Complications in Rhinoplasty

Obstructive Sleep Apnea-Hypopnea Syndrome: Pathophysiology, clinical diagnosis and overview of management

Nasal Fractures: A clinical approach to diagnosis and treatment

Common Manifestations of HIV and AIDS in Otorhinolaryngology

Ethics

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The expanding horizons of Ear, Nose and Throat, Head and Neck Surgery, or Otorhinolaryngology Head and Neck Surgery as we are now officially called, sets the scene for this edition of the SA Ear, Nose and Throat Review. As specialisation, even at specialist level threatens us to become Jacks of all trades but masters of none, it becomes increasingly important to know which skills are available in each speciality to provide patients with the most appropriate treatment options for their specific medical problems. Therefore, in this edition we focus on the expanding field of ORL, H&N Surgery with special reference to our involvement in facio-plastic reconstructive surgery and the relatively new, but increasingly important condition of obstructive sleep apnoea and its long-term medical consequences. The topics are addressed very eloquently by Dr. Jacques Viljoen and Dr. Jannie Kock.

In the article covering nasal trauma by me and Dr. Khumalo, a registrar in our department, we come face to face with a problem that often presents as a minor aspect in a patient involved in major trauma. He describes in a practical way the approach to diagnosis and treatment of the condition to assist general practitioners, doctors involved in emergency medicine and as a review for ENT specialists.

The article by Dr Ian Olwoch about the impact of AIDS in ENT brings reality home and highlights the necessity for ENT specialists to get to grips with this terrible disease that is afflicting our community.

In Prof Chris Joseph’s article on “The Ethical Tariff”, it becomes clear that doctors in private practice and the HPCSA still has a long way to go before this issue is resolved.

I would like to thank my contributing authors and especially Kathy Harrison and her team for their assistance in putting this journal together.

With this issue we hope to catch the interest of General Practitioners as well as other specialities as the practice of medicine becomes increasingly integrated and complicated.

David Stolp  
Issue Editor  
Professor and HOD  
Department of Otorhinolaryngology  
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ARTICLES

COMPlications in RHINoPlASTY

Dr Jacques Viljoen MBChB, MMed (ORL) (Pret)

BACKGROUND

With the development of rhinoplasty over the decades it became clear that the surgeon should not only possess a sense for rhinoplasty and a refined set of artistic skill, but also display a sound knowledge of the science to the skill. More surgeons are trying their hand to rhinoplasty which can be rewarding to both the patient and the surgeon, but it is important to stress that surgeons should have a good understanding of nasal structural anatomy and functionality, as well as the techniques available to alter the shape of the nose. The interaction of soft tissue and structural framework is complex which often makes surgical manipulation difficult and results unpredictable. Positive outcomes render the procedure straight forward, predictable and easy, but when complications arise, it has the potential to create a most unpleasant relationship between the surgeon and the patient. The resulting deformities may be extremely difficult and challenging to correct and may cause angst to even the seasoned rhinoplasty surgeon.

Becker noted in his chapter on *Complications in Rhinoplasty* (Thieme 2009) that “All complications must be addressed with forthright recognition, close attention to the patient, and appropriately timed corrective measures”.

It will be sensible to discuss possible complications as it relates to technical pitfalls that may occur in primary rhinoplasty in this article. Most occasional rhinoplasty surgeons will attend to dorsal deformities, less frequently venturing into deformities of the nasal tip. This article will therefore firstly concentrate on complications that may arise in the dorsum (middle third), the root of the nose (upper third), and lastly the tip. It will focus on prevention rather than the management of acquired complications, as the management of complications provides for several independent topics.

WHAT DEFINES A COMPLICATION?


- Underresection: easier to address, you just “take a little more”
- Overresection: this is more difficult, it is often complicated by scarring and a need for graft material.
- Asymmetry

The incidence of complications in the literature ranges from 8% - 15%.

PRE-OPERATIVE CONSULTATION

It is important to discuss the goals and expectations with the patient, and the risk of complications should be clearly understood. Documentation during the pre-operative assessment by means of diagrams and photography is imperative. Most complications are minor and correctable, but more debilitating complications may occur.

Complications may be minimised by an understanding of the postoperative changes that occur during healing, the pristine recognition of the precise anatomical cause of a nasal feature, knowledge of the technical execution and meticulous attention to surgical detail.

Issues that may arise are:

- Underresection: easier to address, you just “take a little more”
- Overresection: this is more difficult, it is often complicated by scarring and a need for graft material.
- Asymmetry

REDUCTION OF THE DORSAL HUMP (NASAL VAULT)

The tip-supratip relationship is of critical importance in profile reduction. Overreduction of the bony component may lead to a flattened appearance that may simulate hypertelorism. Overreduction of the bony *and* cartilaginous component will create an overly concave appearance resulting in an iatrogenic saddle nose deformity. Failure to preserve the support of the middle nasal vault may give rise to nasal valve collapse or an inverted-V deformity.

Knowledge of skin thickness and anatomical contributions of the bone and cartilage to the dorsum is mandatory to prevent over- or underresection. The bony contribution to the dorsal profile is usually less than the contribution by the cartilaginous structures. The skin/soft tissue envelope (SSTE) is thicker over the nasofrontal angle and the supratip region than over the rhinion, less hump should therefore be resected at the rhinion. Underresection generates a persistent deformity that may result in pollybeak deformity.
### SUMMARY OF COMPLICATIONS ASSOCIATED WITH THE PRIMARY RHINOPLASTY GOAL AND THE SURGICAL PRINCIPLES THAT WILL PREVENT THEIR FORMATION.

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Asymmetrical resection is clearly unsightly and unforgiving, and correction is often challenging. Precise pocket placement is imperative for dorsal onlay grafts when an endonasal technique is deployed, the alternative being the use of an external approach.

**Saddle Nose Deformity**

Saddle nose deformities follow either excessive dorsal hump resection, or failure to preserve an adequate septal cartilaginous L-strut (15 mm), resulting in collapse. It may also be caused by a septal hematoma, septal abscess and severe nasal trauma. Patients with short nasal bones are more at risk.

Excessive resection may also be avoided with proper attention to the variable skin thickness of the nasal dorsum. It can be managed by camouflage with septal or auricular cartilage, onlay grafting and in severe cases by major reconstruction with cantilevered cartilage or bonegrafts.
"The marketplace declares the ultimate winners."

― Coulter Watt

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<tr>
<td>7</td>
<td>Mometasone furoate</td>
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<tr>
<td></td>
<td>(Nexomist)</td>
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<tr>
<td>2</td>
<td>Budesonide</td>
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<td>2</td>
<td>Fluticasone propionate</td>
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<td>0.33</td>
<td>Fluticasone furoate</td>
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― Data of 84 studies were extracted to determine a therapeutic index (TIX) score based on efficacy (ES) and side effects (AES) for 6 substances, with respect to allergic rhinitis.

― Maximum TIX score that can be achieved is 9.

Reference:


Cipla Medtech (Pty) Ltd. Reg. No. 1992/053158/07, Bouchard Heights, Pinetown, Pinetown, Pinetown, Pinetown, Pinetown, Pinetown. Tel: 021 342 4200, Fax: 021 342 4014. E-mail: medtech@ciplamedtech.co.za Website: www.cipla.co.za
Inverted-V Deformity

With inverted-V deformities the caudal edges of nasal bones are visible in the broad relief, the upper lateral cartilages (ULC’s) are displaced medially with respect to the nasal bones. The caudal edge of the nasal bones create a shadow visible as an inverted-V. Patients with short nasal bones and long ULC’s are prone to the problem. It occurs with inadequate support of the ULC’s to the dorsal septum after hump removal, creating an infero-medial collapse of the ULC’s, or inadequate infracture (medialisation) of the nasal bones after dorsal reduction. It can be prevented by preservation of the underlying nasal mucoperichondrium and adequate infracture of the nasal bones.

Nasal Valve Collapse

The internal nasal valve is bounded by the caudal margin of the ULC and the nasal septum. The external nasal valve is the area delineated by the cutaneous and skeletal support of the mobile alar wall, anterior to the internal nasal valve.

Internal nasal valve collapse follows any manoeuvre that weakens the support of the ULC’s and dorsal septum, such as reduction of the dorsal hump. Preservation of the ventral mucoperichondrial envelope will help to prevent internal valve collapse.

External nasal valve collapse may result from overresection of the lateral crura of the lower lateral cartilage (LLC), or postoperative soft tissue contraction. The width of the LLC after cephalic resection should not be less than 6 - 8 mm. Middle vault collapse will effect internal nasal valve collapse.

Pollybeak Deformity

Pollybeak deformities will be discussed under the section on the nasal tip.

AUGMENTATION OF THE DORSUM

Complications of this manoeuvre are related to the use of grafting. Alloplastic grafts, homografts and autogenous grafts are available.

Alloplastic grafts commonly used are Gore-Tex, silastic and Medpor. Infection rates are quoted from 2.5% - 3.2%, extrusion rates can be as high as 50% if used in the columella, and 10% if used on the nasal dorsum.

Allogenic grafts or homografts include irradiated lyophilised cartilage and acellular dermal grafts (Alloderm). Irradiated cartilage has a significant resorption rate of 3% - 11%. Alloderm are mainly used for contouring in thin skinned patients.

Autogenous grafts are ideal and may be sourced from septal, auricular and costal cartilage as well as calvarial bone. Septal and auricular cartilage has a very low resorption rate. Costal cartilage is used for large nasal reconstruction and has a lower resorption rate than irradiated lyophilized cartilage. Warping remains a problem with the larger costal grafts.

DEVIA TED OR TWISTED NOSE

A deviated nose may pre-operatively occur in the upper third, middle third or the tip of the nose, or can be the post-operative result of a previously straight nose. Pre-operative anatomical diagnosis is therefore paramount in establishing a surgical plan to address the correction of a deviated nose.

Post-operative deviation of the upper nasal vault may be caused by persistent deviation of nasal bones as a result of greenstick fractures or inadequate osteotomies.
Inherent deviations of the ULC’s in the middle nasal vault constitute a challenging surgical solution.

Hump resection often reveal underlying asymmetries in the middle nasal vault not catered for during the pre-operative assessment, which necessitates adjustment of the surgical plan.

An occult twisted septum may be encountered after hump reduction that will lead to an iatrogenic twisted nose. It may even require removal of the septum and re-implantation.

Tip asymmetry is often overlooked pre-operatively. Post-operative tip deviation or asymmetry may result from asymmetrical resection of the lateral crurae, asymmetrical placement of an overlong columellar strut, asymmetrical placement of interdomal sutures and failure to correct a dorsal deviating septum.

**SURGERY OF THE UPPER NASAL VAULT (UPPER THIRD OF NOSE)**

**Deformity of the nasal bones**

Underlying nasal bone symmetry irregularities should be noted pre-operatively as multiple osteotomies such as intermediate osteotomies may be required to align the nasal bones.

