Oral and Maxillofacial Surgery

Revision Study Guide

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Springer

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Foreword

Mr. Abdul Ahmed sat the FRCS exit examination in 2013 and has successfully practised as a Consultant OMFS in Northwest London for 8 years. He is now in the "Goldilocks Zone" of his career, being at the peak of consultant practice but having been a trainee sufficiently recently to understand the demands and expectations placed on trainees as they enter the final phase of training.

Abdul is an enthusiastic and innovative surgeon, trainer, and educator introducing Robotic surgery, Sentinel node biopsy, Augmented Reality, and 3D planning to the OMFS department at Northwick Park Hospital. He is the current course Director for the London flap course and Clinical tutor of the Griffin Institute, developing the successful microvascular surgery course at Northwick Park which has trained several generations of surgeons from all surgical disciplines. He is also a senior lecturer for the University of London, an Educational supervisor for the London school of surgery and an international speaker.

This book covers the core curricula of the FRCS (OMFS) exit examination and represents the distillation of many hours of study condensed into 20 chapters of notes. The succinct format is successfully enhanced by diagrammatic illustrations used to emphasise relevant points without surrendering to over-elaboration. As an aide-mémoire, the book succeeds admirably, and as Abdul points out, it is most profitably used during the final weeks of study once the candidate has pored through the standard texts and scientific papers.

Although aimed primarily at Oral/Maxillofacial Surgery Trainees, due to overlapping anatomical areas of practice, it will also prove useful for Oral Surgery, ENT, and Plastic Surgery trainees.

A trainee who digests and retains the information in this book will never have to endure the discomfiture of having to guess an MCQ or discuss with an examiner, an aspect of Maxillofacial Surgery with which they do not possess core knowledge.

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Preface

The book's journey started whilst I was revising for my final FRCS exams in OMFS. I always knew I was a visual learner and liked all my facts crammed into as few pages as possible. To that end, I decided to collate all my revision notes, lectures, courses, and things I had read into bound A3 books. Using my micrographic handwriting, I crammed as much into each page as possible, with the help of Pritt Stick, and uni-ball micro pens.

Following the end of the worst period for most surgeons in training, I had this neatly packed manuscript of 8 months of my life embodied into its pages.

I became involved in running the FRCS revision course in London which, ran for several years, and I always had an idea to publish my manuscript in some form 'one day'. Months became years, and the hustle and bustle of clinical and family life put this on the back burner.

One day, the bounded pages just gnawed at me; One thing led to another, and I was writing to publishers to see if my pages were of any interest. Springer showed interest and the rest they say is history!

This book is primarily aimed at trainees coming up to the final FRCS exams. It is written mainly in short form with the majority comprising of lists and is intentionally not comprehensive. For that, the reader has many volumes of books to browse. Rather it is intended as a succinct aide-mémoire, and something you can read, write on, and make your own.

This book will not be to everyone's taste in its format and style. I would call it a 'dessert' rather than a 'main course' as the book demands a lot of prior reading.

I hope some of you find it helpful as you come up to possibly the easiest, but the most stressful exams of your lives.

Apparently, you should always have an early night before an exam, and so my hope is that this book will eventually only take a few hours to skim-read before the lights go out.

London, UK — Abdul Ahmed

Acknowledgements

Special thanks to Xi Man (Rose) Poon. When I started this journey, I had a set of scribbled notes with my difficult-to-read handwriting, annotations, and micrography. Rose typed the manuscript in its entirety and words cannot explain the amount of hard work that took.

Maryam Bennani, another one of my trainees, provided all the illustrations for the book, which really helps the reader to understand the concepts. Finally, Sarah Sacoor, who has an uncanny knack of spotting errors in the text. She did an amazing job with the final edit. This book would not have been possible without all three.

Finally, the amazing support of my loving, patient wife and two children, who had to bear with me going missing for the last few months whilst I was finalising the book.

London, UK Abdul Ahmed

Contents

Chapter 1
Facial Aesthetics

Abstract Overview of facial aesthetics including initial assessment, surgical and non-surgical treatment.

Key Points

- Indications for facial aesthetic treatment.
- Aetiology of ageing.
- Facial assessment for aesthetic treatment.
- Botulinum toxin mechanism of action, indications, contraindications, and side-effects.
- Treatment with chemical peels and microdermabrasion.
- Hair restoration techniques.
- Surgical techniques for rhinoplasty, blepharoplasty, brow lift, rhytidectomy and otoplasty.
- Use of dermal fillers.
- Fat augmentation.
- Uses of laser.
- Management of facial scarring.

Why?

- Look younger.
- Selection criteria.
- Expectations.
- Complications.
- Scars.
- Health.
- Biological years (not chronological).
- Skin.

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1.1 What Causes Facial Ageing

- Skin—Lower production of collagen.
- Fat—Atrophy.
- Bone—Increase in piriform aperture/Loss of teeth.
- Eyes—Fine lines, wrinkles, dark circles, puffiness, loss of firmness, dryness, and uneven texture.
- Muscles—Decreased tone (stretches/loosens).

Ageing

- Forehead.
- Midface.
- Lower jaw—Jowls (80%).
- Neck → Platysmal banding.

Ageing is a Combination of Gravity, Age, Time and Sun

- Lines (rhytids).
- Orthostatic (e.g. neck lines).
- Dynamic (e.g. crow's feet, procerus).
- Gravitational (e.g. jowls).

Skin

- Sun damage.
- Reduction in elasticity.
- Muscle → Flattened rete pegs.
- Collagen changes.
- Basal cells → Stem cells in skin, increases.
- Increased water loss with ageing → Moisturiser to keep hydrated.
- Subcutaneous fat.
- Atrophy.
- Gravitational descent.
- Malar fat pad descent.

1.2 General Assessment

- Medical history → HTN/NSAIDs → Increased risk of bleeding.
- Social history → Smoking → Affects healing (relative contraindication).
- Photographs → Examine for asymmetry.

1.3 Middle Third Ageing

Midface

- Loss of orbicularis oculi muscle tone.
- Descent of malar soft tissue.
- Illusion of excess in lower lid.
- Nasolabial creases descent.

Lower Third

- Chin ptosis.
- Thinning of subcutaneous fat → excess skin.
- Jowl formation.
- Platysmal banding.
- Loss of cervicomental angle.
- Submental fullness.
- Upper lip lengthening.

1.4 Assessment

1. ***Forehead***
    - Hairline—Especially men.
    - Brow position—Male at orbital rim, female maximum at lateral limbus.
    - Wrinkles.
2. ***Eyes***
    - Brow position.
    - Dermatochalasia → Loose connective tissue, non-elastic.
    - Lid fullness/fat herniation.
    - Lid crease → Position/asymmetry.
    - Lid laxity
        - Snap test → Pull vertical → Snap back in 1 s.
        - Pull test → Pull lower lid down >7 mm from globe.
    - Lagopthalmos → Tilt head back.

1.5 Nose

Examination

- Function → Breathe through nostrils → Cottle test (internal nasal valve) → Stretching cheek laterally.
- Internal nasal valve: junction of the septum with upper lateral cartilage.
- External valve: Lower lateral cartilage → Nostril vestibule.
- Worm's eye view → 'M' arch.
- Photos with 6 main views
  - Full face.
  - Full face R.
  - Full face L.
  - L lateral.
  - R lateral.
  - Basilar.

External

- Upper nose dorsum → Symmetry/deviation/width/radix position/NF angle/dorsal hump deformity.
- Nasal tip → Shape/definition/projection/nasal tip rotation/nasolabial angle.
- Columella → Angle (36–40°), alar-columella relationship.
- Skin thickness → Chin/nose relationship.

1.6 Botulinum Toxin (Botox)

Clostridum Botulinum (Anaerobic Gram +ve Rod)—Seven Serotypes (A–G)

- Toxin binds to pre-synaptic neuron of the neuromuscular junction, enters cell and acts by inhibitory release of acetylcholine.
- Results in neuromuscular blockade and flaccid paralysis.
- Irreversibly binds snap-2s protein.
- Reduction in motor activity and partial muscle atrophy → slowly reversed by reinnervation of muscle and development of extra junctional acetylcholine receptors.

1.7 Types

Botulism Toxin A

- Botox.
- Dysport (Azzalure) → 3 units = 1 unit Botox.
- Vistabel.
- Xeomin: Designed to deliver the toxin without any protein additives

  (a) 1 unit: Median lethal dose (LD50) in female Swiss webster mice, intraperitoneal injection.
  (b) 1 unit: 0.05 ng of toxin.
  (c) Botox reconstituted in sterile bacteriostatic saline.
  (d) 100 units in 5 ml → 24 in 0.1 ml → Each injection diffuses radially, can take up to 7–14 days to take effect → Length action 4–6 months, may need to repeat in 2–3 months (note dilution may vary depending on brand and type of treatment).

1.8 Indications

Function

- Strabismus.
- Blepharospasm.
- Recurrent TMJ dislocation.
- Frey's Syndrome.
- Sialocoele.
- Masseteric hypertrophy.
- TMJD.
- Hypersalivation.
- Facial dystonia/spasms.
- *Off license → Frey's Syndrome, Sialocoele, TMJD, Masseteric hypertrophy.
- *Licensed → Blepharospasm, facial dystonia, hemifacial spasm, spasticity, drooling.

Cosmetic → Wrinkles

- Glabella furrows.
- Forehead.
- Crow's feet.
- Nasolabial folds.
- Upper lip.
- Neck.
- Also mentalis 'pebbling'/'bunny nose'/brow lift.

Contraindications

- Pregnancy, breastfeeding, myasthenia gravis, neuromuscular disorders.
- Allergies, aminoglycans, calcium channel blockers, warfarin/DOACs/bleeding disorders.
- Local infection, inflammation or wounds.
- Body dysmorphic disorder.
- Caution with keloid scarring.
- Within 3 weeks of COVID-19 vaccination.

Side Effects

- Local: Pain, swelling, bruising, unintended relaxation of other muscles → Mild headaches following injection, asymmetry.
- Systemic: nausea, fatigue, rashes, dysphagia.

1.9 Chemical Peels

1. **Superficial** (epidermal loss) → Trichloroacetic acid (TCA) 10–25%.
2. **Medium** → Phenol (88%) TCA (10–25%) → injury to superficial dermis.
3. **Deep** → mid dermal damage → Baker-Gordon solution contains phenol, water, septisol, croton oil

    - Cannot → Reduce pore size/eliminate telangiectasias/eliminate deep scar/affect deep wrinkles.
    - Can → Improve appearance of sun damaged skin/flatten mild scarring/smoothen rhytids, and help with acne.
    - Lower pre-operative Fitzpatrick types translate to lower risk of pigmentation.
    - Avoid if history of Accutane in last 6 months, previous radiotherapy, previous facial cosmetic surgery, rosacea, psoriasis (allow time to think about it).
    - Treatment is non-specific wounding to increase and reorganise cutaneous collagen.
    - Chemical peeling, mechanical peeling, or laser. Aim to injure into papillary dermis or very top of reticular dermis.
    - Glycolic acid 30–70%.
    - Trichloracetic acid (TCA).
    - CO_2 laser.
    - Phenol peel.
    - αHydroxy acid peels (e.g. lactic acid, malic acid or glycolic acid).

1.10 Microdermabrasion

- ***Aluminium oxide crystals***.
- Exfoliate dead stratum using vacuum pressure → To pull blood and nutrients to surface of the skin.
- ***Management***
  - Dry/patchy skin, fine lines, wrinkles, sun damaged skin

    Over several weeks (7–10 weeks).
    Decreases scarring, hyperpigmentation.
    Develop erythema after 24 h.
- ***Dermabrasion***
  - Abrasive brushes to mechanically remove superficial layers → Similar to peels/lasers → May have increased risk of scarring/hyperpigmentation.
  - Best application: Deep scarring, deep rhytids, acne related pits or scars.

1.11 Hair Restoration

- Relocation or transfer of hairs from donor site area to area of hair loss.
- Initial introduction by Orentreich in 1959 on the use of punch grafts for hair loss.
- Micrografting introduced by Tamura in the 80s.
- Origin: Ectodermal.
- Consists of shaft and root.
- Types: Vellus hair (soft, hypopigmented) in the frontal area of scalp and over the body.
- Terminal hair (thick, long, pigmented) over the scalp, eyebrows and pubic area
- With age, vellus hairs replace terminal hairs.
- In bald areas, hair follicles are present but are atrophic.
- Aetiology: Androgenic alopecia. (Common cause). Inherent Secondary hair loss (e.g. stress, chemotherapy, metabolic disorders, trauma, and autoimmune disease.
- Pattern of loss: Men—crown of the scalp; Women—mostly diffuse loss.
- Classification: Norwood (1–7) Ludwig (1–3) for women.
- Pre-Surgery TX: Finasteride 1 mg (5 alpha reductase inhibitor)
  - Topical minoxidil.
  - Scalp massage.
  - Stop smoking.
  - Avoid hair products.
- Donor site: Occipital, parietal and temporal areas preferred.
- 50% can be harvested before its noticeable.

- Frequency:
  - Minimal hair loss 1–2 sessions.
  - Extensive hair loss 3–4 sessions.
- Procedure:
  - Clean the hair.
  - Trim donor area 2–4 mm in length.
  - Harvesting area marked.
  - Tumescent injected.
- Techniques: Local flaps
  - Excision of area of alopecia.
  - Free auto graft: micrograft (1–2 hairs) or mini graft (3–6) are used.
  - Grafts once harvested are kept in cool, saline solution.
  - Needles/mini blades are used for skin preparation.
  - Grafts carefully held below the hair bulbs.
  - Atraumatic placement of grafts into the slits.
- Post-Surgery: Bi-layered protective dressing
  - Wearing headband reduces swelling of the face.
  - Careful washing after 3–4 days.
  - Avoid trauma, strong shampoo.
  - Topical minoxidil.
- Complications: Infection/bleeding/haematoma
  - Folliculitis.
  - Periorbital oedema.
  - Hypoesthesia of the scalp.

1.12 Rhinoplasty

Assessment

- Frankfort plane: bony EAM → Infraorbital rim (good/reproducible reference plane).
- Basic steps
  - Assess fabric nose → Skin quality → Thickness/colour = Thin shows changes more.
  - Assess facial propositions → 1/3 facial height + 1/3 facial width.
  - Break down into following views.

- Proximal 1/3: Nasal bone (Figs. 1.1 and 1.2).
- Middle 1/3: Upper lateral cartilage.
- Lower 1/3: Lower lateral cartilage.
- Nasion: Nasofrontal suture.

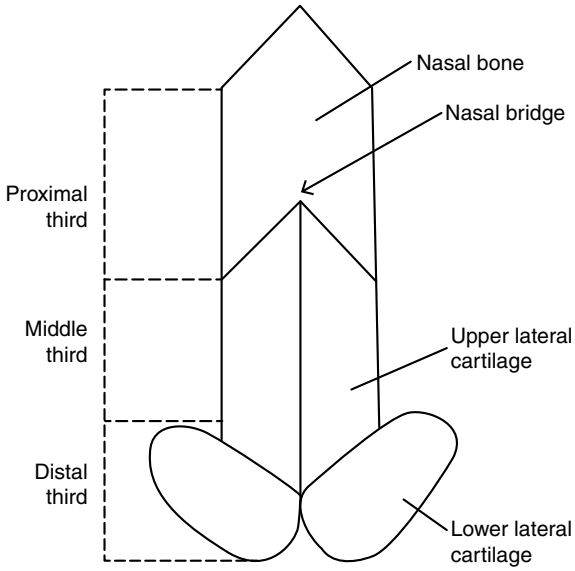

Fig. 1.1 Nasal thirds

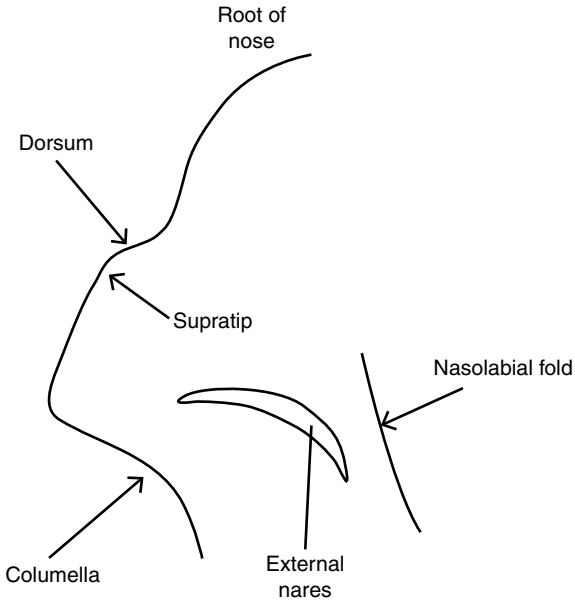

Fig. 1.2 Nasal anatomy

- Stellion: Soft tissue landmarks → Deepest point of nasofrontal angle → Slightly lower than nasion.
- Rhinion: Bony cartilaginous junction of nasal dorsum.
- Radix: Roof of nose.

Frontal

- Alignment: Straight/twisted/C-shaped/angled.
- Widths: Upper 1/3, middle 1/3, lower 1/3.
- Tip: Bulbous, bifid, asymmetric.

Lateral

- Profile: Hump, saddle, tip/supratip relationship, columellar show (2–4 mm).
- Nasal length: Long/short and starting point (high/low).
- Angles: Nasolabial, nasofacial, nasofrontal (120°).
- Tip projection ratio: Simons/Goode/Baum/Powell/Crumley.
- Chin projection: Balanced with the nose.

Base

- Triangularity (triangular/trapezius).
- Columella-lobule rotation.
- Nostrils (shape + size).
- Alar base width (intercanthal distance is the norm).

The Nose Planning

- Planning
  - Access → Open/closed.
  - Hump reduction.
  - Lateral wall osteotomy.
  - Tip: Rotation/projection strut/definition (shield graft).

Access Incisions

1. Inter-cartilaginous
   - Between lower and upper lateral cartilage.
   - Gain access to dorsum in closed rhinoplasty.
2. Trans-cartilaginous
   - Directly beneath the alar cartilage to split the cartilage.
   - Used for conservative volume reduction of lower lateral cartilage.
3. Infra-cartilaginous/Marginal rim
   - Along caudal edge of lower lateral cartilage to gain access to nasal tip.
   - In closed rhinoplasty, the lower lateral cartilage is delivered into direct view.
   - In open rhinoplasty, this incision is continued with trans-columellar to deglove the nose.

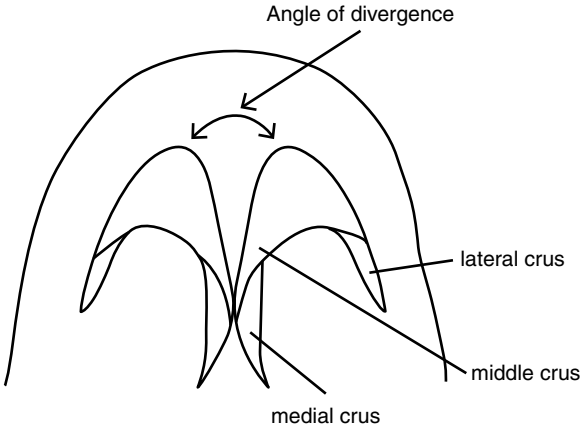

Fig. 1.3 Nasal tripod

4. Trans-columellar
    - Narrowed position.
    - Broken line for best cosmesis.
    - Small risk of shortening.

1.13 Nasal Tripod

Model for alar cartilage and tip support mechanisms (Fig. 1.3).

- Middle leg—Medial crura.
- 2 lateral legs—Lateral crura.
- Tripod apex—Nasal tip.

The tripod legs are held in place by various forces of tip.

3 major tip support mechanisms (Fig. 1.4)

1. Cartilage of medial/lateral crura.
2. Attachment of medical crura to the caudal septum.
3. Attachment of the lateral crura to the upper lateral cartilage.

Most rhinoplasties alter one of these mechanisms

1.14 Tip Correction

Tip suspension sutures to pull alar cartilage lobules in the cephalic direction

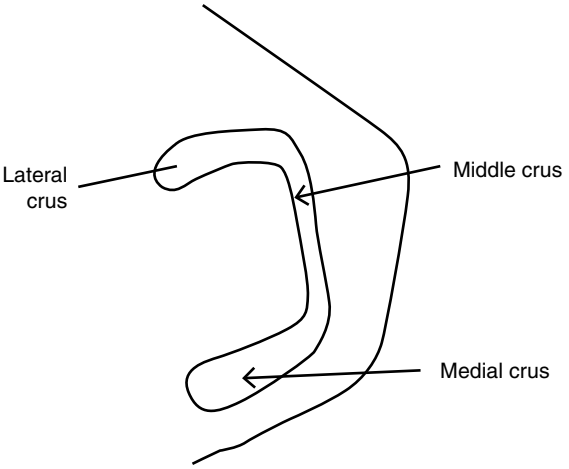

Fig. 1.4 Nasal tip support mechanisms

1. Resect cartilage from cephalic portion of alar cartilage or caudal edge of lower lateral cartilage.
2. Tip projection: Augmented using medial crural strut graft.
3. Tip grafts: Greater projection more contour/tip onlays—(Sheen shield graft).

Under-Projected Nose → Tip Projection → Too Little

High nasal dorsum

- Hypoplastic maxilla.
- Lack of support alar cartilage.
- Short columella.
- Secondary to surgery.

Suturing (Fig. 1.5)

1. Reduced intradermal distance—dome-sparing sutures.
2. Medial crural unification.

Grafting

1. Columellar strut– reinforcement and extension of medial support column.
2. Tip graft—sheen shield graft.

1.15 Dorsum

- Straight nasal dorsum—generic
  - Slightly concave: Female.
  - Slightly convex: Male.

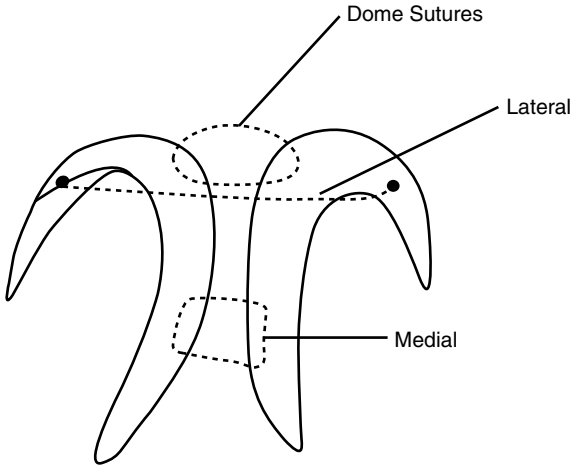

Fig. 1.5 Suturing techniques

- Long-term post-operative → More concave than original post-operative → Thinner skin overlying dorsum.
- Bony raspirator → Reduce nasal bridge without damaging cartilage → Then scalpel to remove cartilage.
- De-humping causes opening of roof
  - Narrow nose—Usually ok.
  - Broad nose—Open-book deformity needs osteotomies.
- Medial osteotomes → Free nasal bone from nasal septum.
- Lateral osteotomes → Free nasal bones from maxilla
  - *Bent medially → Using fine osteotomes.
  - *Internal osteotomy → Low to low/low to high.

Indication for nasal osteotomies: For narrowing wide lateral walls of the nose, closing open roof deformity, straightening nasal bony framework in a deviated nose.

Classifications:

- Described by the starting and ending points along the piriform aperture relative to the midline.
- A low osteotomy is positioned further off the midline while a high osteotomy is positioned more medially.
- Low-low osteotomy: For movement of large segment of lateral wall e.g. movement of bony roof is needed to correct a very wide nasal base or for large open roof deformity.
- Low–high osteotomy: To correct small open roof deformity/medium-wide nasal base.

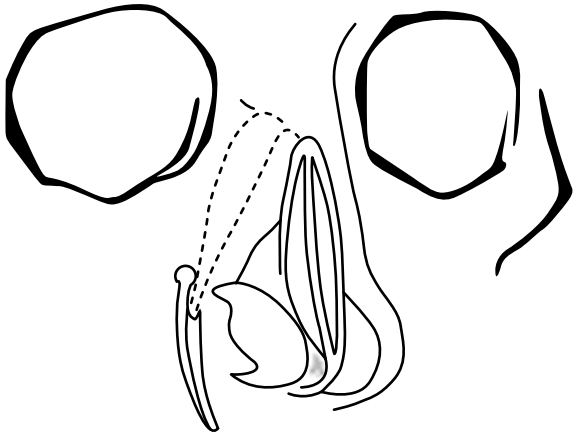

Fig. 1.6 Cartilage grafts

1.16 Nasal Valves

- Area inside the nose that lies beneath caudal edge of the upper/lateral cartilage → Area internal nasal valve.
- Internal nasal valve collapse → Spreader grafts to lateralise the upper lateral cartilage.
- External nasal valve → Alar collapse → Can be corrected via alar batten graft to stiffen and lateralise the lower lateral cartilage.

1.17 Grafts

Septal cartilage/ear cartilage/rib graft (Fig. 1.6)

(a) ***Spreader graft***

- Separate dorsal edges of the upper lateral cartilage from septal cartilage after reduction of dorsum, enabling physical width of dorsum to be maintained.

(b) ***Columellar graft***

- Strut: Fixed between medical crura with sutures.
- Supported/secured to ANS so does not move sideways.
- Alter nasolabial angle.

- Alar reduction → Nasal bone → Wedge excision of alar base
  - Narrows nostril.
  - Shortens ala.

Polly beak deformity: Post-surgical convexity at the supra-tip relative to rest of the nose → Complication of over-resection.
Over-resecting the nasal dorsum or nasal tip supports → Relative protrusion of the supra-tip region.

Correction (Combining Methods)

1. Resection prominent excess.
2. Grafting nasal dorsum.
3. Augmenting tip support/projection.

Anaesthesia (GA/LA) → 5% lidocaine, cotton along floor = 4 ml in adults.

Post-operative (Immediate)

- External splint: Tincture Benzoin/tape/splint/- remove 7 days.
- Internal splint: Septoplasty → Silastic → Keep septum midline and avoid haematoma.
- 6 months → Post-operative final form → Soft tissue takes months to settle.

1.18 Saddle Nose

Trauma

- Augmentation of nasal dorsum—bony part.

Infection

- Reconstruction of nasal dorsum—cartilaginous.
- Both → Augment + septum reconstruction.

Septum Perforation

- Myomucosal vestibular lip flap → Horizontal from under surface of upper lip.

1.19 Twisted Nose

- Significant post-traumatic deviation of the external pyramid → Deviated septum, needs septoplasty.
- Open rhinoplasty
  - Correction deviated bony pyramid → Osteotomies/mobilisation.
  - 2 mm micro-osteotomies with stab incisions.
- If not correctable → Camouflage techniques (overlap/grafts)
  - Septal cartilage.
  - Upper lateral cartilage.

1.20 Blepharoplasty

Anatomy

- **Upper eyelid (Figs. 1.7 and 1.8)**.
- Two fat compartments, medial and nasal that can be accessed through small incisions in the septum.
- **Lower eyelid (Figs. 1.7 and 1.9)**.
- 3 fat compartments. Care is taken not to damage the inferior oblique muscle that separates the medial from the middle fat pocket.

