Also, lateral dissection was limited to the stenotic area to decrease the risk of devascularization of the tracheal stump.

The combined supero-inferior or infrahyoid (*fig.3)* and suprahyoid (*fig.4)* release (*fig.5*) was begun. The body of the hyoid bone was exposed and removed after transecting the muscles attached to its superior surface, its ligamentous attachments to the larynx and its attachment to the greater cornua. The laryngeal drop was accomplished by dissecting the sternohyoid and omohyoid muscles away from the larynx at the level of the thyrohyoid membrane.

The thyrohoid muscles were then incised at the upper edge of the thyroid cartilage to preserve the superior laryngeal nerve, artery and vein, followed by division of the superior cornua of the thyroid cartilage.

The tracheal stumps were approximated. Finger dissection was used to free the anterolateral surfaces of the distal trachea only if tension was present at the anastomotic site. In previous studies, this procedure was usually done prior to any laryngeal release. We avoided dissecting all the way to the carina and preferred limited dissection of the distal trachea due to potential disruption of its segmental blood supply. This procedure only contributed about 10-15% of the lengthening of the trachea.

An orotracheal tube was inserted past the site of anastomosis so that ventilation was still maintained while anastomosis was being done. End-to-end anastomosis was performed with no.2-0 nylon sutures instead of silk sutures because in our experience, there was more granulation formation after using silk sutures. At this time, the neck was partially flexed to again lessen tension at the area. Posteriorly, sutures were placedthrough the full thickness of the trachea, including the mucosa and submucosal layers. Anteriorly, sutures were placed submucosally, making sure no sutures were in the tracheal lumen. Likewise, previous experience made us more confident of use of the submucosal suturing instead of through and through suturing, done by the other authors, due to lesser tissue reaction with this maneuver. The sutures were placed first on one side then the other side of the ET tube going anteriorly. All sutures were left untied until the tracheal stumps were satisfactorily positioned. Three additional sutures were placed submucosally about 1-2 tracheal rings above and below the anastomosis for support. All sutures were then tied starting with the posterior inner sutures making sure all knots were not lying inside the tracheal lumen (*fig.6*).

A nasogastric tube was inserted for postoperative feeding. The patient was then carefully introduced to oral feeding after 2 weeks. Suction drains were placed and brought out laterally though the neck. The wound was then closed in layers.

For 10 days, the neck was placed in maximum flexion using heavy nylon sutures from the chin to the chest skin to add to support of the anastomotic site and prevent hyperextension of the neck. The patient was extubated when fully awake after 24 hours, earlier than in other studies to lessen irritation to the anastomotic site that may lead to re-stenosis. Medimist inhalation was given to lessen the formation of crusts at the anastomosis.

Antibiotics were given post-operatively and prednisone was administered for one week to prevent swelling without impeding healing.

After 2 weeks, we trained the patients to take first liquids then gradually solids by mouth. Transitory dysphagia may be encountered at this time because the combined supero-inferior release would cause a 20-30% tilt of the aerodigestive tract anteriorly and downwards. With proper training and reassurance, the patients would often be able to overcome this symptom.

DISCUSSION

As a complication of assisted ventilation, stenosis might occur in the trachea and subglottis. Although, tracheal stenosis is not a common clinical entity, it still presents with difficult management problems[7]. The best treatment for patients with circumferential stenosis of the cervi-cal trachea is a sleeve resection of the sten-otic portion and end-to-end anasto-mosis[8].

Restenosis and suture disruption results if tension on the suture line develops. To avoid these complications, numerous techniques as well as release procedures have been advocated for gaining additional tracheal length at the time of tracheal anastomosis (*table 1*)[3]. Basically, these are divided into infra-laryngeal and supralaryngeal methods[2].

Thoracic infralaryngeal techniques, advocated by Grillo, involve mobilization of the right hilum, division of the inferior

Table 1. Mobilization Procedure and Relative Lengths Gained

Procedure	Grillo	Montgomery	Miller	Dedo and Fishman
Neck Flexion	4.5-6 cm		5-6 cm	
Suprahyoid release	1.5 cm	5 cm	2.5 cm	
Infrahyoid release			2.5 cm	2.5 cm
Anterior / Posterior tracheal dissection			1.5 cm	1.5 cm
Mobilize right hilum	1.5 cm		3 cm	
Dissect pulmonary artery / vein	1.0 cm	6 cm		
Reimplant left main stem bronchus	2.5 cm		2.5 cm	

pulmonary ligament and intrapericardial dissection of the pulmonary vessels. The left main bronchus is then sectioned distal to the carina and re-implanted to the right bronchial system by end-to-side anastomosis. This procedure carries the danger of mediastinal dissection[2].

Cervical infralaryngeal techniques, described by Som, involve the incision of the annular ligaments between the tracheal rings[2]. Complete circumferential section-ing of the ligaments will disrupt the tracheal blood supply that runs in a vertical direction based on the superior and inferior thyroid arteries. A staggered maneuver, that is, contralateral sectioning of the ligaments on either side of the anastomosis, should therefore be used.

The suprahyoid release by Montgomery[4] has been preferred over the infrahyoid release of Dedo and Fishman[5] because of the decreased amount of postoperative tongue base and pharyngeal edema, and the decreased chance of permanent injury to the superior laryngeal nerve.

Complete laryngeal release involves combining all of the features of these two methods as done by the authors. This includes removal of the hyoid bone and eliminating all of the superior muscular and ligamentous attachments of the larynx. Up to 5 cm of additional tracheal mobilization can be obtained with these steps.

The disadvantages of this technique are the modification in the vertical position of the larynx, the disturbance of normal laryngeal elevation during swallowing, and the risk of superior laryngeal nerve injury. These may lead to dysphagia and aspiration, which are transitory[2]. None of our patients complained of such symptoms.

These laryngeal procedures do not suddenly move the larynx to a lower position in the neck. Instead, transection of ligaments, muscles and fascia reduces the amount of recoil that develops if the larynx is pulled toward the mediastinum and then released[3].

Blunt dissection of the anterior and lateral tracheal walls down to the carina is a

maneuver that is generally considered to offer better exposure of the trachea in the mediastinum and to give some relaxation allowing temporary traction of the distal trachea superiorly during traction[3]. This procedure is usually done prior to any laryngeal release. We think that it offers only little extra length and may lead to tracheal necrosis due to disruption of the tracheal blood supply. we therefore suggest avoidance of this procedure or, if still needed after laryngeal release, limiting this form of dissection.

If tension is still encountered at the anastomotic site after supralaryngeal release, this maneuver can be done only up to the point where tension is released so as not to interrupt the blood supply which may lead to necrosis.

Neck flexion is considered as a short-term guard against excessive tension in the anastomosis during the early phases of healing but not as a long-term preventive measure[3].

We would also like to suggest the use of nylon sutures during anastomosis instead of silk sutures. These sutures must also be placed submucosally without entering the tracheal lumen to lessen the emergence of granulation tissue.

CONCLUSION

The combined supero-inferior release without extensive thoracic dissection is an effective means to reduce tension. Dysphagia may be problematic but only transient. Blunt dissection of the anterior and lateral walls of the distal trachea down to the carina is performed only if tension is still present. Nylon, instead of silk sutures is preferred and should be placed submucosally because it produces less tissue reaction. Early decannulation should likewise be done to prevent irritation at the anastomotic site and therefore avoid postoperative complications.

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Original Studies

EFFECTS OF RETINOIDS (TRETINOIN) ON THE HEALING OF TYMPANIC MEMBRANE PERFORATIONS IN RABBITS

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Objectives: To determine the effectiveness of tretinoin in the closure of tympanic membrane perforations in rabbits and to describe the histologic changes in tympanic membranes treated with tretinoin
Design: Open trial
Subjects: 21 healthy adult rabbits
Outcome measures: Proportions of rabbit tympanic membranes that closed on the 9th, 18th and 27th day after perforation and thickness of tympanic membranes
Results: There were no differences in proportions of tympanic membrane closure among tretinoin-treated and untreated rabbits on the 9th, 18th and 27th day. All tympanic perforations were closed by the 27th day. Tretinoin-treated tympanic membranes tended to be thicker than untreated membranes. The kind and amount of cellular infiltrates were similar between tretinoin-treated and untreated membranes.
Conclusion: There is no added benefit conferred by tretinoin treatment of tympanic membrane perforations in rabbits.

INTRODUCTION

The tympanic membrane is a membranous partition separating the external acoustic meatus from the tympanic cavity.[9] It is composed of three layers. The lateral layer is continuous with the skin lining the external auditory meatus. Medial to this is a fibrous layer or lamina propia composed of outer radial fibers and inner circular fibers.[10] Transversely and parabolically oriented fibers intertwine these two layers. The third layer is the mucosal layer continuous with the tympanic cavity.

Tympanic membrane perforations commonly result from trauma, infection or myringotomy tube placements. This perforations heal by re-epithelialization and fibrous layer proliferation.[1] Studies have shown the beneficial effects of retinoids, tretinoin in particular on the skin. These effects included increase in the speed with which the surface cells are replaced, increase in the overall thickness of the epidermis, increase in the activity of epidermal cells and increase in the production of collagen in the dermis.[4]

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Because of this retinoids are often used to improve the appearance and texture of the wound healing in full skin. Studies on retinoids also have proven its effects in dramatically accelerating thickness wounds.[11] Whether retinoids exert similar effects on the healing of tympanic perforations have not yet been determined.

The objectives of this paper are (1) to compare the effects of retinoids (tretinoin) on the closure of tympanic membrane perforations with untreated tympanic membrane perforations in rabbits, and (2) to describe the histologic changes in tympanic membranes exposed to tretinoin.

MATERIALS AND METHODS

Twenty-one healthy adult rabbits contributing 42 intact tympanic membranes were used. The animals were anesthetized using 25mg/kg ketamine hydrochloride IM.[12] In each tympanic membrane, a small perforation measuring approximately 25% of the tympanic membrane was made over the postero-inferior quadrant using a Storz House Sickle Knife (N1705-H). For all the rabbits, all 21 perforated tympanic membranes on the left were allowed to heal spontaneously while all 21 perforated tympanic membranes on the right were treated with tretinoin 0.025% (Derm A USIP). The tretinoin cream was applied directly over the tympanic membranes using a cotton applicator every three days. The viscosity of the cream maintained the tretinoin solution over the tympanic membrane.