**Rocker deformity**

When the medial and lateral osteotomies are executed to extend to high superiorly, the superior aspect of the osteotomised nasal bone will then “rock” laterally on infracturing. Osteotomies should be delivered in the natural cleavage plane where the nasal bone gradually thickens cephalically into the frontal bone.

**Dorsal irregularities (“open roof”-deformity)**

An “open roof”-deformity after dorsal hump removal will result from inadequate lateral osteotomies and medialisation of the nasal bones. Failure to effectively suture the severed ULC’s to the remaining nasal septum will contribute to the defect. The upper nasal vault appears widened and artificially flat. Rough bony margins should be smoothened with a rasp and loose bony fragments or debris should be removed. Patients with a narrowed nasal base and broad dorsum may require only a medial oblique osteotomy to reduce an open roof. Various authors proposed different techniques to camouflage dorsal irregularities, such as gelatin film, temporo-parietal fascia, Alloderm and the use of a powered drill.

**Greenstick fracture**

Incomplete fracture of the nasal bones after osteotomies may result in recurrence of the deformity due to tissue “memory”.

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*Figures 5, 6, and 7: Images of nasal deformities and osteotomies.*
SURGERY OF THE NASAL TIP

Tip surgery is rightfully considered the most difficult part of rhinoplasty. The nose is a three-dimensional unit subjected to dynamic changes during surgical alteration of the anatomical tip structures. The tip needs a careful approach and unnecessary incisions should be avoided. To understand the implications of surgical incision to tip structures, it will be necessary to review the major and minor support mechanisms of the nasal tip:

- **Major tip support mechanisms:**
  - Size, shape and strength of the LLC’s
  - Medial crural footplate attachment to the septum
  - Attachment of the caudal border of the ULC’s to the cephalic border of the LLC’s (scroll area)
  - Nasal septum

- **Minor tip support mechanisms:**
  - Interdomal ligament (Ligamentous sling spanning of domes of LLC’s)
  - Cartilaginous dorsal septum
  - Sesamoid complex
  - Attachment of LLC’s to the skin
  - Nasal spine
  - Membranous septum

Effect of inappropriate tip surgery

**Overreduction of tip structures:**

Overreduction will violate the tip support mechanisms, resulting in tip ptosis or inadequate tip projection.

- Overresection of the caudal septum:
  - This will cause overrotation of the nasal tip with excessive shortening of the nose
- Overresection of LLC’s may result in overrotation of the nasal tip, bossae formation, alar retraction and alar collapse

**Underresection of tip structures:**

Underresection may give rise to a persistent overprojected state or a pollybeak deformity.

**Asymmetry of tip structures:**

Asymmetry is usually due to unequal reduction of LLC’s, asymmetrical application of dome-binding sutures or unequal scarring.

**Tip ptosis (under-rotation)**

Loss of tip support will result in a ptotic, underprojected droopy nose with an overly acute nasolabial angle. Manoeuvres that will the tip to drop are a transfixion incision, shortening of the medial crura, separation of the LLC from the ULC at the scroll region and loss of septal and columnellar support. It can be avoided by proper pre-operative assessment of the tip anatomy and support, and the application of manoeuvres that will maintain or augment tip support by means of grafts, struts and sutures. Christophel and Park in their article *Complications in Rhinoplasty* made the following statement: “The surgeon should not be afraid to use grafts to replace the structural support of cartilage that required resection to obtain the primary rhinoplasty goal. Although it may seem to defeat the purpose of the initial resection, often times the in-situ cartilage was malformed and the surgeon has more anatomic control over placement and size of the replacement graft.”

**Overrotated tip**

Any manoeuvre adding length to the medial crura and shortening of the lateral crura will cause overrotation (tripod theory). The overrotated tip causes an overly obtuse nasolabial angle and provides an unsightly, shortened appearance. It is usually caused by overresection of the caudal septum, lowering of the septal angle, excessive cephalic trim of the LLC’s and vertical dome division.
Bossae formation is second only to pollybeak formation in complications in rhinoplasty. Knuckling of the LLC’s at the tip usually results from the contractural healing forces acting on the weakened cartilages. Patients at risk are thin skinned patients (young patients and females), patients with strong cartilages and pre-operative nasal tip bifidity.

It may be caused by excessive resection of the lateral crus of the LLC’s, failure to eliminate excessive interdomal width, asymmetric alignment of the domes of the LLC’s, scar contracture on an overly narrowed complete rim strip, misplaced tip grafting and various cartilage splitting techniques.

Alar retraction

Cephalic resection of the LLC’s is commonly performed to refine the nasal tip. If an inadequate amount of the LLC is left, contractile forces of healing will cause the ala to retract and cause the external valve to collapse. Approximately 6 - 8 mm of the complete strip should be preserved.

It should be noted that 20% of patients have a thin alar rim and cephalic resection should therefore be approached conservatively. Vestibular mucosal resection should be avoided and strong support to the alar rim is imperative.

Pinched tip

The appearance is self-defining. It may form as a result of vertical dome division or soft tissue contracture overlying an insufficient tip framework. It can be prevented by reestablishment of the cartilaginous framework at the end of surgery such as columellar struts, interdomal suturing and tip grafts.
**Intervalve area collapse**

This refers to the area between the internal and external valves. It is naturally devoid from cartilage support and subject to collapse from tip manoeuvres.

Again, 6 - 8 mm of LLC width should be maintained to minimise the risk of collapse. Alar batten grafts are usually placed to fill the void in this area.

It is important to assess the pre-operative LLC recurvature, with careful application of medialisation techniques that may exacerbate the curvature. This may be prevented by the use of lateral crural strut grafts.

**Alar-columellar disproportions**

This condition may be of significant concern to the patient. The normal columellar show should be between 2 - 4 mm.

The complexities of the alar-columellar relationship was categorised by Gunter *et al*. They defined the position of the ala and columella to relate to two parallel lines drawn through long axis of the nostril. They conclude by stating that *all* patients have either a hooded, normal or retracted *ala*, and that *all* patients have either a hanging, normal or retracted *columella*.

A post-operative protruding or hanging columella may be caused by a persisting uncorrected deformity which is present as a result of overly long medial crurae or an overly long caudal septum. An increased columellar show may also result from retraction of the alar margins. A deficient or retracted columella can be the result of a pre-existing uncorrected deformity or excessive resection of soft tissue, cartilage or the nasal spine.

**Pollybeak deformity**

This deformity is one of the most common deformities encountered after primary rhinoplasty (40% - 64%). It is defined by postoperative fullness of the supratip region, resulting in an abnormal tip-supratip relationship, causing the illusion of tip ptosis. This is caused by failure to maintain adequate tip support (tripod theory), inadequate cartilaginous hump removal (the anterior septal angle), deadspace or scar formation and overresection of the bony hump.

**Columellar incision**

Failure to meticulous attention of the collumella incision may lead to columellar retraction, a distorted columella and even flap necrosis.

Incision complications can be prevented in being cautious not to bevel the incision, in avoiding notching at the margins when closing and precise alignment of skin edges.
It is better to use 6-0 Nylon mattress sutures for closure on the columella and 5-0 chromic at the edges, whereas the use of single 6-0 PDS subcutaneous sutures may be beneficial.

SKIN/SOFT TISSUE ENVELOPE (SSTE)

Avascular dissection should take place in a well-defined tissue plane which is the submusculo-aponeurotic plane. The vascular supply and lymphatics lie superficial to the nasal musculature.

Alloplastic implants in the nose should be used with caution as it risks skin complications such as extrusion and perforation of the skin. Infection of the SSTE is in general rare.

FUNCTIONAL COMPLICATIONS

1. Local complications that may occur are as follows:
   - CSF-leak
   - Septal perforation
   - Septal haematoma
   - Septal abscess
   - Adhesion formation
   - Toxic shock syndrome

2. Medical issues to be considered relates to:
   - Allergy
   - Sinusitis
   - Rhinitis medicamentosa

3. Technique related complications:
   - Overnarrowing of the bony pyramid will leave the patient with nasal valve obstruction.
   - Overresection of the lateral crus of LLC’s most certainly will give rise to nasal valve collapse.

CONCLUSION

Tardy identifies the following patient characteristics that would need further evaluation prior to surgery:

- The patient with unrealistic expectations
- The obsessive-compulsive, perfectionist patient
- The sudden whim patients
- The indecisive patient
- The rude patient
- The over flattering patient
- The overly familiar patient
- The unkempt patient
- The patient with minimal or imagined deformity
- The careless or poor historian
- The "VIP" patient
- The uncooperative patient
- The overly talkative patient
- The surgeon shopper
- The depressed patient
- The plastic 'surgiholic'
- The price haggler
- The patient involved in litigation
- The patient whom you or your staff dislikes

How should the dissatisfied patient be managed?

- It is most important to listen attentively.
- The secondary procedure should be managed with the same enthusiasm as the initial procedure.
- Make sure that you ask the patient to return visits - don't abandon them!

REFERENCES


COMPLICATIONS IN RHINOPLASTY

Which of the following statements is/are true?

1. Patients with short nasal bones are prone to inverted-V deformities, saddle noses and a rocker deformities.
2. The scroll area forms a major tip support mechanism.
3. Overrotation of the tip, internal valve collapse, bossae formation and alar retraction are complications of excessive LLC resection.
4. Dorsal irregularities, bossae formation and external valve collapse are more prominent in thin skinned patients.
5. Septal, auricular and costal cartilage has a high resorption rate.
Cures* more patients\(^1\) ...more often.

Topical therapy is the first-line approach in Acute Otitis Externa (AOE) and Acute Otitis Media with Otorrhoea (AOM+O)\(^2\)

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- \(\times 2\) daily
- \(\times 7\) days

References:

* For AOM / AOM+O, cure is defined as absence of clinical
* For ADE, cure is defined as absence of signs & symptoms i.e. inflammation, edema, tenderness & otorrhoea

\(^{7295}\)
DEFINITIONS

**Snoring** is a sound originating from the upper airway due to obstruction.

**Apnoea** is the cessation of airflow for a minimum of 10 seconds.

**Hypopnoea** is a 50% reduction in airflow (for a minimum of 10 seconds), terminated by an arousal or followed by a minimum of 2% reduction in oxygen saturation.

**Obstructive sleep apnoea-hypopnoea syndrome (OSAHS)** is defined as excessive daytime sleepiness and an apnoea-hypopnoea index (AHI) of at least 5 (some authors: 10) apnoeas plus hypopneas per hour of sleep.