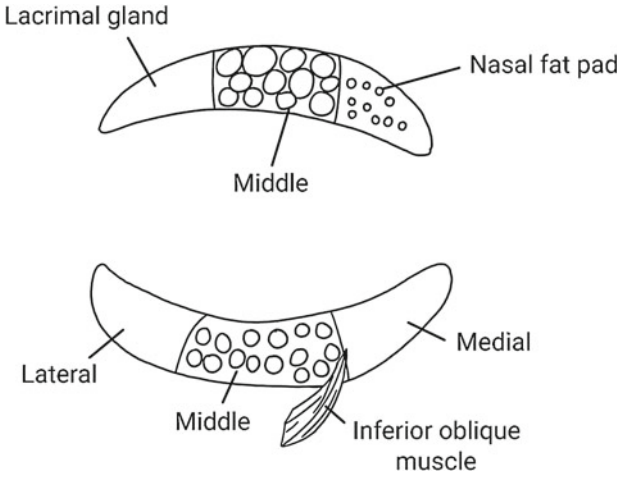

Fig. 1.7 Eyelid fat compartments

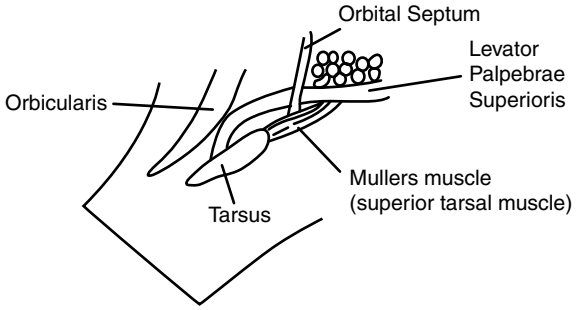

Fig. 1.8 Upper eyelid anatomy

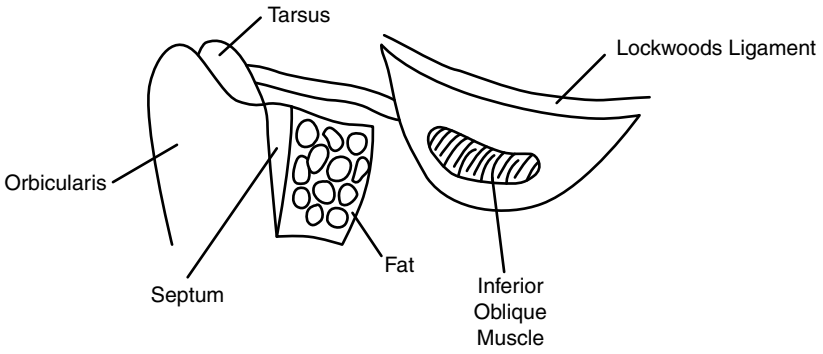

Fig. 1.9 Lower eyelid anatomy

1.21 Assessment

Brow Position

- Male at rim, female maximum at lateral limbus.
- Look for descent of eyebrows when eye closed → Demonstrates brow ptosis (compensated brow ptosis).
- The action of frontalis → Keeps eye elevated when eyes are open → Need brow lift.
- Lid fullness: Amount of redundant skin.
- Symmetry/Fat herniation.
- Lid crease → Create position/symmetry, position of eyelid in relation to eye.
- Lid laxity
- Snap test → Pull vertical → Snap back in 1 s.
- Pull test → Pull lower lid down >7 mm from globe.
- Lagophthalmos: Tilt head back and check.
- Press on globe → Fat herniation location.
- Assess Bell's phenomenon → Eyelid closure → Globe moves upwards/cornea protected. If absent, then risk of damage to cornea.

Procedure—Day Case/LA/Facial Wash (No Make-Up) → Check Eyebrows Stimulus Brow Lift

- Mark eyelids whilst patient is sitting up.
- Lower incision → Pinch excess skin until lids separate slightly.
- Incisions.
- Strip of tissue and orbicularis oculus excised (sutured back).
- Remove fat if required (small stab incision + bipolar).

  Lower Blepharoplasty → Easier to Excise too Much Causing Scleral Show/Ectropion

- Mouth open—tension lower lid → Incise below lower lash (subcillary) either skin/skin muscle flap raised.
- Conservative excision of excess tissue → May get scleral show→ May require canthopexy if too much fat removal → Excess can cause sunken appearance → Less of this done now
  - Fat preservation grooves between lower eyelids and cheek skin cause tear trough deformities.
  - Corrected by transposition of orbital fat into area.
  - Free fat/fillers/tear trough implants.

Transconjunctival Approach

- Remove fat from 3 compartments without scar.
- Does not address skin/orbicularis oculi muscles.
- Complications
  - Bleeding, haematoma, subconjunctival ecchymosis, chemosis, lagophthalmos, dry eye, poor scar, loss of vision secondary to retrobulbar haemorrhage.

Complications

Ocular Damage

- Corneal abrasion/ulceration → Eye protection.
- Dry eyes → Common due to oedema → Artificial tears (ophthalmologist)
  - Haematoma → Meticulous haemostasis/ice-cold compression.
  - Loss eyelashes → Diathermy.
  - Scleral show → Ectropion/round eye deformity → Too much excision.
  - Infection (rare).
  - Diplopia

    Transient—LA, oedema/haematoma (Müller's muscle).
    Inferior oblique damage→ Diplopia on upper and lateral gaze.
- Ptosis → Lid oedema/damage to levator apnoneurosis→ Immediate repair.
- Blindness → 0.04% → Retrobulbar haemorrhage → Inform patient risks
  - Severe pain behind the eye.
  - Proptosis.
  - Reduction in visual acuity.
  - Asymmetry → Over-correction?

1.22 Brow Lifts

Assessment (Fig. 1.10)

- Hairline especially men/shape.
- Brow position → Men at rim, women above rim.
- Wrinkles.
- Height forehead, hair distribution/density.
- Close eye → Ruler + check if brow lift up >2 mm (this suggests ptosis).

Anatomy

- Medial brow level with medial canthus and alar base.
- Men: At supraorbital rim.
- Women: Maximum high point lateral limbus.
- Distance mid-pupil to top of brow—2.5 cm.

Approaches

1. ***Open***
   - High/low.
   - 5 cm behind hairline → Dissect subgaleal plane superficial to deep temporal fascia laterally.
   - Mobilise lateral eyebrow of the supraorbital rim → Avoid supraorbital/supratrochlear nerves.
   - Resect corrugator.
   - Drape skin back → Resect → Sutures/drain.

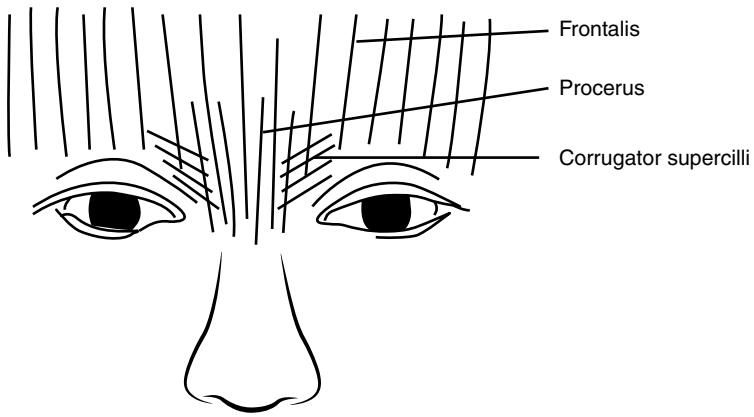

Fig. 1.10 Forehead anatomy

2. ***Mid-brow***

   - Within horizontal crease.
   - Transverse incisions in the galea.
   - Procerus/corrugator divided.

Direct/Suprabrow

- Mild ptosis (bushy eyebrows → scar).

Endoscopic

- Shorter scars, less tissue damage/less hair loss → Less effective (80% of the results, 20% scar).
- Not suitable if brow is very ptotic/very heavy.
- 3/5 ports.
- Tunnel endoscopy → Subperiosteally central, superficial to the deep temporal fascial laterally.
- Resect corrugator muscles, and supraorbital/supratrochlear nerves.
- Treat brows with lateral fascia sutures.
- With glue/screws/through bone tunnel to temporalis fascia.

Complications

- Supraorbital/supratrochlear nerve injury.
- Frontal nerve injury.
- Alopecia/numbness/asymmetry/hairline elevation/recurrent ptosis/scarring.

1.23 Rhytidectomy

Effects of Ageing/Gravity

- Brow ptosis.
- Prominent nasolabial grooves.
- Marionette lines.
- Hollowing of infraorbital regions.
- Jowls.
- Loose submental skin.
- Less elastic skin → wrinkles.

Facelift Anatomy

- Skin.
- Subcutaneous fat.
- SMAS
  - Superficial musculoaponeurotic system (SMAS).
  - Layer fascia → Continuous with frontalis muscles, galea aponeurotica, temporoparietal fascia, and platysma muscle.

  - Tightly bound to zygoma.
  - Less distinct anteriorly at the level of nasolabial folds.
  - Sensory nerves above.
  - Most facial nerves below.

- Muscles of facial expression.
- Thin layer fascia.
- Facial muscles.

Retaining Ligaments (Face) → Secures the Superficial Structures to the Bone

- Osteocutaneous: Zygomatic-mandibular (McGregor's patch).
- Musculocutaneous

  - Thickening of the fascia between muscles/skin.
  - Parotid-cutaneous ligaments.
  - Masseter.

- The fibre composing the zygomatic ligament (McGregor's patch) extends through malar fat pad and attach to dermis of the malar skin to suspend fat over the underlying malar eminence (superficial to SMAS) → Different techniques have been described to allow repositioning of the fat pad cranially.
- ***Platysma***

  - Part of the SMAS/muscle layer in the cervical area.
  - Medial aspects of the muscle becomes stretched with age, giving appearance of bands in the neck.

- ***Nerves***

  - Frontal branch → Facial nerve at risk → Pitanguy's line.
  - Greater auricular nerve → Sensation ear → When crosses sternocleidomastoid (Erb's point is where it crosses posterior border).

Four Basic Types of Skin Lifts

1. **Skin only** → Skin undermined and excess skin removed

   - Useful in very thin face who may be undergoing 2nd/3rd lift.
   - High relapse rate.
   - SMAS plication or lift considered necessary for long-term effects.
   - Can look somewhat unnatural.
   - Access via pre-auricular incisions

     - Temporal.
     - Post-auricular extensions.

2. **Skin + SMAS**—platysma

   - Skin elevated up to nasolabial fold.
   - The SMAS elevated at anterior superficial to zygomaticus to avoid damage to facial nerve.

- SMAS tightened/plicated- augments zygoma.
- As SMAS + platysma the same → Tightens SMAS, elevates platysma.
- The skin is draped over tightened SMAS.
- Additionally → On platysma
  - Platysmal plication medially.
  - Excision + resurfacing.
  - Gives more natural appearance.

3. **Skin + SMAS − platysma + midface suspension**.
4. **Deep plane and/or combination of above**.

Complications

- Haematoma, skin necrosis (beware smokers etc.), unfavourable aesthetic result, Frey's syndrome, facial nerve damage, hypertrophic scarring, alopecia, beware moving sideburn into ear in men, injury to greater auricular nerve.

Technique

- Prep and drape, tumescent solution, skin incision, skin flap, SMAS flap, plicate SMAS, excise skin, platysmal plication if necessary.

Original and Modified Tumescent Fluid Formulas

- Klein (Original Tumescent) Formula.
- 1000 ml NaCl, 50 ml 1% Lidocaine, 1 ml of epinephrine/adrenaline 1:1000, 12.5 ml of Sodium bicarbonate.
- Hunstad (Modified Tumescent) Formula.
- 1000 ml lactated Ringer's solution, 50 ml 1% Lidocaine, 1 ml of epinephrine/adrenaline 1:1000, Solution is warmed to 38–40 °C.

Mask/Subperiosteal Facelift

- Coronal and intraoral incision.
- Dissection subperiosteal over the forehead superficial to the deep temporal fascia to reach zygomatic bone.
- Attachments to zygoma relieved.
- Intraoral dissection precedes subperiosteal over midface.
  - Soft tissues suspended.
- Allows skeletal changes
  - Widening of orbital aperture.
  - Reduction of heavy supraorbital bone.
  - Augmentation of malar region.

MACS-Lift

- Minimal access cranial suspension.
- Shortened scar.

- SMAS suspended with non-absorbable sutures.
- Reduced healing time.

Endoscopic

- Dissected subperiosteally.

Complications

- Haematoma—3–5% commonest usually 10–12 h after surgery
  - Stop smoking, stop aspirin 10 days prior to the surgery.
- Neurological.
- Greater auricular nerve.
- Temporal branch facial nerve → Crosses zygomatic arch and travels along deep surface of the temporoparietal fascia → Penetrates the fascia to innervate frontalis.
- Buccal branch → Cross innervation.
- Marginal mandibular
  - Unable to depress/pull lateral/evert vermillion.
  - Damage via electrocautery/stretching/transection.
  - First 24 h may be due to local anaesthesia.
  - Conservative for 6 months
    - 80% recovery, baseline electromyography 6 months and repeat.
    - Botulinum toxin opposite side.

Skin Loss

- Skin slough.
- Especially smokers → Allow to heal secondary intention
  - Loss hair.
  - Infection.
  - Asymmetry.
  - Hypertrophic scars.
  - Pixie ears.

1.24 Ears

Key Notes

- Ears grown to a suitable size for otoplasty at the age of 5–6 years.
- Deformities classed as lop ear, cup ear, microtia or protruding ear depending on deformity (Rogers classification).
- Most common deformity in protruding ears is absence of antihelical fold and superior crural fold, and over-large conchal bowl.

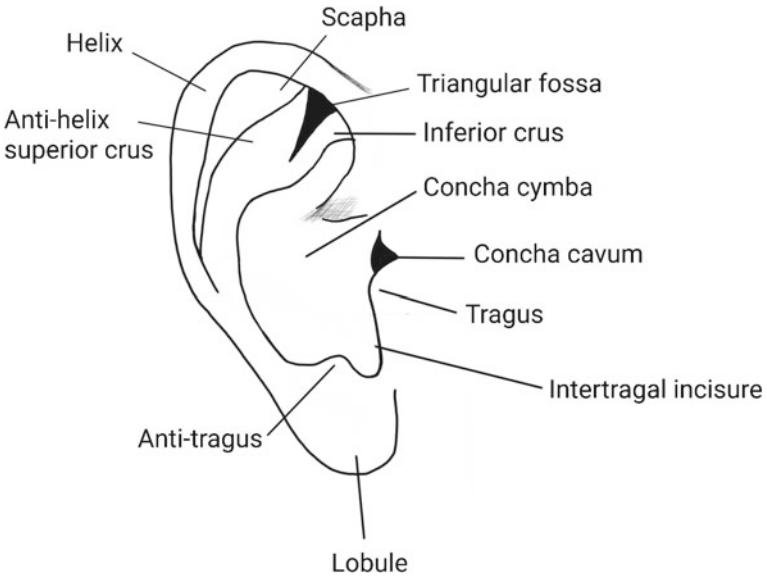

Fig. 1.11 Ear anatomy

- Ideal angle between the ear and the head is the auriculocephalic angle and measures around 30°.
- Helical rim should be at least 15–20 mm from the scalp or it will appear plastered to the head.

Anatomy (Fig. 1.11)

- Auricle formed at birth.
- 85% adult size at age 3 years.
- Adult size at 6 years.
- Blood supply
  - Superficial temporal → Auricular branches.
  - Posterior auricular → Cranial aspect ear.
  - Occipital.

Sensory

- V3 auriculotemporal.
- C3 Greater auricular.
- Vagus auricular branch (Arnold's nerve).
- Lesser occipital nerve.

Assessment

- Compare right to left.
- Level ear → Level of eyebrow.

- Angle to mastoid >30° prominent → Prominent (bat ear)
  - Protrudes excessively from temporal surface.
  - Normal 21–25°.
  - >25° → ear more prominent.
- Conchal depth.
- Helix/antihelix form
  - Poor.
  - Deficient.
  - Normal.
- Photographs.
- Common problem
  - Usually loss of definition of antihelical fold to develop.
  - Excessively deep concha.
  - Combination of above.
- Timeline for surgery
  - Child if obvious—application ear moulds.
  - 5–6 years: Before school if very prominent/psychosocial reason.
  - 14 years with LA/sedation.
- Surgery technique
  - LA infiltration. Skin excision, conchal bowl setback if appropriate. Creation of antihelical fold with either suturing (Mustardé technique) or scoring of cartilage (Converse technique).
  - Cartilage cutting.
  - Cartilage sparing.
  - >100 methods

    1. ***Mustardé***
       - Recreate antihelical fold → Sutures placed from scalp to concha to develop new antihelical folds.
       - Mark where you want to fold with ink from front to back (press on ear medially to create helix).
       - Mattress sutures with nylon.
    2. ***Furnas***
       - Concha-mastoid sutures → Mark with needle from front, concha sutured to underlying mastoid fascia with mattress sutures.

Cartilage Scoring Technique—If Patient Has Thick Cartilage

- Gibson's principle.
- Cartilage when scored → Bends/warps away from scored surface by release of interlocked stressors.
- Score → Tunnel from posterior → Bend needs right angles—score.
- Anterior incision along inside edge of helix.
- Then place sutures.
- Incisional technique
  - Pair of parallel incisions.

Complications

- Relapse (10–24%)/recurrence.
- Reverse telephone/telephone deformity → Inappropriate placement of sutures.
- Haematoma → Immediate draining.
- Wound Infection.
- Asymmetry.
- Keloid scars/hypertrophic scarring altered vascularity.
- Cartilage necrosis.
- Paraesthesia.
- Suture extrusion.
- Plastered look.

1.25 Fillers

Injectable Substances Used to Augment Skin

Used For:

- Wrinkles (static/dynamic).
- Nasolabial folds.
- Marionette lines.
- Depressed scars.
- Augmentation of lips.

Types

- Autogenous fat.
- Autogenous dermal fat.
- Hyaluronic acid derivatives (e.g. Restylane, Juvederm).
- Synthetic Poly-L-lactic acid (e.g. Sculptra).
- Liquid silicone.
- Collagen (used for many years → Drawback allergy risk, skin testing)
  - Hyaluronic acid → Natural carbohydrate polymer found in ground substance of connective tissues—lasts up to 6 months.

- Sculptra → Poly-L-lactic acid → Stimulates production of autogenous collagen → Delayed effect → Needs seen/treatment last for 18–24 months → Useful in aiding lipoatrophy (volumiser).
- Do not use permanent fillers → Complaints/impossible to deal with infections.

Method

- Training specific—inject intradermally into subcutaneous → Good injection technique/fan-shaped manoeuvre/along different directions.
- Fill deeper grooves.
- Massage to get even distribution.

Complications

- Bleeding/infection/uneven injection (subcutaneous lumps).
- Granulomas → May need steroid injections.
- Tissue necrosis.
- Injection into blood vessel (risk of loss of vision).
- Management: Hyaluronidase.

1.26 Fat Augmentation

Coleman's technique → Concentration of fat cells by centrifugation and use by injecting tiny globular amounts in different areas/planes → Layering technique.

Indications

- Contour defects.
- Small facial defects.
- Depressed scars.

Donor Sites

- Abdominal wall.
- Thigh.

Contraindications

- Recent history of DVT, PE.
- Liver function.
- Coagulopathy.

Technical → Tumescent Infiltration

- ± Steroid→ Reduce inflammation → Triamcinolone.
- ± Hyaluronidase → Reduce breakdown connective tissue → Easier to harvest.
- Harvest fat with syringe.
- 10ml using gentle suction (low negative pressure → Move back and forth).
- Syringe then centrifuged 3000 rpm for 2 min.

- Concentrated fat cells collected → Decant top layer.
- Transfer to 2 ml syringe → Inject with 18G needle layering technique.
- Must over correct by 20–30%.

- Post-operative
  - Oedema → Steroids.
  - Ice packs for 48 h.
- Complications
  - Oedema/contour irregular.
  - Infection.
  - Fat embolism
    - Intracerebral.
    - Sudden blindness.
  - Fat cysts/hard nodules.
  - Post-inflammatory pigmentation → Hyaluronic acid 4% for 4 weeks or IPL (Intensive Pulsed Light).

1.27 Laser (Light Amplification by Stimulated Emission of Radiation)

Generated in an optical chamber controlling crystals or gas and mirrors at each end, chamber electronically charged, and laser energy generated

Laser Beam

- Monochromatic → Same wavelength.
- Collimated → Not spread out.
- Coherent → Waveform peaks/troughs aligned
  - Absorbed by active medium → Resonator → CO_2/argon/helium.

Laser

- Pulsed.
- Continuous → CO_2 → More energy to tissue causing thermal damage and coagulation.
- Super-pulsed → vaporise tissue it contacts only → minimised thermal speed.

Chromophores Are Light Absorbing Compounds

- Water/Melanin/Haemoglobin/Collagen/Oxyhaemoglobin.

Advantages

- Vaporisation of tissue and coagulation → Denatures collagen → Haemostasis <0.5 mm diameter vessels.
- Less pain.
- Less scarring.
- Denaturation of proteins (including inflammatory mediators) → Underestimation by body of damage sustained.
- Sterilisation of tissues.

Disadvantages

- Plume→ Hazardous chemicals/biological agents → Need evacuation system—scavenging via high flow two-stage filtration.
- Dangerous laser light → Goggles/laser ET tubes/fires.
- Delayed healing.

Complications

- Infection, bleeding, airway fires, tooth damage (enamel/pulp), hyper- or hypopigmentation, contact dermatitis, acne/milia outbreaks.

Types

A. **CO_2**

- 10,600 nm.
- Chromophore water.
- Excision of skin/mucosal lesions + vaporisation/deep skin resurfacing.

B. ***Pulsed Dye Laser***

- 585 nm.
- Chromophore.
- Target → Haemoglobin, melanin.
- Used on:
  (a) Vascular lesions → Capsular/venous, facial telangiectasia.
  (b) Pigmented lesions → Scar revision, tattoos.

C. ***Potassium Titanyl Phosphate (KTP)***

- 532 nm.
- Oxyhaemoglobin.
- Used on telangiectasia/vascular lesions.

D. ***ND:YAG***

- 1064 nm.
- Chromophore—tissue proteins.
- Used on deep benign vascular lesions.

E. ***Intensive Pulsed Light (IPL)***

- Different to lasers.
- Multiple wavelengths.
- Target specific chromphore.
- Facial scarring/wrinkles.

1.28 Facial Scars

Prevention

- Incise along relaxed skin tension lines.
- Tension free closure.

Hypertrophic

- Scars that are thickened and raised above the skin surface but remain within original borders.
- Wounds heal and over-proliferative response results in excessive ECM, collagen.
- Usually regress spontaneously → More responsive to treatment.
- Common: <20 years.
- Familial inheritance.
- M > F.

Aetiology

- Poor wound healing.
- Infection/wound dehiscence/tension.
- Chest, shoulders, anterior neck.
- Wound healing with secondary intention.

Keloid

- Thickened/elevated, extend/beyond boundaries of original wound.
- Pathophysiology:
  - Build-up of collagen.
  - Excessive production.
  - Decreased in degradation.
  - Collagen less cross-linking.

Usually occur → Up to 1 year after trauma/injury → Reach specific size and do not regress

Epidemiology

- Common <30 years.
- Familial incidence.
- 5–15× more common Afro-Caribbean.
- 2× more common Chinese and Japanese.

Aetiology

- Genetic relationship.
- Poor wound healing
  - Commonly found → Face/ear lobes/anterior chest.
  - Management.
- Assessment: History, medical history, family history.
- Examination: Other scars.
- Investigation: Pre-treatment photographs.

- Conservative → Allow to settle for 1 year or until pale → Symptoms controlled.

1. ***Pressure***
   - Elasticated garments → Increase collagenase → Increase wound metabolism and speed of maturation of scar.
2. ***Silicone gel (hydrogel)***
   - Affect healing, 12–24 h/day → 2–3 months → Action unknown.
   - Combination → Pressure/wound hypoxia/increase skin temperature/direct chemical agents (Dermatix).
   - 80% success hypertrophic scars.
   - 33% success keloid scars.
   - Decreases oxygen tension.
   - Provides pressure.
   - Reduces collagenase activity.
3. ***Tape***
   - Along scar longitudinally for 2 months to prevent scar → Reduced tension along wound.
4. ***Corticosteroids***
   - Intralesional trimacinolone.
   - Side effects → Pain on injection, hyperpigmentation, crystalline deposits, telangiectasia, atrophy.
   - Reduce collagen levels, reduce inflammation, reduce itching and tenderness.

5. ***Topical vitamin A***
   - Inhibits fibroblasts.
6. ***Laser***
   - Reduces pigmentation/inflammation.

Surgery

- Wait until mature scar, revision—consider cause → Prevent recurrence → Warn patient risk of worsening the scar.
- Scar excision → Better surgical closure surgical closure.
- Serial excision → Staged → Allows adjacent skin to relax.
- Break up scar/or alignment → Z-plasty/W-plasty/graft/flap.

Radiation

- 5–6 doses, 15–20 days.
- Use it to prevent it.
- Superficial low dose 10–15 days in very high-risk patients.

Steroids

- 75% success with surgery (at time of surgery).

Chapter 2
Orthognathic Surgery

Abstract Overview of orthognathic surgery, from initial evaluation and clinical examination to some treatment planning techniques.

Key Points

- Goals of surgery and clinical examination.
- Treatment pathways from initial orthodontics to surgical phases of treatment.
- Surgical planning and classical patterns seen in patients.
- Maxillary and mandibular surgical techniques.
- Soft tissue changes, and hierarchy of stability.
- Special cases including anterior open bite, condylar hyperplasia, progressive condylar resorption, and vertical maxillary excess.
- Finally, obstructive sleep apnoea and macroglossia.

Goals of Surgery

- Function: Mastication/speech/respiratory/occlusion/sleep apnoea.
- Aesthetics: Facial harmony/balance.
- Stability: Avoid short-term/long-term relapse.
- Minimisation of treatment: Efficient/effective treatment.

Stability: The long-term maintenance of the achieved post-operative result.
Relapse: Movement towards the pre-operative position (skeletal or dental).
Migration: Continued movement in the direction of the initial movement.

Patient Evaluation

1. Full medical and dental history (Systematic MDT approach: Surgeon, orthodontist, psychologist, restorative dentist, hygienist, and technician in some centres).
2. Patient concerns: Motivation/expectation, specific concerns: what/why/what to expect/previous treatment.

Clinical Examination

- Sitting upright in straight-backed chair.
- Examiner directly opposite the patient at eye level.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_2

- Clinical horizontal plane: parallel to floor—clinical Frankfort plane (tragus—infraorbital rim). Patients often mask asymmetry by adopting a certain posture.
- Seat condyles in glenoid with teeth lightly touching (centric → tongue tip on posterior palate/relax jaw position).
- Patient's lips relaxed.

The aim is to assess the patient in three planes.

2.1 Vertical Anteroposterior (AP) Transverse

Vertical

- Symmetry of face/dental midline.
- Divide the face into thirds (Fig. 2.1).

Frontal View:

- Facial third: Upper hairline (trichion) to glabella, glabella to subnasale, subnasale to soft tissue menton.
- Anterior lower facial third: Often slightly greater than the middle third, particularly in males.
- Anterior lower facial third: Further subdivided into
  - Upper lip (subnasale to stomion) 1/3.
  - Lower lip and chin (stomion to soft tissue menton) 2/3.