Regular examinations of the tympanic membranes via otoscopy (Welch Allyn Otoscope) were done prior to the application of the tretinoin and closure of perforations were recorded. No infection was noted over the tympanic membranes. Seven rabbits were then sacrificed using ketamine hydrochloride (Calypsol 500mg/10mL) 50mg/kg IM on the 9th, 18th, and 27th day.[12] The tympanic membranes were then harvested under magnification 1.5x (Welch-Allyn Lumiview) using microotologic surgical instruments and preserved in formalin 10%. Histologic examination of the tympanic membranes were eventually done. Four normal tympanic membranes from 2 rabbits were also harvested and examined histologically as controls.

RESULTS

After 3 days of tretinoin application, the tympanic membranes had more erythema compared to the untreated tympanic membranes. No observable infection was noted on the perforated tympanic membranes. By the 2nd week of application, there was much less erythema noted on the tympanic membranes.

On the 9th day of this study, the closure of the tympanic membrane perforation was achieved in 57% (12/21) of the tretinoin treated and 47% (10/21) of the untreated animals (Table 1). On the 18th day of this study, closure of the tympanic membrane perforations was achieved in 85% (11/13) of the tretinoin treated and 92% (12/13) of the untreated animals (Table 2). On the 27th day of the study, both groups achieved 100% closure, although 2 deaths occurred with this set, the tympanic membrane perforations were

closed during examination at the time of death (Table 3). At all time points, the differences in the 2 groups were not statistically significant.

Table 1 Tympanic Membrane Status by Treatment Group (9th day)

	Perforated	Healed	Total
w/ tretinoin	9	12	21
w/o tretinoin	11	10	21
total	20	22	42

p.= 0.76

Table 2. Tympanic Membrane Status by Treatment Group (18th day)

	Perforated	Healed	Total
w/ tretinoin	2	11	13
w/o tretinoin	1	12	13
Total	3	23	26

* 1 rabbit died from unknown cause prior to 18th day
p=1.000

Table 3. Tympanic Membrane Status by Treatment Group (27th day)

	perforated	healed	Total
w/ tretinoin	0	5	5
w/o tretinoin	0	5	5
Total	0	10	10

* 5 rabbits died from unknown causes prior to the 27th day however, examination of the tympanic membranes during the time of death showed complete closure.

Histologic Observations:

At 9th day, sections of tympanic membranes not treated with tretinoin showed minimal to moderate infiltrates of neutrophils and few lymphocytes in the subepithelial layer and scattered in the fibromyxoid layer. Few fibroblasts were seen. Tympanic membranes treated with tretinoin showed neutrophilic and lymphocytic infiltrates mostly in the subepithelial layer with few fibroblasts.

Table 4. Mean Thickness of Tympanic Membrane Layers (9th day)

	Without tretinoin	With tretinoin	control
Epithelial layer	0.085um (94%)	0.125um (139%)	0.09um
Fibrous layer	0.20um (67%)	0.20um (67%)	0.30um
Total of 2 layers	0.285um (73%)	0.325um (83%)	0.39um

The epithelial layer was thicker than both control and untreated subjects and attained even 139% of the thickness of the control epithelial layer. The fibrous layer however was similar in thickness in both groups. Total thickness attained by the 2 sublayers in relation to the control was 73% for the untreated and 83% for the treated subjects.

At the 18th day, tympanic membranes without tretinoin had infiltrates that are denser and mostly composed of lymphocytes and plasma cells. Few neutrophilic infiltrates were noted. Tympanic membranes treated with tretinoin had lymphoplasmacytic

infiltrates that are moderate in amount and accompanied by fibroblastic proliferation.

Table 5 Mean Thickness of Tympanic Membrane Layers (18th day)

	without tretinoin	With tretinoin	control
Epithelial layer	0.08um (89%)	0.09um (100%)	0.09um
Fibrous layer	0.175um (58%)	0.20um (67%)	0.30um
Total of 2 layers	0.255um (65%)	0.29um (74%)	0.39um

The epithelial and fibrous layers were thinner compared with the previous group (day 9). This corresponded to an overall decline in thickness.

After 27 days, non-treated membranes had minimal lympho-plasmacytic infiltrates. Those treated with tretinoin had less inflammatory reaction with a fibrocollagenous middle layer.

Table 6 Mean Thickness of Tympanic Membrane Layers (27th day)

	Without tretinoin	With tretinoin	control
Epithelial layer	0.075um (83%)	0.11um (122%)	0.09um
Fibrous layer	0.19um (63%)	0.25um (83%)	0.30um
Total of 2 layers	0.265um (68%)	0.36um (92%)	0.39um

At the 27th day , the only sublayer which declined further in thickness was the epithelial layer of the untreated subject. The rest of the sublayers were thicker compared with the 9th and 18th day subjects. Overall thickness of the 2 sublayers however showed an increase in both groups.

Table 7 Total Thickness Relative to Control of Tympanic Membrane by Treatment Group:

	day 9	day 18	day 27
Without tretinoin	.285/.39um 73%	.255/.39um 65%	.265/.39um 68%
With tretinoin	.325/.39um 83%	.29/.39um 74%	.36/.39um 92%

The tretinoin treated subjects had generally thicker tympanic membranes than the untreated subjects and attained near normal thickness at day 27.

DISCUSSION

Closure of tympanic membrane perforations proceeds in an established pattern, which has been studied in animal models. The mechanism is described as epidermal migration that begins at the margin of the defect and leads to closure of the perforation.[2] A delayed fibrous reaction then occurs. The development of the fibrous layer may be lacking, resulting in a thin replacement membrane.[3] The mucosal layer does not appear to play a prominent role in the closure of the tympanic membrane perforations.[2]

Upon gross histologic measurement using the micrometer, the tympanic membranes of the tretinoin treated membranes tended to be thicker as would be expected from the epithelial hyperplasia that retinoids have shown to induce in previous studies. This result suggests the role of retinoids in the repair of tympanic membrane perforations. It can theoretically promote repair by increasing the epidermal proliferation rate.[6]

Retinoids are related to vitamin A which is a necessary nutrient for growth, vision, reproduction, and the maintenance and differentiation of epithelial tissue for all vertebrates.[5] In mammals, vitamin A activity is fulfilled by three major compounds, retinol, retinal and retinoic acid, and by some of their metabolites. The term retinoid refers to the entire group of compounds that includes retinol and its naturally occurring and synthetic derivatives. All-trans retinoic acid (tretinoin), is a naturally occurring metabolite of retinol. Retinoic acid is an important oxidative metabolite of retinol that can substitute for some vitamin A functions[6]. In vitamin A deficient animals, retinoic acid can replace retinol in supporting growth promotion and in the maintenance and differentiation of epithelial tissues.[7] Retinoic acid enhances the DNA stimulatory effect of epidermal growth factor[13] in vitro. In related studies, examination of skin biopsy samples from tretinoin managed areas yielded significant histologic results[15]. Mean epidermal thickness in forearms increased by 82% with tretinoin versus 8% with vehicle (control) cream. Similarly, the number of cells in the granular layer increased by 117% with tretinoin versus 18% with the vehicle cream. The mitotic rate, as measured by the number of mitotic figures in the epidermis, increased after tretinoin therapy. In most patients, tretinoin induced a compaction in the stratum corneum epidermidis and an alteration from a basket weave pattern of the stratum corneum to a more homogenous appearance. A glycosaminoglycan-like material appeared in the intercellular spaces of the stratum spinosum epidermidis and in the lower portion of the stratum corneum epidermidis with tretinoin therapy. This finding was not seen with the vehicle cream. Additionally, the tretinoin-treated areas showed wider vascular lumens. Under the basal lamina, the collagen of the papillary dermis substantially changed. New collagen was apparently generated and produced by tretinoin induced-fibroblasts that appeared in greater numbers and showed greater activity than the skin managed with vehicle cream.

Adverse side effects may attend the application of retinoids (tretinoin) especially if systemically administered. These include xerosis, liver toxicity, bone pain, signs of increased intracranial pressure and teratogenicity which is by far the most serious of all retinoid side effects[14]. However, this study involved topical tretinoin which may have some hypersensitivity reaction. Tretinoin also renders the skin sensitive to sunlight but this is irrelevant with tympanic membranes usage. Cholesteatoma formation is a more serious concern but was not observed in this study. Induced

proliferation of the epithelial layer could increase migration through the perforation and therefore cause trapping of squamous epithelium and resultant cholesteatoma[8].

CONCLUSION

Topical tretinoin does not seem to improve healing of tympanic membrane perforations. However, both groups showed a high incidence of perforation closure. Tympanic membranes receiving treatment initially showed increased thickness most markedly in the epithelial layer although still thinner than normal tympanic membranes.

The possible use of tretinoin in the field of otology may be promising. Although data arising from this study did not show a statistically significant difference in the closure of tympanic membrane perforations, histologically noted differences in the tretinoin treated and untreated subjects suggest some possible clinical application pending further studies. These studies should include application of systemic or topical tretinoin in tympanoplasty grafts, patch tests, and poorly healing tympanic membrane perforations arising from trauma, infections and myringotomy procedures with ventilation tube placements.

Studies with longer term application and larger sample sizes may give more information regarding possible effects or complications.

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CLEFTS BEYOND THE LIP AND PALATE: A Case Series on Facial Clefts

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Objective: To document 4 cases of facial clefts: Tessier nos. 0, 0/14, 4, and 7 cleft, describe the clinical presentation and the management done
Design: Case series
Setting: Tertiary Government Hospital
Subjects: 4 Female Infants
Results: The patient with Tessier No. 0 cleft had an absent philtrum and columella, absent premaxilla, a vestigial nose with no nasal bone, orbital hypotelorism, craniosynostosis, and a presumptive diagnosis of Down's Syndrome. The patient with coexisting Tessier No. 0 and 14 cleft was a newborn female similar to the first case but with cranial involvement of the frontal bone. The patient with Tessier No. 4 cleft had bilateral incomplete cleft lip lateral to the philtrum and cupid's bow, bilateral lower eyelid colobomata medial to the punctum, incomplete cleft palate, and left preauricular skin tags. This case was initially misdiagnosed as a common cleft lip and subsequently managed inappropriately with cheiloplasty. The patient with Tessier No. 7 cleft had a fissure at the right oral commissure, macrostomia, and right preauricular and cheek skin tags. Commissuroplasty using Z-plasty with reconstruction of the orbicularis oris resulted in an adequately functioning competent commissure.
Conclusion: A high index of suspicion for the existence of these rare cases would increase the number of correct diagnoses and proper prognostication and appropriate surgical planning would follow. The potential for misdiagnosis is great for the uninformed clinician, as had happened in the third case.