**Upper airway resistance syndrome (UARS)** is a sleep breathing disorder in which there are increased breathing efforts during periods of increased upper airway resistance but in the absence of apnoea or hypopnoea. Multiple micro-arousals precipitate cardiovascular strain and excessive daytime sleepiness (EDS). Oxygen saturation is maintained > 90%. UARS may be present in the absence of snoring.

PATHOPHYSIOLOGY

**Hypoxia**

- Increases sympathetic activity, causing peripheral vasoconstriction and therefore pulmonary and systemic hypertension.
- Dysrhythmias are more prevalent in the presence of hypoxia.

The increase in inspiratory efforts during obstruction stimulates vagal activity, resulting in bradycardia.

Airway obstruction also increases the negative intra-thoracic pressure during the inspiratory phase, thus causing arousals and excessive daytime sleepiness.

Obstructive sleep apnoea-hypopnoea syndrome (OSAHS) is prolonged and become more severe during REM sleep.

**Obesity**

Obesity leads to:
- Compression of the pharyngeal airway by the excess cervical adipose tissue
- Decreased lung volumes
- Compromised pulmonary and pharyngeal reflexes

Apnea induces a decreased cardiac output, increased sympathetic nervous system activation and increased systemic vascular resistance. Upon termination of apnea, there is an increase in venous return to the right side of the heart with a concomitant increase in cardiac output. This increased flow against an already increased vascular resistance causes an abrupt and substantial increase in blood pressure.

Repeated episodes of apnea, hypoxemia and arousals cause further increases in sympathetic nervous system activation. The increased sympathetic nervous system activation persists in the waking state (Studied by Virend Summers at Mayo Clinic with electrodes in the peroneal nerve) and is believed to be one of the mechanisms of daytime blood pressure elevations.

Morning headaches are probably caused by night time increases in levels of CO2, a known trigger of migraine.

**Insulin sensitivity and OSA**

Hyperinsulinemia, glucose intolerance, dyslipidemia, central obesity and hypertension are all risk factors for vascular disease.

Mechanisms of insulin resistance in OSA:
- Altered adrenergic function
- Direct effects of hypoxemia on glucose regulation
- Release of pro-inflammatory cytokines that affect metabolism
Prospective data from two separate studies indicate that habitual snoring is associated with more than a 2-fold risk of developing DM type II over a ten year period independent of BMI and other confounders.

**Sleep related cardiac risk**

- Marin *et al* published results of a 10 year observational study:
  The endpoints (myocardial infarction, stroke, or acute coronary insufficiency requiring invasive management, death of myocardial infarction or stroke) were 3 times as high in patients with untreated severe apnea as in the healthy control individuals.

OSA has been strongly implicated, when severe, in the aetiology of hypertension, ischemia, arrhythmias, MI and sudden death in people with coexisting ischemic heart disease.

Atrial fibrillation may be triggered by autonomic or respiratory disturbances during sleep, in certain patients.

Sleep in heart failure patients is highly fragmented; arousals and state changes may occur at a rate of up to 50 per hour. More than half of heart failure patients suffer from sleep-related breathing disorders, and the common appearances of Cheyne-Stokes respiration can impair cardiac function and increase mortality.

The AHI has been determined to be a powerful independent predictor of poor prognosis in clinically stable congestive heart failure patients.

CPAP may improve sleep, exercise tolerance, cognitive function and left ventricular ejection fraction.

Sleep apnea has been strongly implicated in the occurrence of myocardial infarction. When apnoeas exceed a frequency of 20 per hour, the risk of death is greatly increased, particularly in patients less than 50 years.

**RISK FACTORS**

Major independent risk factors for snoring include:

- Male gender
- Age 40 - 64 years
- Obesity (BMI > 40 kg/m²)
- Cigarette smoking: more than 40 per day is one of the most important risk factors.

Snoring prevalence was slightly increased in subjects who regularly used alcohol or hypnotics. This is due to a reduction in the activity of the genioglossus muscle by alcohol.

Snoring and OSAHS are progressive conditions. A person usually snores at the age of 30 and progresses to OSAHS at the age 45.

Other risk factors for OSAHS include:

- Family history of snoring
- Neck circumference (NC): male > 43 cm; female > 40 cm
- Nasal obstruction
- Medical conditions, e.g. diabetes, cardiac failure, Parkinson’s disease
- Endocrine disorders – hypothyroidism, acromegaly
- Hypertrophied tonsils and adenoids
- Hypoplasia mandible/maxilla (Marfan-, Pierre-Robin- and Down syndromes)
- Neuromuscular diseases

**PREVALENCE**

The incidence in the USA is 4% of adult males and 2% of adult females, thus just as common as asthma. The incidence among NFL players is 14% (prof. Somers - Mayo Clinic).

In an epidemiological polysomnographic (PSG) study done by Madison in Wisconsin, 9% females and 24% males are estimated to have OSAHS. (A1 > 5 and EDS). This would make it the most prevalent disease of men in America.

OSAHS among patients with essential hypertension was found to be in excess of 25%. The minimum prevalence of clinically significant OSAHS is 1%.

**Obesity as a risk factor for snoring and OSAH:**

A body mass index (BMI) > 28 kg/m² is present in 60 – 90% of patients with OSAHS. With a BMI < 28 kg/m², central obesity measurements (hip - waist ratio: normal values for males < 0.95 and females < 0.85) and NC (neck circumference) exhibit a better correlation with OSAHS than does BMI.

Katz reported neck size to be more closely related to the severity of OSAHS than the BMI. Neck size is a useful indicator of upper body obesity.

**Snoring and brain infarction**

The prevalence of habitual snoring was 23.3% among patients with stroke and 8.3% among controls. Even after adjustment for age, sex, arterial hypertension, cardiac arrhythmias and obesity, the odds ratio of habitual snoring for stroke remained statistically significant.

**Snoring and sudden death**

Cardiovascular cause of death was more common among habitual snorers and often snorers, than among occasional or never snorers. Habitual snorers died more often while sleeping.
Snoring and dementia

Reynolds (also Vitiello & Prinz) found a higher prevalence of OSAHS among female patients with Alzheimer’s disease compared to control groups.

Repetitive hypoxic episodes in severe apnoeics contribute to a general intellectual deterioration (impairment of verbal fluency, attention, memory and executive functions) that may not be fully reversible.

CLINICAL SYMPTOMS

<table>
<thead>
<tr>
<th>Night time:</th>
<th>Daytime:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoring</td>
<td>Sleepiness</td>
</tr>
<tr>
<td>Witnessed apnoea</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Choking</td>
<td>Morning headaches</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>Poor concentration</td>
</tr>
<tr>
<td>Restlessness</td>
<td>Decreased libido or impotence</td>
</tr>
<tr>
<td>Nocturia</td>
<td>Decreased attention</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>Depression</td>
</tr>
<tr>
<td>Reflux</td>
<td>Decreased dexterity</td>
</tr>
<tr>
<td>Drooling</td>
<td>Personality changes</td>
</tr>
</tbody>
</table>

Although up to 30% of pregnant women snore, overt OSAHS is uncommon during pregnancy.

Other findings often encountered include:
- Conjunctival injection, usually in the mornings
- Peri-orbital and eyelid oedema
- Peripheral oedema
- Floppy eyelid syndrome (uni- or bilateral) with the upper lid being lax and easily everted with ptosis.
- Lower lid ectropion and rarely corneal complications

Pulmonary hypertension is diagnosed in 15% of patients with OSAHS.

Nocturnal symptoms

It is important to distinguish heart failure from OSAHS. The two disorders may co-exist. Dyspnoea resulting from OSAHS usually quickly resolves on awakening.

Oesophageal reflux is frequently observed among patients with OSAHS. Reflux results from increased gastric pressure during episodes of upper airway obstruction and a subsequent increase in breathing effort and abdominal pressure.

Patient reports awakening with heartburn. Chronic reflux may lead to lingual tonsil hyperplasia, resulting in further airway obstruction. These patients often present with symptoms of dysphagia.

Nocturia is common. Studies showed up to 28% of patients making 4-7 nightly trips to the bathroom.

Dry mouth (74%) and drooling (36%).

Daytime symptoms

Excessive daytime sleepiness (EDS) is the most common symptom. EDS is seldom experienced with a RDI < 20. There is an increased risk of car accidents with an AHI >15.

The Epworth Sleepiness Scale is used to determine the severity of EDS. It may be subtle, such as a mid afternoon drowsiness during a group meeting or occasional naps, or severe such as falling asleep while driving. Some may have difficulty distinguishing sleepiness from fatigue.

Clumsiness in tasks requiring dexterity, concentration, attention, memory or judgment.

Personality changes such as aggressiveness, irritability, anxiety or depression and even psychosis may be observed. Family and social life may suffer considerably. Alienation may lead to depression.
A third of patients report **decreased libido or impotence**.

**Morning or nocturnal headaches** usually last 1–2 hours.

**CLINICAL EXAMINATION**

**Obesity and neck circumference (NC)**

BMI and NC should be measured. NC is measured at the superior border of the cricothyroid membrane with the patient in the upright position. Kushida found the cut-off level of 40cm as having a sensitivity of 61% and a specificity of 93% for OSAHS regardless of sex.

**Upper airway**

- Examine the patient seated and supine
- Retrognathia and dental overbite should be identified
- Tongue - macroglossia is a predisposing factor
- Uvula and velum - look at the size, length and height. Oedema, hyperaemia, mucosal folds (telescoping of uvula) are also indicative of possible OSAHS
- Tonsillar hypertrophy and the size of the tonsillar pillars must be evaluated
- Make a visual estimate of the retroglossal area
- Nose-septum, turbinates, polyps etc. Nasal obstruction itself cannot be a main factor causing OSAHS, but a co-factor of OSAHS during sleep
- Nasal obstruction may cause habitual snoring, sleep fragmentation, sleep deprivation and daytime tiredness
- Nasal CPAP treatment is doomed to fail when the nose is obstructed!

**Lower airway**

- Larynx is examined for cysts, webs, tumours or vocal fold paralysis/paresis. Signs of reflux are commonly seen
- Epiglottis - look for the omega shaped epiglottis and the "toilet lid" epiglottis. High upstream resistance causes the latter
- Evaluate tongue base for hyperplasia of lingual tonsils

Floppy redundant supraglottic mucosa may also contribute to OSAHS.

A fibre optic nasopharyngolaryngoscopy is performed to assess the above-mentioned anatomical areas properly.

**Dictum:** CPAP treatment should not be initiated without adequate endoscopic examination of the complete airway.

Müller manoeuvre: first described by Dr. Johannes Müller (1801-1858) as an inspiratory effort with a closed glottis, at the end of expiration. This demonstrates lateral pharyngeal collapse.