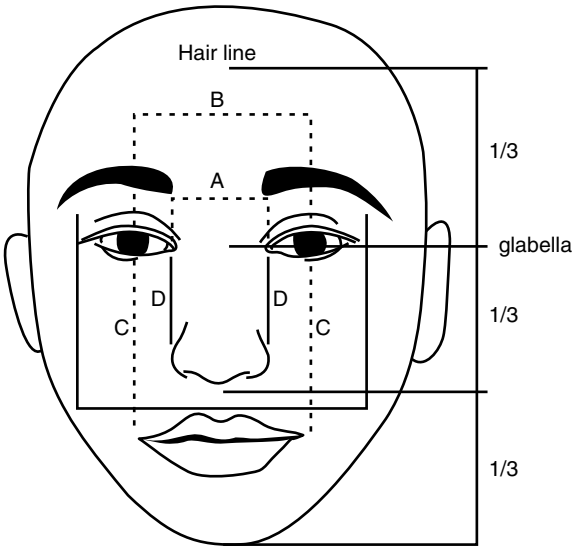

Fig. 2.1 Frontal facial thirds

- Rule of fifths—each fifth is approximately the width of an eye.
- Brow tip aesthetic line.
- Centre lines: Nasal/dental upper and lower/chin point.
- Upper lip length 20 mm for female/22 mm for male: Incisor show at rest 3–4 mm and dynamic approximately at the gingival margin.
- Dental centreline relationship.
- Occlusal plane/cants.
- Intraoral
  - Intercanthal distance (same as the alar base width)

    32 ± 3 mm (Caucasians).
    35 ± 3 mm (Afro-Caribbean/Asians).
    Intercanthal distance = alar base width- vertical line through medial canthus should coincide with the alar base ±2 mm.

  - Interpupillary distance

    65 ± 3 mm = width of mouth.

- Easily remembered as '33 mm' (intercanthal width) '66 mm' (interpupillary width) '99 mm' (lateral canthal width).

Upper Lip Subnasale → Upper Lip Stomion

- 20 ± 2 mm = Female.
- 22 ± 2 mm = Male.
- Tooth/lip—exposes 2.5 mm ± of incisal edge with lip in repose.
- Smiling vermillion—fall at cervicogingival margin no more than 1–2 mm exposed gingiva.
- Assess crown length upper central incisors for microdontia/tooth surface loss.

⇒ If patient lips over closed (generally skeletal III) → Open jaws until lips just begin to separate to evaluate lip length/tooth to lip relationship.

Amount of upper lip elevation during smiling affected by

- AP relationship: maxilla/mandible to cranial base.
- Overbite/overjet, angulation of teeth, clinical crown length, neuromuscular function.

Anteroposterior View (Fig. 2.2)

- When maxilla normal AP with normal upper lip thickness, ideal chin projection is 3 mm posterior to line through subnasale that is perpendicular to clinical Frankfort plane—zero meridian.
- Line perpendicular to clinical Frankfort plane and tangent to globe should fall on infraorbital soft tissue ±2 mm.
- Upper labial superioris → 1–3 mm anterior to subnasale.
- Cervicomental angle—110°. Chin throat length average 4 cm.

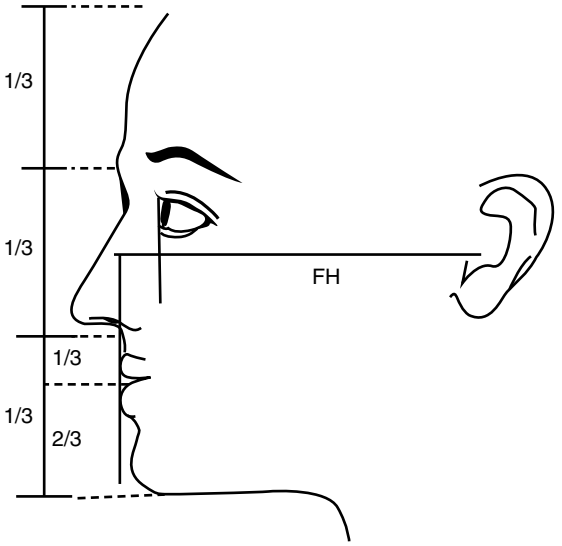

Fig. 2.2 Lateral facial thirds

- Nasolabial angle—100° ± 10°
  - Comment on frontal bossing, nasal shape, nasofrontal, nasolabial, labiomental, cervical fascial.
  - Dorsum of nose (nasal tip rotation).
  - Maxilla hyper-/hypoplastic–(paranasal hollowing).
  - Gross skeletal pattern (I, II, III), convex/concave.
  - Infraorbital rim: The globe 2–3 mm in front of the orbital margin.

2.2 Transverse View (Intraoral Evaluation)

- Maxillary and mandibular centrelines coincident with the facial midlines including chin point.
- Intraoral view → Maxillary or mandibular cants (Can be assessed with a spatula/occlusal plane guide. Measure from the medial canthus to the canine tip).
- Buccal corridor and evidence of vertical maxillary excess or deficiency.
- Arch form, U or V-shaped, wide or narrow.
- Missing teeth/crowding arch width/teeth questionable prognosis.
- Molar relationship = Angle's Class 1 → Mesiobuccal cusp upper 6 rests on buccal groove lower 6.
- Occlusion in retruded contact position (RCP → tongue to the roof of the mouth), edge to edge, traumatic bite, scissor or crossbite, displacement, posturing.
- Measure: Overjet/overbite.
- Missing teeth/crowding/arch width/teeth questionable prognosis.
- Wisdom teeth.

- Comment on curve of Spee.
- Incisor angulation and amount of labial bone.
- Lip competence and position.
- Oral hygiene, restorative and periodontal status.
- Tongue position.
- Formal temporomandibular joint assessment if indicated by the patient's history.

2.3 Special Investigations

- Orthopantomogram–general dental assessment for caries, bone levels, 3rd molars.
- Lateral cephalogram taken in the Frankfort horizontal position.
- PA cephalogram–can be considered for asymmetry cases generally of historical interest.
- Occlusal or periapical views.
- Cone beam CT for unerupted teeth or mandibular third molars with an intimate relationship to the inferior alveolar nerve.
- Cone beam or Medical CT for asymmetry cases (permits 3D printing for model surgery or digital planning).
- Study models.
- Digital intraoral scanning.
- Photographs.

2.4 Management Sequence

- Initial assessment and diagnosis (as above).
- Joint clinic discussion and options.
- Orthodontic goals.
- Surgical objectives.
- Desired facial profile.
- Definitive plan.
- Vertical position of maxillary incisor.
- Anteroposterior position of the maxillary incisor.
- Occlusal plane angulation.
- Presurgical orthodontics → Surgery → Post-surgical orthodontics.

2.5 Cephalometric Measurements (Fig. 2.3)

Frankfort Horizontal Plane (Porion Through Orbitale), Go to Me Mandibular Plane

- A point (deepest point of maxilla).
- B point (deepest point of mandible).
- Na(nasion) anterior point of the intersection between nasal and frontal bones.
- Orbitale (Or) lowest point of orbit.
- Sella (S) midpoint of sella turcica.
- Pogonion (Pog) most anterior point on chin.
- Gnathion (Gn) centre of the anterio-inferior point on the mandibular symphysis.
- Menton (Me) most inferior point mandibular symphysis.
- Condylion most posterior and superior point on the condyle.
- ANS tip pf anteripr nasal spine.
- PNS tip pf the posterior spine of the palatine bone
  - SNA: 82 (relation of the maxilla to the cranial base) STEINER.
  - SNB: 80 (relation of the mandible to the cranial base).
  - ANB: 2 (relation of the maxilla to the mandible).
  - UI: 109 ± 6 (upper incisor angle).

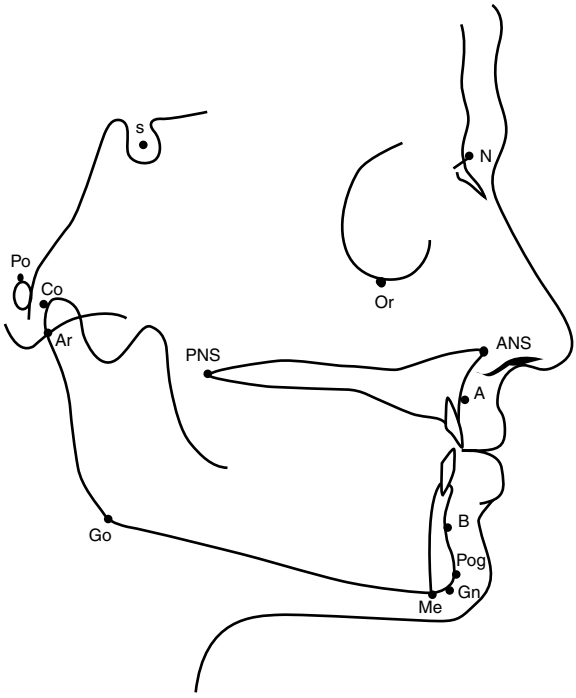

Fig. 2.3 Cephalometric landmarks

- LI: 93 ± 6 (lower incisor angle).
- I/I 135 (interincisal angle- increased in class II).
- AFH (Na-Me) 124 =/− 8 (anterior facial height).
- AUFH (Na-ANS) (anterior upper facial height).
- ALFH (ANS to Me) 34 =/− 5 (anterior lower facial height).
- PFH (S-Go) 99 =/− 6 (posterior facial height).
- FMPA = 25 (high angle >40) (Frankfort mandibular plane angle) TWEED.
- Occlusal plane angle = 10 (compared to Frankfort horizontal) DOWNS.

2.6 Pre-surgical Orthodontics

- Decompensation.
- Arch alignment-relief of crowding.
- Arch levelling
  - If increasing lower anterior face height in class II with mandibular advancement: maintain or increase curve of Spee–3-point landing.
  - Maintain face height.
- Level arch pre-operatively.
- Or segmental surgery with levelling of occlusal planes.
- Anterior open bite.
- Usually involve impaction of posterior maxilla → Retrocline upper incisors, consider proclination.
- Consider segmental (3 part) maxilla, root divergence at surgical sites.
- Arch coordination/expansion.
- Quadhelix: proclines tipping, can get differential expansion.
- Surgical assisted—SARPE (surgical assisted rapid palatal expansion), MARPE (miniscrew assisted).

2.7 Hierarchy of Stability: (Profitt/Turvey 2007)

- Maxillary impaction.
- Mandibular advancement Highly stable >90% less than 2 mm (relapse).
- Genioplasty any direction.
- Maxillary advancement.
- Maxillary asymmetry stable >80% less than 2 mm.
- Maxillary impaction and mandibular advancement.
- Maxillary advancement and mandibular setback stable with rigid fixation only.
- Mandibular asymmetry.
- Mandibular setback.
- Maxillary set down major relapse possible.
- Maxilla widening.

Assessments

Plan the maxillary position to the cephalometric McNamara vertical and the clinical incisor edge at rest

Normal incisors show: 2–4 mm at rest with gingival margin on smiling.
Normal upper lip length 22 ± 2 mm (male): 20 ± 2 mm (female).

Profile Assessment

- Zero meridian:
  - Vertical line from nasion (perpendicular to Frankfort plane).
  - Chin: 2–3 mm behind line.
  - Upper lip: 2–3 mm Infront of line.

2.8 Surgical Planning

Determine anteroposterior position of the maxilla and upper incisor show

- 2–3 mm show → Advancement to increase show, soft tissue moves 80% of the advancement.

Determine position of posterior maxilla

- AP/rotational movement, axis of rotation must be specified.
- AOB–differential impaction.
- Bilateral crossbite—SARPE versus orthodontic expansion (limited to 5 mm).

Mandible to Class 1

(1) Autorotation–centre of condyle.
(2) Affects autorotation.
(3) Decrease anterior face height.
(4) Increase Posterior upper face height.
(5) Levels mandibular plane.
(6) Upright mandibular incisors.
(7) Increase Chin prominence–soft tissue/bony/combination.
(8) Clinical freeway space re-established through proprioceptive feedback mechanism.
(9) Counterclockwise rotation.

Chin

- Soft tissue pogonion moves 1:1 with the skeletal movement.

2.9 Classical Patterns (Table 2.1)

Class III:

- With maxillary hypoplasia–average face height, no open bite ± mandible prognathism—bimaxillary osteotomy.

Class II division II:

- Retrognathia.
- Deep overbite, upper central retroclined.
- Reduced lower anterior facial height.
- Orthodontics to covert to class II division I, maintain curve of Spee.
- Mandibular advancement to class 1 with 3-point landing.

Class II:

- Vertical maxillary excess (VME).
- Retrognathia.
- AOB.
- Maxilla posterior impaction to close AOB with autorotation of mandible.

Table 2.1 Classical patterns of plane angles

Plane angles			
>32° High angle	Counterclockwise	– VME – AOB – Intrude posterior molars – Increase curve of Spee upper arch – Evaluate tongue base – Obstruction of oropharyngeal airway	– Increase chin projection – Increase posterior facial height – Increase oropharyngeal airway – Increased pressure on condyle due to soft tissue stretch – Increased pterygomasseteric sling
≤32° Low angle	Clockwise	– Excessive growth in the vertical vector – Enlarged and often irregularly shaped condylar head – Neck of condyle can be thickened and/or elongated	– Improve function/aesthetics – Chin retruded – Reduced posterior face height – Pterygomasseteric sling same length

2.10 Physiological Changes

Occlusion

- Masticatory efficiency difficult to measure.
- Patients with open bite/Class III → Better incisive stability.
- Reduced wear and enhanced periodontal health.

TMJ

- Decrease in TMJ dysfunction and masticatory muscle pain.
- Velopharyngeal insufficiency (VPI).
- Maxillary osteotomy—VP activity usually accommodates → Normal resonance and quality is achieved.
- If beyond compensation → Hypernasality.
- Even large movements do not result in VPI in the absence of cleft or previous palatal surgery.

Respiration

- Greatest contributors to nasal resistance are the nostril openings and the internal nasal valves (formed by insertion of the upper lateral cartilage to the nasal septum).
- Ability to breathe through nose improves after maxillary osteotomy even with impactions (widening base of nose → opening of nasal valve).

Speech

- Generally improved, multiple variables.

Head Posture

- Head returns to presurgical posture within 1 year.
- Some remain flexed.

Tongue

- Mostly adapt → Mandible setback (hyoid moves inferior and anterior- adaption of pharyngeal airway).
- Maxillary osteotomy → Tongue posture changes to maintain its position relative to anterior teeth and palate.
- Neurosensory.
- Dysaesthesia → <1% More common in middle aged and female.

2.11 Consent

- Anaesthetic risks.
- Bleeding which may require blood transfusion/interventional/surgical intervention.

- Infection.
- Scar (with some types of mandibular surgery and requisite facial nerve injury).
- Plate removal.
- Neurosensory → Damage to inferior alveolar nerve (IAN)—10–20%, lingual nerve—3%.
- Bad split → 4–6 weeks Intermaxillary fixation (IMF)—2%.
- Dental injury.
- Non-union, malunion, delayed union.
- Malocclusion.
- Asymmetry.
- Need for revision → Early/late.
- Avascular necrosis—rare.
- Nasal deformity → Septal deviation/nasal tip over-rotation/widening of the alar base.
- Stability/relapse → Less than 2 mm—1-year post-operative, causes:
- Orthodontics unstable → extrusion/arch expansion.
- Fixation of jaw.
- Osteotomy design—Profitt's hierarchy of stability.
- Neuromuscular function and soft tissue factors → Clockwise rotation of the mandible, tongue thrust.
- Condylar resorption/condylar growth → 2–7% with mandibular advancement.
- Blindness and skull base fracture with Le Fort osteotomy.

2.12 Maxillary Surgery

- Blood supply → Branches of ECA (Fig. 2.4).
- 1969–1975—Bell's research on rhesus monkeys demonstrated that as long as the maxilla is pedicled to the palatal mucosa and the labial gingival mucosa, complete down fracture and mobilisation of the maxilla can be accomplished.
- Further studies demonstrated that you can:
  - Stretch the palatal soft tissue pedicle.
  - Segmentilise the maxilla.
  - Bilateral transection of the DPA (Descending palatine artery).
- The palatal vessels contribute to the mobilised maxilla.
- DPA (can be ligated and still preserve adequate maxillary perfusion).
- Ascending palatine branch of the facial artery.
- Ascending pharyngeal artery.
- Posterior superior alveolar artery.
- Dental pulp blood flow → Long-term pulpal sensitivity (>12 months)—100% → as along as osteotomies 5–6 mm above the apex of the teeth.
- Maxillary sinus—incidence of sinonasal disease appears low and may even improve.

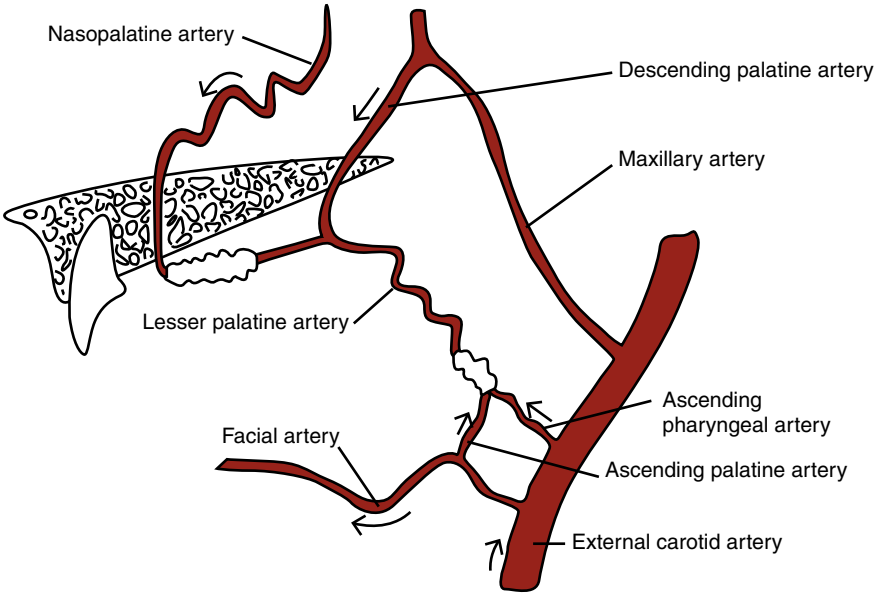

Fig. 2.4 Blood supply to maxilla

- Reduce vascularity if ligate DPA, avoid segmentalisation if possible and avoid or minimise transverse expansion.

Once Osteotomised the Le Fort 1 Segment is Supplied by (Fig. 2.5)

- Descending PALATINE artery (DPA-branch of the maxillary artery).
- Ascending PALATINE artery (APA-branch of the facial artery).
- External carotid artery (ECA) → maxillary artery via pterygopalatine fossa.

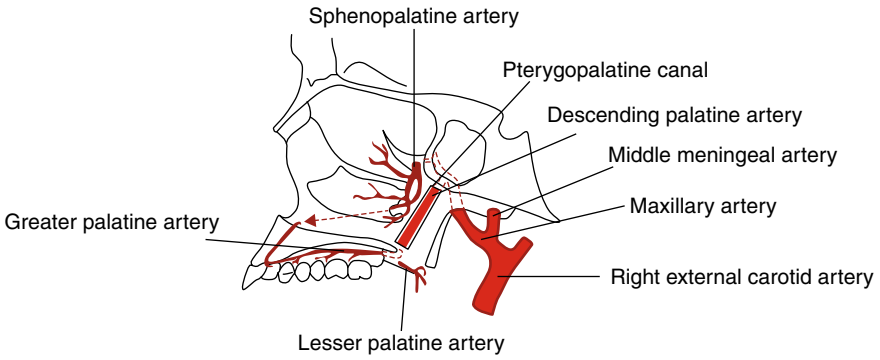

Fig. 2.5 Le Fort 1 segmental blood supply

- (External carotid branches—superior thyroid, ascending pharyngeal, lingual, facial, occipital, posterior auricular artery, superficial temporal artery).
- Ascending pharyngeal artery gives rise to:
- Lesser palatine artery → Supplies soft palate.
- Greater palatine artery passes though the incisive foramen and → Supplies hard palate and mucosa.
- Facial artery gives rise to the ascending palatine artery which anastomoses with the tonsillar branch of the facial artery AND the ascending pharyngeal artery.
- Posterior superior artery (branch of the maxillary artery) → Alveolar/teeth/mucosa.
- Infraorbital artery branch of the maxillary artery) → Maxilla/premolars/canines.

Management of Vascular Compromise

- Suspect → Abort operation and fix maxilla to original position.
- Cyanosis post-operative (hrs/days).
- Release IMF.
- Remove splints.
- Irrigation several times a day—with/without nasal packing.
- Hyperbaric oxygen.
- Antibiotics.
- Allow demarcation after attempting debridement.
- Severe cases → Treat as intraoral free graft.

2.13 Surgical Techniques to Prevent Bleeding

- Hypotensive anaesthesia (aim for a systolic blood pressure 100 mmHg or less).
- Consider I.V. tranexamic acid and dexamethasone.
- Local anaesthesia with adrenaline.
- Diathermy.
- Subperiosteal dissection.
- Protect nasal mucosa/infraorbital nerve.
- Pteryogomaxillary separation.
- Controlled down fracture.

Management

Pre-operative

- Haemoglobin.
- Clotting screen.
- Group and Screen is generally not required.
- Intra-operative: Vasoconstrictor.
- Pressure.
- Pack.

- Direct ligature (DPA if bleeding).
- Bipolar (usually pterygoid venous plexus after down fracture)
  - Torrential bleeding.
- Interventional radiology → Superselective embolisation.
- Tie off ECA at the level of hyoid or branches such as facial artery.

Post-operative

- Nasal packs.
- Posterior pads.

2.14 Surgically Assisted Rapid Palatal Expansion (SARPE)

- Widen arch and correct posterior crossbite.
- Widen arch as a prelude to offer orthognathic surgery.
- Avoid segmental, increased avascular necrosis (AVN) risk of segmental (cleft patient).
- Movement >8 mm not stable with segmental.

Tooth Borne

- Hyrax device → Primary premolar and first molar.
- Intra-operative bone-borne device uses miniscrews.
- Tooth borne.
- Tipping teeth.
- Periodontal problems → Buccal root resorption.

Palatal Bone Borne

- Higher in palatal vault.
- Orthopaedic maxillary expansion.
- Sometimes arch too small to place bone borne.

Principles

- Le Fort I cuts with no down fracture unless posterior arch expansion required.
- Ostetomize maxilla between central incisors → Paramedian → Avoid.
- Palatal mucosa → Saw/chisel.
- Engage expansion device → To check then return to start position.

Controversy → How It Works

- Mid palatal + pterygomaxillary junction → Sites of greatest resistance.

SARPE Versus Segmental Surgery

- Single surgery.
- Simultaneous AP/transverse/vertical correction.
- Immediate placement into post-operative position.

Disadvantage

- Increased risk of ischaemic compromise.
- Risk of damage to teeth in osteotomy site.
- Oroantral fistula.
- Potentially mobile segment (if non-union).
- Movement is limited to 8 mm.

2.15 Le Fort I Osteotomy

History

- Total mobilisation of the maxillary segment.
- 1859 Van Langenbeck.
- 1927 Wassmund.
- 1934 Axhausen.
- Pterygomaxillary separation.
- 1942 Schuchart.
- Tension free repositioning and bone graft between pterygoid plate and tuberosity.
- Obwegeser.

Surgical

- Incision and dissection.
- Maxillary–buccal incision (3–3 minimal to 6–6 maximal).
- Subperiosteal dissection.
- Identify infraorbital nerve.
- Extend to pterygomaxillary junction.
- Expose piriform aperture.
- Nasal floor dissection.
- Osteotomies—reciprocating saw → Cut zygomaticomaxillary buttress → Piriform aperture.
- Septal and pterygoid dysjunction with osteotomes.
- Down fracture, mobilisation and repositioning.
- Removal of interferences.
- Fixation.
- Closure.

Complications

Intra-Operative

- Unfavourable osteotomy (pterygomaxillary separation)
  - Fracture of junction of horizontal process of palatine bone with palatal process of maxilla.
  - Horizontal fracture of pterygoid plates.
  - Sphenoid bone fracture → Transmitted from pterygoid plates.
- Bleeding
  - Often due to high fracture of pterygoid plates.
  - Lateral pterygoid/medial pterygoid and pterygoid venous plexus.
  - Posterior superior alveolar artery.
  - Greater palatine artery.
  - Maxillary artery and terminal branches.
- Maxillary artery → 23–28 mm above pterygomaxillary junction.
- Margin of 8–10 mm exists if osteotomy directed inferior-medially.
- Management
  - Pack and identify cause → Ligature/clip/cauterise.
- Improper maxillary repositioning
  - Failure to seat condyle (inadequate removal or posterior bony interferences).
  - Inaccurate vertical positioning → Check septum/turbinates.
- Trigeminocardiac reflex
  - Stop maxillary manipulation → atropine/glycopyrrolate.

Post-operative

Early

- Loss of vision.
- Cranial nerve palsies.
- Damage ICA.
- Loss of lacrimal function/epiphora.
- Bleeding/vascular compromise.
- Loss attached gingiva/teeth/bony segments.
- Nerve injury.
- ION 96% → Normal in 3 months; II–VIII.
- Nasal septum deviation.
- Fistulas—in segmental (2.6%).

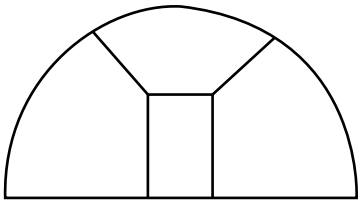

Fig. 2.6 Segmental osteotomy

Late

- Bleeding.
- Neurosensory → ION.
- AVN.
- Nasal deformity.
- Stability and Relapse → Growth/orthodontic relapse/TMJ remodelling.
- Infection.
- Progressive condylar resorption (PCR).
- Malunion, non-union.

Segmental Indications

- Multi-plane maxilla with vertical steps (Fig. 2.6).
- Transverse maxillary deformity along with discrepancies in AP/sagittal plane.

Basic Principles

- Maximum palatal and labial mucoperiosteum preserved.
- Aim for largest dento-osseous segment in order to maintain large soft tissue pedicle.

Anterior Segmental

- Described by Wassmund and Wunderer, Correction AOB.

SARPE and Rapid Maxillary Expansion (RME)

Transverse Maxillary Deficiency

- Congenital.
- Developmental—thumb-sucking >6 years.
- Iatrogenic (post CLP repair) Clinical.

Clinical Findings

- Paranasal hollowing and narrow alar base.
- Narrow, V-shaped maxillary arch, high palatal vault.
- Crossbite (uni-/bilateral >2 teeth, crowded/rotated teeth).
- Often accompanied by hypoplasia in other planes.

Rapid Maxillary Expansion (RME)

- Below 15 years old: Circummaxillary sutures and midpalatal suture not fused.
- Above 15 years old: Sutures fused → RME causes tipping molars with little expansion.

Indication for Surgery

- Transverse maxillary expansion >5 mm in non-growing patients.
- Failed RME.
- Significant nasal stenosis.

2.16 Mandibular Surgery

- Vasculature → ECA and branches.
- Inferior alveolar artery (centrifugal).
- Periosteum (especially muscle attachments centipedal).
- Lingual artery → Facial artery, maxillary artery branches (masseteric/buccal).
- Bell and Levy demonstrated blood flow through mandibular periosteum maintain supply to teeth and bony mobile segment even when labial periosteum degloved.
- Bell and Schendel → Showed reduced incidence of bony necrosis if avoid stripping of pterygomasseteric sling.

2.17 Anatomy

- Inferior dental nerve enters mandibular foramen → 8 mm inferior to the lingula.
- Lingula, 5 mm above occlusal plane.
- Foramen 20 mm inferior sigmoid notch.
- Canal 2–2.5 mm diameter → Furthest point from inferior border is in region 1st/2nd molars—7.5 mm.
- Lingual nerve injury → BSSO 19% (including impaired taste).
- Positioning.
- Tooth damage.