INTRODUCTION

Cleft lip and palate are common congenital anomalies. The incidence is approximately 1:700 live births [1]. Cases are frequently seen in the clinics. These clefts are familiar enough for surgeons to manage with confidence.

Occasionally, clefts involve defects beyond the lip and palate. Soft tissue defects may extend towards the nose, cheek, or eyelid. Alveolar clefts may extend to the cranium, maxilla, or orbit. These unusual clefts are called facial clefts, cranial clefts, or craniofacial clefts depending on the extent of involvement.

Craniofacial clefts are classified anatomically into 15 locations by Paul Tessier (Tessier Nos. 0 to 14) using the orbit as the reference point [2] (Fig 1a and 1b).

Tessier Nos. 0 to 6 are the facial clefts situated through the lower half of the orbit, lower eyelid, cheek, and lip. Tessier Nos. 8 to 14 are the cranial clefts situated through the upper half of the orbit and upper eyelid.

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Tessier No. 7 cleft is both facial and cranial. A combination of clefts may occur in one patient and the extent of involvement, whether soft tissue and/or skeletal, may vary (Fig. 1a and 1b).

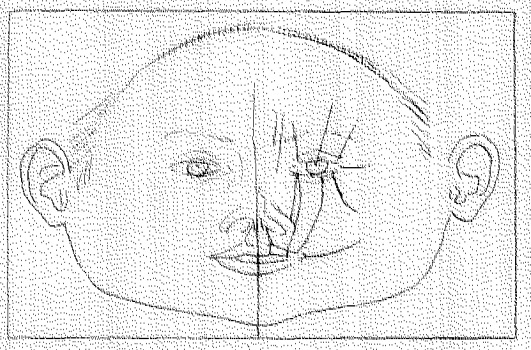

Fig 1a. Facial and Cranial Clefts: Localization on the Soft Tissues [2]

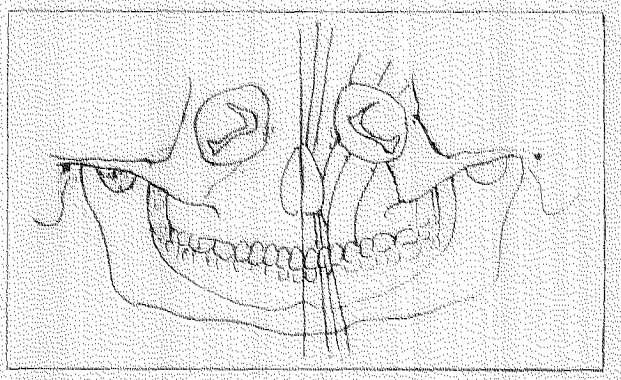

Fig 1b. Facial and Cranial Clefts: Localization on the Skeleton [2]

Facial clefts are rare. In foreign literature, the incidence of craniofacial clefts is placed at 1.43 to 4.85 per 100,000 live births and between 9.5 to 34 per 1,000 common clefts[3]. Another author puts the incidence of oblique facial clefts (i.e. Tessier Nos. 3, 4, and 5) at 0.75 to 5.4/1,000 common clefts [4].

Chiong, Guevara, and Zantua [5] described two Filipino cases of clefts extending from the upper lip to a coloboma in the ipsilateral lower eyelid. These cases are equivalent to Tessier No. 4 clefts.

This paper documents four cases of facial clefts: Tessier nos. 0, coexisting 0 and 14, 4, and 7. The clinical presentation and management of each case are presented. It aims to promote a high index of suspicion for these rare clefts. A proper assessment would lead to appropriate surgical planning and management. Conversely, an uninformed clinician may mistake a Tessier cleft for the usual cleft lip and palate. Subsequently, the surgical management would be inadequate and the cosmetic results disastrous.

CASE HISTORIES

Case No. 1

This was a newborn female with the following congenital anomalies: absent philtrum, absent premaxilla, rudimentary nose, absent columella, absent nasal bone, orbital hypotelorism, and craniosynostosis with hydrocephalus (Fig. 2). There were no cranial defects noted on x-ray. She was born full term to an 18-year-old G2 P1 (1001) mother with an unremarkable prenatal and maternal history. The working impression was Facial Cleft: Tessier No. 0, craniosynostosis with hydrocephalus, and a presumptive diagnosis of Down's Syndrome. She died of severe pneumonia at 3 months of age before any surgery could be done.

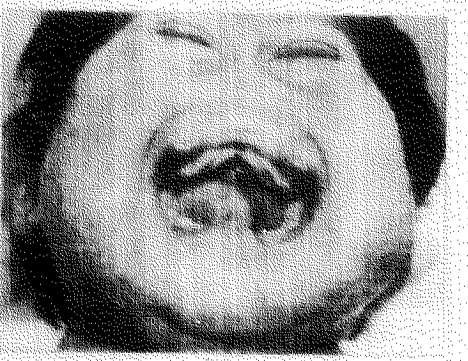

Fig. 2. Case No. 1: Tessier No. 0 Facial Cleft

Case No. 2

This was a newborn female with congenital anomalies similar to case no. 1: absent philtrum, absent premaxilla, rudimentary nose, absent columella, absent nasal bone, and orbital hypotelorism (Fig. 3). Plain radiography showed a midline cleft through the frontal bone and the cranial ultrasound was suggestive of alobar holoprosencephaly. She was born to a 27 year old G2 P1 (1001) mother who had an unremarkable prenatal and maternal history. The working impression was Facial Cleft: Co-existing Tessier No. 0 and 14, alobar holoprosencephaly, congenital hydrocephalus, and a presumptive diagnosis of Trisomy 18. (Note: at the time of writing of this paper, this case was still under neurosurgical evaluation and subsequent clinical course was not known).

Fig. 3. Case No. 2: Coexisting Tessier No. 0 and 14 Craniofacial Cleft

Case No. 3

This was a 1-year-old female with bilateral incomplete clefts of the lip lateral to the cupid's bow and philtrum, alveolar cleft lateral to the right maxillary medial incisor, unilateral incomplete cleft palate, bifid uvula, bilateral lower eyelid colobomata medial to the puncta, two left preauricular skin tags, and a constriction band in the left leg. Skull X-rays (antero-posterior and lateral views) revealed no cranial bony defects (Fig. 4). She was born to a 23 year old G1 P0 mother who had an unremarkable prenatal and maternal history. The impression was Facial Cleft: Bilateral Tessier No. 4 Cleft, left preauricular skin tags, and constriction band on the left leg (Fig.4). This case was initially misdiagnosed as a common bilateral incomplete cleft lip and palate with colobomata and was inappropriately managed with cheiloplasty (Millard Technique). Consequently, the cosmetic result was poor. Unfortunately, the patient did not follow-up and the definitive cosmetic surgery is yet to be performed.

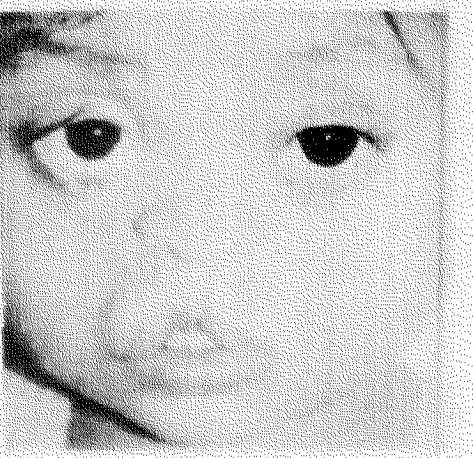

Fig. 4. Case No. 3: Tessier No. 4 Facial Cleft.

Case No. 4

This was a 9-month-old female with a fissure at the right oral commissure, macrostomia, and three skin tags (two preauricular and one at the right cheek). X-ray studies (skull antero-posterior and lateral views) revealed no bony defects (Fig. 5). She was born to a 26 year old G2 P1 (1001) mother with an unremarkable prenatal and maternal history. The impression was Facial Cleft: Tessier No. 7. She underwent repair of the right oral commissure (via Z-plasty with approximation of the orbicularis oris) and excision of the skin tags. Post-operative results showed adequate repair of the right oral commissure.

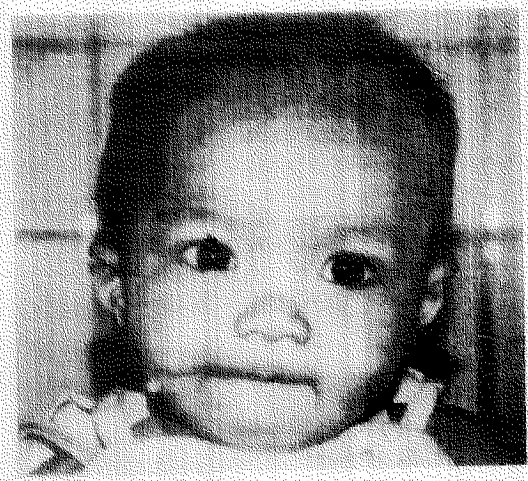

Fig. 5. Case No. 4: Tessier No. 7 Cleft.

DISCUSSION

The etiology of craniofacial clefts is attributed to multiple environmental factors: radiation, infection, metabolic abnormalities, and certain drugs and chemicals. These environmental factors have produced clefting in animals, however, only Influenza A2, toxoplasmosis, abnormal maternal phenyl-alanine metabolism, anticonvulsants, tretinoin, thalidomide, and amniotic bands are associated with increased human craniofacial deformities.[3] With regard to the effects of smoking, alcohol intake, drugs, and infection, Shepard et al pointed out that although these may increase abortion rates there is a lack of documentation for an increase in congenital defect rates[8].