Score collapse of airway:

- $1^+ : 0\text{-}25\%$
- $2^+ : 26\text{-}50\%$
- $3^+ : 51\text{-}75\%$
- $4^+ : > 75\%$

Sleep-endoscopy (jaw lift) and sedation-endoscopy are also utilised to assess the upper airway.
ARTICLES

SPECIAL INVESTIGATIONS

Blood

- Hematocrit
- Thyroid function test
- Blood gas analysis

Pulmonary function test

Lateral cephalometric radiographs

Limitations are that the patient is evaluated while awake and seated and is not a dynamic study obtained during sleep. Overuse of this technique by itself to determine the exact surgical intervention is not recommended.

Cephalometric abnormalities in non-obese and obese OSA patients:
- Non-obese - more craniofacial pathology
- Obese - more soft tissue problems

Non-obese:
- Short anterior skull base
- Narrow retromaxilla
- Longer posterior nasal spine (PNS) to vallecula
- Short mentum
- Lower hyoid

Obese:
- Higher, longer and larger tongue
- Longer and wider palate

Individual variability in the ability to recruit pharyngeal dilator muscles will explain much of the variability in OSA severity. The individual must stay asleep long enough for the muscle to be recruited.

CT and MRI scanning

These procedures are reserved as investigational tools.

The history and physical examination can predict OSAHS in only about 50% of patients. Definitive diagnosis requires a polysomnogram (PSG).

Polysomnogram (PSG)

A negative PSG most often will rule out OSAHS. If the history is strongly suggestive of OSAHS (snoring, observed apnoea and EDS) and the PSG is negative, the test may have to be repeated.

Overnight pulse oximetry is not a substitute for PSG.

TREATMENT

Treatment dilemma

Need to utilise the least invasive, most successful, most predictable, lowest morbidity, most cost-effective techniques (in as few anaesthetic procedures as possible) for the given severity of apnoea, in order to achieve increased patient acceptance and increased surgical success.

MEDICAL THERAPY

1. Obesity

It has been well documented that either medical or surgical weight reduction often has a substantial ameliorative impact on this disorder. In one series only 4% of patients had a "cure" of apnoea related to weight loss.

2. Sleep deprivation (SD)

Absolute deprivation as well as repetitive sleep disruption may also predispose or worsen existing OSAHS.

SD is associated with blunted hypoxic and hypercapnic ventilatory chemo-responsiveness and may prolong apnoea and hypopnoea with consequently greater oxyhemoglobin desaturation, by depressing the arousal responses.

Patients should be encouraged to maintain good sleep hygiene.

3. Modification of body position

A bed partner’s prompting constitutes one of the oldest interventions for snoring and OSAHS.

4. Pharmacological agents that adversely affect upper airway function during sleep

a. Alcohol
b. Benzodiazepines
c. Narcotics and anaesthetics
d. Barbiturates

Beta-blockers and alpha-methyldopa (Aldomet®) act centrally and may worsen the apnoeas. Change to alternatives like ACE-inhibitors.

5. Mechanical devices and techniques for maintenance of upper airway patency during sleep (exclusive of CPAP)

Oral appliances fall into two main categories: those that hold the tongue forward and those that reposition the mandible (and attached tongue) forward during sleep.
Oral appliance therapy should only commence after a complete medical assessment.

The American Sleep Disorders Association (ASDA) recommended in 1995 that oral appliances be used in patients with
• Primary snoring or Mild OSAHS
• Moderate to severe OSAHS who are intolerant of CPAP or refuse treatment with CPAP.

Oral appliance therapy is simple, reversible, quiet and safe to use. The appliances should be used for life.

Effectiveness: 62% partial response with 50% reduction of AHI. Complete response in 37% with AHI < 5.

**PHARMACOLOGICAL THERAPY**

Agents with an uncertain or limited therapeutic role:
• **Stimulants** - some patients remain unacceptably and perhaps dangerously sleepy or inattentive. Stimulants may provide symptomatic benefit.
• **Modafinil (Provigil)** – only indicated as an adjunct to standard treatment of the underlying obstruction.
• **Ritalin** – not generally recommended.

**CPAP**

This is an established form of treatment for OSASHS and for some forms of central apnoea. Nasal CPAP was first described in 1981 in Sydney. It acts as a pneumatic splint to prevent collapse of the pharyngeal airway. With correct CPAP levels, there is often “rebound” slow-wave and REM sleep, lasting about a week. Inadequate CPAP levels are diagnosed when there are continued arousals and continued snoring.

**SURGICAL THERAPY FOR OSAHS**

Surgical treatment philosophies:
• Treatment to cure
• Logically directed management
• Full disclosure of options and risks
• Staged surgical management
• Follow-up of all treatment

1. **Surgery for obesity (Bariatric surgery)**
   This is only considered in patients with a BMI > 40 kg/m² or a BMI > 35kg/m² in combination with other co-morbidity including life-threatening cardiopulmonary problems, severe diabetes mellitus, obesity-related pulmonary hypertension and degenerative joint disease.

2. **Surgery involving the upper airway**
   I. **Nasal**
      Septal repair
      Turbinate reduction
      Polypectomy
      Nasal valve surgery
      Nasopharynx: choanal atresia/stenosis, cysts, adenoids, tumors
   II. **Palate**
      Injection snoreplasty
      Radiofrequency somnoplasty
      Cautery-assisted palatal stiffening procedure (CAPSO)
      Uvulo palato pharyngoplasty (UPPP)
      Laser-assisted uvulo palatoplasty (LAUP)
      Uvulo-palatal flap (UPF)
      Z-palatoplasty
      Palatal advancement surgery
      Expansion-/Lateral pharyngoplasty
   III. **Tongue**
      Radio frequency tongue volume reduction (RFTVR)
      Lingualplasty (LP):
      “open” LP (coblation),
      Submucosal Minimal Invasive Lingual Excision (SMILE procedure)
      Transcutaneous LP
      Lingual tonsillectomy
      Tongue suspension
      Genioglossus advancement
      Hyoid advancement

In a group of patients with severe OSAHS, pharyngeal manometry demonstrated that 35 – 50% of patients have tongue base or hypopharyngeal obstruction. Endoscopic studies during sleep suggested that 30 – 40% of cases obstruct at the tongue base or hypopharynx. This might explain the high failure rate of UPPP and other palatal procedures

3. **Surgery involving the lower airway**
   I. **Tracheostomy or tracheostoma**
   This is done in cases of severe life-threatening sleep apnoea i.e. a RDI > 60 and SaO2 < 60%, who are intolerant of CPAP.
4. Nasal surgery

This is suggested for any patient with notable obstruction of the nasal airway.

5. Uvulopalatopharyngoplasty (UPPP)

This procedure was proposed by Ikematsu in 1964 for the treatment of habitual snoring.

Fujita (1981) adopted the UPPP for the treatment of OSAHS as well as snoring. He only excised the tonsils and redundant tissue with the uvula.

A meta-analysis by Sher et al reported a 39% success rate of UPPP for correcting OSAHS. UPPP has seldom been credited with curing moderate or severe OSAHS.

Woodson advocates careful selection of patients. The main effects of UPP are an increase in airway size in a lateral diameter and reduced collapse of the lateral oropharyngeal walls. UPPP stiffens, shortens and anteriorize the velum. Patients with large tonsils are good candidates. When using more stringent criteria to reduce OSAHS severity to clinically acceptable levels, UPPP fails in 50 – 70% of unselected patients.

Myatt and MacKay described “a simple surgical solution to snoring” in 1996. They used a monopolar cutting diathermy to shorten the soft palate to make it, and the posterior pillars, stiffer without destroying the natural palatal contour.

I prefer a “modified MacKay” procedure performed with the coblation Evac 70 wand. Selection criteria include the presence of tonsils (size 1 to 4), palatal webbing and/or an elongated palate with a “gothic arch” appearance. A standard coblation tonsillectomy is done. The web of the palate is incised (in cutting mode on coblator) at a 45 degree angle, aiming for the hamulus.

In the case of a very long velum, the incision is extended all the way through the velum. The lateral edges of the neo-uvula and distal edge of the “neo-velum” is coblated (in cautery mode) to obtain hemostasis and eventually induce fibrosis. The neo-uvula is not amputated.

Eventual fibrosis leads to retraction of the uvula and a normal appearing free margin of the velum. The mucus sweeping function of the uvula is not compromised with this procedure. Nasopharyngeal competence is also maintained by keeping the uvula intact and not excising normal tissue from the velum. Redundant mucosal folds covering the posterior pillars are also coblated (in cautery mode) as a “debulking” procedure to increase the diameter of the airway.

Dictum: Even the best palatal surgery cannot stand against severe retrolingual obstruction.

The incomplete response to UPPP is almost always due to the fact that other regions of obstruction exist.

Michael Friedman: Patients with mild disease (based on PSG data) do not have a better chance of successful treatment than patients with severe disease, thus severity of disease should not be incorporated in the staging system.
The Friedman Staging System based on anatomic factors is superior when compared to severity of disease as a predictor of successful UPPP.

“We suggest that UPPP is probably overused as an isolated procedure by those who have failed to identify other existing obstructive sites” - Principles and Practice of Sleep Medicine p 918. (Third edition)

“Thus we cannot recommend this (UPPP) as sole therapy in patients with severe sleep apnoea.” - Principles and Practice of Sleep Medicine p 946. (Third edition)

Transpalatal advancement procedure (TPA)

TPA increases the oropharyngeal size by palatal advancement and not by soft tissue excision. This procedure is indicated when the soft palate is of normal length, but with a narrow retropalatal space, for mild to moderate disease.

Good lateral wall movement is a prerequisite for TPA and UPPP. An endoscopic examination of the patient needs to be done.

Patient selection

• In cases of UPPP failure
• Severe OSAHS (RDI > 50)
• Morbid obesity
• Significant collapse of the retropalatal space on endoscopy.

Results

The retropalatal area increased by 321% in patients following; TPA and the closing pressures decreased by an average of 8.4 cm H2O.

In a study it was found that TPA alters the retropalatal airway characteristics more than UPPP alone. In UPPP failure with persistent retropalatal obstruction, TPA provides a method of further modifying and improving the airway.

Contraindications

When bimaxillary advancement is a possibility, then TPA is not recommended.

GENIOGLOSSUS ADVANCEMENT (GA)

The genial tubercle and genioglossus–hyoid complex are advanced.

An osteotomy is performed at the genial tubercle.

The limiting factor is the thickness of the anterior portion of the mandible.

HYOID ADVANCEMENT (HA)

HA was first described in 1984.

The hyoid complex is an integral part of the hypopharynx, both statically and dynamically.

![Figure 9: Transpalatal advancement surgery](image-url)
Forward movement of the hyoid improves the posterior airway space (retro lingual space), down to vallecular level. Numerous reports have supported the concept that surgical intervention at the hyoid complex level improves the hypopharyngeal airway.

In case of a “toilet lid” epiglottis, the HA is very effective. The HA procedure is totally reversible.