2.18 Mandibular Osteotomies

Vertical Subsigmoid Osteotomy (Fig. 2.7)

History

1954—Caldwell and Letterman (extraoral).
1964—Moose (intraoral).

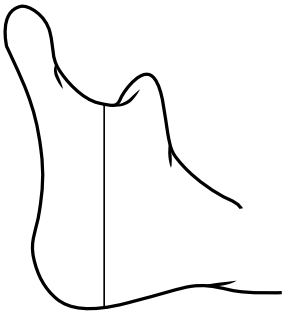

Fig. 2.7 Vertical subsigmoid osteotomy

- Indications: Mandibular setback.
- Contraindications: Advancement, large movements.

Advantages

- Decreased inferior alveolar nerve damage.
- Potentially easier technique.
- Helpful in patients with TMJD who require setback.

Disadvantages

- Difficult to fit rigid fixation.
- IMF.
- Cannot control condyle position.
- Increased healing time.

Technique (Intraoral)

- Confirm IAN position on OPG relative to posterior border.
- Incision should be made midway up anterior border ramus to 1st molar.
- Periosteum reflected laterally to expose ramus.
- Posterior/inferior borders cleared of periosteum except angle, channel retractor posterior border.
- Osteotomy → 5–7 mm anterior to posterior border → Midway up to notch/angle.
- Oscillating saw with 120° blade → Depth 6 mm (avoid damage medial tissues).
- Proximal segment lateralised→ Limited medial muscle strip and prevent condylar sag.
- MMF, if setback >1 cm → Consider coronoidectomy or stripping of temporalis.

Complications

- Stability: Over-correction.
- Nerves: Marginal mandibular branch (MMB) of the facial nerve in external approach, IAN—reduced by 1/3.
- Vascular necrosis: Proximal segment. This is prevented by maintaining muscle attachment at the angle.

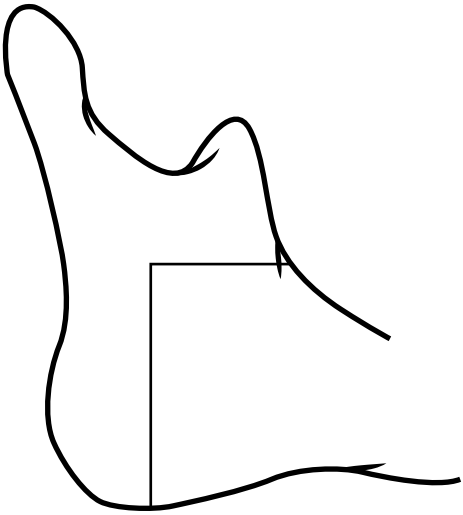

Fig. 2.8 Inverted L osteotomy

Inverted L Osteotomy (Fig. 2.8)

History

1927—Wassmund.
1948—Pichler and Trauner—recommended bone grafts for advancements.

Indications: Increase ramus height.

Contraindications: Advancements without grafting.

Advantages

- Ramus lengthening.
- Able to use rigid fixation.

Disadvantages

- Bone grafting for advancement.
- Extraoral–risk MMB damage.

Technique

- Risdon approach.
- Dissect to inferior border mandible, incision through pterygomandibular sling.
- Periosteum/masseter → Stripped of lateral cortex to level of sigmoid notch.
- Vertical cut—inferior point just in front of the angle (7 mm anterior to posterior border).
- Horizontal cuts—above lingula (intraoral approach).
- IMF.
- Setbacks—lateral decortication to distal fragment.

- IMF for 6–8 weeks.
- IAN—damage is higher than VSSO due to horizontal cut–6%.

2.19 Bilateral sagittal Split Osteotomy (BSSO)

History (Fig. 2.9)

(1) 1849—Hullihen–anterior mandibular subapical osteotomy.
(2) 1889—Blair–body osteotomy.
(3) 1957—Obwegeser–sagittal osteotomy.
(4) 1961—Dal Pont–vertical cut at 2nd molar.
(5) 1968—Hunsuck–lingual cut shortened.
(6) 1970—Epker–lower border cut.

Every move possible.
Contraindication: Severely decreased posterior body height.

Advantages

- Good bony contact for healing.
- Disadvantages
  - Difficult technique.
  - IAN damage.
  - Unfavourable splits.

Technique → Fixation

- Rigid bicortical screws → Via transbuccal → Not lag screws.
- Plates monocortical.
- Possible IMF for 4–6 weeks.

Nerve Damage

- IAN.
- Westermark 1998 → Nerve dysfunction in 40% of the cases.

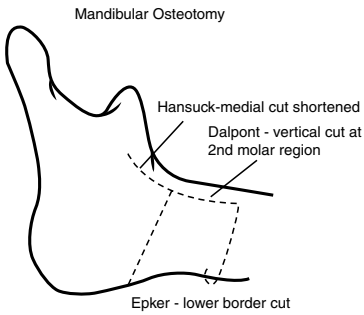

Fig. 2.9 BSSO modifications

- Von Sickes 2002 → Concomitant genioplasty → Increase loss of sensation.
- Lingual nerve.
- 19% of the patients reported.
- 69% resolved within 1 year.

TMJ Dysfunction

- Low, although risk worsening—sometimes with improvement.
- Wolford 2003 → Only negative report of effects of BSSO on TMJ.
- Pre-existing TMJ pathology before/during orthognathic surgery → Recommend treatment before start.
- Condylar resorption.

Other Side Effects

- Blood loss.
- Airway compromise.
- Aseptic bone loss.
- Infection.
- Facial nerve damage.
- Bad splits—1.9%–Tucker–3% bad splits, 4% with lower 8 s present, no evidence that removing 8 s lowers the risk.

Others-Variations

- Symphyseal—narrow arch BSSO.
- Subapical Body procedures
  - Anterior subapical → Close AOB.
  - Posterior subapical → Treat super eruption or ankylosed posterior teeth.

Total Body Osteotomy

- Level occlusal plane.
- Close AOB, without affecting chin position.
- Horizontal subapical cut below IAN → Lower 8 s via degloving incision.
- May need to exteriorise the nerve → Caution, there may not be enough space between apices and canal.
- Bone grafting if closing AOB.

Complications

- Unfavourable split (3.3–6.6%)
  - Buccal plate fracture → Usually due to inferior border not completely cut.
  - Completion of split versus posterior vertical body osteotomy.
  - Through-through osteotomy → Free condyle-ramus segment.
  - Then IMF for 3–4 weeks/physiotherapy.
  - Or abort and allow to heal.

- Why?
  - Patient factors: Age >40.
  - Anatomical: Wisdom teeth, bone quality, thin ramus, high lingula.
  - Surgeons: Technique.
- Nerve injuries.
- Bleeding
  - Maxillary artery and branches (masseteric, inferior alveolar artery).
  - Facial artery/vein → Pressure packing.
  - Retromandibular vein.

Post-operative

Early

- Bleeding/airway compromise.
- Haemorrhage (floor of mouth).
- Nausea and vomiting.
- Infection (6–16%).
- TMJ.
- Relapse.
- Early—hardware failure.
- Condylar malpositioning.
- Neurological.
- IAN → Transient (20–60%).
- IAN → Permanent (15–20%).
- Lingual → (0.6–3%).
- Facial → SSRO (0–67%).
- Facial → IVRO (0–38%).

Late

- Neurology: IAN (14%) Westermark 1998.
- Relapse.
- Growth.
- Orthodontic.
- TMJ remodelling.
- TMJ → Some quote improvement (Devries 2002) → Progressive condylar resorption (2–7%).
- Dental/periodontal.
- Condylar resorption.

  ⇒ **Relapse** → Return to original position.

  ⇒ **Stability** → Bones/teeth stay in same position.

2.20 Genioplasty

Anatomy

- Dissecting only the amount of soft tissue needed is imperative.
- Resuspending the mentalis and layered closure prevents chin ptosis.
- If inferior segment degloved → Intense inflammatory process with osteonecrotic results.
- Studies → Degree of bone resorption and necrosis is in direct proportion to the amount of the pedicle left attached to the segment (if only lingual attachments are left, buccal cortex resorption is observed).

History

(a) 1942—Hoper: Extraoral approach.
(b) 1957—Trauner and Obwegeser: Intraoral approach.

Classification of Chin Deformities

- Functional
  - Chin ptosis.
  - Lower lip incompetence.
  - Gingival recession anterior mandible.
  - Obstructive sleep apnoea (OSA).
- Aesthetics
  - Macrogenia.
  - Microgenia.
  - Retrogenia.
  - Asymmetry.
- Examination
  - Chin: Lip competence.
  - Lower lip length to upper lip length ratio 2:1.
  - Labiomental fold, chin throat angle, mentalis muscle strain, overlying soft tissue envelope.
- Cephalometric
  - Zero meridian → 2–3 mm behind line.
- Designs (Fig. 2.10).
  - Horizontal sliding osteotomy → advancement.
  - Double horizontal (stepped genioplasty) → large advancement.
  - Sagittal, horizontal vertical step, transverse reduction.

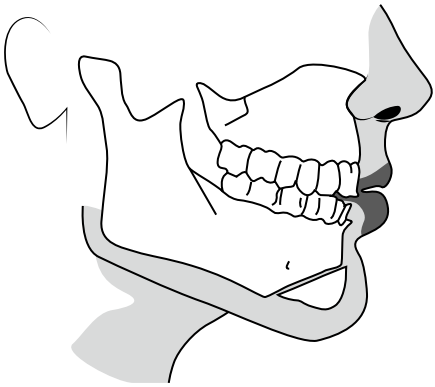

Fig. 2.10 Genioplasty

- Augmentation
  - Sagittal Plane: Horizontal sliding advancement.
  - Vertical Plan: Horizontal osteotomy + interproximal graft.
  - Coronal Plane: Lateral expansion with midline osteotomy + outward rotation of genial segments.
- Reduction
  - Shave osteoplasty.
  - Anterior chin protection (AP excess → reduced by using parallel osteotomies → remove middle segment).
  - Vertical plane → Horizontal chin excess treatment by moving inferior fragment posteriorly.
- Complications
  - Mental nerve (3–5%).
  - Chin ptosis → Mentalis stripped and not reattached.
  - Lip incompetence → Mentalis not reattached.
  - Poor aesthetics → AVN, haematoma, infection.
  - Relapse.
  - Dental damage.
- Propeller genioplasty: asymmetry.

2.21 Soft Tissue Planning

⇒ Most studies give ratios which are an average value, hence they do not apply to all groups.

⇒ Method:

- Lateral cephalogram tracing.

- Photographic prediction → Using software such as Dolphin.
- Computerised video imaging.
- 3D → Computer assisted.

⇒ Multiple factors

- Surgery.
- Fixation.
- Closure.
- Individual variation.
- Patient → Age/soft tissue drape/tone/growth.
- General trend, horizontal changes more predictable then vertical.

⇒ Maxilla

- Widening of the alar base.
- Uplifting of nasal tip: due to elevation of the periosteum and muscle attachments adjacent to the nose without adequate replacement.
- Flattening/thinning of upper lip.
- Opening of nasolabial angle.
- Reduction in visible vermillion borders -Increased bony support in advancement.
- Loss of normal lip support.

Technique to Control Soft Tissues

⇒ V–Y closure

- Always AP thinning of upper lip and loss of vermillion unless V–Y.

⇒ Alar cinch

- Reduce alar base widening.

⇒ Contouring the ANS

- Reduction if undergoing large advancements/impactions who have good nasal tip projection.
- Not in patients with poor tip projection.

⇒ Septoplasty

- Cartilaginous nasal septum should be reduced during impaction >3 mm → to prevent deviation, the nasal septum should be freely mobile when the maxilla has been fixed in position.

⇒ Subspinal osteotomy

- Preserve musculature/reduce widening of the alar base.

2.22 Stability

Depends on CMFDOG

- ***C***ondylar position/remodelling.
- ***M***uscle pull and function
  - Soft tissue stretching.
  - Neuromuscular adaptation.
  - Muscle orientation.
- ***F***ixation method.
- ***D***irection and magnitude of surgical movements.
- ***O***cclusal relationships.
- ***G***rowth
  - See Profitt 1996 → Hierarchy of stability.
- Surgery → Avoid stretching pterygomasseteric sling.
- Functional/adaptation of muscles is required for stability → Relapse related to presence/absence of intermediate fibres → Reduce relapse increase intermediate fibres.

2.23 Anterior Open Bite (AOB)

Negative Overbite

Causes:

- Skeletal
  - Excess vertical posterior maxillary growth.
  - Mandibular ramus shortening.
- Condylar resorption
  - Congenital → 1st branchial arch.
  - Acquired → Rheumatoid arthritis (RA), Juvenile Idiopathic Arthritis (JIA), Osteoarthritis (OA), PCR.

Habits

- Chronic nasopharyngeal obstruction → Mouth breathing.

Post-traumatic

- Bilateral condylar fracture.
- Midface fracture.
- Pathophysiology → two theories.

Morphogenetic theory: AOB is result of aberrant genetic control of morphologic growth patterns.
Adaptive theory: AOB is the malformation secondary to functional aberrations of the naso-oropharyngeal apparatus.

- Contributing factors
  - Habits: Non-nutritive, sucking (dummies) >6 years old.
- Environment →
  - Naso-oropharyngeal obstruction.
  - Triggers adaptive neuromuscular response

    Causing open rotation mandible.
    Inferior/anterior repositioning of tongue.
    Extended head posture → Classic adenoid facies.
- Genetic control of skeletal growth.

AOB → predominantly the result of alterations in mandibular growth patterns.

Diagnosis

- Identify cause of AOB (Fig. 2.11)
- Dental versus skeletal versus combination.
- Habit versus environment versus genetic.
- Identify location discrepancy.
- Maxilla versus Mandible versus Both.
- Excessive vertical development in the maxilla.
- Shortening of mandibular ramus.
- Combination.

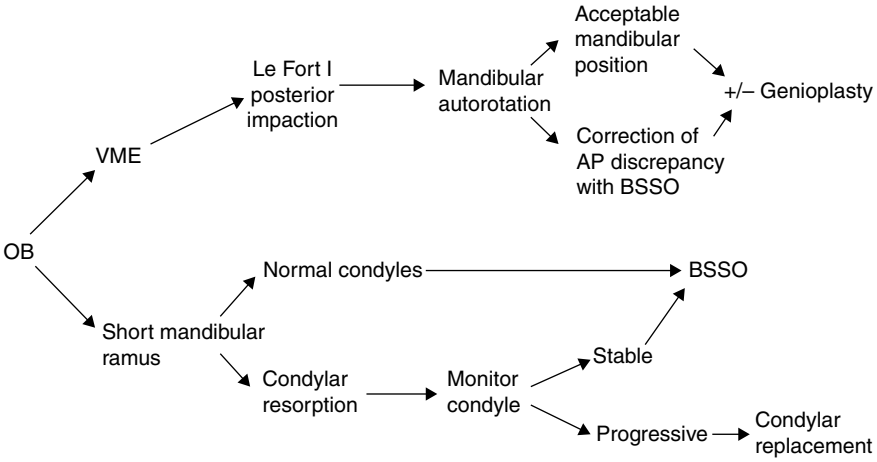

Fig. 2.11 Treatment pathway for anterior open bite

Clinical

- History, change/development.
- Patient factors
  - Trauma.
  - Orthodontic treatment.
  - Previous orthognathic surgery.
- PMH: RA/OA/JIA.
- Family history: AOB in relatives.

Management

- Growing individual
  - Habit cessation.
  - Maxillary dental expansion → Corrects crossbite/assists closing AOB.
- Orthodontic → Unpredictable stability/compromised aesthetics
  - Extrusion of upper and lower incisors (limited).
  - Intrusion of molars.
  - Expansion of maxillary arch.
  - Temporary anchorage devices (TAD) for intrusion of buccal segments.

Combined ortho + surgical treatments → AOB secondary to VME.
Clinical features due to VME

Aesthetic Features

- Lower third of the face almost always elongated.
- Excessive incisor exposure under the upper lip.
- Increased interlabial gap.
- Gummy smile.
- Obtuse nasolabial angle.
- Retrusive chin.

Dental Characteristics

- Open bites may be associated with all types of malocclusion, however, relative or absolute mandibular deficiency and class II malocclusion are most common.
- Tendency for the maxillary arch to be V-shaped and the mandibular arch to be U-shaped.
- Posterior cross bites.
- Flat or reverse mandibular occlusal plane curve.
- Stepped maxillary occlusal plane.

Cephalometric Features

- Increase anterior facial height.
- Steep mandibular and occlusal plane angle.

- Normal mandibular ramus height.
- Saddle cranial base.
- Increase distance from tooth apices to the nasal floor.
- Palatal plane is tipped up anteriorly and down posteriorly.

Presurgical Orthodontics

- Alignments.
- Avoid any mechanics to close bite → If open bite even more improves stability as relapse incisor intrusion serves to further close AOB.
- Extractions → Crowding/incisor angulation.
- Patients with severe reverse curve of Spee in the mandible → Consider surgical levelling mandibular arch with segmental surgery.

Surgery

- Superior reposition of maxilla–differential.
- Final AP/vertical position maxilla → Important → Incisor/upper lip.
- Position of mandible after autorotation → AP position of mandible determines if mandible surgery is indicated.

AOB Secondary to Short Mandibular Ramus

Aesthetics

- Normal incisor/lip.
- Normal paranasal/alar base width.
- Convex profile, slightly retrusive chin.

Dental

- Class II occlusion.
- Normal occlusal curves.

Cephalogram

- No VME, short mandible ramus height.

AOB Due to both VME and Short Mandible Ramus
AOB due to short mandibular ramus with condylar resorption

- Cause of condylar resorption should be investigated.

Idiopathic Condylar Resorption

- AOB develops progressively with no pain, hypomobility, self-limiting (6–24 months).
- Risks factors
  - Young.
  - Caucasian.
  - Women (chronic excessive loading of condyle).

- Management
  - Ensure inactive → dental records/cephalogram/radiograph imaging/occlusal models.
  - Surgical correction focus on maxilla, if mandibular surgery is unavoidable → 3–4 weeks IMF rather than rigid fixation.
- Orthognathic surgery with joint replacement
  - Degenerative joint disease: Progressive/uncontrolled degeneration of the condyle → Pain/crepitus/hypomobility.
  - Total joint replacement combines with orthognathic surgery → Final treatment solution.
  - Limited amount of mandible advancement (7–8 mm with replacement joint prosthesis).

2.24 Facial Asymmetry

- Varying degrees all individuals, mandibular asymmetry.
- 75% of patients have deviation of chin point.
- Difficult to restore symmetry in all 3D distributions of the facial skeleton.

Aetiology

- Congenital
  - Hemifacial Microsomia (HFM).
  - Cleft lip and palate (CLP).
  - Plagiocephaly.
- Developmental
  - Intrinsic jaw growth → Hemifacial atrophy/HH/HE.
  - Secondary growth deformity → Torticollis, dentomandibular disproportion, cleft palate, Möbius syndrome.
- Acquired
  - Trauma.
- Pathology
  - Unilateral TMJ disease → degenerative joint disease/ankylosis.
  - Neoplasms

    Osteoma/osteosarcoma/osteochondroma.
  - Fibro-osseous lesions.
  - Odontogenic tumours.
  - AVM.
  - Cystic lesion (jaw).

Assessment

- History: trauma.
- Head posture.
- Displacement.

Clinical

- Presence of maxillary cant.
- Orbits.
- Skeletal/dental/both.
- Displacement.
- Midline position.

Hemimandibular Hyperplasia (Rare)

- Enlargement of
  - Condyle.
  - Condylar neck.
  - Ramus.
  - Body of mandible.
  - Increase in mandibular height on affected site.
- If occurs before puberty
  - Maxilla compensates → Occlusal cant.
- Occurs later in life and/or develops quickly
  - Lateral open bite.
- Teeth appear to tilt to affected side.
- Chin deviated away from affected side.
- Bowing lower mandible.
- Inferior dental canal displaced as body is thick.
- Asymmetry → Generalised increased in size of one 1/2 of the mandible → Ipsilateral posterior open bite.
- Rotated facial appearance, bowing lower border of mandible → IAN displaced inferiorly.
- Chin deviated away from affected site.
- Maxillary and mandible → Alveolar overgrowth → Compensatory canting occlusal plane and tilting lip tissue line.
- Enlargement of all parts of mandible, usually at earlier age.

Hemimandibular Elongation (Common) (Table 2.2)

- Elongation of the affected side of the mandible.
- Deviation of chin/lower dental midline to the normal side.
- Buccal crossbite on unaffected side.
- Typically, no occlusal cant.

Table 2.2 Mandibular assymmetry

Type	Clinical findings	Histological findings
Type I (Hemimandibular elongation)	– Chin deviation to the contralateral side – Midline shift to the contralateral side – Lingual deviation of contralateral mandibular molars – Posterior crossbite	– Excessive growth in the horizontal vector – Condyle often unaffected – Elongated mandibular ramus – Misshapen and slender condylar neck
Type II (Hemimandibular Hyperplasia)	– Sloping rima oris with minimal chin deviation – Super-eruption of the maxillary molars on the affected side – Possible open bite – No midline shift	– Excessive growth in the vertical vector – Enlarged and often irregularly shaped condylar head – Neck of condyle can be thickened and/or elongated
Type III (Combination of Type 1 and Type II)	– Chin deviation towards the contralateral side with sloping rima oris – Midline shift – Possible open bite/crossbite	– Excessive growth in the horizontal and vertical vectors – Enlarged condylar head neck and ramus – Irregularly shaped condylar head, neck and/or ramus

- Mandibular body is slender and elongated without increase in body height (on involved side).
- Horizontal displacement mandible/chin → Towards unaffected side (midline shift unaffected side/crossbite on unaffected side).
- Slender form → Condyle elongated and slender → Ramus thin.
  Table 2.2.
- No slender form → Normal size/width.

Management: Await cessation growth → Combined orthodontic treatment and orthognathic surgery.

- BSSO/VSSO ± maxillary → Asymmetric.
- Camouflage genioplasty instead.
- Secondary procedures to camouflage asymmetry such as Coleman fat transfer/onlay graft.
- Can occur in hybrid forms HH + HE.

Obwegeser and Makek Classification

Clinical

- Lateral cephalogram/PA cephalogram.
- 99 Technetium (Tc) scan Increase uptake → Suggests activity→ Can get false negative/positive results.
- SPECT → Single Photon Emission Computed Tomography can provide quantitative assessment of uptake of 99 Tc; difference of 55:45% indicates active growth.
- 3D CT.
- Serial study models.
- Photographs.

Histopathology (Obswegser)

- HE → Hyperactive growth focus located in centre of condyle.
- HH → Actively proliferating cartilage incorporating entire fibrocartilaginous layer condylar head.

Management

- Growing → Monitor/wait for cessation.
- Early surgery → Condylectomy/rib graft/distraction
  - Functional appliances for asymmetric growth modification.
- Usually, self-limiting → Active growth usually completed by mid-20's.
- Perform orthognathic surgery after growth.
- If continuation of growth
  - High condylectomy → Remove 4–5 mm top condylar head → reposition disc.
  - Delay surgery until growth stops → Worse facial deformity → Asymmetry + dental compensation.

Torticollis

- Shortening/excessive SCM → Decreased motion/altered length.
- Clinical
  - Chin position.
  - Interpupillary line canted.
  - Cant of the occlusal plane.
- Management
  - Physiotherapy.
  - 5–10% → Surgical excision → Removal of SCM.

2.25 Condylar Hyperactivity (CH)

Condylar overgrowth with elongation of the condylar neck occurs in isolation or as part of HE or HH.

- F > M → Hypermetabolic growth centre in affected condyle.
- Cause of growth remains unknown
  - Hormonal.
  - Hypervascularity.
  - Trauma.
  - Genetics.
- Classification (Table 2.3).
- Obwegeser 1986 → CH seen as separate entity to HE and HH, also described hybrid forms that combine HE/HH.
- Wolford 2002
  - Type I

    Horizontal growth vector, condyle retains relatively normal architecture. Increase in condylar head/neck/and mandibular body seen (HE).
  - Type II

    Vertical growth vector.
  - Condylar head and neck much larger in length and diameter than normal (HH).

Clinical

- Typically born with symmetrical jaw, onset during puberty, progressive slowly, after variable period of progression, the deformity ceases.

Differential

- Mild HFM.
- Hemi-mandibular hyperplasia → Not accompanied by hyperactive condylar region.
- Osteoma/osteochondroma/osteosarcoma.
- Hemifacial hypertrophy, unilateral > dysplasia.
- Arterio-Venous Malformation (AVM), Sturge Weber syndrome.

Investigations

- OPG–Diffuse enlargement condyles/ramus/body but not coronoid.
- Nuclear medicine.
- Planar scintigraphy, Technetium phosphate (99Tc) scan → Increased uptake active areas.
- Sensitive not specific, HFM decreased uptake both condyles.
- SPECT bone scintigraph → Increased uptake.

Table 2.3 Condylar hyperactivity

Type of condylar hyperactivity	Clinical findings
Type I (prognathic) IA-bilateral IB-unilateral	– Occurs in adolescence, initiated during puberty – 1/3 genetic, 2/3 spontaneous – 60% female – Accelerated growth of the normal condyle in a horizontal vector (mandibular prognathism) – Continuation of growth until early to middle 20s – Elongation of the condylar neck and mandibular body Bilateral – Class III skeletal and occlusal relation – Anterior and posterior crossbites – Maxillary incisors proclined and mandibular incisors retroclined – If surgery performed prior to cessation of growth can relapse – Bone scintigraphy usually normal Unilateral – Facial asymmetry with the mandible shifting towards the slower growing side – Unilateral anterior and posterior crossbite on slower growing side – Class III on the faster growing side – TMJD
Type II (Unilateral mandibular condylar enlargement caused by osteochondroma (OC) that vertically lengthens the ipsilateral mandible) IIA enlarged, elongated, deformed ipsilateral condyle IIB exophytic extensions of the tumour off the condyle	– Osteochondroma-common benign tumour of bone – Not self-limiting relative to growth – Growth rate varies – Develops at any age, 20s commonest – Female preponderance – Requires histopathology – Increased unilateral mandibular height, condyle, neck, ramus, body and dentoalveolus – Increased soft tissue volume – Chin deviation – Supraeruption of the maxillary molars on the affected side – Possible open bite – Occlusal cant – Mandibular anterior teeth crowns may be tipped – TMJD and internal derangement
Type III	– Other rare benign tumours

(continued)

Table 2.3 (continued)

Type of condylar hyperactivity	Clinical findings
Type IV	– Malignant tumours

*normal facial growth normally completed at 15 years for females and 17–18 years for males

- Monitor changes → Every 6 months.
- Clinical serial evaluation/models.
- Lateral cephalogram/OPG/CT.

Management

- Active phase
  - Observe until growth complete.
  - Growth arresting procedure (high condylectomy).
  - High condylectomy + orthognathic surgery.
- Pre-adolescent and active CH, Type 1B
  - Obwegeser 1986, Wolford

    Unilateral high condylectomy + functional appliance → Avoid overgrowth disturbance (maxilla).
    Unilateral disc repositioning if indicated.
  - Wolford 2002

    Cease growth → Malocclusion/asymmetry may be in early to middle 20s.
    Delay surgery → Age 15 (female); age 17 (male).
    High condylectomy + orthognathic surgery.
- Adolescent
  - If very severe → High condylectomy to prevent progression.
- Adult
  - Non-active CH → Orthognathic.
  - Active CH → Condylectomy ± reconstruction and orthognathic as single stage.