Heredity plays only a minor role. [3,6] An association of facial clefts to Trisomies 13 and 18 was noted 1 and, specifically, a 6.5 % incidence of trisomy 13 and 18 was noted among fetuses with a facial cleft[9]. Furthermore, Nicolaides et al [1] had observed a 7 % incidence of facial defects among 2086 fetuses that underwent karyotyping because of antenatally diagnosed fetal malformations and growth retardation. And of this 7%, 64 (44%) had facial clefts, of which about half (48%) had chromosomal abnormalities.

A change in residence had no effect on the chances of having a second child with a facial cleft but a reduction of incidence was noted with a change of male partners [10].

Another study noted that the acetylator phenotype of the mothers of children with malformations did not differ from those of the control. [11]

All of the four cases presented had mothers with an unremarkable prenatal and maternal history. There was also no family history of clefting.

There are several theories on the pathogenesis of facial clefts. Failure of fusion of the different facial processes during gestation, [5] failure of the neuroectoderm to penetrate the unsupported epithelial wall [4,5,6], inadequate arterialblood supply during gestation [4,5,6], amniotic bands [4], and premature amnion rupture [4] have all been postulated. These multiple pathways plus the interaction of environmental and heredity factors make parental counseling a difficult task.

Patients suspected of craniofacial clefts should have thorough radiologic studies. Plain radiographs delineate the extent of bony involvement when present. If available CT scans (including 3-D reconstruction) give a more comprehensive picture aside from delineating the extent of associated brain malformation. In the absence of a CT Scan, a cranial ultrasound may reveal evidence of brain malformation and hydrocephaly.

Karyotyping is recommended. It could screen for genetic abnormalities frequently associated with craniofacial clefts, i.e. Trisomy 13 and 18. Appropriate genetic counseling could, then, follow.

In the cases presented, the diagnostic examinations were limited to plain radiography and ultrasound due to financial constraints.

The first case fits the textbook description of a No. 0 cleft: total absence of the philtrum (prolabium) and premaxilla, vestigial nose with an absent columella, and orbital hypotelorism [2]. This cleft carries with it a poor prognosis due to the associated brain malformation. And most patients die

within the first three months [6], as in the case mentioned. There was no associated cleft of the lower lip and mandible.

The usual reconstruction entails accurate alignment of the vermilion border and coaptation of the orbicular muscle. A simple V excision may be done. However, a zigzag form of closure may be preferred to prevent contracture. The absence of the premaxilla may be treated with bone graft and orthodontic treatment. Hypotelorism is treated using osteotomies. [6]

Certain facial clefts may occur with cranial clefts: Clefts No. 0 and 14, 1 and 13, 2 and 12, 3 and 11, 4 and 10, and 5 and 9 [2,6]. The second case exhibited this phenomenon: a coexisting Tessier Nos. 0 and 14 cleft. The midline cleft involved not only the soft tissue and facial bones but also the frontal bone. The usual surgery is similar to the first case.

The third case belongs to one of the rarer clefts [7]: Tessier No. 4 Cleft. This typically presents with clefts of the lip lateral to the cupid's bow (midway between the philtral crest and labial commissure) terminating in the lacrimal portion of the lower eyelid [2]. However, the cleft did not extend to the cheek and maxilla in this case. In contrast, the common cleft lip begins at the arch of the cupid's bow.

With inappropriate surgery using the usual cheiloplasty technique, the midfacial anomalies and colobomata are not adequately addressed. A soft tissue defect of the cheek may be repaired using a series of z-plasties and a bony defect augmented with bone grafts. [12]

The two cases reported by Chiong, A.T. et al [5] fit the description of Tessier no. 4 clefts. The definitive procedures done were reconstruction of the lower eyelids and cheek using rotation and transposition local skin flaps and silicon implantation for the defect of the maxilla.

The fourth case belongs to the least rare of the atypical craniofacial clefts: Tessier No. 7 cleft. The defect varies from a widening of the oral commissure to a complete fissure extending horizontally towards the ear [2]. And although clefts of the lateral side of the midface are more frequently associated with bone involvement, there was no skeletal deformity detected in this case.

Reconstruction is tailored to suit the extent of the defect. Macrostomia is addressed using a Z-plasty with reconstruction of the orbicularis muscle.[12] Bony defects may be corrected using a combination of osteotomies and inorganic implants or bone grafts. [6]

SUMMARY AND CONCLUSION

Four cases of facial clefts were presented: Tessier No. 0, coexisting 0 and 14, 4, and 7 clefts. Patients with congenital clefts should be evaluated not only for soft tissue involvement but also for bone involvement. The possibility of a rare craniofacial anomaly should be kept in mind. A high index of suspicion for these rare cases would increase the number of correct diagnoses and proper prognostication and appropriate surgical planning would follow.

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HYPOTHYROIDISM AFTER TREATMENT OF LARYNGEAL CARCINOMA: A Preliminary Report

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Objectives: To determine the incidence of hypothyroidism after surgery alone and after surgery with postoperative radiotherapy
Design: Case series
Setting: Tertiary medical center
Subjects: 24 patients with squamous cell carcinoma of the larynx stage III who were admitted for definitive management from January 1996 to June 1998. Surgical management consisted of laryngectomy with partial thyroidectomy. Radical neck dissection was performed in 16 patients.
Outcome measures: Postoperative thyroid function as reflected by T4 and TSH levels
Results: 13 out of 24 patients (54%) had low thyroid function tests. Five of 9 (56%) who underwent surgery alone and 8 of 15 (53%) who underwent surgery with postoperative radiotherapy had low thyroid function tests.
Conclusion: There is a high incidence of hypothyroidism after surgery and radiotherapy for laryngeal carcinoma.

INTRODUCTION

Cancer of the larynx is predominantly a disease of the elderly: its peak incidence is at the sixth and seventh decades of life. There is some evidence that the frequency of the laryngeal cancer may be increasing by as much as 4 percent per annum with the Asian races by far showing the highest rates.[2] However, it does have a generally favorable prognosis, the overall 5-year survival rate being 67% with adequate treatment.[3]

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At present there are several modes of managing carcinoma of the larynx: chemotherapy, surgery, or a combination of the two for advanced tumors (Stage III and IV). Total laryngectomy with and without neck dissection, either alone or in combination with postoperative radiotherapy, has long been considered the primary mode of treatment for advanced laryngeal carcinoma.

Although neoplastic invasion of the thyroid or the cricoid cartilage in laryngeal carcinomas is not always detectable by diagnostic procedures, the risk is real enough to warrant removal of the homolateral lobe of the thyroid gland combined with the total laryngectomy[4]. The proximity of the

thyroid gland to the postcricoid area, the hypopharynx and, more intimately, to the thyroid lamina of the larynx places this organ at risk for neoplastic invasion; either through the lymphatic network or by direct extension. Therefore, the thyroid itself is ultimately involved in any treatment modality used.

The untoward effect of post-operative radiotherapy on the remaining thyroid lobe is another factor that should be considered. The ensuing hyperthyroidism, in turn, may significantly limit the overall recovery and quality of life of the patient.

Many foreign studies have documented the occurrence of hypothyroidism after treatment of laryngeal cancer. As far as we know we have not come across any local literature regarding the subject.

This investigation has been brought about by our own experience of several patients with squamous cell carcinoma of the larynx who underwent total laryngectomy with or without neck dissection Radiotherapy was administered either as a postoperative procedure or in some cases as the sole treatment. In one particular case, signs and symptoms of hypothyroidism such as easy fatigability, cold intolerance, sluggish movements, and constipation were noted postoperatively.

This paper aims to present our own experience in managing hypothyroidism after surgery for laryngeal carcinoma with or without postoperative radiotherapy.

METHODOLOGY

This is a descriptive study of cases treated for squamous cell carcinoma of the larynx from January 1996 up to June 1998 at our institution. Included in this prospective study were those cases who underwent surgery with and without postoperative radiation. They had been informed of the study and all agreed to cooperate.

Excluded in this study were patients who underwent concomitant total thyroidectomy or any prior thyroid surgery, those with prior history of thyroid disease, including those who are presently taking medications; and those with abnormal pretreatment thyroid hormone levels.

We diagnosed primary hypothyroidism on the basis of high serum thyroid stimulating hormone (TSH) and low serum thyroxine (T4)[1c] levels taken before and at least one month after the completion of therapy for the malignancy.

The patients were grouped according to the treatment modality they underwent. Group 1 patients were treated with surgery alone; Group 2 patients were treated with combined therapy (surgery and postoperative radiotherapy.

The data were analyzed using frequency distributions and cross tabulations .

RESULTS

A total of twenty-four cases were followed up from January 1996 to June 1998 in our institution. Twenty-three were males and one female; the mean age was 66.5 years (Table 11, Fig. 1).

All patients had squamous cell carcinoma of the larynx. Nine patients underwent surgery alone (Group I) while 15 patients underwent surgery plus post-operative radiotherapy (Group 2)

Seven out of the 9 Group 1 patients and 9 out of the 15 Group 2

patients underwent radical neck dissection.

Thyroid function tests were taken within one month after surgery in 66% (6/9) of Group 1 patients. In Group 2, 4/15) patients had their tests taken on the 5th month

Thirteen of the 24 patients (54.2%) had elevated TSH levels (Table 1).

Table 1. Distribution of patients with elevated TSH

	Number	Percent
Normal	11	45.8%
Elevated	13	54.2%
Total	24	100%

More than half of Group 1 and Group 2 patients had elevated TSH levels (Table 2).

Table 2. Distribution of patients with elevated TSH by group

	No. with elevated TSH	Total no.	% with elevated TSH
Group 1	5	9	55%
Group 2	8	15	53%

Most of Group 1 and Group 2 patients were found to have elevated TSH values in the one-month and 5 months into the postoperative period, respectively.

Seven out of the 24 patients had decreased T4 values (Table 3).

Table 3. Distribution of patients with decreased T4 levels

	Number	Percent
Normal	17	70.8%
Decreased	7	29.2%
Total	24	100%

In both groups, the proportions of patients with low T4 levels were lower than those with elevated TSH (Table 4).

Table 4. Distribution of patients with decreased T4 levels by group

	No. with low T4	Total no.	% with low T4
Group 1	2	9	22.2%
Group 2	5	15	33.3%

We next determined the proportions of patients with both elevated TSH and decreased T4 levels. In Group 1, only 1 out of 5 patients with high TSH had low T4 levels. In Group 2, 5 out of 8 patients with high TSH had low T4 levels.