**Lateral pharyngoplasty procedure (expansion pharyngoplasty)**

Lateral pharyngoplasty means transforming the superior pharyngeal constrictor muscle into a dilator pharyngeal muscle. UPPP means reconstruction of the superficial pharyngeal layers.

Lateral pharyngoplasty repositions pharyngeal muscles, giving support to the lateral pharyngeal wall and producing subjective and objective improvement in sleep in OSA patients.

**ORTHOGNATIC SURGERY**

The practice of maxillo-mandibular advancement as primary treatment modality is generally not advocated due to acceptable cure rates with the less invasive phase I surgery.

The long-term follow up of phase II surgery was a mean of 39 months (range of 12 – 110). There were no progressive snoring, no EDS and absence of apnoea.

**PROCEDURES INVOLVING THE TONGUE**

1. **Radio frequency tongue ablation (RFTA)**

   - Algorithm – every 3 weeks X 4 at level of 750 – 1000 Joule, then reassess. This may continue to a total of 8 treatments
   - RFTA is performed percutaneously under ultrasound guidance

![Figure 10: Pre (top) - and post Pharyngoplasty (bottom)](image)
2. Glossectomy procedures

- Posterior midline glossectomy (laser, cautery or coblation)
- Lingualplasty: Keyhole (anterior) Suprahypoid (open)
  Suprahypoid (submucosal)
- Ablational (somnoplasty)
- Local Intra-oral plasma excisional
  - Midline glossectomy: requires tracheostomy
  - Repose” tongue suspension
  - Chabbolt technique

REFERENCES


CPD ACCREDITATION

If you are a subscriber to SA Ear Nose and Throat Review please register on-line at www.samedicalreviews.co.za to access your CPD questionnaire and get your CPD points and relevant certificate.

**OBSTRUCTIVE SLEEP APNEA-HYPOPNEA SYNDROME**

Which of the following statements is/are true?

1. Habitual snoring is not associated with an increased risk for developing diabetes type 2.
2. Alcohol reduces the activity of the genioglossus muscles, thus aggravating snoring and sleep apnea.
3. Pulmonary hypertension is diagnosed in 15% of sleep apnea patients.
4. Overnight pulse oximetry may be a substitute for a polysomnogram.
5. Patients with mild disease (based on polysomnographic data) have a better chance of successful treatment than patients with severe disease.
INTRODUCTION

The prominence and delicate structure of the nose makes it vulnerable to a broad spectrum of injuries. Nasal trauma and especially nasal fractures have a long history of misdiagnosis and inadequate or inappropriate management.

In a country where contact sports, assault and motor vehicle accidents are part of our daily lives, it can be expected that nasal trauma is a common occurrence. As it often forms part of the bigger picture of a severely injured patient, nasal injuries and fractures are often overlooked or regarded as minor injuries leading to long term complications as a result of inadequate or incorrect treatment. Examples include cosmetically unacceptable nasal deformity, nasal airway obstruction due to septal deviation, septal perforation and in children growth retardation of the nose and mid-face. In severe facial injuries nasal fractures can also form part of more extensive maxillofacial fractures and these should be recognised and adequately treated.

ANATOMIC CONSIDERATIONS

The skeletal component of the nose includes the frontal process of the maxilla, the nasal process of the frontal bone, the ethmoid, the vomer and the nasal bones. Fractures of the nasal bones occur more commonly distally where they are broader and thinner. The cartilaginous structures include the two lower lateral cartilages the two upper lateral cartilages and the central septal cartilage. (See figures 1A and 1B)
Nasal bone fractures are the most common type of facial fractures. Understanding the process by which nasal fractures occur and how injuries to key areas of support can alter appearance and function are essential to appropriate treatment. Blunt force trauma is the most common mechanism. Most fractures result from laterally applied forces as greater force is required to fracture the nose with a blow from the front. The bony vault is a pyramid shaped structure composed of paired nasal bones centrally and the frontal process of the maxilla laterally.

On the inner surface of the nose the perpendicular plate of the ethmoid fuses in the midline with the nasal bones to form a tent like structure with the ethmoid plate representing a pole and the nasal bones representing the side walls of the tent. This interrelationship helps to explain the susceptibility of the septum to injury during nasal fractures.

The bony vault is a pyramid shaped structure composed of paired nasal bones centrally and the frontal process of the maxilla laterally.

The mid-portion of the nose is composed of the upper lateral cartilages and the quadrangular cartilage of the septum which supplies structural support to this part of the nose.

The mechanism of nasal fractures is illustrated in figure 2. These fractures are caused by low to medium velocity trauma. The range of fractures includes:

a) Isolated nasal bone fracture
b) Bilateral nasal fracture with septal dislocation
c) Dorsal widening
d) Comminuted nasal fracture

This excludes more complicated naso-orbito-ethmoidal fractures caused by high velocity blunt trauma which falls outside the scope of this article.

**DIAGNOSTIC ASSESSMENT**

**History**

A detailed account of the events surrounding the nasal trauma is helpful in determining the type and severity of injuries that may be present. In addition to changes in appearance, the patient’s history should be evaluated for functional changes in nasal breathing as well as bleeding and watery discharge which could indicate cerebrospinal fluid leak. Severe nasal airway obstruction is often an indication of septal haematoma which requires urgent surgical evacuation. Associated epistaxis often stops spontaneously, but significant amounts of blood can be lost. Other injuries may also be present and may have been overlooked. Diplopia, epiphora and visual disturbance suggest orbital trauma.
Look out for loose teeth and other facial fractures which may require a maxillofacial opinion. Finally enquire about previous injuries as it is possible that the nasal deformity is due to a previous nasal fracture.

**Physical Examination**

The diagnosis of nasal fractures is made primarily by physical examination. The presence of epistaxis, nasal swelling as well as periorbital ecchymosis are signs suggestive of significant nasal trauma and warrant a thorough internal and external examination. Deviation and asymmetry may be masked by soft tissue swelling and severe nasal airway obstruction may be indicative of a septal haematoma. The use of topical decongestants and local anaesthetic will aid visualisation especially if nasal endoscopes are available for examination. The presence of crepitus on palpation confirms fracture, but is not always possible due to pain and swelling. Particular attention should be paid to the status of the septum. One should note any mucosal tears or septal deformity indicative of significant septal trauma. The importance of recognising and optimally treating septal deformity cannot be overemphasized, as a fractured septum unfavourably affects the alignment of the nasal bones during the healing process, leading to future nasal bone deviation.

**Radiography**

There is clear evidence that plain radiographs do not influence the management of nasal fractures due to the high percentage of false positive readings. However, it is helpful to evaluate surrounding structures like the orbit and other facial bones and can play a part in medico-legal cases. In cases of severe trauma and where other facial bones are involved, computed tomography should be used to assess the extent of the bony injury. Most information is derived from thin cut coronal and axial bone window views. Special attention should be given to the orbit as blow out fractures can accompany nasal fractures and requires separate surgical intervention. Surgical emphysema is indicative of fracture of the lamina paparacea and ethmoid which is suggestive of more severe trauma which could involve the cribriform plate with subsequent cerebrospinal fluid leak.

Although thin sectioned computed tomography is a helpful diagnostic tool for septal fractures, it is not accurate enough to predict the severity of the fracture due to its low correlation with operative findings. Therefore it should not be used as a definite diagnostic modality in terms of deciding whether a septoplasty is needed.

**MANAGEMENT**

It is mandatory that all evaluations begin with the ABC’s (airway, breathing and circulation) as well as examining the patient for other facial and neck injuries.

Epistaxis from nasal trauma may range from mild bleeding from mucosal tears to life threatening large vessel haemorrhage. Most bleeds are not serious and stop spontaneously, but others need more active treatment like endoscopic cauterization, anterior and posterior plugging or vessel ligation.

Lacerations of the skin and soft tissues should be cleaned and sutured primarily with minimal debridement. The rich blood supply to the nose allows for excellent wound healing.

Proper management requires the consideration of the following four important factors:

1. Severity of injury
2. Timing of intervention
3. Use of local versus general anaesthesia
4. Closed versus open reduction

**Severity of injury**

Several classifications of nasal fractures exist, none of which are accurate as the initial clinical findings and perioperative findings often differ. The following factors have to be taken into account when planning surgery. One has to try and determine whether the fracture falls into any of these categories.

- Soft tissue injury alone
- Simple unilateral or bilateral fracture without displacement
- Fracture with displacement.
- Injury to the nasal septum with or without haematoma
- Comminuted nasal fracture
- Complicated naso-orbito-ethmoidal fractures with or without associated facial bone fractures

### Timing of intervention

The timing of surgical assessment and subsequent reduction is crucial as there is a narrow window of opportunity to correct the deformity. Failure of timely referral is a common cause for litigation in nasal fractures. Patients with suspected septal haematoma should be seen urgently as delay can result in septal abscess and resorption of the quadrangular cartilage of the septum leading to severe saddle deformity of the nose.

In a minority of patients seen within the first four hours after injury, before swelling sets in, the fracture can be reduced immediately. Surgical assessment is ideally done about four days after the injury when the swelling has subsided somewhat leaving time for reduction of the fracture in the next few days.

The timing for surgical intervention to correct the deformity is therefore crucial as further delays make effective reduction less likely and sometimes impossible without doing osteotomies or a complete septorhinoplasty.

### Anaesthesia

Both local anaesthesia and general anaesthesia are acceptable. A well administered local nasal block through a combination of topical and injected anaesthetic gives excellent anaesthesia. For more severe fractures especially those involving the nasal septum, general anaesthetic is preferred.

### Method of Reduction

The general principle of fracture reduction is to mobilise and disimpact the fractured bone, followed by closed reduction until the nose is restored to its original position. A combination of digital pressure and elevation of the fragment with a Freer elevator is often sufficient.

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**Table 1**

**NASAL TRAUMA ALGORITHM**

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<td>- Mechanism of injury</td>
<td>- Signs/symptoms of fracture:</td>
</tr>
<tr>
<td>- Time since injury</td>
<td>- CSF rhinorrea</td>
</tr>
<tr>
<td>- Patient age</td>
<td>- Epistaxis</td>
</tr>
<tr>
<td>- Differentiation of acute vs. chronic/pre-existing deformity</td>
<td>- Hypoesthesia</td>
</tr>
</tbody>
</table>

**History and Physical**

- NO NASAL
- NASAL

**NO SURGICAL TREATMENT**

- Soft tissue injury
  - Ice
  - Control Epistaxis
  - Follow-up

- Assess timing of injury
  - < 4 hours
  - Closed reduction if no swelling or septal fracture
  - Re-assess 4-5 days
  - Planned surgery 7 days after injury

**DEPENDING ON SEVERITY OF INJURY:**

- Closed reduction
- Closed reduction & open reduction septum
For severe deviation and impaction special instruments are required e.g. Walsham’s forceps (nasal bones) or Ashe’s forceps (septum).