2.26 Progressive Condylar Resorption (PCR)

Progressive alleviation of condylar shape and reduction in mass with resultant:

- Decrease in posterior face height.
- Retrognathism.
- AOB malocclusion.

Pathogenesis

- Condylar head normal undergoes remodelling with both resorption and deposition (part of calcium haemostasis and in response to alterations of the local environment).
- PCR occur when resorption > deposition.

Causes

- Traumatic.
- Idiopathic/Infective (condylar overload orthognathic surgery/orthodontic treatment).
- Neoplastic.
- Autoimmune disease (RA/SLE).
- Degenerative disease (OA).
- Unknown.

Risk Factors

- Young, Female:Male 9:1.
- Class II malocclusion.
- High FMPA.
- Posteriorly inclined condylar head.
- Orthognathic surgery → Pre-existing TMJ dysfunction—24%.
- Especially mandibular advancement → 7.1%.
- Counterclockwise rotation if normal neck of condyle.
- IMF.

Clinical

- Usually, bilateral → May be unilateral → 1st sign occlusal relapse.
- Mandibular retrognathia, loss of posterior face height → Development of AOB.
- TMJD symptoms may be present.
- End point of PCR → Condyle resorbed to sigmoid notch.

Investigations

- Plain films.
- Bone scan (increase uptake) → If undergoing resorption → Can be relapsing–remitting.

Management (Controversial)

- Orthognathic surgical correction but only if PCR has ceased.
- TMJ symptoms must be treated and stabilised.
- Condylectomy and reconstruction with autogenous/alloplastic.
- Radiographs/photos/models/99Tc scan.
- Management of TMJ with splint/muscle relaxants/NSAIDs.
- Longitudinal observation → 2 years → Response to treatment/stability.
- Stable → Orthognathic.
- Progressive TMJ symptoms → Condylectomy + reconstruction.

Why Females?

- No specific cause identified → Hormonal/medication.
- Oestrogen known to mediate cartilage and bone metabolism in the female TMJ.
- Increased receptors → May predispose to exaggerated response to joint loading from parafunctional habit/trauma/orthognathic surgery.

2.27 Vertical Maxillary Excess (VME)

Clinical Features of VME

1. Excessive tooth display.
2. Excessive gingival exposure on smiling.
3. Open bite especially if steps in maxillary occlusal plane → Seen in Class I, II, III malocclusions.
4. Long facial height.
5. Chin rotated downwards/posteriorly.

Management

- Early orthodontic intervention (age 8–12 with high-pull headgear).
- Adults → Le Fort I/superior repositioning maxilla.

Treatment Considerations

- Vertical growth of maxilla → Last to cease → Delay surgery until growth ceased.

Posterior VME

- Excessive maxillary vertical growth → Associated with open bite.
- Clinical
  - Step maxilla occlusal plane.
  - Increase facial height → Downward/backward rotation of mandible.

Management → Segmental osteotomy

2.28 Obstructive Sleep Apnoea (OSA)

Partial/complete upper airway/obstruction leading to potential cessation of breathing.

- Apnoea: Absence breathing > 10 s despite an effort to breathe.
- Hypopnoea: 50% reduction in airflow ≥ 10 s with >3% oxygen desaturation.
- Apnoea Hypopnoea Index (AHI) = apnoea + hypopnoea's/total sleep time × 60, represents the number of obstructive respiratory events per hour of sleep
  - Mild: 5–15.

Table 2.4 Risk factors for obstructive sleep apnoea

Predisposing risk factors for obstructive sleep apnea	
Age (40–70 years)	Obesity (body mass index >35 kg/m^2
Commercial motor vehicle driver	Postmenopausal woman not taking hormone therapy
Family history of obstructive sleep apnea	Pre-operative for bariatic surgery
Male sex	Retrognathia

  - Moderate: 16–35.
  - Severe: >36.

- Arousals associated with breathing that do not meet the technical criteria for apnoea or hypopnea.

Risk Factors (Table 2.4).

- Obesity, neck circumference > 17 inches, nasal obstruction.
- Skeletal → Craniofacial abnormalities → Retro-/micrognathia.
- Soft tissues → Long soft palate, Mallampati III/IV, macroglossia, tonsillar hypertrophy.

Pathophysiology

- Increased upper airway resistance and airway closure due to anatomical/physiological and postural factors often multiple site obstruction.

Effects

- Immediate.
- Hypoxia.
- Arousal during sleep.
- Negative airway pressure.

Long Term

- Cardiac: Hypertension, sudden death, stroke, cor pulmonale.
- Neurological: Daytime somnolence, memory, learning, depression.
- Haematological: Polycythaemia.
- Impaired glucose metabolism.

Clinical History

- Choking/snoring.
- Arousal.
- Memory fatigue.
- Daytime somnolence–Epworth Sleepiness Scale.
- Headaches.

Investigations

- Diagnosis/severity/level of obstruction.
- Polysomnography (PSG) → Gold standard → Multiparameter including.
- Electroencephalography (EEG)–brain activity.
- Electrooculography (EOG)–eye movement.
- Electromyography (EMG)–limb movement.
- Electrocardiography (ECG).
- Pulse oximetry.
- Respiratory rate.
- Heart rate.
- Flexible nasendoscopy (FNE).
- Lateral cephalogram → Hard and soft tissues abnormalities related to soft palate and physiological airway.
- Sleep nasendoscopy.

Management

- Lifestyle modifications.
- Alcohol.
- Smoking.
- Weight loss.
- Exercise.
- Avoid sedatives.
- Sleep position.
- Sleep hygiene.
- Non-surgical:
  - Oral appliances.
  - Tongue retaining device.
  - Mandibular reposition device–47% effective.
  - CPAP/BiPAP

    Increases posterior oropharyngeal airway space 100% effective but poorly tolerated.
    60–80% success → Decreases cardiac mortality.

Disadvantages

- Compliance 50–80%.
- Dry mouth.
- Claustrophobic/anxiety.
- Bed partner intolerance.

Fujita classification in patients with OSA

Type	Site	Anatomically
I	Retropalatal and normal tongue soft palate, uvula, tonsil, pillars	
II	Retropalatal + Tongue Above and tongue base	
III	Retrolingual, supraglottic, Retrognathic, floppy epiglottis, lingual tonsil	

Hypopharyngeal obstruction Normal Palate.

Features

- Type I—Narrow oropharynx (retropalatal), large tonsil, uvula, and pillar webbing.
- Type II—Oral and hypopharyngeal obstruction (retropalatal and retrolingual), low-arched palate and large tongue.
- Type III—Hypopharyngeal obstruction (retrolingual only) retrognathia, floppy epiglottis, enlarged lingual tonsils Most OSA patients have a combined problem: 20% discrete type II OSA.
- 10% discrete type I.

Surgical Treatment

- If morbid obesity, consider bariatric surgery.
- Not tolerated CPAP/unsuccessful non-surgical with RDI > 20.
- Multilevel obstruction of maxilla/mandibular hyperplasia.
- Identify obstruction → Site specific surgery.

Stage 1

- Nasal surgery.
- UPPP (Uvulopalatopharyngoplasty).
- Removes the uvula, soft palate, tonsils and adenoids.
- Success rate <25% (one site of multiple problems→ decreases snoring).
- Soft tissue changes unpredictable → can cause narrowing of pharyngeal airway.
- Tongue base (genioglossal advancement).

Stage 2

- Upper airway reconstruction → MMA/mandibular advancement (1 cm), acceptance of the existing occlusion versus presurgical orthodontics/Bariatric surgery.

Other

- Tracheostomy → Only definitive cure for OSA.
- Mandibular advancement.
- Repositions several muscles pulling tongue forward, upwards and away from pharynx.
- Advance maxilla → Pulls soft tissue at the palate forward and upward as well as palatoglossal muscles, increase tongue support.
- Goal → Advancement of deficient structure → Maintains normal facial balance → Ideal MMA 10 mm (may need segmental maxilla).
- Li and Pilly 1999 → (90% success, often no longer require CPAP).

Disadvantage

- VPI (low incidence).
- Negative effects on facial aesthetics.
- Success >50% in RDI (respiratory distress incidents) reduction and RDI <20.

2.29 Macroglossia

Enlargement of tongue → Can cause dentoskeletal deformities → Instability of orthodontics/surgery).

- Causes disturbance to speech/masticatory problems/airway management issues.

Aetiology

- Muscle hypertrophy.
- Congenital
  - Vascular malformation (lymphangioma/haemangioma), congenital hypothyroidism, Down's Syndrome, Neurofibromatosis, Multiple Endocrine Neoplasia 2B, lingual thyroid, Hemifacial Hyperplasia, Beckwith-Wiedemann syndrome.
- Acquired
  - Commonest = lymphoma > extend into neck (7%) > 1st year life slow growing, fibrosis may occur.
  - Glandular hypertrophy.
  - Amyloid.
  - Haemangioma.
  - Lymphangioma.
  - Down syndrome.
  - Beckwith-Wiedemann.
  - Acromegaly.
  - Myxoedema (severe hypothyroidism).

Most AOB (not related) → Closing AOB will adapt tongue, true macroglossia → Instability of orthodontics/surgery.

Clinical Features True Macroglossia

- Grossly enlarge/wide/flat tongue/large mandible/Class III.
- Buccal tipping of maxillary posterior teeth (increase curve of Wilson/scalloping).
- Reverse curve of Spee mandible, speech articulation, problems drooling.

Diagnosis

- History and examination.
- MRI/CT/Angiogram.
- Scintigraphy lingual thyroid.

- Biopsy

Management

- Speech/swallowing/breathing.
- Partial glossectomy.
- Posterior keyhole.
- Wedge.
- Laser reduction–lymphangioma.
- Embolisation–resection–vascular.
- Sclerotherapy–lymphovascular.

Surgery:

- Keyhole procedure (Fig. 2.12), shortens the tongue length. Midline elliptical incision, anterior wedge resection, tissue removed and midline closure.
- Post-operative swelling common.

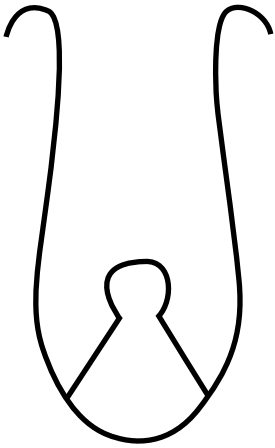

Fig. 2.12 Keyhole procedure

Chapter 3
Vascular Anomalies

Abstract Overview of vascular anomalies.

Key Points

- Classification of vascular lesions (vascular tumours and vascular malformations).
- Assessment and investigation vascular tumours.
- Indications for treatment of vascular tumours.
- Management of vascular tumours.
- Assessment of vascular malformations.
- Management of vascular malformations.
- Complications of treatment.
- Majority clinical diagnosis (>90%).
- History and examination.
- Discussion at MDT.

Classifications (Mulliken and Glowacki 1982)

A. Vascular tumours (endothelial hyperplasia)

- Haemangioma, not present at birth.
- 2/3 weeks old, grow rapidly, 70% at 7 years involute.

B. Vascular malformation (normal endothelial turnover)

- High flow: AVM.
- Low flow: Capillary–port-wine/venous.
- Lymphatic: Microcystic (lymphangioma), Macrocystic (cystic hygroma).
- Combined
  - Capillary lymphatic—Klippel-Trenaunay Syndrome.
  - Capillary venous with AV shunting—Parkes Weber Syndrome (PWS).
  - Sturge-Weber Syndrome.

Clinical Examination

- Colour—bluish.
- Pulsatile—high flow.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_3

- Valsalva manoeuvre—tilt head down 30 s → Increase in dimension → Venous.
- Skin change → Temperature → High flow → Warm.
- Bruit.
- Normal investigation if intraosseous.
- Cardiovascular status if high flow (cardiac failure).

Investigations

- MRI/MRA + gadolinium contrast.
- Plain radiograph/CT → Bony changes in relations to vascular lesions.
- Angiography only before endovascular treatment
  - 1% Mortality—no longer first line.
  - Indicated in high flow lesion needing embolisation.
  - Venous for direct sclerotherapy

Ultrasound doppler/duplex.
Histology.

Treatments

- 35% Sclerotherapy.
- 30% Conservative.
- 15% Surgery.
- 20% Other
  - Laser/radiotherapy.
  - Camouflage/embolisation/chemotherapy.

Multi-disciplinary Team

- Interventional radiologist/ultrasonographer.
- Surgeons (OMFS, Head and Neck Reconstruction).
- Dermatologist.

3.1 Vascular Tumours

1. Infantile haemangioma (GLUT-1 positive)
   (a) Superficial (Campbell de Morgan spots, cherry haemangioma, strawberry haemangioma).
   (b) Deep.
   (c) Mixed.

Endothelial Tumours of Infancy → Rapid Growth → Skin Regression → Often Involutes.

Theory: Implanted Placental Tissue (MALAT1 Placental Vasculature).

Clinical

- 3:1 Female: Male → Week 1–4 after birth → Grow rapidly 1st year of life → Proliferate → May ulcerate/bleed.
- Remain stable → Involute over time
  - 70% by 7 years.
  - 80% by 8 years.
  - Most by 9 years.

Management

- Conservative versus treatment.
- Functional problems
  - Bleeding.
  - Feeding.
  - Vision (if over eye)

    Discuss in MDT and ophthalmology.
    Risk of amblyopia → Visual pathway not forming.
  - Airway obstruction.
  - Aesthetics.
  - Coagulopathy/platelet consumption.

Treatment

- Propranolol → Younger age gives better results
  - 3.5 mg/kg–can cause hypertension, bradycardia–start gradually.
- Intralesional Steroids → Superficial.
- Systemic steroids → Deep → Prednisolone.
- Laser → Pulsed dye, Nd:YAG, interferon alpha.
- Surgical excision—lip.
- Systemic interferon/vincristine.

Haemangioma or Tumour

- Failure to respond to treatment with steroids.
- Unusual growth.
- Histology—markers e.g. placental GLUT-1.
- Consider biopsy.

Congenital Haemangiomas

- GLUT-1 negative.

- Rare vascular tumours that occur when fully grown.
- ***Two types***

  1. Rapidly involuting → Gone 6–10 months—RICH.
  2. Non-involuting congenital → Excision—NICH.

Management

- Observe/wait → For involution → For small haemangiomas that cause no harm/disfigurement.
- Pulsed dye → Residual telangiectasia that persist after involution.
- If destructive/bleeding/ulcerating/obstructing → Intralesional steroids if <2 cm versus systemic.
- Surgical excision + purse string closure.

  – Kaposiform haemangioma.
  – Pyogenic granuloma.
  – Tufted haemangioma.

Vascular MDT

- Complications of haemangiomas

  – Intralesional thrombosis.
  – Superficial skin necrosis.
  – Kasabach-Merritt phenomenon—coagulopathy.
  – >5 lesions—liver haemangiomas—Ultrasound liver.

Vascular Malformation

Clinical Features

- Soft, easily compressible.
- Non-pulsatile in soft tissues.
- Can occur centrally in bone.
- Can get periods of accelerated growth during puberty/pregnancy/trauma.

Venous (Commonest)

- Dark blue hue.
- Soft and compressible.
- Non-pulsatile.
- Increase size with increased venous pressure → Lying down/Valsalva manoeuvre.
- May have coagulopathy → Elevated D-dimer/fibrin degradation products, decreased fibrinogen and platelet levels.
- INR/APTT usually normal → Check pre-operatively.

Management

- Aspirin.
- Large VM → Prevent thrombosis and control pain.
- Sclerotherapy
  - STD (sodium tetradecyl sulphate) → Ultrasound-guided.
  - Absolute ethanol—toxic.
- Resection → Carried out after completion of sclerotherapy when there are no remaining large venous channels.

Orthognathic surgery can be undertaken ± protection of airway and tracheostomy if oral cavity is involved.

Cystic Hygroma

- Macrocystic (usually below hyoid) → Surgical excision.
- Microcystic
  (a) Neck → Suboptimal excision ± sclerotherapy hard to remove completely.
  (b) Oral cavity → Radiofrequency ablation ± sclerotherapy.

3.2 Lymphatic Malformation (LM)

Diverse forms → Isolated mucosa/cutaneous—large channels/multilocular lesions

- Classifications
  - Microcystic

    Lymphangioma.
    Very small size, not physically possible to inject every single cavity.
  - Macrocystic

    Cystic hygroma → transilluminates.
  - Macrocystic—combined.

Clinical

- Appear as unilateral/bilateral hypertrophy, macrocheilia and macrotia.
- Tongue/FOM → Cause macroglossia → Dark stained vessels on dorsal surface.
- Cervical facial → Associated with overgrowth of the mandibular body and result in open bite and anterior crossbite.
- Usually mirror child's growth → Can suddenly expand as a result of infection or intralesional haemorrhage.

Management

Infection

- Broad spectrum antibiotics.

Sclerotherapy

- Doxycycline/bleomycin, OK-432, absolute ethanol → For macrocystic.

Complications

– Skin breakdown.
– Nerve damage/extravasation.
– Haemoglobinuria → Need fluids.

Microcystic

- Intralesional bleomycin → Risk of lung fibrosis.

Resection

- Large LM → Often incomplete.
- Staged resection → Define anatomical areas → Recommended for diffuse microcystic LM.
- Vesicles of the mucous membrane/dorsal tongue → Laser or radiofrequency ablation (RFA).

3.3 Capillary Malformation (CM)

Known as Port Wine Stain.

Clinical

- At birth as macular pink stain that blanches on pressure.
- Can be localised or extensive—facial CM usually.
- Tend to darken to an intense red hue during young adulthood → Deep purple in middle age.
- The skin thickens/becomes raised and studded → With nodular and fibrovascular lesions, gives appearance of cobblestones.

Two Related Conditions

Sturge-Weber (Encephalotrigeminal Angiomatosis)

- CM in V1 ± V2 distribution → Leptomeningeal capillary and venous anomaly and vascular lesion of the ocular choroid (seizures/cognitive impairment).

Klippel-Trenaunay Syndrome

- Unilateral lower extremity angio-osteohypertrophy.

Management

- Pulsed dye laser → Significant lightening of the CM occurs in 80% in patients.
- Staged excision and skin graft if cutaneous overgrowth occurs.

3.4 AV Malformation

- High flow → Erythematous/warm/pulsatile/bruit → Thrill.
- Due to abnormal communication between arteries and veins without normal intervening capillary bed.
- Expansion of AVM because of collateralisation and recruitment.
- Present at birth → Not clinically evident or misdiagnosed → Grow proportionally with child in late childhood phase.
- Primary skeletal involvement → Tooth bearing bones → Expanded by malformation.
- AVM—jaws → Asymptomatic or present as pulsatile swelling/localised throbbing pain/toothache/earache or unexpected haemorrhage after dental extraction/exfoliation.

Schobinger Staging

1. **Quiescence**
   - Cutaneous bluish, warm, AV shunting on doppler.
2. **Expansion**
   - Bruit, pulsatile thrill, tense.
3. **Destruction**
   - Dystrophic skin changes, ulceration, tissue necrosis, pain, bleeding.
4. **Decompensation**
   - High output—cardiac failure.

Investiggations

- Ultrasound/Doppler.
- Intraosseous AVM
  - OPG

    Uni-/multilocular radiolucency → Soap bubble to fine mixed radiopaque appearance.
    Like fibrous dysplastic ± periodontal bone loss around teeth.
- MRI/MRA
  - Defines extent.

- Angiography
  - Assess number of feeders, size, location, parent feeding vessel, time required until venous phase.
- **Nidus Concept**
  - Latin for "nest".
  - Nidus fed by one or more arteries and drained by one or more veins.
  - Shunting arterioles that bypass capillary beds.

Management

- Usually pre-operative selective embolisation → Surgical resection within 48 h (coils/gel foam/PVA).
- Complications
  - CVA.
  - Non-target embolisation.
  - Necrosis.
- Resection → Like treating AVM in bone
  - 8 units cross match.
  - Hypotensive anaesthesia.
  - Tracheostomy.
  - Wide access.
- Or decortication and curettage
  - Superficial dissection → Bony resection → Bone wax.
  - Recon plate → 3 months later → Cancellous marrow graft.

Nidus Obliteration

- Prevent inflow/outflow.
- Not to get revascularisation of collateral supply.
- Onyx
  - Inject nidus/sets hard.
  - Problems → Wound breakdown.

Prognosis

- Resected/embolised tend not to re-occur.
- Unresected → Serial embolisation
  - 10% Recurrence.
  - 60% Prevent progression.
  - 30% Uncontrollable bleed.

Emergencies

- Life-threatening bleed
  - Socket packed → Surgicel + gauze.
  - Resuscitate → Angioplasty ± embolisation.

Chapter 4
Facial Pain

Abstract An overview of the anatomy, aetiology, assessment and management of facial pain.

Key Points

- Trigeminal neuralgia.
- Glossopharyngeal neuralgia.
- Headache disorders including migraines, cluster headaches and temporal arteritis.
- Burning Mouth Syndrome.
- Atypical facial pain and atypical odontalgia.

Aetiology of Facial Pain

- Local—teeth/jaws.
- Psychogenic—psychosomatic/atypical facial pain/Burning mouth syndrome → associated anxiety/TMJ.
- Referred—Angina/oesophagus/neck/chest.
- Neurological—Trigeminal/MS/space-occupying lesion/herpes zoster.
- Vascular—Migraine/temporal arteritis/cluster headaches.

Trigeminal Neuralgia

- Disorder of cranial nerve V: intense paroxysmal pain with one or more divisions.
- 3–6/100,000: more common in females—incidence and severity increase with age → Usually 50–70 years.

Causes

- Compression of trigeminal nerve root by artery in middle cranial fossa (pons): 80–90%, rarely by aneurysm/AV malformation.
- Nerve demyelination: suspect MS in younger patients.
- Others: CNS.
- Achondroplasia.
- CPA Lipoma/schwannoma/pituitary tumour/sarcoid.
- Charcot-Marie-Tooth.

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Symptoms

- Paroxysmal pain: Intense, lancinating, burning pain "like electric shock"—lasts seconds/minutes.
- Frequency varies—may be multiple times, almost always unilateral.

  Many patients have trigger points—stimulated every day, with period remission and relapse, worsen overtime.

- Eating.
- Talking.
- Washing.
- Shaving.
- Toothbrushing.

Investigations

- Complete neurological examination, look for signs/symptoms of multiple sclerosis (multiple defects):
  - Vision.
  - Weakness.
  - Limbs.

MRI brain if:

- Atypical features.
- Patient age <50, look for intracranial lesions/MS.
- Consider microvascular decompression.

Management—Medical First Line Treatment

- Most effective anticonvulsants: carbamazepine 100 mg BD (up to 1600 mg in 3–4 divided doses), follow-up with wean after six months, early side effects decrease with time, must monitor FBC, U&Es and LFTs.
- Leucopenia.
- Rashes/Nausea and vomiting/dizziness/headaches/diplopia.
- Ox carbamazepine: induces hepatic enzymes to a less extent.
- Unlicensed use for trigeminal neuralgia.
- Usually 300 mg/BD up to 600 mg/daily.
- Usually 600 mg–2400 mg/divided doses daily.
- Review in facial pain clinic and wean eventually.
- Gabapentin/lamotrigine also used as second-line agent.
- Severe cases I.V. phenytoin in a crisis.
- Start with small initial dosages and then increase. A few patients may be unresponsive or unable to tolerate side effects. Consider surgery adjuvant with neurosurgical assessment.

Peripheral Procedures

- Cryotherapy/chemical destruction/radiotherapy ablation.
- Stereotactic (gamma knife).

Options:

Operative:

– Nerve blocks with alcohol/phenol—temporary relief (up to 2 years).
– Avulsion of the superior/inferior orbital nerves—prolonged pain relief.
– Fogarty balloon inflation—ganglion in Meckel's cave (or glycerol injection).
– Radiofrequency thermocoagulation—site of facial tingling produced by electrical stimulation of needle inserted into trigeminal ganglion. When site of tingling corresponds to trigger spot, thermocoagulation produces analgesia of the appropriate area.
– Microvascular decompression—exploration of cerebellopontine angle (CPA) reveals blood vessel in contact with trigeminal nerve root—Separation using non absorbable (Teflon) sponge can produce relief in symptoms.

Risks

- Permanent.
- Paraesthesia/anaesthesia.
- Dolorosa (severe continuous pain within distribution of nerve).
- Pain relief = 80–85% remain pain free for 5 years.
- Young = microvascular decompression (MVD).
- Elderly/frail = interventions.

Surgery Risks:

- Damage to cranial nerves V, VII, VIII.
- Vascular damage.
- Post-operative weakness.
- If sensory loss to cornea = risk of scarring, have eyelid shut until able to test corneal reflex—need special glasses with sides.
- Can get relapse after MVD—consider re-operation as vessels move or material dislodged.

Glossopharyngeal Neuralgia

- Rare—similar to trigeminal = pain felt at base of the tongue and fauces on one side.
- TN:GN 100:1.

Symptoms

- Poorly localised—affects tonsils, tongue base, ear/and intra-auricular area.
- Patient often point to behind angle of mandible—symptoms often treated as TMJD.
- Trigger point difficult to identify = yawning/swallowing.

Investigations

- Topical LA to ipsilateral tonsils/pharynx—immediate relieves symptoms, but short acting *Diagnostic.
- MRI—Space-occupying lesion in cranial cavity or jugular foramen/MS.

Management

- Medical similar to management of trigeminal.
- Surgical—MVD.
- 75% works.
- Risks—morbidity/mortality.
- Headaches: many causes.

Migraine

- Primary recurrent headache disorder.
- More common in females.
- Usually commonest in adolescence.
- Termed 'hemicrania' as it affects half of the head.

Cause

- Possible relationship to abnormal 5-HT (serotonin) activity—leading to initial vasoconstriction of portions of cerebral arteries, followed by compulsory vasodilation, with cerebral oedema and pain.

Precipitant

- Hormonal factors including Oral Contraceptive Pill (OCP).
- Diet—chocolate/bananas, stress, sleep deprivation, bright/flashing lights.

May Have Preceding Aura

- Visual hallucination—flashing lights/decrease in color, visual disturbance.
- Motor—temporary motor palsy.
- Speech—aphasia.
- Severe unilateral headache—initially poorly localised, becomes localised to temporal/frontal/orbital region.
- Photophobia/nausea/vomiting.
- Attacks decrease with age—may totally resolve.

Management

- Acute attacks: Triptan (5-HT antagonist) plus NSAID/paracetamol.
- Prophylaxis: Topiramate or propranolol, acupuncture.

Cluster Headaches

- Exquisite pain of midface/upper face—centred around eyes.
- Attacks occur in temporal groups/clusters—extended periods of remission between attacks.

- Often positive FH=Family history.
- 80% are smokers.
- 30–40 years.
- Less common than migraines.
- Male:Female 6:1.
- 1:10,000 males/year.

Cause

- Unknown, allergic basis with mast cell release and vasodilatation.
- Associated with sleep apnoea, and low oxygen saturation.
- Alcohol, cocaine, GTN spray.

Symptoms

- Severe unilateral episodes of burning/lancinating pain in and around the orbit/frontal/temporal.
- Abrupt onset—lasts for 15 min to 3 h—often wakes patient at night.
- Begins same time every day (alarm clock headache—may have multiple episodes every day).

May be Associated with Autonomic Symptoms

- Conjunctival vessel congestion.
- Eye watering.
- Nasal stiffness.
- Facial Flushing.
- Diagnosis.
- Clinical/MRI.