Based on the definition of overt hypothyroidism---high TSH with low T4--the overall incidence of overt hypothyroidism in our case series was 25% or 6 out of 24 patients. The incidence in Group 1 was 11% (1 in 9) and in Group 2, 33% (5 in 15).

The overall incidence of compensated hypothyroidism---high TSH with normal T4--- was 26% or 7 out of 24 patients. The incidence in Group 1 was 44% or 4 out of 9 and that in Group 2 was 20% or 3 out of 15 (Table 5).

Table 5. Distribution of T4 Levels among 13 patients with high TSH by group

	No. of patients with high TSH	T4 levels	
		Low	Normal
Grp 1	5	1	4
Grp 2	8	5	3
Total	13	6	7

Sixteen out of the 24 patients (66.7%) underwent ipsilateral radical neck dissection. (Table 10). In Group 1, all 5 patients with high TSH levels underwent radical neck dissection while in Group 2, half of the patients with high TSH (4 out of 8) underwent radical neck dissection.

DISCUSSION

Local information regarding the possibility of hypothyroidism after the management of laryngeal carcinoma is relatively scarce. Because of this, otolaryngologists may become only aware of a thyroid dysfunction once overt hypothyroidism has already set in.

Partial or total thyroidectomy is primarily performed during the total laryngectomy when treating carcinoma of the larynx. Several potential routes of involvement of the thyroid gland are possible. Lymphatic spread of the tumor and possible direct extension through the lamina of the thyroid cartilage or the cricothyroid membrane has been extensively studied by Harrison et al.[4] who advocated that total laryngectomy should always be combined with the removal of the homolateral lobe of the thyroid gland. All our cases underwent total laryngectomy, isthmusectomy, and ipsilateral thyroid lobectomy with and without radical neck dissection and some with postoperative radiotherapy.

Hypofunction of the thyroid gland may develop from any combination of the commonly employed treatment modalities for laryngeal carcinoma most especially when a part of the thyroid gland is removed and when the remaining thyroid gland is subjected to radiotherapy post-operatively.

Our data clearly indicate that the treatment modalities customarily used for laryngeal carcinoma affect thyroid function in a significant number of cases.

This study aims to underscore the significant incidence of hypothyroidism after treatment of laryngeal carcinoma. Utigar et al.[5] stated that TSH levels are almost invariably elevated in patients with primary hypothyroidism and that "this finding must now be considered a sine qua non for this diagnosis". Hence, an elevated TSH value was used as the principal screening parameter for primary hypothyroidism.

Depression of the T4 level accompanied by an elevated TSH is traditionally recognized as diagnostic of overt primary hypothyroidism. However, it is not clear if the TSH level is always elevated before the depression of the T4 values. It is during this period that the pituitary gland compensates by increasing the TSH levels to maintain a normal T4 level. Therefor, patients with elevated TSH with normal T4 levels are considered to be in the compensated hypothyroid state.

Surgical manipulation results in devascularization of the remaining thyroid gland. Radiotherapy on the other hand can produce direct epithelial injury, necrotizing vasculitis and

thrombosis which compromises further the viability of the remaining thyroid lobe. The additive effect of these modes of therapy would explain why the incidence of overt hypothyroidism was higher among Group 2 than Group 1 patients. The critical decrease in functioning thyroid tissue would also explain why there were more compensated hypothyroid patients in Group 1 than in Group 2.

In Group 1, majority of those patients with hypothyroidism were noted on the first month postoperative period and the remaining 20% two months postoperatively. It is also in the first month postoperative interval wherein there is a high incidence of compensated hypothyroidism. This data suggests that compensatory thyroxine elevation as a response to increased TSH occurs early after reduction of thyroid mass.

In Group 2, 25% of the population with hypothyroidism were mostly noted on the 5th month. It is also at the 5th month when most cases of overt hypothyroidism were seen. This suggests that even if the TSH levels were high, normal T4 levels could not be sustained for long. The time relationships between the completion of the treatment modality and the appearance of elevated TSH levels are yet to be fully explored ideally in a much larger series of patients

The high number of radical neck dissection cases among those with elevated TSH confirms the observations made by Cannon, C.[6] that radical neck dissection increases the surgical time and the degree of surgical manipulation of the neck which may inflict further damage to the vasculature of the remaining thyroid gland.

CONCLUSION

There is a high incidence of hypothyroidism in cases treated for laryngeal carcinoma. Surgery combined with radiotherapy predisposes to more severe cases of hypothyroidism than surgery alone.

This is a preliminary report. Our on-going studies include not only laryngeal carcinoma cases but also other forms of head and neck cancers that will be treated with neck surgery and postoperative radiation. We believe that these procedures on other head and neck malignancies will also have deleterious effect on thyroid homeostasis.

We are going to prolong the study to determine the time of onset at which hypothyroidism sets in and the possible early management needed in these cases.

We recommend screening for hypothyroidism among postoperative patients with laryngeal cancer using TSH level determinations starting 4 weeks after surgery. The increase of TSH level will alert the physician to institute early medical intervention.

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Original studies

A SIMPLIFIED NO-BONE GRAFT TECHNIQUE IN CRANIOFACIAL DEFORMITIES

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OBJECTIVES: To describe the qualities of a newly available, alloplastic material for craniofacial contour correction, and to describe our experience with an improved technique of using the material to correct craniofacial deformities.
DESIGN: Case series
SETTING: Tertiary private hospital
PATIENTS: A series of more than 40 patients, male and female, ages ranging from 6 - 55 years from 1996 - 1998 were included in the study. These patients all underwent reconstruction of craniofacial defects and correction of contour deformities at the institution using this new implant material. The defects included full-thickness cranial defects and midfacial deformities. Two sample cases both male patients are described in detail.
RESULTS: The patients who underwent reconstruction of craniofacial deformities using the implant were followed up at the institution and had significant aesthetic and functional improvement. There was negligible morbidity and neither were there surgical nor implant related-complications.
CONCLUSION: The properties of porous polyethylene make this implant an excellent choice for the existing methods of repair of small to medium-sized cranial defects and correction of maxillofacial contour deformities as compared to other alloplastic implants presently available in the market.

INTRODUCTION

One of the major concerns that confront an ENT - head and neck or craniomaxillofacial surgeon is the repair of facial skeletal defects due to bone loss. These defects involve full-thickness craniofacial bone loss arising either as a congenital deformity or as a result of tumor resection or trauma. Several potential donor sites for bone grafts are available: cranium, rib, scapula, fibula and iliac crest.[1] However, bone grafting procedures have potentially significant morbidity associated with the harvesting of bone grafts. This disadvantage has prompted the search for alternative procedures for

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reconstruction of defects of the facial skeleton.[2] Because of this, alloplastic and allograft materials continue to be popular as evidenced by their widespread use.[1]

The purpose of this presentation is to describe the qualities of a newly available alloplastic material for craniofacial contour correction and to describe our experience with an improved technique using this material to correct craniofacial deformities.

MATERIALS AND METHODS

A. The Implant:

As an alternative to traditional bone grafting techniques, we sought to apply the Medpor surgical implant. This implant is made from pure medical grade high-density polyethylene. While it has been used abroad since 1985, it has been only recently available in the Philippines. Medpor is available as blocks or as preformed anatomical implants.[1,2]

B. The Technique

1. Pre-operative Planning

In our patients, pre-operative planning consisted of available diagnostic aids such as plain, contrast and 3-D reconstructive CT scans.[3] Just as importantly, we relied on our innate aesthetic sense to assess facial contour and defects. (The latest pre-operative planning modality in craniofacial reconstruction is stereolithography, involving the fabrication of 3D models made up of laser-treated liquid resin.[3] However, with a price tag of US $15,000 - 20,000, this process is financially impractical in our setting).

2. Surgical procedure

The craniofacial defect is first approached and exposed using standard incisions appropriate to the site involved. Whenever possible, we transfer a pattern of the defect onto the smooth surface of the implant. We then cut and carve the implant using scissors and scalpel blade only.[1,4] No power instruments are used so as not to obstruct the pores of the implant.[1]

Bending and shaping of the Medpor material is done after heating the implant in boiling normal saline solution. Then we cool and set the newly shaped implant in cold normal saline mixed with antibiotic powder such as gentamicin.[1,2,5]

We then strive to achieve correct fitting of the implant, feathering the edges with a scalpel blade if needed so as to make it less obvious. When we have good edge to edge contact, we now fix the implant onto the recipient site using titanium microplates and screws.[1,4] We may also use sutures to stabilize the implant to soft tissues around the defect. We always try to attain good fixation of the implant for adequate bone and soft tissue ingrowth into it.[1]

We have used this implant and technique for various cases of craniofacial bone deformities in forty patients over the past two years (1996 - 1998). The following are accounts of two of the patients who have undergone this procedure.

Patient 1: A 26 year-old, male, single, came in for episodes of severe headaches and slight blurring of vision of the left eye. A CT scan requested by the neurosurgeon revealed fibrous dysplasia of the left cranial and temporal area. The left supra-orbital ridge and frontotemporal bone including roof of the orbit were resected. The bony defect was repaired using Medpor porous polyethylene. Postoperative period was unremarkable.

Patient 2: A 48 year-old, male, was admitted for repair of a hacking wound sustained two years ago. The bony defects involved the right lateral orbital wall, floor of the orbit, zygomatico-malar complex and right anterior wall of the maxilla. Only a small segment of the zygomatic arch, lateral rim of the orbit and a collapsed right naso-maxillary segment were retrieved, osteotomized, and repositioned. The rest of the bone loss was reconstructed using prefabricated Medpor porous polyethylene implant. Scar revision was done and an orbital prosthesis was used to replace the lost right eyeball. Recovery was uneventful.