In a study by SeungChul Rhee et al it was found that 78.8 percent of simple nasal bone fractures required septoplasty as part of the procedure. The final position of the nasal dorsum reflects the deformity of the underlying septum.

In more severe fractures where a c-shaped fracture of the septum is present, open reduction and functional repair of the nasal septum is required, to prevent persistent displacement due to overlapping segments of the fractured perpendicular plate of the ethmoid and septal cartilage, which can only be repositioned by open reduction.

There can be no question that open reduction has historically been underused in the treatment of nasal trauma. Some nasal injuries simply cannot be managed by closed reduction. This includes comminuted fractures, fractures with a loss of support that nasal shape cannot be maintained, severe septal injuries, and nasal fractures with substantial soft tissue trauma. There is no rationale for delaying definite management in these cases for the often quoted 4-6 months. During this period, the remodelling and secondary changes that occur in the nasal shape may make restoration of the pre-injury appearance more difficult.

In short, any basic rhinoplasty sequence may be used, but because the septum is the keystone of the correction, it should be addressed early in the procedure.

Good exposure can be obtained via the open rhinoplasty approach. Both the septum, the nasal bones and the upper lateral cartilages can be accessed and deformities be dealt with accordingly. Where necessary, precise planned osteotomies may be required to correct any residual loss of alignment.

A relatively new technique developed by Burm et al for comminuted fractures describes indirect open reduction and intranasal Kirschner wire splinting. Basically a Kirschner wire is inserted through an intercartilaginous incision between the upper and lower lateral cartilages and pushed cephalad along the posterior surface of the reduced nasal bone until it comes to rest against the cephalic portion of the nasal bone. On the surface anatomy the distal tip of the Kirchnner wire is placed below or at the nasion. The end result is internal splinting, preventing internal collapse of comminuted nasal fragments.

The nasal fracture is stabilised with a dorsal nasal splint which is kept in position for 7 days. Contact sports are to be avoided for 6 weeks. Most fractures can be handled in this way. More complex fractures involving other facial bones often require open reduction and fixation, working together with maxillofacial surgeons. Occasionally the bones are fixed if the fracture is old, requiring formal septorhinoplasty (functional nose reconstruction) to correct cosmetic and functional nasal defects. The importance of addressing the septum during surgery cannot be overemphasised.

Complications

Poor cosmetic results are due to:

- **Time delay in surgical reduction**
  The nasal fracture becomes fixed and unreducible within two to three weeks, which will result in cosmetic deformity and functional impairment, requiring septorhinoplasty to correct it.

- **Unrecognised septal haematoma**
  The nasal septum receives its blood supply from the overlying mucosa. A septal haematoma separates it from the supply. If the vascular supply of the nasal septum becomes comprised it will lead to necrosis and absorption of the cartilaginous portion of the septum, resulting in collapse of support for the lower part of the nose. It manifests itself as a saddle nose deformity or collapse of tip support accompanied by tip ptosis and columellar retraction.

- **Pre-existing deformity.**
  As nasal fractures are common and often left untreated, it is important to try and establish whether the patient had previous nasal trauma. The result of the reduction of a fracture on a previously fractured nose will be unsatisfactory unless this is taken to account.
This will then require additional rhinoplastic procedures for example osteotomies, dorsal augmentation and correction of saddle deformity by using cartilage grafts and other rhinoplastic techniques to restore both function and a cosmetically acceptable result.

- **Septal fracture unrecognised and untreated**
  The eventual cosmetic and functional result is totally dependent on the position of the septum after the trauma and the surgery. A deviated septum not only leads to nasal obstruction but to external deformity. Remember the saying “where the septum goes, there the nose goes”.

- **Post-operative scarring and fibrosis**
  Some patients inevitably require formal septorhinoplasty (functional nose reconstruction) to correct residual functional and cosmetic defects due to post operative fibrosis and scarring.

- **Septal perforation**
  Septal perforation can result from untreated septal haematoma or as result of surgical trauma by tearing the nasal mucosa or by excessive removal of cartilage. It is often very difficult and sometimes impossible to close surgically with nasal crusting and bleeding as permanent sequelae.

- **Septal abscess**
  An undrained septal haematoma will lead to a septal abscess with destruction of the nasal septum and the systemic toxic consequences of an abscess as well as the feared complications of meningitis and brain abscess.

**SUMMARY**

Nasal fractures are common and often form part of other injuries and is therefore easily overlooked. Early recognition, referral and appropriate surgical intervention are imperative to prevent complications and unacceptable cosmetic results.

**Key points:**

- Nasal fractures are common and awareness of the basic principles of management ensures good outcomes.
- All significant nasal injuries should be assessed by a primary practitioner within 24-48 hours to exclude septal haematoma, and specialist referral made if there is cosmetic deformity and/or nasal obstruction.
- It is important to assess each patient carefully and be vigilant over potential complications.
- Document all details carefully in case of subsequent litigation.
- Not all referred patients actually have fractures and even some with fractures do not need surgery.
- Most cases can be reduced adequately with closed techniques, unless the fractures are complex or a significant septal fracture-dislocation is present.
- There are no significant differences between the outcomes of local versus general anaesthesia: more important predictors of outcome include pre-existing deformity, severity of injury, delay in assessment and poor surgical technique.
- Residual cosmetic deformity and nasal obstruction are relatively common complications and it is wise to counsel patients about these from the outset.
- A formal septorhinoplasty can be performed at least six months after the injury.

**BEST CLINICAL PRACTICE**

- Prompt medical review within 24-48 hours of all significant nasal injuries, with urgent ENT referral if a septal haematoma or a complex naso-frontal -ethmoidal fracture is present.
- Exclusion of related injuries to face, orbit, jaws and central nervous system (CNS) at initial assessment and appropriate specialist referral if present.
- ENT assessment at four to five days if cosmetic deformity or nasal obstructions are present.
- Closed reduction at seven days, or open reduction if more complex.
- Review seven days postoperatively with removal of external nasal splint.
- No contact sports for at least six weeks postoperatively.

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*Figure 5: Saddle deformity*
REFERENCES


NASAL FRACTURES

Which of the following statements is/are true?

1. The diagnosis is made primarily by plain radiographs of the nasal bones.
2. Septal haematoma is a medical emergency and should be drained promptly.
3. The fracture is stabilised with a dorsal splint which is kept in position for 6 weeks.
4. Septal fractures often accompany nasal bone fractures.
5. In the majority of patients immediate reduction of the fracture is required.

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COMMON MANIFESTATIONS OF HIV AND AIDS IN OTORHINOLARYNGOLOGY

Dr Ian Paul Olwoch, Department of Otorhinolaryngology, Medical University of Southern Africa

INTRODUCTION

Human Immunodeficiency Virus (HIV) is an RNA lentivirus, a genus of the Retroviridae family, whose growth is typically slow and characterised by a long incubation period and the unique ability among retroviruses of being able to replicate in non-dividing cells. HIV together with its associated Acquired Immune Deficiency Syndrome (AIDS), is a global pandemic affecting about 33.3 million people worldwide and with an estimated 2.6 million new infections occurring each year.1

South Africa ranks amongst the worst affected countries in the world, with the total number of persons living with HIV having risen from an estimated 4.10 million in 2001 to 5.24 million by 2010, and about 310 000 AIDS-related deaths occurring each year.1,2 The prevalence rate of HIV amongst South African adults, aged 15 to 49 years, is estimated to be between 10.5 and 18.8% whilst the prevalence amongst women in their reproductive years is about 20%.2 These worrisome statistics reflect the overall presence of HIV infection in the general population and the challenge currently faced by medical practitioners within the country (Table 1).

The vast majority of HIV-infected patients will eventually develop head and neck signs and symptoms of the disease. The spectrum of diseases includes all the common problems encountered in head and neck disease and, in addition, a unique variety of opportunistic infections and malignancies hardly ever occurring in non-infected patients.3,4 Over the years, the likelihood of early detection of otorhinolaryngologic manifestations of HIV by health care providers has increased, probably as a consequence of the overall level of awareness of the disease. General practitioners, and otorhinolaryngologists alike, perform an essential service in the diagnosis and management of otorhinolaryngologic manifestations. It is essential that these healthcare providers are familiar with the early manifestations so as to effect early detection and intervention. In addition, an understanding of both the pathogenesis and various pathological findings in patients infected by HIV is fundamental to the appreciation of the diverse clinical manifestations of this disease.

PATHOGENESIS

Primary inoculation of the virus most frequently occurs through the reproductive and/or gastrointestinal mucosa by the transfer of blood, semen, pre-ejaculate, vaginal fluid or breast milk. The infection becomes entrenched by way of local replication of virus particles within targeted T-lymphocytes, macrophages and dendritic cells in the affected mucosa that express CD4+ and chemokine receptors, with a particular predilection for T-lymphocytes. CD4+ T-lymphocytes modulate the immune responses to invading pathogens, tumour cells and foreign antigenic matter by promoting the maturation of B lymphocytic cells into plasma cells and memory cells, and activating CD8+ T cells (cytotoxic T cells) and macrophages. The role of activated CD8+ T cells is to destroy virally infected cells and tumor cells. The hallmark of HIV infection is the progressive depletion of CD4+ T lymphocytes (also known as T helper cells) which translates into failure of the immune system and the subsequent emergence of opportunistic infections and tumours which eventually result in death.5

Clinically, HIV infection begins as an acute symptomatic illness that lasts for a few weeks and manifests with symptoms that range in severity from a mild illness, characterised by fever, sore throat and fatigue, to a life-threatening encephalopathy. During the acute phase there is rapid viral multiplication and subsequent release of virus particles resulting in a viraemia associated with a high viral load causing widespread dissemination of the virus (Figure 1). Furthermore, a precipitous drop in peripheral blood CD4+ T cell counts occurs, a reservoir of latently infected CD4+ T cells becomes established, and an HIV-specific immune response develops.6,7,8

During the 6th to 12th week of the infection there is a 100 to 1000-fold fall in viral load and a transient increase in CD4+ T cell counts.9 This is followed by an asymptomatic phase of chronic infection that lasts about 10 years, during which there is a partial recovery in CD4+ T cell counts which is followed by a gradual unrelenting decline and a concomitant rise of viraemia.
Although seemingly quiescent, the chronic phase is a highly active period of complex interplay between the virus and the immune response system characterised by high levels of activation of both CD4+ and CD8+ T cells and viral multiplication. Direct viral killing of infected cells, increased rates of apoptosis in infected cells and destruction of infected CD4+ T cells by CD8+ cytotoxic lymphocytes eventually lead to decreased CD4+ T cell counts.

When the peripheral blood CD4+ T cell count declines to less than 200 cells/µl, and the total number of CD4+ T cells in the body is reduced by 50%, AIDS defining opportunistic infections and tumours occur.