Management

- Acute attacks.
- Oxygen may abort attacks and its effectiveness diagnostic.
- Subcutaneous triptan.
- Prophylaxis.
- Verapamil drug of choice.
- Nifedipine.
- Lithium.
- Ergotamine.
- *Distinguish from chronic paroxysmal hemicrania—respond to indomethacin.

Giant Cell Arteritis

- Multifocal vascular affecting the cranial arteries.
- Unknown aetiology.
- Systemic vascular—large/medium vessels especially branches external carotid.
- Females > Males.
- Average onset—70 years.

- Related to polymyalgia rheumatica (PMR).
- Genetic predisposition possible.

Symptoms

- Unilateral headache—initial burning—throbbing.
- Usually temporal/or occipital artery.
- Lingual/facial/maxillary arteries may get involved leading to claudication on eating/drinking.
- Affected vessels feel hard/tender.
- Tongue ischaemia if lingual artery.
- TIA/CVA.
- Left untreated.
- 25% develop visual problems.
- Due to central retinal artery involvement—which may be bilateral—loss vision.

Diagnosis

- Increased ESR 60–100 (although can be normal), normocytic normochromic anaemia.
- Temporal artery biopsy with granuloma formation.

Management

- Prednisolone >50 mg daily.
- I.V. methylprednisolone first if visual loss.
- Urgent ophthalmological assessment.
- Consider rheumatology referral.
- Decrease steroid with resolution of headache—ESR may take month/years to normalise.
- PPI.
- Osteoporosis cover—bisphosphonates.
- Calcium/Vitamin D.
- Aspirin (decrease CVA).

Burning Mouth Syndrome

- Burning sensation of oral mucosa (usually tongue—glossopyrosis) in absence of any pathology.
- Common 5 in 100,000—much higher middle aged and elderly.
- Female:Male 16:1.
- Age >50 years.

Aetiology

- Unknown.
- Psychogenic.
- Increased in anxiety/depression/stress.
- Obsession.
- Cancerphobia.

- First exclude organic cause of burning.
- Local.
- Xerostomia/chronic mouth breathing/mechanical trauma/referred pain/trigeminal neuralgia/atypical facial or neuralgia/angioedema/candidiasis/TMJD/submucous fibrosis/trauma lingual nerve.

Systemic

- B vitamins, folate, zinc, iron.
- Diabetes mellitus / GORD/ hypothyroidism/ oestrogen deficiency/ Parkinson's / AIDS.

Symptoms

- Dry mouth/altered or bad taste.
- Burning sensation affecting tongue and anterior palate, less common in lips.
- Aggravated by certain food—usually bilateral.
- Does not wake patient, but often present on waking.
- Normal examination.

Investigations

- FBC/haematinics/swab candida—all normal.
- Tricyclic antidepressants—side effects.
- Arrythmias/heart block.
- Postural hypotension/tachycardia.
- ECG changes.
- Dry mouth/blurred vision/constipation/urinary retention.

Management

- Reassurance.
- Avoid stimulating factors.
- Cochrane review: Amitriptyline 10 mg daily (max 75 mg daily titrated/Clonazepam 1 mg TDS mouthwash.
- Tricyclic antidepressant—same patients respond.
- 2nd line: Pregabalin 150 mg divided doses daily/75 mg BD.
- Tricyclic antidepressants: nortriptyline: 10–25 mg once at night (maximum 75 mg once at night); less sedative effects than amitriptyline.
- CBT: shown in RCTs to help patients manage symptoms—as medical management often disappointing.

Atypical Facial Pain

- Diagnosis of exclusion.
- Constant chronic dull ache.
- Most common in females—middle age/elderly.
- Can get intermittent/severe episodes, may be bilateral.

Clinical

- Often difficult for patient to describe—deep constant ache or burning.
- No anatomical bundles—cross midline moves to different sites.
- Maxilla > mandible.
- Does not stop sleeping, but may awaken with pain.
- Often exacerbated/initiated by dental treatment.
- Often have other complaints: IBS/TMJD/back pain/neck pain/dry mouth—diagnosis from trigeminal neuralgia is difficult (lack of trigger, vague distribution).

Investigations

- MRI/Bloods/Biopsy—nerve.

Management

- Tricyclic antidepressant—some effect on symptoms—no adequate RCT to show this.
- 30% respond gabapentin. Dose regime

  (a) Day 1: 300 mg/OD
  (b) Day 2: 300 mg/BD
  (c) Day 3: 300 mg/TDS

- Pain team/CBT.

Atypical Odontalgia

Pain tooth/site of extraction—sharp/aching/throbbing, burning—exclude pathology

Management Includes:

- Topical capsaicin—some benefit, need for several weeks.
- EMLA.
- Tricyclic antidepressant.
- Gabapentin.

Chapter 5
Cleft Lip and Palate

Abstract An Overview of the Aetiology, Diagnosis and Management of Cleft Lip and Palate.

Key Points

- Embryology relevant to cleft lip and palate.
- Aetiology of cleft lip and palate.
- Diagnosis of cleft lip and palate.
- Multidisciplinary care in cleft lip and palate.
- Surgical management of cleft lip and palate.

5.1 Cleft Embryology

Week 6

- Two medial nasal swellings (become philtrum) and the maxillary swellings (lateral upper lips) fuse to form upper lip.
- Nasal swellings merge at deeper levels as well = inter maxillary segment—primary palate (4 incisor teeth).
- Cleft of lip almost invariably associated with CLP.

Week 8–9

- Secondary palate develops—medial growth of palatal shelves of maxilla, which normally fuse together as the tongue is pushed down.
- Stage I: Formation of palatal shelves from maxillary process.
- Stage II: Vertical palatal shelves elevate to horizontal position above dorsum of tongue.
- Stage III: Fusion of medial edge of epithelium.
- 1 in 700 live births: Lower in Africans, higher in Asians.
- 50%: CLP and palate M:F.
- 35%: Palate only 1 M:2F.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
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- 15–20%: Cleft Lip 2 M:1F; L > R; unilateral > bilateral.

Embryology—2 Theories

- Failure of fusion.
- Lack of mesodermal penetration (breakdown of fusion).

Aetiology

Multifactorial—genetic/environmental

1. ***In utero/maternal environmental factors***
   - Anti-convulsant—phenytoin (risk × 10).
   - Alcohol, smoking (risk × 2), folic acid deficiency, steroid (risk × 3), hypoxia.
2. ***Genetic/hereditary***
   - 40% genetic, especially syndromes, but not classical Mendelian, not single gene disorder.
   - Genes included: MSX-1, FOXE1, MTHFR, IRF-6.
3. ***Genetic/environment interaction***
   - TGF-a genotype and smoking increases risk by 6 times.
   - Increase in folate deficiency if have MTHFR.

Coincidence rate monozygotic twins (60%)—15–30% syndromic

- HFM/22q11 velocardiofacial (Di George) syndrome/Stickler syndrome (eye examination—retinal detachment).

Diagnosis

- Ultrasound at 12 weeks—transvaginal ultrasound = full anatomical survey indicated for syndromes.
- Detection: Depends on experience of the operator/age/study/technology—45% antenatal diagnosis (2007—44%/Now—80%).
- Not always positive, diagnosis at birth, decrease in detection if over 20 weeks, only cleft lip detectable.
- National standard—think cleft = cleft nurse—patient to see within 24 h, explain to patient/show picture.
- Psychologically—antenatal diagnosis is better as more prepared, patients tend to be happier, but some would consider termination.

At Birth

Aim diagnosis at birth, parents to be contacted by members of cleft team within 24 h with designated cleft nurse available for counselling/support to the family—telephone.

MDT clinic soon after birth by team members involved in care

- Cleft surgeons.
- Paediatrician/psychologist/social worker.
- Clinical coordination/specialist nurse.
- Audiologist.
- ENT/OMFS/Orthodontics.
- Speech and Language Therapist.

Details of surgery/familiar with ward

Genetic Advice

Genetic Counselling at Birth—Especially in Syndromic

- Risk of any child—0.1%.
- Risk of 2nd child—4%.
- Risk of 3rd child—10%.

Risk If a Member of Family Has CLP:

- Father + child—13%.
- Father + 2nd child—17%.
- 1st degree relative—2%.
- 2nd degree relative—0.7%.
- 3rd degree relative—0.3%.

Pathway Overview

- 1 year: arrange patient to meet SALT.
- 18 months: Speech assessment—2 year SALT (if VPI—investigate).
- 3 years: SALT.
- 5 years: Audit.
    - Study model.
    - Speech.
    - Psychologist.
    - Audiologist.
    - Photographs.
- 7.5 years think about Alveolar bone graft (ABG).

    (1) Orthodontics

        – Primary: Difficult to graft.
        – Secondary: Early 2–4 years increase burden care

            (1) Secondary: Late - canine 1/2–2/3 root formation. Tooth eruptive potential.
            (2) Pre-surgical orthodontics: Expand cleft to base in bone graft not always required.

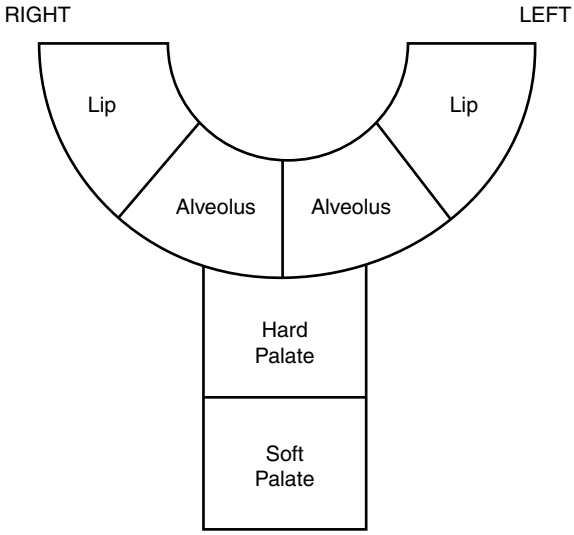

Fig. 5.1 LAHSAL classification

- 8–9 years
  - 6 months after ABG—occlusal x-ray, CBCT Check for facial growth.
- 10 years AUDIT.
- 12 years: Definite orthodontics unless orthognathic.
- 15 years: AUDIT.
- 16–18 years: Orthognathic ± rhinoplasty.
- 21 years: END—no formal appointment.

Classification

LAHSHAL (Fig. 5.1)

- Submucous small case.
- Isolated soft palate small case.
- Complete cleft upper case.
- Incomplete cleft lower case.

Submucous

- Notch hard palate absent posterior nasal spine.
- Blue line (translucent zone = zona pellucida).
- Bifid uvula.

5.2 Neonatal Care

Airway

- Especially: Pierre Robin Sequence
  - Nurse prone.
  - NP airway.
  - Tongue suture.
  - Tracheostomy.

Feeding

- CP babies cannot create negative pressure, usually unable to breast feed.
- Cleft nurse specialist.
  - Modified teat—longer, reach base of tongue.
  - Soft silicone squeeze bottle—expressed breast milk—1/3 cleft palate undiagnosed.
  - Syringes.
  - Swallow safe will still get regurgitation.

Surgical Pathway

(1) 3 months

- Lip repair in 3 months—10 (lb)/10 (weeks old)/10 Hb (as per specialized service dashboard by 183 days).

(2) 9–12 months

- Palate repair (as per specialized service dashboard by 396 days 13 months).
- Sommerlad repair is UK standard. If very wide use Von Langenbeck + intravelar veloplasty.
  - Intravelar veloplasty, avoids lateral releasing incisions as much as possible, very wide cleft usually have von Langenbeck repair.

(3) Latest 18 months (speech results not favorable if later)

- Speech versus growth—babbling patients: more glottal/pharyngeal speech, difficult to eliminate once developed = maladaptive speech patterns.

- Same time: ENT grommets as all have middle ear effusions. Reduced insertion in recent years, only approx. 1/3 receive grommets, usually at the time of palate repair, hearing test before palate repair if hearing loss significant.

(4) Age 18 months—5 years: CLP revision.
(5) 4–5 years: VPI assessment.
(6) 6–14 years: Orthodontic treatment phase.

(7) 6–9 years: Consider canines—Alveolar bone graft/ ± ONF closure.
(8) 16–18 years: Orthognathic treatment.
(9) Adult: Cleft rhinoplasty—early if function/psychosocial issues/closure any residual defect.

Hearing

CP—Eustachian Tube Dysfunction Related to Tensor Veli Palatini/Levator Veli Palatini

- Both attach to Eustachian tube and assist in tubal dilation: creates negative pressure during swelling—otitis media (may need grommets/adenoidectomy).

Cleft Lip Repair

1. ***Basic premise 3-layered closure—reconstruction of orbicularis oris (critical)***
   - Skin.
   - Muscle.
   - Mucosa.
2. ***Orbicularis oris abnormally attached to lower lateral cartilage cleft side and into septum on the medial site***
   - Anatomy—clefting results.
   - Abnormal attachment into nasal spine and alar base.
   - Opposing forces.
   - Splaying of alar cartilages.
   - Shortening columella.
   - Shortening philtrum—1/3 cupids bow.
3. ***Timing = 10 lb/10 Hb/10 weeks old***
   - Pre-surgical orthopaedics—(abandoned in UK (Adali et al. 2014) in most centres, no long-term difference, some may do PSO in bilateral cases.
     - Passive dental plate.
     - Active—Latham's device.
   - NAM device: Nasal alveolar molding—use like denture, no screws (USA with elastic band worn 24 h, mold soft tissues). Mainly to align nasal elements to improve outcome in primary rhinoplasty—long-term results pending, currently no evidence.
4. ***Unilateral cleft lip***
   - Millard—half the surgeons in the UK do a modified Millard repair with a lip triangle and often a second triangle in the dry mucosa.
   - Fisher repair—anatomic subunit repair.
   - Most important is muscle reconstruction.

5. ***Millard—rotation advancement flap***

- Midpoint cupid.
- Height.
- Mirror of 2.
- Peak on cleft side
  (1) A: Advancement.
  (2) R: Rotation.
  (3) C: Columellar advancement.

6. ***McComb's dissection***

- Correct nasal deformity.
- Free and reposition nasal cartilage in as normal position.
- Scissors—upper/lower to release lower lateral cartilage to allow skin re-drape-no effect on growth.
- Facial release.

Bilateral Lip Repair Bilateral Cleft Lip

- 10% cases.
- Short columella: tenuous blood supply to pre-maxilla.
- No muscles across midline: Difficult to bring muscles across, may need pre-surgical orthopaedics.
- Nose is symmetrical.
- No consensus on treatment options.
- Mulliken repair: prolabium is used for the lip repair, a "shield" is created to philtrum.

Cleft Palate Repair

- Closure of secondary palate, minimal effect on growth potential.
- Speech versus growth—optimal age unresolved.
- Timing based on child's speech development = 9–12 months
- Mostly—9–12 months to minimise pathological compensatory speech patterns while minimising surgical risks and potential for growth restriction.
- Principles.
- Soft palate.

Levator Palatini: *Abnormally inserted on posterior aspect of hard palate*

Must release, reconstruct midline, closed in 3 layers (nasal/levator muscle/oral mucosa)

Hard palate: Closed in 2 layers usually single layered vomer flap Oslo type repair Pichler-1934-using nasal mucosa/oral mucosa (vomer).
Sommerlad repair with intravelar veloplasty to release muscles for the soft palate.

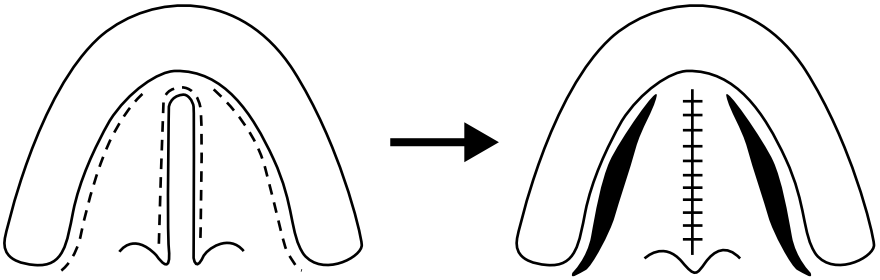

Fig. 5.2 Von Langenbeck lateral release

VF Very Wide: *then Von Langenbeck for lateral release (*Fig. 5.2*).*

Easier to Furlow Z-Plasty as Only 2 Layers to Dissect

- Separate Z-plasty on oral and nasal side.
- Flaps transposed—lengthen soft palate and closed in 3 layers.
- The elevator is oral mucosa flap on one side and nasal mucosa on one side.
- Hard palate close with vomer flap—nasal mucosa closure.

VPI (Velopharyngeal Insufficiency) (Fig. 5.3)

- Inadequate closure of nasopharyngeal airway during speech, mechanism combination (soft palate + pharyngeal wall musculature—functions as sphincter valve to regulate airflow between oral and nasal cavities).

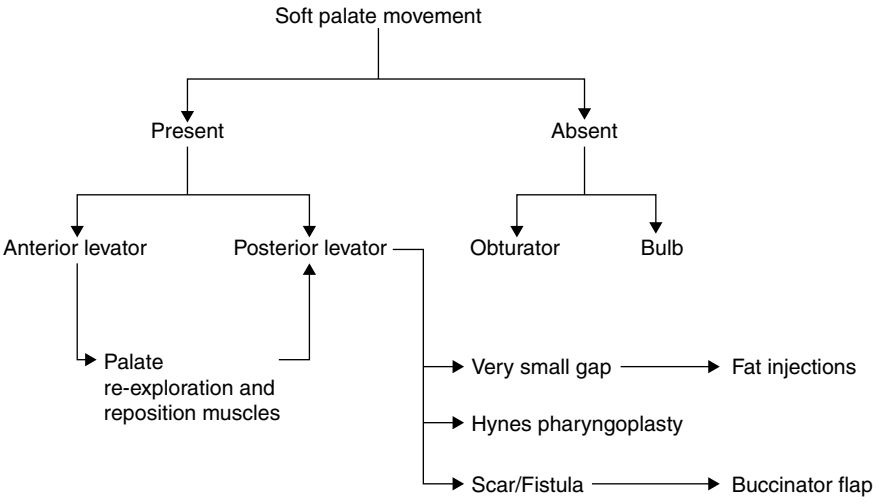

Fig. 5.3 VPI treatment pathway

- Lateral/posterior pharyngeal wall motion: continue to VPI = compensation—activation of passavant's ridge (nasal airway dynamics).
- Audible air escape with hyper nasal speech and lack of intelligibility of phonetic consonants (p, t, k) associated with VPI.

Indication/Timing

- Hypernasal resonance—leaks, air escape down the nose, quieter consonants.
- Articulating errors—put tongue in wrong place/lateralisation—use tongue in wrong way.
- If glottal sounds, it would be difficult to correct, would require speech assessment.

Assessment

- Speech/nasopharyngeal ± videofluoroscopy.
- Difficult to fully assess until age 5.
- GOSH—MRI experimental.

Options

- Redo original/palatal repair or Z-plasty.
- Avoid pharyngoplasty: increase snoring/decrease nasal airway—worsening sleep apnoea.
- Static:
  - Hynes: tonsillar posterior pillar as myomucosal flap—bilaterally (superior places—palatophayngeus).

Post-wall Augmentation

(1) Dynamic: Orticochea.
(2) Sphincter pharyngoplasty—superior/inferior based.

Complications

- Causes congenitally short despite adequate repair.
- Excess scarring following palatal repair.
- Improper re-approximation of muscle during repair.
- Cleft palate with neurological dysfunction.

Alveolar Bone Grafting

Aims

- Aesthetics: Osseous support lip/nose.
- Function.
- Functional nasal airway support, close ONF.
- Maxillary arch continuity—facilitate orthognathic surgery.
- Eruption/support/orthodontic alignment teeth.

Timing

- Primary: < age 2—inhibit growth.
- Age 2–5: Early secondary (no long-term growth effects)—GOSH: secondary mixed dentition when canine root 1/2–1/3 formed around 8–12 years.
- Age 6–12: Mixed dentition-UK.
- Tertiary > age 12: Poor results.

Individual Factors

- Dental versus chronological—Evaluate when maxillary central incisions erupt.
- Lateral incisors: Early grafting beneficial/distal to cleft usually (early grafting).

Use Iliac Crest

- Autogenous (osteogenic/osteoconductive/osteoinductive).
- High volume, 2 teams.
- Theory anterior crest: Growth not completed until 20—no reported growth alleviations.
- Use trephine—morbidity (gait distributes/pain/sensory—neuropraxia of lateral cutaneous nerve).

Imaging: OPG/Occlusal/CBCT

Most Preferred Method

- Pre-surgical expansion to align arches and prepare defect placement.
- Improves access to cleft—closure ONF.
- Better post-operative hygiene and less change of reopening ONF.

GOSH Bovne + Sands

- Post-surgical orthodontics (>3 months)—improve bone consolidation, small defect, less difficulty getting bone amount in patients with resolve arch form.
- Little sense in delaying grafting to expand arch.
- If unilateral cleft with collapsed arch, may be easier to have pre-surgical orthodontics.

Technique

- Advancement buccal gingival flap + palatal flap.
- Lateral sliding flap—raised on lesser segment: good blood supply, buccal incision with periosteal incisions.
- It brings keratinised mucosa, provide tension free closure/some shortening of buccal sulcus.
- Watertight and tension free closure are important.
- Palatal flaps: separate from nasal mucosa with sharp dissection scissors from anterior/posterior to preserve maximal palatal tissue for closure.
- Nasal mucosa: reflected/sutured, buried knots with 4-0 vicryl.

- Bilateral same: be careful with stripping the pre-maxilla as it relies on labial blood supply.
- Graft placed (bone—fill up to piriform rim, overfill as resorption can occur).

Post-operative

- Antibiotics/chlorhexidine/oral hygiene instruction/soft diet for 5 days.
- Imaging in 8 weeks.
- Bergland

  (1) Type 1: Normal interdental alveolar height.
  (2) Type 2: >75%.
  (3) Type 3: <75%.
  (4) Type 4: Nil.

Kindelan score in some units

Buccinator Flaps

- Pedicle, 3 weeks need to disconnect, may use bite block to protect.
- Myomucosal flap—axial pattern, based on facial or buccal artery.

VPI

- Poor palatal function.
- Undiagnosed sub mucous clefts.
- Palatopharyngeal disproportion.
- Neuromuscular dysfunction.

Cleft Speech (Complex)—Characteristic

- Hypernasal.
- Hyperresonance.
- Nasal emission.
- Nasal turbulence.
- Facial grimace.

Speech Problems Related to

- VPI.
- Nasal obstruction.
- Dental malocclusion.
- Jaw skeletal abnormality.

Speech Therapy

- Articulation therapy—main.
- Improve speech.
- Modify place of articulation- to move.
- Anterior positions consistent with normal speech.

Resonance

- Little evidence to modify hypernasality.

Oronasal Fistulas

- Usually hard palate/sometimes soft palate—abnormal communication between oral cavity (nose).
- Problems.
- Nasal regurgitation.
- Speech: nasal air escape.
- Hygiene: food impaction, bad taste from nasal secretion, halitosis.
- Classification Pittsburgh I–VII.

Usually After Primary Cleft Repair

- Infants: 1-4 mm—Leave until late childhood, if not functional speech/feeding concerns.
- >5 mm: Earlier closure indicated if functional concerns.
- Closure versus mucoperiosteal stripping—Impede growth, must allow 4–6 months after primary palatal repair.
- Consider obturators—Need speech therapist monitoring.

Treatment Principle

- Separation of oral and nasal side soft tissues each other.
- Closure hard palate 2 layers (nasal and oral mucosa).
- Closure of soft palate 3 layers (nasal side, elevator musculature, oral side mucosa).

Techniques Local Flaps Rotational *(*Fig. 5.4*)*

- Creation turnover flaps (single/double) around defect for nasal side closure.
- Elevation palatal finger flap and rotation flap for coverage defect.
- Exposed bone donor site heals by secondary intention.
- High failure rate: previous surgeries (difficult to mobilise without reduced tension).

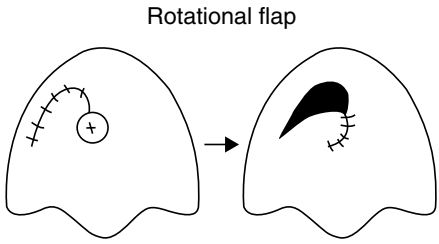

Fig. 5.4 Palatal rotational flap

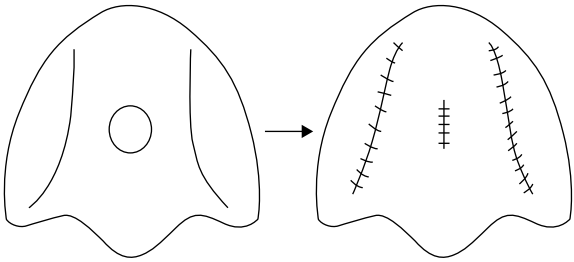

Fig. 5.5 Lateral releasing incisions

Modifications Von Langenbeck: for small defect in hard palate—lateral releasing incisions (Fig. 5.5)

Combined

- Local/regional flaps.
- Palatoplasty incorporating superior pharyngeal flap.
- Tongue based flaps (anterior, posterior and lateral).
- Large palatal defects, scarred palate—cases with previous ONF failure.
- Anterior based tongue flap better tolerated, allows greater degree of tongue mobility.
- Nasal side closure with turnover flap with 4-0 vicryl.
- Tongue flap—5 cm long 1/2–1/3 width of tongue (wide length to cover defect, elevated with 7–10 mm of underlying tongue musculature with rich vascularity.
- Tongue closed primarily.
- 2–3 weeks (GA—flap sectioned, stump at donor site freshened and inserted into tongue).
- Buccal mucosa/fat pad.
- Buccinators myomucosal.
- Temporalis.
- Free flaps.

Cleft Orthognathic

- Distraction ontogenesis—RED frame.
- Le Fort I cuts internal/external distraction.

Advantages

- Less relapse.
- May affect speech less or adjust if speech changes.

Disadvantages

- Unsightly frame 6–8 weeks of external—psychological consequences.
- Expensive—need > 1 surgical procedure.

VPI

No Method to Predict VPI

- Not possible to predict palatal function reserve.
- Not always related to distance moved.
- Careful planning.
- Bimaxillary surgery—minimize movement.
- Distraction.

Consent For

- Relapse.
- VPI.
- Bone graft in pre-maxilla. Alveolar site free flap.

Cleft

- Bilateral—cleft osteotomies: blood supply to pre-maxilla is compromised.
- Full labial incision—risk of vascular compromise.
- Tunneling approach—small vertical incision in the midline to get access to pre-maxilla bone to enable full mobilisation.

Issues

- Maxillary hypoplasia: vertically short (often), canted occlusal plan (up on cleft side).
- Class III malocclusion.
- Residual ONF ± bone facts, cleft dental gap.
- Prognathism uncommon—only if facial asymmetry, occlusal plane cants/AP discrepancy.

VPI

- SALT assessment prior to surgery—patients counselled (VPI—may worsen).
- Vascular supply: compromised vascular pedicles due to previous surgery, influences incision design/advancement/and treatment planning.
- Some cases accept—cross bite/other occlusal imperfection rather than risk necrosis.

Surgical

(1) If pharyngeal flap in place, oral intubation required

    - With maxillary advancement—remove flap only if not able to adequately mobilize maxilla.

(2) If maxilla grafted successfully:

    - Standard Le Fort I.
    - Maintain adequate buccal pedicle on mobilized maxilla.