DISCUSSION

The acknowledged gold standard in facial skeletal reconstruction is autogenous bone grafting. However, several difficulties have been encountered in employing this type of graft. There is usually limited graft availability. There are potential difficulties in shaping the bone graft tridimensionally to fit the defect. We expect significant donor site morbidity, as well as some intra- or postoperative complications. Bone grafts are known to be prone to some degree of resorption and late deformation.[5]

These recognized disadvantages have led to the development and use of alternative graft materials. The ideal implant should be biocompatible with long term stability.[5] It should have low potential for infection. It should also be easy for the surgeon to use. It should also provide a suitable matrix for cells to infiltrate and produce new bone through "osteoconductive" and "osteoinductive" healing pathways.[1]

Many implants are now available in our country. However, most of them have serious limitations:

1. *Methylmethacrylate* has a high potential for infection because of a toxic monomer released during the healing, which causes local and systemic tissue damage.[1,4]
2. *Silicone* has a high reported incidence of bone resorption. Also, being non-porous, no soft tissue and bone ingrowth and revascularization occurs, leading to rejection and infection.[1]
3. *Hydroxyapatite* is very brittle and difficult to use. Also it is prone to resorption after implantation.[1]
4. *Proplast* has been found to be unstable when implanted because there is no soft tissue ingrowth for revascularization.[1,4]
5. *HTR Polymer* contains methylmethacrylate, sharing the same limitations. Also its extreme hardness makes it very difficult to use.[1]
6. *Allograft - Freeze-dried and Radiated Bone* has a high failure rate

because it does not promote osteoinductive healing process. There is no transformation of multipotent mesenchymal cells into osteoblasts and chondroblasts, therefore no bone regeneration occurs.

Medpor, on the other hand, is made of high-density polyethylene. This material is highly stable, yet maintains some flexibility. It is a porous alloplast which exhibits rapid soft tissue and bone ingrowth for revascularization. For the surgeon, it is easy to shape and fit into various defects in the craniofacial skeleton.[1]

In our experience with forty patients in the past two years, we did not encounter any major complication with the use of Medpor and this technique. Patients were highly satisfied with our post-operative results. Moreover, we found that this technique significantly cuts down operating time and technical difficulties, compared to our previous experiences in using autogenous bone grafting techniques.

CONCLUSION

We find that Medpor porous polyethylene possesses several properties, which make it ideal for small to medium-sized craniofacial deformities. It is easy to use, strong and stable due to good soft tissue and bone ingrowth and revascularization.[1]

We presented two of our 40 Filipino patients to illustrate Medpor's significant advantage over any of the conventional bone grafting techniques. Using Medpor eliminates the surgical difficulty, post-operative morbidity and other attendant disadvantages of bone grafting.

We conclude from our experience that the best interests of both patient and surgeon would best be served with the use of the Medpor implant in place of bone graft.

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GRAND ROUNDS CASE

CONDYLAR NECK FRACTURE: SURGICAL VERSUS NON-SURGICAL MANAGEMENT

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INTRODUCTION

The condylar neck fracture is a relatively common fracture of the mandible. It is believed that this fracture is actually a protective mechanism to prevent the condyle from being driven into the auditory canal and the middle cranial fossa.

At present. management of these fractures still vary much from institution to institution and from one otolaryngologist to another. There is still lack of agreement and common protocol on how to address this special type of fracture.

Therapeutic modalities ranged from surgical treatment to non-treatment at all. The Edwin-Smith papyrus written in 3,000 BC suggested lacing through a bandage in the lower jaw and immobilizing it. This was also the same management found in the Arabic records of Albacusis. Hippocrates would place leather frills around the head of patient with lower jaw fractures which he called "funda hippocratis".

In the early part of this century, this same principle has been advocated by Ruedi. He believed that the risk of complications posed by an open surgical technique was very high and that results might be very disabling and disfiguring for the patient.

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However, various studies conducted through the years have concluded that conservative management was not devoid of complications either. These complications may include pain, malocclusion, temporomandibular joint dysfunction, and sometimes, even deformity. For this reason, Perthes and Wassmund advocated primary surgical treatment. They agreed that complications arising from the open technique are almost nil. Under skilled hands, the procedure was very safe.

With the advent of the metal plate osteosynthesis in maxillofacial surgery, a new era in the treatment of these fractures began. However, we are still haunted by this seemingly endless controversy: to treat condylar neck fractures surgically or conservatively.

Given this divisive scenario, where then do we stand? We hope that this paper would offer some guidelines toward building a consensus in treating this problem.

CLINICAL HISTORY

S.S., a 32 year old male, was admitted for the first time at St. Luke's Medical Center for painful mouth opening. The condition started about four days prior to admission when patient accidentally slipped from a boat while it was being docked. The underside of his chin hit a concrete pavement causing him to sustain a laceration on the chin and avulsion of

the right lateral incisor. He also experienced painful mouth opening. He was immediately brought to a hospital where the wound was sutured. CT Scan of the mandible revealed a fracture on the condylar neck of the left mandible. Patient was subsequently referred to St. Luke's Medical Center for further management.

On physical examination a 2 centimeter sutured wound on the mentum was noted. There was also pain and difficulty in opening the mouth with maximum opening of 10 millimeters measured at the inter-incisor area. The lower jaw closed with a slight open-bite on the right side. No step deformity was noted along the borders of the mandible.

Panoramic view of the mandible showed a displaced fracture of the condylar neck on the left with decreased height of the left ramus as compared to the right. CT scan revealed left mandibular condylar neck fracture with displacement and luxation of the distal fractured segment laterally and a medial rotational displacement of the proximal segment. He was referred to a dentist for consultation and occlusal splint measurement and was started on flucloxacillin, mefenamic acid, eperisone and ranitidine.

The patient then underwent open reduction with internal fixation using rigid miniplates of the left condylar neck and intermaxillary-mandibular fixation using arch bars and ligature wires.

Under general nasotracheal anesthesia, a skin incision was done initially on the subtemporal and pre-auricular area extending into the infra- and post-auricular area in a lazy S fashion. The skin incision was then carried down to the subperiosteal layer at the level of the zygomatic root and dissection proceeded anteriorly to expose the zygoma and the underlying temporomandibular joint capsule.

The main trunk of the facial nerve was then identified and traced anteriorly to expose the more proximal branches. The nerve with some parotid tissue clinging to it was then carefully retracted out of harm's way laterally from the operative site.

The temporomandibular joint capsule was then incised and dissected to visualize the fracture site. A complete transverse fracture from the condylar neck posteriorly extending to the condylar head anteriorly was noted. The condylar head was still in place within the glenoid fossa but was medially displaced. The distal fractured neck segment was laterally displaced and subluxated into the lateral aspect of the proximally fractured condylar head. This situation decreased the ramal height on the left with an obtuse angle subtended by the distal and proximal fractured segments.

Intermaxillary-mandibular fixation was done using arch bars and wires to immobilize the mandible. An occlusal splint was placed to approximate pre-traumatic occlusion. Afterwards, the fractured segments were reduced and rigid fixation was accomplished with an L-shaped seven-hole titanium mandibular miniplates. Hemostasis and wound closure in layers completed the procedure.

The patient was started on clindamycin and ketoprofen. Dexamethasone was given for 3 days. He was started on liquid diet per straw. Routine wound care was given daily.

On the 5th post-operative day, sutures were removed. The intermaxillary-mandibular wires were replaced with elastic bands and patient was started on mandibular mobilization

exercises. The rest of the hospital stay was uneventful and patient was discharged dramatically improved.

DISCUSSION

The mandible is highly predisposed to trauma because it occupies a vulnerable position in the face. The chin is a favored target for injury by virtue of its anterior projection in the facial skeleton. Likewise, the thin condylar neck is prone to fracture because it is an area where the traumatic forces are mostly transmitted especially in cases where the chin is injured with an upward force as in this case.

There have been many attempts to classify condylar neck fractures but the most accepted classification was the one made by Spiesl and Schroll who divided fractures into displaced, dislocated and undisplaced. They also classified fractures as high or low depending on their location in relation to the sigmoid crest.

Using this classification, our patient falls under Type III, that is, high fracture with displacement. This type of fracture is usually treated non-surgically.

What then has influenced us to finally decide to treat this patient surgically?

In 1983, as an attempt to standardize treatment for these types of fracture, Zide and Kemp proposed guidelines for the surgical treatment of condylar neck fractures. They proposed that a fracture pattern which precludes reduction is an absolute surgical indication. This meant that a fracture pattern that makes it infeasible to achieve normal occlusion utilizing simple closed techniques such as immobilization should be operated. We firmly believe that this was the case in our patient. We must therefore prove that the patients' pre-morbid occlusion would not be obtained using closed reduction techniques.

Let us start with the more obvious reason. This particular patient had severe pain and limitation of movement on mouth opening. The maximal interincisor distance was only 10 millimeters. This could be explained by the fact that on mouth opening the distal fractured segment of the condylar neck and the maxilla impinge on the proximal fractured segment and both act as impediment to further movement of the mandible.

The limited mouth opening was also noted intraoperatively when we tried moving the mandible. It was clear then that simply immobilizing the jaw would not relieve the trismus because the problem was both anatomic and patho-physiologic. The fracture had to be directly approached, reduced, and fixed so that the proximal fractured segment and the maxilla would not get in the way when the mandible moves. This is an instance when a structural defect has to be primarily corrected by anatomic reduction so that better function will ensue.

A less obvious reason why we opted for surgery was related to musculoskeletal adaptation. Normally, adaptations occur in the musculoskeletal system that change the biomechanics of mandibular function to help the patient establish normal occlusion. When these adaptations would be sufficient to restore function, then, non-surgical therapy might suffice.

However, there are clinical parameters to be considered to ensure that these musculoskeletal adaptations will ultimately be effective so as to obviate the need for surgery. Let us

discuss each adaptation and parameter in detail.

The mandible acts as a type III level system with the mean force vector of the elevator muscles located between the fulcrum (TMJ), and the load (bite point). When there is condylar fracture, there is loss of posterior vertical dimension as evidenced in this case by the decrease in ramal height. To allow the mandibular ramus and its enveloping musculature to maintain the normal position without gross foreshortening, the neuromuscular system will make the temporalis muscle only minimally active. With this adaptation however, the mandible can not generate enough biting force. As a result of loss of posterior vertical height, the mandibular plane becomes steeper bringing the condylar stump closer to the cranial base for establishment of a new articulation.

The establishment of a new articulation is another important adaptation. In the young, this happens through a process termed restitutional remodeling where the TMJ after trauma still appears anatomically and physiologically normal. In the immature skeleton, the adaptive capacity of the developing occlusion in response to the undamaged functional matrix of the face is considerable. Even when the growth potential of the condylar region is destroyed by early trauma, significant asymmetry is rare before puberty if function is maintained.