In the terminal stages of AIDS there is a sharp increase in viraemia and a fall in peripheral blood CD4+ T cell counts.

**Otorhinolaryngology-Head and neck manifestations of HIV/AIDS**

The head and neck region is probably the area of the body that most often presents with manifestations of HIV infection that present early in the course of the disease, are clearly visible and readily identifiable. It is therefore important for primary care physicians and otorhinolaryngologists to become familiar with common

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**HIV prevalence estimates and the number of people living with HIV 2001-2010**

(Source: Stats SA. 2010)

<table>
<thead>
<tr>
<th>Year</th>
<th>Population 15 - 49 years</th>
<th>Percentage of the total population</th>
<th>Total number of people living with HIV (in millions)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percentage of women</td>
<td>Percentage of the population</td>
<td></td>
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<td>2001</td>
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<td>2010</td>
<td>19.7</td>
<td>17.3</td>
<td>10.5</td>
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Table 1
presenting lesions in order to ensure timely diagnosis and commence early treatment. HIV-infected patients are highly susceptible to the spectrum of diseases that are common in head and neck. In addition they are more likely to succumb to wide range of opportunistic infections (viral, bacterial, fungal and protozoal) and malignancies that are rarely encountered in non-infected populations. Some of the more common lesions are listed in Table 2.12

Figure 1. Changes in peripheral blood CD4+ T cell count and HIV viral load [source: http://www.microbiologybytes.com/virology/AIDS1.html]

### Head and Neck Manifestations of AIDS

<table>
<thead>
<tr>
<th>Otologic</th>
<th>Nasal and Sinus</th>
<th>Oral</th>
<th>Neck</th>
</tr>
</thead>
</table>
|  • Otitis externa  
  • Serous otitis media  
  • Kaposi’s sarcoma  
  • Eustachian tube dysfunction  
  • Sensorineural hearing loss |  • Cutaneous  
  - Kaposi’s sarcoma  
  - Herpes simplex ulcers  
  - Herpes zoster  
  • Non-cutaneous  
  - Adenoid hypertrophy  
  - Eustachian tube obstruction  
  - Acute/chronic sinusitis |  • Recurrent aphthous ulcers  
  • Condyloma  
  • Candidiasis  
  • Herpes simplex  
  • Gingivitis  
  • Stomatitis  
  • Periodontitis  
  • Kaposi’s sarcoma  
  • Non-Hodgkin’s lymphoma  
  • Squamous cell carcinoma |  • Generalised lymphadenopathy  
  • Neck mass (due to infection with Mycobacterium tuberculosis, Mycobacterium avium complex, cryptococcosis, histoplasmosis or coccidioidomycosis)  
  • Parotid gland cyst |

Table 2
NECK MANIFESTATIONS

As many as 91% of HIV-infected patients present with neck masses that have a broad and extensive differential diagnosis which include both infective and neoplastic aetiologies.13 Patients that are profoundly affected by HIV, with peripheral blood CD4+ T cell counts below 200 cells per µl, are especially vulnerable to opportunistic infections. In such patients the clinical course of the infection is often severe, persistent and recurrent despite appropriate therapy.14

Often the presenting symptom and sign is that of a neck mass which is either due to the infection or a reactive lymphadenopathy. Isolation of unusual microorganisms such as fungi, atypical mycobacteria and Pneumocystis carini, should always raise the suspicion of HIV/AIDS (Table 2).

HIV is now probably the most common cause of parotid pathology in Sub-Saharan Africa (Figure 2). HIV-related benign lymphoepithelial cysts of the parotid gland are typically non-tender bilateral and multiple. The cysts vary in size and can grow to become very large and disfiguring. They occur during the latent phase of HIV infection when the CD4+ count is between 300 and 600 cells/µl. Treatment by aspiration, sclerotherapy or surgery is largely of cosmetic value.15

Laryngeal tuberculosis the most common granulomatous disease of the larynx that occurs in HIV-infected patients, therefore a high index of suspicion must be maintained for all patients who present with laryngeal lesions.16 A history of hoarseness, stridor and dysphonia will direct attention towards the larynx. Radiological upper airway assessment using lateral neck x-ray, coronal tomogram and CT scan will demonstrate narrowing of the airway with asymmetrical thickening of the epiglottis and circumferential mucosal thickening of the laryngeal supraglottis and glottis. The clinical profile of laryngeal TB in adults is indistinguishable from that of laryngeal carcinoma and the two entities can occur simultaneously.

SINONASAL MANIFESTATIONS

Rhinosinusitis is the most common sinonasal feature in HIV-infected patients.17 The common causative organisms, clinical features and complications (Figure 3) are similar to those that occur in non-HIV infected patients, but with a greater tendency to develop multiple sinus pathology.18,19 However, as the CD4+ T cell count falls below 200 cells per microlitre patients become more susceptible to a variety of exotic opportunistic bacterial, fungal and protozoal infections such as Pseudomonas aeruginosa, Entebacter species, Escherichia coli, Serratia marcescens, Aspergillus species, Alternaria alternata, Cryptococcus neoformans, Candida albicans, Acanthamoeba castellani and Legionella pneumophila.18,19 HIV infected patients with sinusitis, including those with low CD4+ counts and opportunistic microbial pathogens, usually progress to full recovery with appropriate medical (antibiotics and decongestants) treatment and surgery if indicated.20

Nasal congestion and obstruction occurs in HIV-infected patients as a result of adenoid hypertrophy, allergic rhinitis and a variety of sinonasal lymphomas.17,20,21,22 Adenoid hypertrophy, the most common cause, may be due to adenoidal infection with viruses (including HIV, cytomegalovirus and Epstein-Barr virus) that cause proliferation of B lymphocytes in the lymphoid adenoidal tissues or occur as part of a generalised HIV-related lymphadenopathy. It is reasonable to suggest that finding an enlarged adenoid in an adult should raise the suspicion of HIV infection. Persistent B cell activation in HIV-infected patients leads to increased production of circulating immune complexes and immunoglobulins (IgA, Ig G and IgE). A two-fold an increase in the frequency and intensity of IgE-mediated allergic rhinitis has been observed in HIV-infected men.22

Adults require a mandatory definitive diagnosis of laryngeal TB by histological examination. Laryngeal TB is a treatable disease that responds well to antituberculous drug therapy.
Symptomatic relief of rhinorrhoea, nasal congestion and obstruction may be achieved with use of topical nasal steroids and systemic antihistamines. However, in cases where adenoid hypertrophy is refractory to medical therapy, an adenoidectomy may be performed.

HIV-infected patients who develop nasal malignancies tend to have severe immunodeficiency, extranodal disease, aggressive histological findings, and a poor treatment response. Non-Hodgkin’s lymphomas of the B cell variety are the most common histological type and patients typically present with epistaxis, nasal obstruction and rhinorrhoea. A multidisciplinary management approach is required and this will include confirming the histological diagnosis by needle aspiration or tissue biopsy, the administration of systemic chemotherapy and local radiotherapy.

**ORAL AND PHARYNGEAL MANIFESTATIONS**

Oral lesions occur in up to 100% of HIV-infected patients and, when present, can serve as indicators of the progression and severity of the infection. Common lesions include oral candidiasis, herpes simplex infection, oral Kaposi’s sarcoma, oral hairy leukoplakia, parotid gland enlargement, gingival diseases, xerostomia and recurrent aphthous ulcers. Oral candidiasis, caused by *Candida albicans*, is the most common oral pathology in patients with HIV infection. The lesions may involve any part of the oral mucosa and often extend to involve the mucosa of the pharynx, oesophagus and larynx. It is usually one of the earliest manifestations of HIV/AIDS and can occur in patients with CD4+ T cell counts between 200 and 500 cells per µl. The lesions may present in different forms, the most common of which is a pseudomembranous white plaque that is easily scrapped off to reveal an erythematous haemorrhagic base. The diagnosis is made by microscopic examination of unstained wet mounted, or Gram-stained mucosal scrapings which reveal characteristic yeasts and hyphae. Topical and systemic antifungal agents, such as clotrimazole and ketoconazole respectively, are effective therapeutic agents.

Oral hairy leukoplakia is an AIDS-defining mucosal disease associated with Epstein–Barr virus infection which occurs in 50% of patients with severe immunosuppression. The lesions appear as painless hairy white plaques that form on the lateral surface of the tongue and resembles candidiasis except that they cannot be scraped off and do not respond to antifungal therapy. Oral hairy leukoplakia can be diagnosed clinically and a confirmatory biopsy is not always required. Although there is no specific treatment, the lesions may resolve with antiviral therapy.

Oral neoplasms occur with greater frequency in patients with severe immune suppression and include tumours such as Kaposi’s sarcoma (KS), non-Hodgkin’s lymphoma and squamous carcinoma, of which KS is the most common. KS is an AIDS-defining systemic disease caused by Human Herpesvirus 8 which causes neoplastic proliferation of lymphatic epithelium with the formation of blood filled channels. All mucosal surfaces of the oral cavity may be affected and advanced lesions appear as dark erythematous lobulated masses. Clinical symptoms are due to pain and mass effect of the tumour and may include odynophagia and dysphagia. A tissue biopsy is required for a definitive diagnosis and treatment options will be tailored to the patients needs may involve HAART, chemotherapy, radiotherapy and surgery.
**Figure 7** illustrates an uncommon benign myxoid fibrous tumour that was present in an HIV-infected female patient in our institution.

**OTOLOGIC MANIFESTATIONS**

The incidence of otological diseases in HIV-infected patients is about 33%. The most common otologic complications are listed in Table 1, and the list is by no means exhaustive. Immunosuppression in HIV-infected patients predisposes them to the development of a wide variety of bacterial and fungal infections of the external and middle ear.

Otitis externa is an infection of the auricle and the cartilaginous portion of the external auditory canal. It is usually caused by *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Proteus mirabilis* and *Aspergillus* species. These microorganisms gain entry into the body through the skin and their entry is greatly facilitated by excessive irritation and mechanical trauma.

Patients will present with auricular pain that is aggravated by manipulation, pruritus, purulent otorrhoea, inflammation of the auricle, occlusion of the external auditory canal and a conductive hearing loss. If left untreated the disease may advance further to malignant otitis media with bone involvement of the skull base. Otitis externa is treated by aural toilet and by instilling topical antibiotics (e.g. ciprofloxacin) and steroids. Careful cleaning to remove infected debris must be done under good illumination with cotton wool on a wool carrier, or by suction under a microscope. Early diagnosis and prompt treatment cures the majority of cases without complication.