(3) If buccal pedicle (difficult)—may need limited/small access

- Cleft: Greatest resistance to mobilization—vertical portion of palatal bone, posterior-medial aspect of maxillary sinus.
- Failure to weaken these structures—prior to mobilization may result in unfavorable fracture extending to base of skull or orbit, may cause blindness.
- Segmental osteotomy: Could compromise vascularity, except crossbite.

Lateral Incision/Canine: Not Present/Dysmorphic (4 Options)

- Removable prosthetic.
- 3-unit bridge.
- Endosseous implant.
- Orthodontic substitution—ipsilateral canine (eliminate need for any prosthetic component).

Lip/Nasal Form

(1) Unilateral cleft lip/palate

- Typically, nasal deformities: Splaying alar base, inferior displacement alar rim, deviation of nasal tip, base columella displaced towards non-cleft side.
- Timing.

(2) If maxillary advancement—delay 6 months post-orthognathic.
(3) Early: if severe airway/nasal airflow problems/psychological (school teasing), but after ABG—stable foundation, along piriform rim and nasal base exist first.
(4) Reconstruction—often requires

- Dorsal reduction.
- Lower lateral cartilage sculpting.
- Cartilage grafting.
- Nasal osteotomies.

(5) Bilateral cleft lip/palate

- Asymmetry less problematic.
- Short columella.
- Timing rationale—similar to UCLP.
- Technique.

(6) Posnick: Septal cartilage strut grafts attached to caudal nasal septum and lower lateral cartilages.
(7) Correction of underlying cartilaginous anatomy with stretching of overlying soft tissue envelope.
(8) Cleft lip scar revision

- Vast majority require it—ideally one lip scar revision between age 5–15 (staged as late as possible).
- Earlier if severe/psychological.
- Excision of residual scar: re-approximate key anatomical landmarks.

Hearing

- Eustachian tube dysfunction—common in cleft palate.
- Palatal surgery improves velopharyngeal closure.
- High %—otitis media with effusion (OME).
- Need careful audio logical follow-up.
- Only put ventilation tube if significant hearing loss on auditory evoked brain stem response.
- NICE: lack of evidence on optimal treatment.
- Insert ventilation tube at time of cleft lip/palate repair.
- Can place hearing aids.

Cleft Outcome

- Facial growth.
- Speech GOS.SP.ASS Great Ormond Street Speech Assessment.
- Dental.
- Hearing.
- Appearance.
- Psychological.

Chapter 6
Oral Medicine

Abstract An overview of oromucosal conditions and their management, and oral manifestations of systemic diseases.

Key Points

- Oral ulceration including Behçet's Syndrome.
- Lichen planus and lichenoid reactions.
- Conditions causing oral ulceration.
- Erythema multiforme, Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis.
- Reactive arthritis.
- Syphilis.
- Tuberculosis.
- Management of opportunistic and deep fungal infections.
- Acute Necrotising Ulcerative Gingivitis.
- Systemic Lupus Erythematosus.
- Graft-Versus-Host Disease.
- Oral candidal infections.
- Geographic tongue.
- White sponge naevus.
- Fordyce Granules.
- Oral hairy leukoplakia and EBV.
- Oral manifestations of HIV.
- Nicotinic stomatitis.
- Actinic cheilitis.
- Erythroplakia.
- Pyogenic granuloma and pregnancy epulis.
- Peripheral giant cell granuloma.
- Vesiculobullous disease.
- Infections including herpes simplex, varicella zoster, herpangina and hand foot and mouth disease,
- Ramsay Hunt Syndrome, measles and mumps.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_6

- Verrucopapillary lesions.
- Pigmented lesions.
- Granulomatous conditions.

6.1 Oral Ulceration

- Defect in continuity of epithelium, exposing underlying connect tissue, causes:
- Traumatic
  - Physical.
  - Chemical.
  - Burns.
- Infective: Bacterial/Viral.
- Idiopathic Recurrent oral ulceration.
- Drugs: e.g. Nicorandil.
- Mucocutaenous
  - Erosive lichen plans.
  - Vesicobullous disease.
- Neoplastic.
- Systemic disease
  - Haematological.
  - GI disease.
  - Autoimmune.
  - Autoinflammatory (Behçet's Syndrome).
- Clinical
  - Diagnosis and history: site/size/shape/symptoms (painful/duration/number).
- Investigations
  - Biopsy if >3 weeks.
  - Bloods/immunology/HLA.

6.2 Recurrent Oral Ulceration

- 25% population.
- T cell mediated immunological reaction.

Following All Implicated

- Trauma.
- Genetic: Increase HLA—A2, A11, B12, and DR2, family history.

- Nutritional: Increase in Crohn's/Coeliac (associated with HLA types), secondary to malabsorption.
- Hematological: thinning epithelial barrier.
- Immune dysregulation: HIV, chemotherapy, IgA deficiency.
- Hormonal: decrease progesterone in luteal phase of menstrual cycle - pregnancy.
- Stress.
- Drugs:
  - Nicorandil: mimics SCC/very painful—also have skin (cutaneous)/perianal.
  - ACE: angioedema.
  - Decrease/withdrawal drugs may help.

6.3 Classification

Minor: 80%

- Increase in females.
- Non-keratinised (buccal/labial).
- <5 mm; 1–5 at a once.
- Extremely painful.
- Yellow/white membrane/erythematous halo.
- Last 10–14 days, health without scarring, espies few weeks–years.
- Prodromal phase—burning/itching.

Major: 10%

- Post-puberty.
- >10 mm up to 4 cm; 1–2 ulcers.
- Deeper extension.
- Keratinised/non-keratinised: posterior mouth/soft palate/oropharynx.
- Last up to 6 weeks, may heal with scarring.

Herpetiform

- More common in females.
- Occur in adulthood.
- Any mucosal surface.
- up to 100 small (1–3 mm ulcers).
- May coalesce, resemble primary herpetic stomatitis.
- Last 7–10 days.
- Frequent recurrence (may be almost continuous for 2–3 years).

Investigations

- FBC, haematinics, zinc.
- Immunology.

  - Anti-endomyosial—Coeliac.
  - Tissue transglutamate antibodies—Coeliac.
  - Need to do serum immunoglobulins as a lot of people have low IgA.
  - ANA/ENA.
- Biopsy: Major to exclude SCC.

Management

- Chlorhexidine: RCT decrease length of ulceration.
- Benzydamine hydrochloride 0.15% (Difflam).
- Topical: betamethasone mouthwash.
- Covering agent: Topical local anaesthetic (Xylocaine).

6.4 Other Topical Treatments Include

- Prednisolone.
- Doxycycline.
- Fluticasone nasules.
- Beclometasone inhaler.
- Triamcinolone
  - Intralesional—triamcinolone.
  - Systemic—Severe ulceration: predisolone/colchicine/DMARD e.g. azathioprine.

6.5 Cochrane Review (2012)

- 25 trials.
- No single treatment found effective.
- Individual drugs seem to work on individual patients.

6.6 Behcet's Syndrome

- Rare, multisystemic, autoinflammatory disorder
  - Oral ulceration.
  - Genital ulceration.
  - Ocular disease—uveitis (hypopyon in severe disease), retinal vasculitis.
  - Cutaneous—folliculitis, pustules, acneiform lesions, erythema nodosum-like lesions.
  - Arthropathy.

  - Neurological.
  - Vascular.
  - Gastrointestinal.
  - Pericarditis (rare).

- More common in individuals with Silk Route ancestry.
- Young adults.

Aetiology

- Unknown.
- Associated with HLA-B51.

Investigations

- Pathergy test: 1–2 days after injection saline—sterile pustule develops.
- Bloods—FBC, ESR, autoimmune serology to exclude other systemic disease.
- Not always diagnostic.

Management

- Multidisciplinary: Oral medicine, immunology, ophthalmology, neurology, rheumatology, psychology, gastroenterology.
- Oral ulcers: Triple mouthwash (betamethasone, doxycycline, nystatin).
- Genital ulcers: Topical steroids.
- First line management colchicine, azathioprine.
- Other DMARDs: mycophenolate mofetil, ciclosporin, 6-mercaptopurine.
- Severe disease: Anti-TNFa e.g. infliximab, adalimumab.

Prognosis

- Periods of disease activity and inactivity.
- Mortality low unless CNS/or significant vascular involvement.
- Risk of blindness—ophthalmology review essential.

6.7 Lichen Planus

- Immunological-mediated disease of strained squamous epithelium of mucocutaneous tissues.
- Affects oral mucosa, genital mucosa, skin, scalp and nails.
- T cell mediated autoimmune response: basal layer keratinocytes attacked.

6.8 Epidemiology

- 1–2% of adult population.
- More common in females.
- Associated with Hepatitis C in certain population.

6.9 Clinical Features

- Oral: buccal/labial mucosa/tongue/gingivae—usually bilateral, can be asymptomatic or painful, particularly if erosive.
- 6 clinical subtypes, can have more than one type at a time.
- Reticular—70–80% raise white lines/stria.
- Erosive—9% painful, slow healing, ulcer/erosions, desquamative gingivitis.
- Atrophic—red atrophic areas, desquamative gingivitis.
- Papular—white papules.
- Plaque-like—white plaques.
- Bullous.
- Other manifestations
  - Cutaneous—pruritic, purple, polygonal, planar papules.
  - Often with white Wickham's striae on flexor surfaces of wrists, arm, ankles, legs and trunk.
  - Can have Koebner's phenomenon—trauma induces skin lesions.
  - Nail involvement: Longitudinal ridges, occasional destruction of nail.
  - Scalp: Hair loss due to scarring alopecia.
  - Glans penis/vulva: similar to oral lesions.

6.10 Histology

- Hyper- or parakeratosis.
- Variable thickness spinous layer.
- Rete ridges classically saw tooth.
- Liquefaction degeneration of basal layer.
- Intense band-like infiltrate T cells.
- Civatte bodies.
- In atrophic lesions can have thinning of epithelium, destruction of basal cells and a subepithelial inflammatory infiltrate.

6.11 Treatment

- Asymptomatic—no active treatment.
- Pain/ulceration—escalating treatment
  1. Topical anaesthetic and/or barrier agent—benzydamine hydrochloride 0.15% spray/mouthrinse or 2% lidocaine gel to painful area.
  2. Anti-septic mouthwash Cholrohexidine.
- Treat superimposed candida infections.

6.12 Other Factors

- Toothpaste (SLS), medication, amalgam restoration.

6.13 Topical Corticosteroids

- Soluble prednisolone 5 mg dissolved in 10 ml water, used for 2–3 min.
- Betamethasone sodium phosphate 500mcg dissolved in 10 ml water, used for 2–3 min.
- Fluticasone propionate nasules 400mcg dissolve in 10 ml water, used for 2–3 min.
- Triple mouthwash (500mcg betamethasone, 100 mg doxycycline, 100,000 units nystatin dissolve in 10 ml water, used for 2–3 mins, 4 times daily, no eating or drinking after use).

6.14 Review Intervals

- No evidence base/consensus.
- MLE guidance: patient with oral lichen planus monitored for oral cancer as part of routine dermal examination.
- 1 year asymptomatic/6 monthly for severe forms/3 monthly if dysplasia present on biopsy.

6.15 Prognosis

- May persist for many years, unknown malignant potential—controversial, many restrospecitive studies, lack clarify for initial diagnosis—present evidence may develop SCC (0.5–2% over 5 year period).

- Lichen planus is common, as association with SCC coincidental, as there would be SCC if true.
- Advised to keep under review with GDP every 6 months, patient must be informed, advised to reduced alcohol intake, and stop smoking, frequent consumption of fresh fruits and vegetables.

6.16 Lichenoid Reaction

- Closely resemble lesion of erosive LP, but due to drug reaction, hypersensitivity to amalgam/toothpaste.
- Biopsy may aid diagnosis, dental materials allergy testing.
- Common: gold salts/beta blockers, anti-malarias, thiazide diuretics, furosemide, spironolactone, metformin.

6.17 Desquamative Gingivitis

- Chronic mucosal desquamation, seen as part of numerous diseases especially erosive lichen planus/pemphigoid.
- Common in females >40.

Clinical

- Affects gingival tissues from margin to alveolar mucosa.
- Loss of gingival stippling/erythema.
- Vesicles/bullae form—rupture = long term: areas of ulceration/erosion/atrophy.
- Usually anterior mouth, may be asymptomatic or burn/aggravated by food.

Causes

- Lichen planus.
- Vesiculobullous disease.
- Allergies: toothpaste.
- Psoriasis.
- Drugs.

Investigations

- Biopsy + immunofluorescence.
- Patch testing.

Treatment

- Oral hygiene.
- Topical steroids as above.

6.18 Ulcerative Conditions—Underlying Causes

- Traumatic.
- Recurrent aphthous stomatitis.
- Autoimmune: Reiter's/Erythema Multiforme/Granulomatosis with polyangiitis (Wegener's) and other connective tissue diseases.
- Inflammatory: Crohn's, Ulcerative Colitis, Coeliac, Behçets Syndrome.
- Infective:
  - Viral (HSV/VZV, Coxsackie).
  - Bacterial: TB, syphilis, actinomycosis, gonorrhea, leprosy.
  - Fungal.
- Drugs.
- Neoplastic: SCC, salivary gland tumours.

6.19 Traumatic Ulceration

- Mechanical, thermal, chemical, radiation (<60 Gy/fraction).

Clinical

- Acute: Pain, redness swelling, yellow white fibrinous exudate—heal in 7–10 days.
- Chronic: Eosinophilic ulcer—sharply demarcated, indurated.

Differential Diagnosis

- Infection: TB/Syphilis, deep fungal.
- Neoplastic.

Histopathology

- Acute: Loss epithelium, containing neutrophils.
- Chronic: Granulation tissue base with scar in deeper tissue, dense macrophage infiltrate.

Management

- Remove aggravating factor.
- If symptomatic = topical steroids.

6.20 Traumatic Eosinophilic Ulceration

- Chronic: clinically resembles SCC.
- Commonest site is tongue.
- Middle painful or asymptomatic, long duration without healing.

Differential Diagnosis

- Infection: TB/Syphilis, deep fungal.
- Neoplastic: SCC.

Investigations

- Incisional biopsy.
- CT/MRI—resemble SCC.

Management

- Excision—0.5 cm margin to avoid recurrence.

6.21 Erythema Multiforme

- Sudden onset hypersensitivity reaction affecting the skin and mucous membranes.
- Characterised by cutaneous target lesions.

Epidemiology

- Young, age 20–30 or children.
- M > F, often with family history.

Causes

- Drugs: Carbamazepine, phenytoin, penicillins, NSAIDs—old drugs—barbiturates/sulphonamides.
- Infection: Herpes simplex, mycoplasma pneumonia.
- Ultra-violet light, chemicals (food colours, perfumes, pregnancy, malignancy, immune conditions).

Clinical

- Wide spectrum
  - Prodromal: Headache, malaise, arthralgia.
  - Oral: ulceration, haemorrhagic, circumoral crusting—similar lesions affect genitalia.
- Eyes: Photophobia, conjunctivitis, symblepharon.
- Skin: Target lesions (hands/feet)—concentric rings with central papule that becomes ulcerated, surrounded by red then pale oedematous ring = usually subsides after 2 weeks can last up to 6 weeks (25% cases occurs).
- Severe:
  - Stevens-Johnson syndrome—often drug related, may have either eye/genital involvement.
  - Toxic Epidermal Necrolysis: Severest part of spectrum, can be lethal.

Treatment

- Withdraw any triggers, supportive therapy: nutrition/I.V. rehydration.
- Oral: Oral hygiene, chlorhexidine mouthwash.
- Triple mouthwash: 500 mcg betamethasone, 100 mg doxycycline and 100,000 units Nystatin dissolved in 10 ml water and used for 2–3 min.
- Systemic: Prednisolone/immunosuppression—MDT—immunology/dermatology.
- Acyclovir if herpes simplex.

6.22 Reactive Arthritis (Reiter's Syndrome)

- Rare post STI.
- Triad of = arthritis/urethritis/conjunctivitis ± aphthous ulcers and systemic symptoms.

Clinical

- Cutaneous—macules/vesicles/pustules.
- Oligoarthritis: affects large/small joints of lower limbs (self-limiting with remission in 3/12).

Diagnosis

- Clinical, 70% have HLA-B77 genotype (tissue typing), increased ESR.

Management

- Ophthalmology/immunology/rheumatology.
- NSAIDs.

Syphilis

- Sexually transmitted infection by Treponema Pallidum (gram negative spirochaete).
- Incidence: Increasing numbers, STI, parenteral/vertical transmission.

Primary

- Large painless ulcer (chancre) + lymphadenopathy heals in 7–10 days.
- Primary lesion develops at site of infection
  - Genitalia/anus—commonest site entry.
  - Upper lip/oral cavity rarely.

Secondary

- 4–8 weeks post infection.
- Systemic flu like symptoms: fever. Headache, weight loss.

- Widespread generalised maculopapular cutaneous rash on trunk, plantar and palmar surfaces.
- Lymphadenopathy.
- 'Snail track ulcers' (serpiginous ulceration) commonly on buccal mucosa and palate.
- Condylomata lata may appear in the genital, perineal and anal areas.
- Resolve by 12 weeks, may recur for up to 1 year.
- Latent if secondary syphilis resolves without treatment.

Tertiary

- 1/3 patients, latent period up to 30 years multi system pathology usually secondary to endarteritis obliterates affecting vascular system including aneurysms LUH, CCF.
- CNS: Paralysis, tabes dorsalis.
- Gumma: Granulamatous inflammation—widespread sites
  - Oral: gumma—tongue/palate (focal/necrotic inflammatory lesion). Can cause communication with sinus/nares.
  - Interstitial glossitis: tongue becomes lobulated/irregular as muscles contact and gumma heals.
  - Syphillitic glossitis: atrophy and loss tongue papillae (malignant change), syphilitic leukoplakia.

Quaternary

– Cardiovascular and neurosyphilis.

Congenital Syphilis

- Frontal bossing, short maxilla, saddle nose, saber chin.
- High arched palate, mandibular prognathism.
- Hutchinson's triad
  (a) 8th nerve deafness.
  (b) Ocular interstitial keratitis.
  (c) Moon's/Mulberry molars.
  (d) Notched incisors.

Differential Diagnosis

- 'Great mimicker'.
- Primary: SCC, TB.
- Secondary.
- Tertiary: T cell lymphoma, mucormycosis.

Investigations

- Haematology: VDRL, (venereal disease research lab) PRP, ELISA.
- Park ground microscopy of fluid/scraping.
- Histopathology silver stain of biopsy.

- TPHA: treponema pallidum.
- FTA-abs: Treponema pallidum absorption test.

Management

- Penicillin/doxycycline/erythromycin.
- Contact tracing recommended, notifiable disease.
- When treatment: Jarisch Herxheimer reaction (as cells die)

  (a) Fever.
  (b) Muscle pain.
  (c) Tachycardia.
  (d) Headache.

6.23 Tuberculosis

- Mycobacterium (aerobic, non-spore forming AFB), rare M. bovis.
- Caseating granulomatous infection.
- 1 billion infected worldwide: droplet spread (M. tuberculosis) via pasteurised milk (M. bovis).

Clinical

A. Primary
   – 5–10% infection reactivated.

B. Secondary
   – Systemic: low grade fever, night sweats, weight loss, malaise, anorexia, cough—haemolysis/pleuritic chest pain.

Head and Neck

- Cervical lymphadenopathy (collar stud abscess/scrofula).
- Indurated, chronic, non-healing ulcers: usually painful (tongue/palate/fissures/granular areas).
- Rarely osteomyelitis.

Investigations

- Sputum: Ziehl–Neelsen stain—acid fast bacilli.
- Biopsy: 6 weeks (take OPG) = Löwenstein-Jensen slope.
- Chest XR/CT.
- Quantiferon gold, Mantoux test.
- Interferon assay to detect latent TB.

Treatment

- Chest.
- RIPE—at least 9 months = isoniazid, rifampicin, pyrazinamide, ethambutol. Check local policy.

6.24 Opportunistic Fungal Infections

- Mucormycosis.
- Aspergillosis.
- Transmitted by GIT/respiratory tract.

Clinical Features

- Invades—arterial walls/thrombosis (death of tissues/necrosis), haematogenous spread.
- Pain/swelling—precede ulceration.
- Muscles: nasal cavity, paranasal cavity, oropharynx.
- Maxilla: overlying skin necrosis, swollen eyes, sinusitis, headache, black discharge.

Differential

- Syphilis, T cell lymphoma.
- Granulomatosis with polyangiitis (Wegener's), sinus SCC, salivary gland.

Investigations

- Mucocutaneous/biopsy.

Treatment

- Anti-fungals: Amphotericin-B.

6.25 Deep Fungal Infections

- Characterised involvement of lungs—disseminate to involve any organ.
- Histoplasmosis, Cryptococcosis, Coccidiomycosis, Blastomycosis.

Clinical

- Lung, cough, fever, night sweats, weight loss, chest pain, hemoptysis.
- Oral lesions: chronic non-healing ulcers.

Investigations

- Infectious disease referral, anti-fungal (Amphotericin-B, ketoconazole).

6.26 Granulomatosis with Polyangiitis (GPA)

- Previously known as Wegener's Granulomatosis.
- Multi-system inflammatory disease: characterised by granulomatous inflammation + necrotising vasculitis, classically: URT, lung, kidneys (renal function).

Clinical

- Red hyperplastic, granular lesions on attached gingiva.
- Necrosis, perforation of the nasal septum/palate.

Management

- Immunology/rheumatology referral.
- Steroids or other immunomodulatory drugs.

6.27 Acute Necrotising Ulcerative Gingivitis

- Acute onset, necrotic ulceration of gingival tissues, due to:
- Fusobacterium.
- Spirochaetes: Borrelia Vircenti.
- Anaerobic rods.

Risk Factors

- Males, smokers, recently systemically unwell, malnutrition, poor oral hygiene, stress, immunosuppressed (HIV), local trauma.
- <0.1% population.

Clinical

- Interdental papillae initially oedematous/haemorrhagic = then punched out ulcers and necrosis spreads along gingival margin.
- Halitosis, mild lymphadenopathy—if severe spread to bone/skin = NOMA.

Treatment

- Oral hygiene/debridement.
- Irrigation (3% hydrogen peroxide 1:1 water or 0.12% chlorhexidine).
- Metronidazole + tetracycline.
- Investigate underlying immune problem.

NOMA

- Extension ANUG.
- Mainly affect malnourished children in developing countries after illness.

- Cancrum oris: gangrenous necrosis of gingiva = continue to involve oral mucosa/skin, underlying bone may be affected seen in developed countries with underlying immunosuppression (leukaemia/chemotherapy).

Treatment

- Rehydration.
- Antibiotics: penicillin/metronidazole.
- Conservative debridement.
- Reconstruction after disease more than 1 year.

6.28 Systemic Lupus Erythematosus

- Multisystem, autoimmune connective tissue disease.
- Classification: Discoid (skin/mucous membrane)/and systemic.

Clinical

- Skin: red, scalp patches—malar butterfly rash.
- Mucosal: resemble OLP (more common in palate).
- Rheumatological: arthritis ± Sjögren's/Raynaud's.
- Cardiac, renal, hematological, CNS.

Investigations

- FBC/increased ESR/ANA/anti-ds-DNA/anti-Sm/biopsy IgG on BM (granular layer).
- Management: Refer to rheumatology for systemic treatment.

6.29 Graft-Versus-Host Disease

- Complex multi system immunologic phenomenon occurs 70% allogenic bone marrow transplants.
- Classification: Acute (<100 days since BMT) versus Chronic (>100 days).
- Aetiology: Transplanted immunocompetent donor T cells—attacks various recipient tissues, patient receives immunosuppression to prevent this.

Clinical

- Most often affect skin/liver/GIT/lung/eye.
- Mimic other autoimmune: SLE/Sjögren's/Primary biliary cirrhosis/burning mouth.
- Oral manifestation in 80%: LOOXE
  - Lichenoid: Clinically/histologically similar.
  - Opportunistic: Candida/aspergillus/HSV.

  - Oral SCC.
  - Xerostomia: Mucosal affected, later major salivary glands.
  - EM/TEN: scleroderma.
- Investigations: Biopsy exclude SCC.

Management

- Xerostomia: hydration, saliva substitutes, fluoride.
- Treat infections.
- Triple mouthwash (500 mcg betamethasone, 100 mg doxycycline, 100,000 nystatin dissolved in 10 ml water and used for 2–3 min).

6.30 Oral Candidal Infections

- Candida albicans: 50% oral commensal = unicellular yeast in 2 forms:
- Yeast—benign.
- Hyphae—ability to invade host tissue.
- Risk factors—diabetes, immunosuppression, smoking, antibiotics, dentures.

Classification

- Pseudomembranous
  - Acute: thrush.
  - Chronic

    Steroid inhaler immunocompromised—creamy white plaque, rubs off leaving erythematous areas.
    Affect palate/tongue/buccal mucosa with bleeding points/maybe burning sensation/bad taste.
    Seen neonates, patient with chronic diseases.
- Erythematous
  - Acute—antibiotic sore tongue.
  - Chronic—denture stomatitis.
  - Often severe burning/pain affects hard palate/buccal/dorsum of tongue.
  - Median rhomboid glossitis—pseudoepitheliomatous hyperplasia.
  - Filiform papillae lost if tongue affected.
  - Angular cheilitis: Staph aureus present 60%—associated with reduced occlusal vertical dimension in edentulous individuals.
  - Tongue: smooth/red.
- Hyperplastic
  - Inflammatory papillary hyperplasia.

- Candidal leukoplakia
  - Considered pre-malignant lesion occurs mainly smokers/drinkers.
  - Often decreased ferritin, high-rate conversion to SCC.
  - Buccal mucosa adjacent commissures, commonest in white plaques/speckled leukoplakia.
  - Candidal type seen in lesions, increase dysplasia in speckled lesions.
  - May regress dysplasia with fluconazole.
  - Candida infection superimposed on leukoplakia.
- Chronic mucocutaneous
  - Candidiasis (CMCC): rare group of immunological disorders with superficial mucocutaneous candidiasis, affect skin/nails/oral mucosa.
  - Usually, thick white plaques that cannot be removed.
  - May develop endocrine abnormalities in time.

Investigations

- Oral swabs from 3 separate sites.
- Candidal rinse = 10 ml sterile water gargled—micro smear PAS stain gram + ve type.
- Biopsy mandatory—candidal leukoplakia.
- Check FBC, haematinics, zinc—medical investigations underlying systemic cause.

Management

- Oral hygiene/denture care—keep it out at night.
- Antifungals for 6 weeks.
- Polyenes.
- Nystatin 100,000 units/g—topical.
- Amphotericin lozenges 10 mg.

A. Imidazoles
   - Miconazole gel (also anti-staph).
   - Clotrimazole—topical.
   - Ketoconazole.

B. Triazole: Fluconazole 50 mg once daily for 7–14 days
   - Avoid pregnancy.
   - Multiple drug interactions.
   - Monitor liver if high dose/long treatment.
   - Can cause rash/Stevens-Johnson Syndrome.
   - Severe cutaneous reactions in AIDS.

6.31 Geographic Tongue

- Erythema migrans.
- Migrating pattern: resemble the Earth of atrophic patches and hyperkeratotic margins on the tongue.

Investigations

- Clinical.
- Biopsy if atypical micro—candida (PAS).
- FBC, haematinics, zinc.

Management

- Reassurance asymptomatic.
- If candida, Nystatin 100,000 u/ml.
- Zinc—some evidence.

6.32 White Sponge Naevus (Cannon's Disease)

- Autosomal dominant, often appears before puberty.
- Asymptomatic while folded lesion, spongy consistency, almost always bilateral and symmetrical (buccal mucosa > tongue > vestibular mucosa).
- Histopathology: Epithelium grossly ticked with spongiosis/parakeratosis.
- Management: None.