After the growth spurt however, a different pattern is observed as the healing pattern in untreated cases adapt to functional requirements with perman anent anatomical deformity. Therefore, in older individual, as in our patient, this happens by functional remodeling where the articulation appears to be structurally abnormal but may or may not function quite well.

TMJ remodeling is therefore a functional of age where remodelling is inversely proportional to age. This phenomenon of functional remodeling may also explain why studies have shown that morbidity associated with closed reduction is significantly higher for skeletally mature individuals compared with children. Several authors have suggested that in skeletally mature individuals, an open reduction might be useful in preventing dysfunctional problems after traumatic injury.

If the mandibular plane becomes steep by virtue of loss of posterior vertical height, then another adaptation would be the orthopedic movement of bone and teeth in the dentoalveolar area. There should be extrusion of the anterior teeth and intrusion of the posterior teeth. If this particular adaptation fails, then an anterior open bite deformity may occur.

Malocclusion ensues when these adaptations do not occur in sufficient degree or quality. In our patient, gross displacement of the fractured segments brought about a severe loss of posterior vertical height in a skeletally mature individual. As such, we believe that these adaptations might not be sufficient to correct the malocclusion we saw in this patient.

Moreover, studies that have done analysis of condylar fractures by means of geometric modeling suggest that the vertical regenerative power is limited when an angle of dislocation is over 37 degrees. This was evident in our patient.

Among the objectives of the surgery that we did in this patient was therefore to maintain the vertical ramus

dimension and to restore normal occlusion.

The rapid restoration of function that surgical therapy affords compared with plain immobilization was another reason why we decided to operate on this patient. Plain immobilization would entail a lot of patience and sacrifice on the part of the patient and the therapist. The mandible has to be immobilized for an average of six weeks with rather unpredictable outcome.

Our patient was anxious not to miss much of his work and did not receive well the idea of a wait and see policy. On the fifth post-operative day, our patient could move his lower jaw significantly and was already able to start with rehabilitation. He could already tolerate soft diet which he could not do pre-operatively due to trismus.

CONCLUSIONS

We have presented a patient with displaced high condylar neck fracture on whom we performed an open surgical procedure. The patient was greatly improved after surgery with no complication noted. The surgical procedure was done for several reasons discussed previously.

From this experience, we recommend that the decision-making process in cases of condylar neck fractures be approached in a more scientific way based on theory and evidence and not merely on pragmatic reasons, wherein we apply practical methods adapted to the circumstances. There should be harmony between these two approaches.

Moreover, we should have evidence-based guidelines in order for us to come up with realistic protocols for management.

Case Report

PRIMARY NASOPHARYNGEAL WEGENER'S GRANULOMATOSIS PRESENTING AS COMBINED "PETROSPHENOIDAL CROSSWAY" AND COLLET-SICARD SYNDROME

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Wegener's granulomatosis is a necrotizing, granulomatous vasculitis that classically involves the upper and lower respiratory tract and kidney. It is a distinct clinicopathologic entity with unknown etiology. The most frequent onset is during adulthood. Males and females are affected equally. Classic and limited forms have been recognized. The limited form of Wegener's granulomatosis involves focal disease manifestations without glomerulonephritis. The gold standard for the diagnosis has been pathologic finding of granulomatous vasculitis. c-ANCA serves as a marker of the disease. Neurologic involvement has been reported in 22-54%[8] and can occur in either classical or limited form of the disease. A diffuse peripheral and cranial neuropathy particularly of cranial nerves I,VII,VIII have been described, but previous studies made no mention of involvement of cranial nerves V,VI ("petrosphenoidal crossway") and IX,X,XI,XII (Collet-Sicard Syndrome) involvement in association with Wegener's granulomatosis. The major portion of this report is devoted to describing a patient with this unusual pattern of neurologic lesions and tracing the clinical evolution of the disease. The importance of intimate anatomic knowledge to localize the disease can not be overemphasized. This case offers practical insights for better understanding and treatment of Wegener's granulomatosis. Comprehensive management of this disease must extend beyond the traditional confines of any single subspecialty.

INTRODUCTION

Wegener's granulomatosis is a necrotizing, granulomatous vasculitis that classically involves the upper and lower respiratory tract and the kidney. It is a distinct clinicopathologic entity with unknown etiology. The first case was reported in 1931 by a German investigator named Klinger[1], who described a patient with destructive sinusitis, nephritis, and disseminated vasculitis. In 1936 Wegener clearly defined the clinicopathological spectrum of the syndrome that now bears his name.

Wegener's granulomatosis is an uncommon, but not rare, disease that affects males and females equally. It can occur at any age, but most frequently presents in adulthood. The mean age of onset is 40 years. The gold standard for the diagnosis of WG has been pathologic finding of necrotizing granulomatous vasculitis.

The diagnosis and classification of Wegener's granulomatosis was revolutionized by the discovery and

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characterization of serum antibodies that react with certain neutrophil cytoplasmic proteins. The existence of these autoantibodies was first reported in 1982 by Davis et al[2]. In classic Wegener's granulomatosis, as defined by granuloma-tous inflammatory disease of the respiratory tract and active glomerulo-nephritis , more than 90% of patients will have a positive c-ANCA. However, in the absence of renal disease ("limited" WG) [2,3,4], the sensitivity of the c-ANCA test may be as low as 65%. Thus, a significant number of patients have localized granulomatous vasculitis in the absence of c-ANCA[3].

If untreated Wegener's granulomatosis runs a rapidly fatal course, with a mean survival of 5 months. In the early 1970s, Fauci and Wolff[4] developed a therapeutic regimen that combined steroids with cyclophosphamide. This regimen has dramatically improved the survival of patients with the disease. More than 90% of patients treated with this regimen will experience marked relief, and 75% will achieve a complete remission.

Classic and limited disease forms have been recognized[6]. The classic form of Wegener's granulomatosis is a multi-system process that includes respiratory tract granulomas, gener-alized vasculitis, and glomerulo-nephritis, whereas the limited form of WG involves focal disease manifestations without glomeruloneph-ritis and with few or no systemic features.

Neurologic involvement has been reported in 22 to 54% of patients and can occur in either the classic or limited form of the disease[8]. It involves the nervous system, usually in the form of peripheral or cranial neuropathy. Involvement of the brain and meninges is reported in only 2% to 8% of patients. Three major mechanisms have been proposed for nervous system involvement: granuloma-tous invasion by contiguous extension from sites external to the central nervous system (CNS) (e.g. extension of nasal, paranasal, nasopharyngeal granulo-mata). The nasopharynx provides an easy pathway into the cranium. Seven foramina are adjacent to the walls of the nasopharynx into the meninges or brain; CNS granulomatous vasculitis; and remote granulomatous lesions.

Neurologic involvement is most typical with mononeuritis multiplex, foot drop and/or wrist drop or both, with patchy sensory and or motor abnormalities. Headaches, hypothalamic or pituitary disease with clinical diabetes insipidus, and cerebral or subarachnoid hemorrhage have been reported infrequently. A diffuse peripheral neuro-pathy and cranial neuropathy, particularly of cranial nerves I, VII, VIII[14,16] , have been described but no there is no mention in current and recent literatures about cranial nerves V, VI (accessible through the "petrosphenoidal crossway")[12,13,14] and IX, X, XI, and XII (constituting the Collet-Sicard Syndrome)[12,13,14,15] involvement in association with Wegener's granulomatosis.

Patients with multiorgan involvement secondary to Wegener's granulomatosis present probably the most complex diagnostic and therapeutic challenges encountered in almost all fields of medicine. Comprehensive management of these patients reaches beyond the traditional confines of any single subspecialty.

The otorhinolaryngologist, by virtue of his skills in head and neck medicine and surgery, is naturally prepared to contribute significantly to

the study and care of this uncommon disease entity.

The major portion of this report is devoted to identifying this unusual pattern, explaining the clinical evolution of this disease entity and reiterating the importance of "knowing your anatomy".

It is hoped that the presentation of this case will lead to practical clinical applications for a better understanding of Wegener's granulomatosis.

CASE HISTORY

A previously healthy 34-year-old man experienced a prodrome of flu-like illness, described as malaise with fever, and anorexia. One week later this was followed by recurrent bouts of left sided frontal headache described as pins and needles sensation, nasal congestion and epistaxis. Two weeks later, the patient experienced limitation of lateral ocular movement and bilateral ear fullness. These later became associated with dysphagia to solid foods and change in voice quality such that the patient developed nasal twang. His symptoms were unresponsive to analgesics, decongestants and multiple courses of oral antibiotics.

Two months later, the relatives noted both of his eyes to be deviated inward. At about the same time he felt a numbing sensation in the perioral area. The patient had bouts of nasal congestion with mucopurulent to blood tinged discharge and decrease in taste sensation specially to bitter foods. Several physicians were consulted. The patient was prescribed antibiotics and steroids but was not relieved of his ailment.

Two months later, the patient noted worsening of the diplopia and his nasal twang. The nasal discharge became foul smelling. A local EENT specialist was consulted. Cranial CT scan revealed an infiltrating neoplasm in the nasopharynx with extension to the base of the skull particularly at the region of the foramen lacerum bilaterally. The enhancing mass also involved the area of the jugular foramen and hypoglossal canal.

He was then advised admission for further work-up and management. On admission the patient was noted to have bulging tympanic membranes with air-fluid levels. Turbinates were congested, however there were no crusting, nor ulceration noted. There was no appreciable oral lesion. On indirect laryngoscopy the right true vocal cord was in the cadaveric position. Audiometric findings were consistent with bilateral conductive hearing loss, mild. Tympanograms showed Type B tracings.

Upon nasal endoscopy, the entire nasopharynx was noted to be filled up with an ulcerated granulomatous, hard mass lesion with suppuration; both eustachian tubes were hyperemic and edematous.

Biopsy showed ulcerated granulomatous inflammation with vasculitides and secondary suppuration, consistent with Wegener's granulomatosis. The results of culture for mycobacteria and fungus were negative.

Upon referral to the ophthalmology service, forced duction test was found to be negative. On red glass test, more than 30% restriction on right lateral gaze and more than 50% restriction on left lateral gaze was noted.