Serous otitis media and acute otitis media frequently occur in HIV-infected patients (Figure 8). The high incidence of middle ear disease is largely as a consequence of eustachian tube dysfunction secondary to recurrent viral and bacterial infections, hypertrophic adenoids, nasopharyngeal tumours. The symptoms (pain, hearing loss), the common causative microorganism (*Haemophilus influenzae*, *Streptococcus pneumoniae*) and management are the same as in non-infected patients. β-lactamase antibiotics and decongestants are the mainstay of treatment and surgery (tympanocentesis, myringotomy, adenoidectomy) may be considered in patients who do not respond to medical therapy.

Sensorineural hearing loss occurs in 20 to 50% of patients. A complete audiological evaluation should be undertaken and management should be directed to the underlying pathology. Aural rehabilitation with hearing aids should be considered for HIV-infected patients with no identified cause for hearing loss.

**CONCLUSION**

An exhaustive discussion of the otorhinolaryngologic-head and neck manifestations of HIV and AIDS is beyond the scope of this article. Due to the influence of numerous intrinsic and extrinsic factors, patients infected with HIV will differ widely in the clinical manifestations and progression of the disease. The pathogenesis of HIV infection is characterised by the progressive deterioration of the immune system and the subsequent emergence of opportunistic infections and tumours which eventually result in death. The head and neck region is an easily accessible area of the body, and manifestations of HIV infection are readily exposed. “Tell-tale” clinical signs and symptoms tend to develop in the head and neck during the early stages of the disease. It is therefore important for primary care physicians and otorhinolaryngologists to become familiar with common presenting lesions in order to ensure timely diagnosis and commence early treatment.

**REFERENCES**


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**COMMON MANIFESTATIONS OF HIV AND AIDS IN OTO RhINOLARYNGOLOGY**

Which of the following statements is/are true?

1. A 100 to 1000-fold rise in peripheral blood viral load occurs during the 6th to 12th week of HIV infection.

2. AIDS-related Kaposi’s sarcoma typically occurs when the peripheral blood CD4+ T cell count lies between 600-800 cell/µl.

3. Oral hairy leukoplakia does not respond to treatment with topical and systemic antifungal drugs.

4. Laryngeal tuberculosis of the larynx in HIV-infected patients typically presents with epistaxis, nasal obstruction and rhinorrhoea.

5. Effective treatment of otitis externa, caused by Pseudomonas aeruginosa, can be achieved by aural toilet and topical administration of ciprofloxacin ear drops.
THE ETHICAL TARIFF

Prof C A Joseph, Honorary Associate Professor, University of Limpopo (Medunsa) and the Witwatersrand.

The Ethical Tariff was introduced by the HPCSA in an attempt to set an upper limit to fees charged by doctors. This implied that fees above those proposed in the tariff would be unethical. This attempt to define an ethical tariff based on a particular fee level set in 2006 is incorrect.

The ethics of tariff is concerned with the doctors’ behaviour with respect to how fees are charged rather than the actual level of fees. Doctors may be unethical even when charging below the “Ethical Tariff” set by the HPCSA, conversely charges above the HPCSA tariff are not necessarily unethical.

Today medical ethics is essentially a code of moral behaviour that has developed through centuries of caring by a profession that is placed in a unique position of trust by their patients.

We can debate on an ethical dilemma or question based on:
1. Irrational approach
2. Rational approach

IRRATIONAL APPROACH

This may be difficult to understand or explain, but it “just feels right”. Doctors develop this over years of work with colleagues and patients where trust is paramount. Ethical behaviour is essential to maintaining this trust, without which high standards of professional care are impossible. Working in this environment endows the “just feels right” ability.

RATIONAL APPROACH

The rational approach consists of:

a. Principilism
b. Consequentialism

Principilism is essentially an application of the Hippocratic Oath encompassing “non nocere (first do no harm)”, justice and autonomy.

Consequentialism is the consideration of the consequences of the behaviour on quality of care, the greater good (altruism) and cost effectiveness (fiduciary responsibility).

Hippocrates 400BC recorded a code of behaviour (ethics) to be taken as an oath by doctors. It remains largely intact though rewritten by different institutions to suit the modern idiom.

An ethical tariff, therefore is one that “feels right”, is just, cost effective and respects a patient’s autonomy. It is about moral behaviour regarding the tariff and not the actual fee level. The fee level may vary according to quality of service, outcomes and practice costs.

Practice costs vary from group to group, specialist to specialist and geographically, so attempting to set a “ceiling” would be arbitrary. It could also have a negative effect on the quality of care and would therefore be unethical.

An “Ethical Tariff” is about behaviour with respect to fees and coding rather than a set fee or “ceiling”.

A doctor may charge a “low” fee, but still act unethically with respect to tariffs by behaving in any of the following ways:

• Overservicing ie performing unnecessary services (tests or procedures) for a fee.
• Padding accounts ie adding charges for services that are not rendered.
• Scheduling repeat visits or hospital admissions that are not indicated.
• Entering into a payment arrangement with an administrator of a fund or funds.

Most doctors would agree with the first three comments, but some may have a problem with payment arrangements.
Unfortunately these fail the ethical test on numerous levels, for example:

- Autonomy: both patient and doctor autonomy is negated or diminished.
- Quality care: compromised by time constraints and arguable protocols. Current payment arrangements completely ignore quality assessment and even fail to ensure all necessary services are reasonably available. Doctors are exposed to possible dual loyalty between what is best for the patient and what is best for the payment partner.
- Justice: this is not served by permitting 3rd party interference with patient management. These arrangements are aimed at benefiting 3rd parties and possibly doctors at the expense of the patient.
- Altruism: many plans lose money on payment arrangements resulting in patients usually on lower option plans cross subsidizing these losses (this is the poor paying for the rich). 50% of patients on the largest payment arrangement in SA are on losing plans. The greater good is not served, but the administrator of this largest payment arrangement profited by more than R1billion last year.
- Fiduciary responsibility: our ethical duty is to care for the patient and costs that they incur, not the profits of a 3rd party.
- Finally it just does not "feel right" to have a for profit administration partner in the doctor - patient relationship.

Charging set fees per procedure based on a patient’s funding plan (“payment arrangements”) rather than quality, experience and outcomes is unjust. Many, if not all of these payment arrangements are not cost effective (50% of members in the largest payment arrangement are in plans with operating losses), they are not altruistic (for the good of 3rd party profits rather than for the greater good) and do not measure quality or outcomes. Patient and doctor autonomy is ignored. Despite the quantum in these payment arrangements being below (up to 70% lower) the “ceiling” set by the HPCSA “ethical tariff” they are unethical.

An ethical tariff is less concerned with quantum than the application of codes and charges. However there could be situations where charging a higher quantum would be unethical.

Charging higher fees for PMB conditions based on the requirement that PMBs must be paid in full is unjust and therefore unethical. Fees should be based on experience, complexity and practice costs, not the patient’s medical plan or whether the fund must pay in full or not.

Generally doctors charge the same or less for PMB conditions (B Steenekamp, CMS research), which “feels right”. 67% charge NHRPL for non PMB conditions, but 70.3% charge NHRPL for PMBs.

These findings contradict the BHF claims that doctors abuse PMBs and that the PMB legislation is a threat to the financial stability of medical schemes. Furthermore the following graph demonstrates that the payout to consulting and surgical specialists is relatively small.

Table 1: Percentage of doctors charging NHRPL, 1x - 3x NHRPL and >3x NHRPL (PMB vs non PMB conditions). B Steenekamp

Table 2: Only 10.59% (VAT incl) is spent on consulting/surgical specialists. Approximately half of this is on surgery. Take out the VAT and only 4% of every rand paid to medical aids by patients is spent on surgeons.
The NHRPL tariff was declared unreasonably low, irrational and set aside by Judge Ebersohn, July 2010. Furthermore, the judgement found the failure to introduce tiered consults and the distinction between specialist groups’ remuneration was unreasonable and irrational.

Whether it is ethical to charge fees according to an irrational and unreasonable NHRPL tariff is arguable. The NHRPL is certainly unjust in its differential payments to specialist groups and could therefore be unethical. However it may be impractical to charge otherwise under conditions where funders refuse to pay doctors unless the NHRPL (or similar so called fund tariff) is charged. As the NHRPL is unreasonably low (Ebersohn judgement), then Rule J (for disproportionately low fees) may be applied to all NHRPL rates. Indeed, this may be the ethical answer to such unfair and unjust circumstances.

Ethics and the law are not always good bedfellows (it may be legal to disclose confidential patient information, but unethical). A moral compass demonstrates where these may differ with reference to tariff. Behaviour should remain in the red segment. Though the yellow segment is legal, it is unethical and should be avoided.

With the promulgation of the Consumer Protection Act in April, doctors who charge different fees, based only on the particular medical plan that a patient belongs to, for the same procedure or consultation are in contravention of the Act. This shifts payment arrangements from the unethical/legal quadrant to the unethical/illegal quadrant.

Doctors who believed such payment arrangements were both ethical and legal are now faced with the possible shift of payment arrangements to the illegal quadrant.

Managed care has created some confusion and misunderstanding. Legal processes are used to cloud ethics.

The doctor’s leadership is undermined by the use of “service provider”, thereby weakening the doctor’s role in disease management and the “feels right” ability to assess 3rd party interference. Managed care attempts to place “case managers” in control of disease management. Interference in the doctor-patient relationship is inevitable.

Managed care sets tariff at differential rates according to plans (unjust!), selects doctors and medicines (autonomy!).

![The Moral Compass](image-url)

Table 3

<table>
<thead>
<tr>
<th>ETHICAL</th>
<th>ILLEGAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Payment arrangements</td>
<td>Payment arrangements</td>
</tr>
<tr>
<td>Unnecessary procedure</td>
<td>Convenience admission</td>
</tr>
<tr>
<td>Overservicing</td>
<td>Overservicing</td>
</tr>
<tr>
<td>Padding accounts</td>
<td>Padding accounts</td>
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</tbody>
</table>

LEGAL

UNETHICAL

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and places cost before quality to ensure 3rd party profits and returns for shareholders. Unethical, yet it is a successful R2billion business in South Africa, all from patients’ premiums with little or no opposition from doctors. It surely contravenes the Consumer Protection Act and is currently under investigation. Hopefully the law will succeed where doctors have regrettably failed to protect their patients from this abuse.

CONCLUSION

The Ethical Tariff is not what you charge, but how you charge. It is a fair and just fee applied with regard to your patient’s right to autonomy, freedom of choice and quality medical care. It is a tariff that honours the doctor patient relationship without 3rd party interference. It takes cognisance of our fiduciary responsibility to cost effective care without sacrificing quality. The quantum is based on cost, experience and outcomes, respecting your patient’s right to time, respect and compassion.

REFERENCES

1. CMS report May 2010, B Steenkamp.
3. The High Court of South Africa case no. 37377/09, 28 July 2010, The Honourable Mr Acting Justice Ebersohn.
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Ethics