On thorough neurological examination, a 50-75% sensory loss on the mental region (mandibular branch of the trigeminal nerve distribution), was noted. There was limitation of eye movement on lateral gaze; decreased taste sensation on the posterior third of

the tongue, right; absent gag reflex, and absent palatal elevation on phonation, right. There was also loss of palatal arch on the right, slight deviation of the uvula to the left, poor shoulder shrug on the right, a depressed resistance on head movement to the right, and slight deviation to the right on tongue protrusion. Impression was multiple cranial nerve palsy (Cranial nerves V, VI bilateral,IX, X, XI, XII ,right) secondary to nasopharyngeal mass with extension to the skull base.

Renal function tests were normal. Complete blood count revealed normocytic normochromic anemia; erythrocyte sedimentation rate was elevated. Radiographic study of the chest was likewise normal. At this time a cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA) determination was negative.

The patient was diagnosed as having primary nasopharyngeal Wegener's granulomatosis with multiple cranial nerve palsy (V,VI or bilateral petrosphenoidal crossway syndrome; IX,X,XI,XII,right or Collet-Sicard Syndrome. Therapy with prednisone 60mg/d, was begun, and the patient was offered monthly doses of cyclophosphamide 1g/m2, intravenously. He was asked to follow-up on a regular basis.

DISCUSSION

This case illustrates several important points about the clinical presentation, diagnosis and usual mode of treatment of Wegener's granulomatosis. Persistent inflammatory nasal disease, recurrent bouts of epistaxis, headache in association with systemic symptoms of fever, malaise, and anorexia are part of the typical clinical presentation of Wegener's granulomatosis. This constellation of signs and symptoms should prompt a thorough search for evidence of pulmonary and renal disease.

Although the classic form of of Wegener's granulomatosis involves the lungs (~95%), paranasal sinuses (90%), kidneys (85%) nose and nasopharynx (65%) and joints (65%) *virtually any organ system can be affected by vasculitis and granulomatous inflamm-ation* Most patients with this disease first seek medical attention for upper and /or lower respiratory airway symptoms. Nasal involvement is characterized by inflammatory rhinitis and mucosal ulceration that often lead to recurrent bouts of epistaxis.

Until relatively recently, laboratory features of Wegener's granulomatosis were nonspecific. A typical laboratory profile included normocytic normochromic anemia, elevated erythrocyte sedimentation rate, leukocytosis, with or without urine sediment abnormalities or elevated serum creatinine.

In the past 16 years, c-ANCA and its relationship to Wegener's granulomatosis has been studied. Independent investigators reported that antibodies reactive with the cytoplasm of of human neutrophils and could be detected in 80-85% of patients with active WG. The characteristic cytoplasmic staining produced by these autoantibodies is now referred to as c-ANCA . The target antigen responsible for c-ANCA was eventually shown to be a serine proteinase generally referred to as proteinase-3.

The specificity of c-ANCA test result for Wegener's granulomatosis is high, especially in the setting of active glomerulonephritis. However, false positive c-ANCA results have been reported in a number of infectious and

neoplastic disorders. Thus, a positive ANCA test does not obviate the need for tissue diagnosis in a patient with clinical manifestations suggestive of Wegener's granulomatosis, especially if signs of glomerulonephritis is lacking.

Moreover, c-ANCA is considered a supportive evidence of the disease as well as marker of disease activity but is less common in patients with "limited" form of the disease.

Nasopharyngeal biopsy yielded the necrotizing granulomatous vasculitides which is the hallmark of Wegener's granulomatosis. Vasculitis can occur in three forms: (1) microvasculitis, (2) necrotizing vasculitis, and (3) granulomatous vasculitis that involves small or medium sized arteries or veins.

Any of these forms of vasculitis can result in thrombosis and occlusion of the blood vessel lumen, which in turn lead to another histopathological hallmark of Wegener's granulomatosis. The granulo-matous inflammation may be mimicked by mycobacterial and fungal infections. Thus, when these histological features are found in a biopsy specimen from a patient with suspected Wegener's granulomatosis, it is essential that appropriate tissue stains and cultures be performed to exclude mycobacterial or fungal infections.

Neurologic involvement occurs in 22-54% of cases. It commonly involves the nervous system in the form of peripheral or cranial neuropathy. Our patient had neurological findings that were sufficient to warrant evaluation by cranial CT scan. From this, the mechanism that is evident in our patient is granulomatous invasion by contiguous extension from the nasopharynx.

The nasopharynx is a roughly cuboidal open chamber located below the base of the skull and behind the nasal cavity. Its posterior wall is made up of four layers: (1) mucous membrane of the pharynx, (2) the pharyngeal aponeurosis, (3) the superior constrictor muscle of the pharynx, and (4) the buccopharyngeal fascia. The muscular wall of the nasopharynx is incomplete. In the upper nasopharynx, the lateral wall consists of only two layers: the mucous membrane and the pharyngeal aponeurosis. This area of muscular deficiency is called the sinus of Morgagni.

The pharyngeal fascia of the posterior and lateral wall of the nasopharynx is attatched to the pharyngeal tubercle on the bassiocciput just in front of the foramen magnum. This fascia is continuous with the fibrous tissue occupying the foramen lacerum, which is separated from the middle cranial fossa only by fibrocartilagenous tissue. Five other foramina are adjacent to the wall of the nasopharynx": (1) the foramen ovale (transmits the mandibular branch of CN V), (2) the foramen spinosum, (3) the carotid canal, (4) the jugular foramen (CN IX, X, XI), and (5) the hypoglossal canal(CN XII). The foramen lacerum and the foramen ovale constitute the " petrosphenoidal crossway"[14,16], provide an easy pathway into the cranium, and are in close relationship to the cavernous sinus.

Anterior to the eustachian tube, the fossa of Rosenmueller is in close relationship with the lateral pharyngeal space. It contains the lateral pharyngeal nodes, the internal carotid artery, the internal jugular vein, and the glossopharyngeal, vagus, spinal accessory, and hypoglossal nerves as well as the cervical sympathetic nerve as they emerge from the base of the skull.

Hence, impaired hearing of the conductive type, with or without

tinnitus and serous otitis media, occurs as a result of obstruction and/or edema of the eustachian tube orifice due to the granulomatous inflammation.

Speech with nasal twang occurs as a consequence of nasal obstruction, loss of nasopharygeal resonance, mechanical interference with normal movement and depressed function of the soft palate.

Nasal discharge, epistaxis, and coughing of blood tinged phlegm from postnasal drip are symptoms resulting from the local effect of the granulomata.

Hypesthesia on the mental area and bilateral lateral rectus palsy can be explained by the anatomic proximity of the nasopharynx to the foramen ovale and the foramen lacerum where CNVI courses at the base of the skull. Both of these foramina constitute the "petrosphenoidal crossway" thus, providing easy access to the cranium. Involvement of these nerves constitutes the "Petrosphenoidal Crossway " Syndrome[14,16].

Paralysis of the trapezius and sternocleidomastoid muscles (CN XI) as evident by the poor shoulder shrug, and poor resistance on head flexion to the right, paralysis of the vocal cord (CNX) occur. The right vocal cord paralysis in cadaveric position] and the pharyngeal paralysis (CNIX), hemiparalysis of the tongue (CNXII), loss of taste on the posterior third of the tongue (CN IX) , and hemianesthesia of the palate, pharynx and larynx (CNIX,X), and loss of palatal arch ipsilateral to the site of the lesion comprise the *Collet-Sicard Syndrome*[12,13,14,15]. Again, the relationship of the nasopharynx to the foramina which these nerves are transmitted can not be over emphasized enough.

Optimal treatment of active Wegener's granulomatosis consists of cyclophosphamide and corticosteroids. Cyclophosphamide is initiated in a dose of 1-2 mg/kg/day or 1gm/m2 single dose with initially weekly monitoring of complete blood count. The drug is generally continued for approximately 1 year beyond clinical remission.

Corticosteroids are used at the time of diagnosis, initially at 1 mg per kilogram per day then tapered to an alternate day regimen preferably discontinuing this drug by 3 to 6 months[3,4,5,6].

Wegener's granulomatosis is a chronic disease and patients deserve close follow-up, patient and provider education, and sometimes creative therapeutic strategies.

CONCLUSION

This unusual case has been presented. The pertinent literature has been reviewed.

Wegener's granulomatosis can present with multiple cranial cerve involvement as eevidenced by this case, but its clinical evolution can easily be explained by knowing the anatomical structural relationship. It appears therefore that a variation could exist in the spectrum of Wegener's granulomatosis.

The otorhinolaryngologist's expertise is increasingly sought in the identification and management of patients with this chronic disease. It may be said, however, that no single subspecialty is eminently qualified to deal with the multidimensional problems presented by Wegener's granulomatosis.

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Why should I join?

Our speciality has a long tradition of promoting the practice of the art of surgery on the basis of sound scientific principles. Using "evidence" which is the "best" available has always been a cornerstone of our decision-making. As individuals we are committed to providing our patients with the best available care. As a professional body we are equally committed to evidence-based practice. For these reasons alone we should be cognisant of the need for this type of work. There are more pragmatic reasons why we, as otolaryngologists, should get involved. If we do not, others (public health physicians, healthcare providers, etc) will do it for us. Whilst the perspectives of these groups are important, they are unlikely to have the necessary "content expertise", or the practical experience of day-by-day patient contact, to address all the relevant issues.

How do I join?

Joining the group is simple. There is no subscription nor do you have to wait to be invited. Otolaryngologists, audiologists, speech therapists, patients – anyone with an interest in the treatment of ENT disorders can become a member. The only requirement is an interest in the aims of the group and a willingness to participate in some way.

How can I participate?

Individuals can participate in various ways; here are a few examples:

- Undertake a systematic review (or support and encourage a more junior member of the team to do so)
- Volunteer to help peer review the systematic reviews produced by members of the group
- Help improve the Trials Register by hand-searching journals, congress proceedings and abstract books
- Promote awareness and understanding of the value of systematic reviews and their role in the formulation of clinical practice guidelines

Where do I go from here?

If you are interested in any aspect of the Group's work, please contact Dr. Jose Acuin (email: jmacuin@pworld.net.ph). As one of the Editors of the Group, he will send you more information on the activities of the Group and how you can best begin your involvement. He can also keep you informed of the Group's activities.

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