6.33 Fordyce Granules

- Sebaceous glands.
- 80% of population—no treatment required.
- Reassure.

6.34 Oral Hairy Leukoplakia

- Opportunistic infection related to EBV in mainly HIV/AIDS
  - EBV: Human Herpes Virus 4.
  - Infectious mononucleosis.
  - Burkitt's lymphoma.
  - Nasopharyngeal cancer.

- Well demarcated white lesions: plaque like—lateral surface tongue, usually asymptomatic.
- Differential diagnosis:
  - PVL/verrucous cancer/carcinoma/hyperplastic candidiasis.
- Management
  - HIV diagnosis: anti-viral therapy.

6.35 HIV

- RNA virus incorporates in DNA host.
- Antibodies developed, lifelong infection.
- Affects C4 receptors, mainly T-helper, monocytes, dendritic cells = decrease CD4 count and thus T cell-mediated immune response.
- Patients prone to infection—fungi, encapsulated bacteria, often viruses.

Clinical

I. Acute infection
   - Often subclinical, acute self-limiting viral illness, seroconversion occurs 1–2 months.

II. Asymptomatic stage
   - 8–10 years: increase with modern treatment, lifelong asymptomatic infection.
   - May have generalised persistent lymphadenopathy.
   - Biopsy if persistent increase ESR/or localised bulky nodes (increase NHL in HIV).

III. HIV disease
   - Symptomatic infection: viral/fungal infection.
   - e.g. Pneumocystis pneumonia/Histoplasmosis/virally induced malignancies/CNS—dementia, weight loss.

IV. AIDS (acquired immunodeficiency syndrome)
   - Variable presentation CD4 < 200—poor prognosis.

Oral Manifestation—Strongly Associated

- Candidiasis: often in oropharynx—systemic antifungal.
- Hairy leukoplakia: see above.
- HIV periodontal disease.
- Initially linear erythema along gingival margin (ANUG to NOMA).
- Gross vertical bone loss, not responding to oral hygiene instructions.

- Kaposi sarcoma.
- Hard palate/gingiva.
- HHV-8 related.
- Multifocal neoplasm of vascular origin: red/brown non blanching lesions (common).
- Treat only if causing symptoms: RT/intralesional vincristine/local excision/ cryotherapy.
- All palliative only.
- Non-Hodgkin Lymphoma
  - Less commonly associated.
  - Aphthous ulceration.
  - Salivary gland disease.
- HIV associated lymphoepithelial cysts or focal lymphocytic sialadenitis affecting both parotid glands.
- Viral infections
  - e.g. GSV/VZV/CMV/HPV.
  - Increase dysplasia in oral papilloma caused by HPV.
  - Idiopathic thrombocytopenia purpura.
  - Possibly associated with HIV.
  - Other bacterial/fungal infections.
  - Hyperpigmentation.
  - Neurological problem: trigeminal neuralgia, dysaesthesia, palsies.
  - SCC oropharynx: same risk factors, but develop earlier.
  - Diagnosis.
  - Counselling mandatory prior to testing.
  - HIV antibodies develop by 3 months—98% sensitivity, negative early (antigen testing/PCR).
  - Decrease in CD4 count, CD4/CD8 ratio used for monitoring.
  - Present with unusual pathology: hairy leukoplakia, Kaposi.
  - Treatment.
  - Combination anti-retroviral therapy—HAART.
  - Increased resistance and side effects—facial lipoatrophy.

6.36 Nicotinic Stomatitis

- Tobacco related keratosis typically with pipe/reverse smokers.
- Palatal mucosa with red dots (inflammation of salivary gland excretory ducts) surrounded by white keratotic rings.
- Management: not pre-malignant, smoking cessation, although the rest of mouth increased risk of SCC.

6.37 Actinic Cheilitis

- Sun damaged premalignant lesion of the lip (lower > upper); UVB > UVA.
- Lips atrophic, pale silver-grey glossy appearance ± fissuring.
- When severe—epidermalisation of vermillion and superficial scaling/cracking/ulceration.

Management

- Lip protection (PABA sunscreen).
- Biopsy if ulceration/induration—periodic examination.
- Topical 5-FU—2% twice daily for 3 weeks or vermilionectomy (laser/surgical).

6.38 Erythroplakia

WHO: bright red velvety plaques, not characterised clinically/pathology by anything else.

Clinical

- Well-demarcated erythematous plaque/papule often associated with leukoplakia.
- FOM > tongue/soft palate.
- Waldron and Shafer 1974: 91% showed severe dysplasia/CIS/or invasive carcinoma.
- Very high-risk transformation.

Management

- Biopsy depends on histology: excision (laser/surgery).
- CIS/mod dysplasia: 5–10 mm margins = No evidence of recurrence/margins (Van der Waal 2009).
- Cochrane 2004: no evidence that topical treatment prevents transformation—majority undergo transformation.

6.39 Pyogenic Granuloma/Pregnancy Epulis

- Exuberant tissue response to injury: modified by hormonal features.
- Gingiva > lower lip, buccal mucosa, tongue—typically red/bleed excessively, initial rapid growth then stated soft/smooth/lobulated surface—red/purple.
- Investigations: OPG = exclude malignancy.
- Management: Surgical excision with 2 mm margin to depth periosteum (may recur if incomplete) remove cause if traumatic cusp etc.
- Pregnancy: Oral hygiene, may resolve after childbirth. If not, consider excision.

6.40 Peripheral Giant Cell Granuloma

- Uncommon gingival hyperplastic connective tissue response to injury.
- Usually young adults, red/blue broad-based mass, unlike PG, may arise from edentulous ridge.
- Management: Surgical excision = can recur if incomplete.

6.41 Vesiculobullous Disease

I. ***Congenital:***

- Epidermolysis bullosa (inherited CT disease, skin mucous membrane—can be lethal).

II. **Acquired**

- Infective: herpes complex, varicella zoster, hand/foot/mouth disease, herpangina, measles.
- Autoimmune: pemphigus, pemphigoid, dermatitis herpetiformis, linear IgA disease.

6.42 Mucous Membrane Pemphigoid

Chronic autoimmune vesiculobullous disease

- Predominately affects oral and ocular mucous membranes.
- Presents 10 years earlier than bullous pemphigoid, more common in females.

Clinical: affect mucous membranes commonly, skin lesions rare, same as bullous

1. Oral

- Desquamative gingivitis.
- Bullae or vessels often seen—thick walled, less likely to rupture than pemphigus breakdown to form extensive painful ulcers that may take weeks to heal.
- Nikolsky sign, affect any part of oral cavity but tend to recur at the same site.

2. Ocular

- About 25% of patients.
- Initially conjunctivitis and erosions, as well as subconjunctival scarring.
- Xerophthalmos can occur as lacrimal gland function decrease with scarring.
- Cornea procedures keratin as protective layer: decrease vision.

- Symblepharon (scarring between lids, decreased vision, may lead to blindness if untreated).

3. Other manifestations

- Skin: large bullae: flexural skin disease.
- Genital/nasal/oesophageal/laryngeal tissues—airway obstruction from large bullae.

Investigations: 2 Biopsies

- 1 × formalin: histology—sub-epithelial clefting. Chronic inflammatory infiltrate.
- Michel's medium/fresh: direct/indirect immunofluorescence—linear IgG and C3 deposits at basement membrane (direct).

Management

- Topical: Triple mouthwash, steroid ointment for genital lesions.
- Systemic: Prednisolone, DMARDs e.g. mycophenolate mofetil, azathioprine.
- Rituximab—anti-B cell CD20, stops antibody production early stage.
- Ocular: all patients need ophthalmology review even if no eye problems.
- Prognosis.
- Mostly controlled with systemic therapy.

6.43 Bullous Pemphigoid

- Commonest, M = F; age 60–70.
- Usually affect skin (limbs/abdomen). Oral lesions can occur but not frequently.
- Diagnosis/histology: similar to mucous membrane.
- Treatment: same systemic steroids.

6.44 Pemphigus Vulgaris

- Potentially life threatening vesiculobullous autoimmune disorder affecting skin/mucous membrane.
- Most commonly found in middle aged.
- High incidence—Ashkenazi Jews/Mediterranean descent.

Aetiology

- Circulating IgG auto-antibodies against desmoglein 3—prevent epithelial cell adherence, may lead to intra-epithelial cleaving.
- Rarely drug induced, paraneoplastic.

Other Subtypes

- Vegetans, foliaceus, erythematous, paraneoplastic.

Clinical

- Vulgaris commonest: affects skin/mucous membranes.
- Oral lesions precede skin lesions by up to 1 year—sometimes only clinical sign.
- Painful ulcers preceded by bullae (rupture easily), Nikolsky sign positive (digital pressure causes epithelial separation).

Desquamative Gingivitis

- Involvement of ocular mucosa.
- Investigation.
- Biopsy
  (a) Fresh histology = intra-epithelial vesicles 'tombstone appearance' free floating Tzanck cells.
  (b) Immunofluorescence = intra-epithelial IgG, IgM, C3 on direct, indirect +ve 80–90%.
- Serum: Indirect immunofluorescence, anti-desmosomal antibodies—levels indicate disease activity DSG-3.

Treatment

- Untreated—fatal: secondary infection/fluid/protein loss from extensive ruptured bullae.
- Mortality 10%.
- Swallowing difficulties with oesophageal involvement.

MDM: Ophthalmologist/Dermatologist

- Fluid resuscitation/prevention of infection.
- Initially high dose prednisolone: increase BP may need anti-hypertensives, PPI/osteoporosis prophylaxis.
- DMARD e.g. azathioprine, mycophenolate mofetil.
- Topical: Triple mouthwash.

Steroid Side Effects

- Candidiasis/atrophy/telangiectasia/striae/acne.
- DM/myopathy, increase lipids, moon face, buffalo hump, weight gain, hypertension, adrenal atrophy, cataract/glaucoma.
- Once settled then decrease to maintenance dose.
- DMARDs help to reduce steroid dose.

6.45 Angina Bullosa Haemorrhagica

- Acute/benign/generalised subepithelial oral mucosal blisters filled with blood that have no cause.
- Isolated/rapid healing/rare occurrence.
- Rare, possible trauma can cause airway obstruction if in posterior pharynx/epiglottic region.
- Middle aged, blisters initially burning/tingling—last for few minutes, and it burst shallow ulcer heals without scarring/discomforting or pain.
- Bloods (normal): difficult to get histology—sub epithelial bullae filled with blood, non-specific inflammation.
- Conservative treatment—benign.
- Bloods: FBC, clotting.

6.46 Linear IgA Disease

- Chronic autoimmune disease of skin that commonly affects mucous membranes (ocular/oral/skin).

Clinical

- Skin: urticaria, annular, targeted, bullous.
- Oral: ulcers preceded by bullae.

Histopathology

- Subepithelial blisters, IgA in homogenous linear pattern at basement membrane.
- Neutrophil microabscesses.

Management

- Ophthalmology referral.
- Mild: no treatment ± topical steroid.
- Severe: systemic steroids, DMARD.

6.47 Dermatitis Herpetiformis

- Chronic disease with symmetrical and intense rash.
- Papular/vesicular lesions on extensor surfaces/face/scalp often associated with Coeliac disease.
- Young/middle aged adults ‘herpetiform’ lesions.
- Oral: vesicles and bullae that rupture leaving non-specific ulcers on keratinised and non-keratinised mucosa.

Management

- Investigate for Coeliac disease: anti-TTG Ab, anti-EMA Ab—endoscopy + duodenal biopsy.
- If Coeliac diagnosed, gluten free diet once investigations completed.

Herpes Simplex

- DNA virus only found in humans—oral HSV-1/HSV-2 usually genital (can be oral).
- 60% developed world have antibodies infection prior to infection.

Primary Herpetic Gingivostomatitis

- Incubation: 3–9 days, transmitted by direct contact via saliva/other body fluid.
- Often subclinical (80%), clinically—malaise, fever, anorexia, tender lymph node anterior triangle neck.
- Vesiculobullous eruption mucosal surfaces, pin head ulcers fuse to form large painful erosions affecting free/attached mucosa: punched out lesions at midpoint of gingival margins.
- Lasts 5–10 days: children (gingivostomatitis)/older (pharyngotonsillitis).

Diagnosis

- Clinical, increased HSV antibody titres, PCR detection HSV DNA, cytology, culture.

Management

- Supportive therapy: bed rest/fluids/soft diet/analgesia.
- Anti-viral: if immunosuppressed or presented early.
- Antiviral: Acyclovir within 48 h: 200 mg 5 times/day–5% topical acyclovir.

Secondary–Recurrent

- Virus remains latent in dorsal root (autonomic or cranial nerves)—trigeminal or geniculate.
- Can be reactivated—may cause clinical signs—some patients have asymptomatic shedding.
- Incidence: 15% population.
  - Risk factors:
    - Stress.
    - Trauma.
    - Illness.
    - Immunosuppression.

Clinical

- Herpes labialis.
- Prodromal burning/sensation.

- Vesicles for 3–4 days.
- Heal with no scarring.

Management

- Topical 5% acyclovir if applied during prodromal period.
- Systemic: if frequent/immunosuppression (apply early), prophylaxis if severe, increase resistance in immunocompromised.

6.48 Herpangina

- Coxsackie infection type A virus (A1-6, A8, A10).
- Spread faeco-oral route.
- Commonest in <age 4.

Clinical

- Incubation 2–6 weeks, periodic epidemic, usually every few years.
- Mostly mild. Subclinical.
- Fever, malaise, vomiting, headache, 2–4 mm vascular: ulcerated lesions of oropharynx.
- Sites: pathognomonic = uvula, palate, fauces.

Treatment

- Bacterial throat swab to rule out streptococcal, pharyngitis, viral ulcer.

6.49 Hand, Foot and Mouth Disease

- Coxsackievirus (usually A16), endemic in young children, via droplets/faeco-oral.
- Similar to herpangina—except throughout oral cavity.
- Also get maculopapular rash on palm of hands/feet.
- Treat if symptomatic

6.50 Varicella Zoster—Human Herpes Virus Type 3

- Primary: Varicella (chicken pox).
- Mainly children with peak age 5–9 years.
- Spread via mucosa upper respiratory tract droplets or through vesicular fluid.
- Incubation 14–21 days: Prodromal illness with malaise, pharyngitis, rhinitis.
- Oral: buccal mucosa/vesicles/ulceration.
- Skin: itchy erythema > vesicles > crusted lesion = begins face—extremities.
- Contagious until all lesions crusted.

- Diagnosis: Clinical/increased antibody titres, viral culture.
- Management: supportive, antivirals if early diagnosis or immunosuppressed—acyclovir 800 mg 5 times daily.

Secondary (Herpes Zoster—Shingles. Resides in Sensory Ganglia After Infection.)

- Common in adults: elderly, alcoholic, immunosuppressed.
- Virus dormant in dorsal and cranial root ganglion.
- Shingles usually affects 1 dermatome.
- Prodromal period with neuralgia 1–4 days prior to rash > vesicles > pustules > ulceration > crusting > 10 days.
- Skin may heal with scarring and hypopigmentation.
- If ophthalmic division: corneal ulceration—visual loss. Look for lesion on tip of nose 'Hutchinson's sign'.
- 15% develop post-herpetic neuralgia—increase with older patients and respond to gabapentin or tricyclic antidepressants.
- Management: Acyclovir if seen within 3 days of first vesicle: decrease neuralgia incidence; ophthalmology opinion ophthalmic branch involved—conjunctivitis/epidermis/iritis/ocular palsies.

6.51 Ramsay Hunt Syndrome

- Rare—unilateral VII nerve palsy.
- Herpes zoster vesicles in the EAM and ipsilateral pharynx—geniculate ganglion.
- Vertigo/hearing loss/rhinitis secondary to VIII involvement.

Chicken Pox in Adults—More Serious Complications

- Reye's syndrome.
- Secondary skin involvement.
- Encephalitis.
- Cerebellar ataxia.
- Pneumonia—vomiting/diarrhoea.
- Viral—pneumonitis (serious/commonest).

6.52 Epstein-Barr Virus

- Human Herpes Virus type 4—spread saliva.
- Oral: infectious mononucleosis—often subclinical, prodromal lethargy prior to symptoms.
- Sore throat/tonsillar enlargement/fever/rhinitis/cough.
- Generalised tender lymphadenopathy in >90%—hepatosplenomegaly.
- Skin rashes: also seen if given amoxicillin.

- Petechiae hard/soft palate, ANUG, may be protracted recovery over many months
  - Remains latent in pharyngeal or salivary epithelial cells.
  - Atypical lymphocytes in peripheral blood which resemble monocytes.

Other Manifestations of EBV

- Hairy leukoplakia: hyperkeratosis lateral border tongue.
- Nasopharyngeal cancer associated with immunosuppression (transplant/HIV).
- Burkitt's lymphoma: No treatment, biopsy identify EBV—HIV testing.
- Lymphoproliferative disorder.

Diagnosis

- Monospot/Paul Bunnell test for antibodies—90% positive in adult cases/less useful in children.

Management

- Supportive, life-threatening airway obstruction secondary tonsillar enlargement.
- Treat with systemic steroids.

Measles

- High contagious paramyxovirus—triad of = cough + coryza + conjunctivitis.
- Koplik spots: small erythematous macules with white necrotic center on buccal mucosa.
- Maculopapular skin rash: head and neck > trunk > extremities.
- Systemic symptoms.
- Investigations: Viral culture/serology.
- Management: supportive/analgesia.
- Prognosis: 1:1,000—encephalitis/thrombocytopenia purpura/otitis media/pneumonia.

Mumps

- RNA paramyxovirus, mainly affecting parotid glands.
- Increased incidence in UK/decrease MMR > decrease herd immunity—especially young adults.

Clinical

- Droplet incubation—16 days, 30% subclinical infection.
- Prodromal: fever, headache, anorexia, myalgia.
- Swelling lower pole parotids (unilateral 25%)—peaks at 2–3 days—submandibular gland in 1-% with redness/oedema of ducts (both).

Adults Involves Other Tissues

- Epididymo-orchitis—25% usually unilateral, infertility rare.
- Oophoritis and mastitis—post-pubertal females.

- Pancreatitis/deafness/thyroiditis/meningoencephalitis.
- Diagnosis: Clinical, increased mumps specific IgM.
- Management: Supportive, hospital admission may be required in adults.

6.53 Verrucopapillary Lesions

- Congenital: White sponge naevus.
- Acquired
  - Traumatic: papillary hyperplastic, Heck's disease, verruciform, xanthoma.
  - Infective: Skin papilloma/wart, condyloma.
  - Neoplastic: PVI/verrucous carcinoma.

6.54 Squamous Papilloma (Oral Wart, Verruca Vulgans)

- Genetic term described benign papillary and verrucous lesion, some of which are HPV related (2, 6, 11, 57).
- Clinical: Predilection for palate/uvula—pink, white exophytic granular/ cauliflower like surface.
- Superficial removal: laser/excision.

6.55 Papillary Hyperplasia

- Cobblestone lesion hard palate—candida related to wearing prosthesis.
- Management: Excision/topical anti-fungal/removal of denture at bedtime.

6.56 Condylomata Lata

- Secondary syphilis: Exophytic, papillary, polypoid lesions, red/mushroom like mass.
- Incisional biopsy VDRC/RPR.
- Treatment: Antibiotics, contact tracing, notifiable disease.

6.57 Condylomata Acuminata

- STI due to HPV (6, 11)—anogenital may involve oral mucosa.

Clinical

- Numerous pink nodules coalesce to form a soft/broad based exophytic papillary growth.
- Occurs 1–3 months after viral transmission.
- Management: Excision, must treat anogenital lesions.

6.58 Focal Epithelial Hyperplasia (Heck's Disease)

- Numerous nodular soft tissues masses, distributed over the mucosal surfaces, especially buccal mucosa, labial mucosa, tongue—asymptomatic. HPV13 related.
- Management: Can spontaneously regress, reassure. Consider excision of any bothersome lesions.

6.59 Keratoacanthoma

- Benign lesion: sun exposed skin—rarely mucous membrane.
- Red macule = firm papule, increase rapid over 4–8 weeks to firm hemispheric elevated nodule with central ulceration plug, may spontaneously regress.
- Management: Surgical excision no recurrence.

6.60 Proliferative Verrucous Leukoplakia

- Field cancer development phenomenon
  - White, irregular, asymptomatic—slow growth.
  - Large: Verrucous appearance.
  - Eventually, malignant change.

Treatment

- Surgical excision: Lasers—rapid recurrence (not recommended).
- Chemoprevention
  - Beta-carotene 30 mg QDS.
  - Fluconazole 100 mg OD for 2 months.

6.61 Verrucous Carcinoma

- Superficial spreading, non-metastasizing, non-invasive type papillary and exophytic cancer.
- Some consider variant of SCC.

Clinical

- age 50 M = F often arise from PVL.
- Buccal/gingival/tongue.
- Investigations: Biopsy—deceptively benign microscopic pattern—absence cellular atypia.

Management

- Surgical excision 1–1.5 cm margins.
- If too extensive—radiotherapy.

Prognosis

- Cure rate 80, 6% with XRT.

6.62 Verrucous Xanthoma

- Uncommon benign, wart-like = HPV related.
- Common in oral mucosa—genital lesions.
- Histopathology: Lipid laden foamy histiocytes.
- Management: Surgical excision.

6.63 Pigmented Lesions

I. Congenital: Physiological. Melanotic macule, naevus, melanotic neuroectodermal tumour (MNET).
II. Acquired
    - Trauma: Smokers, post-inflammatory hyperpigmentation.
    - Infection.
    - Neoplasm: Melanoma, Peutz-Jegher.
    - Metabolic: Addison's.
    - Iatrogenic:
        - Local: Amalgam.
        - Generalised:

            Drug induced: Minocycline/chloroquine/amiodarone.
            Heavy metal: lead/mercury/arsenic/bismuth.

6.64 Melanotic Macule

- Flat brown—black mucosal pigmentation.
- Management
  - Single: excision biopsy.
  - Multiple: 1–2 biopsy confirmation.
  - Addison's: decreased adrenal function

    Decreased BP.
    Addisonian crisis.
    Convulsions.
    Bloods—9am cortisol.

  - Peutz-Jegher: Autosomal dominant: polyps (increased risk of cancer), pigmentation on lips.

6.65 Melanocytic Naevus

- Pigmented lesion composed of naevus cells <5 mm papule, most common palate.
- Histopathology
  - Junctional (epithelium-CT junction).
  - Intra-mucosal/dermal (in CT).
  - Compound (both epithelium and CT).
  - Blue (spindle shaped cells in deep CT).
- Management: Excisional biopsy.
- Prognosis: Malignant transformation highly improbable.

6.66 Smoker's Melanosis

- Focal increase in melanin pigmentation in the oral mucosa, not dysplastic or premalignant but indicated patient increased risk of SCC.
- Management: Lesion suggestive melanoma—must biopsy
  - Induration.
  - Thickening.
  - Ulceration.
  - Darkening.

6.67 Amalgam Tattoo

- Grey/blue—irregular borders, macular lesions, stable; occasionally XR can confirm metallic nature—often particles too fine for XR, biopsy if unsure or lesion changing.
- Mucosal Melanoma.
- Invades/spreads/metastasizes more quickly than cutaneous melanoma—poorer prognosis.
- Clinical features
  - Palate > gingival, lip, FOM.
  - Superficial spreading vs. nodular ± satellite lesions.
  - ABCDE (Asymmetrical, border irregular, color variation, diameters enlargement).
- Management: WLE ± prophylactic neck dissection.
- Prognosis: 5 years 20% survival.

6.68 Melanotic Neuroectodermal Tumour in Infancy

- Benign neoplasm of neuroectodermal origin: almost always from anterior maxilla.
- Emerge during first year of life as blue/black gingival mass, growly rapidly—usually around primary central incisors.
- XR: irregular resorption of anterior maxilla and displacement of tooth buds.
- Investigations: Incisional biopsy.
- Differential diagnosis: Neuroblastoma/sarcoma/hemangioma/congenital granular cell tumour.

Management

- Excision 2.5 mm margin including overlying margin, maxillary resection not necessary.
- Scarring may restrict maxillary growth.

6.69 Orofacial Granulomatosis

- Idiopathic.
- Crohn's disease, sarcoidosis.
- Hypersensitivity—Sodium benzoates (toothpaste)/cinnamon.
- Mycobacteria not like angioedema = very shiny with fluid inside lips.

- Melkersson-rosenthal syndrome: Neurological disorder—facial paralysis/swelling of the lips/folds in tongue.
- Uncommon: presenting with diffuse enlargement of lip/cheeks/face, linked to abnormal immune reaction.

Clinical

- Intermittent or permanent enlargement lips/cheeks/diffuse facial swelling (angular cheilitis/aphthous ulcers).
- Intraoral: oedema, ulcers, hyperplastic palatal tissues, cobblestone mucosa, mucosal tags, staghorning.

Investigation

- Biopsy: Buccal down to underlying muscles (must include), non-caseating granulomas/oedema.
- Patch test: allergens.
- Bloods: FBC/haematinics/albumin/ESR/calcium/ACE.
- CXR—TB.
- Gastro referral if abdominal symptoms/malabsorption.
- GIT symptoms: mucosal tagging, cobblestone, hyperplastic gingivitis.

Epidemiology

- Any age, usually teenagers/young adults.
- Related to sarcoidosis/Crohn's.

Treatment

- Exclusion diets.
- Intralesional steroids.
- Surgical reduction—comes back.
- Topical corticosteroid if oral ulceration.
- Fexofenadine to reduce lip swelling—autoimmune.
- DMARDs and biologics e.g. Infliximab anti-TNF depending on aetiology.

Crohn's Disease

- Chronic granulomatous disease of unknown cause that may affect any part of the GIT from mouth to anus.
- Diagnosis: young adults, often positive family history.

Clinical

(a) Oral

- Cobblestone buccal mucosa with mucosal tags and folds.
- Oral ulceration, diffuse swelling lips and cheeks.
- Raised, granulomatous gingival lesions.
- Staghorning floor of mouth.

(b) GIT

- Similar appearance thought out GIT with cobblestoning, scarring, ulceration, infection, obstruction, fistulae.

Investigations

- In conjunction with gastroenterologists.
- Stool samples, sigmoidoscopy, colonoscopy and biopsy
  - Non-caseating granuloma with macrophages, epithelial cells and occasional giant cells.
  - Barium enema.
- Treatment.
- Topical steroids may control oral lesions, usually systemic (steroids/DMARD e.g. azathioprine/biologics).

Sarcoidosis

- Systemic granulomatous disorder of unknown aetiology.
- Young adults/Afro-Caribbean F > M.

Clinical

- Bilateral hilar lymphadenopathy, pulmonary infiltrates, enlarged salivary glands, xerostomia.
- Cervical lymphadenopathy, erythema nodosum (skin), anterior uveitis.
- Oral: rare, erythematous submucosal nodules/OFG.

Investigations

- CXR, labial gland biopsy—non-caseating granuloma in 60%.
- Bloods: increased ESR, ACE, hypercalcaemia, hyperproteinemia.

Management

- Can resolve spontaneously over a few months or years.
- Steroids, immunosuppressants.

6.70 Heerfordt Syndrome

- Acute presentation of sarcoidosis.
- Sarcoidosis + uveitis/parotitis/CN VII nerve palsy/lacrimal gland swelling.

6.71 Melkersson-Rosenthal Syndrome

- Rare: neurological disorder, recurring facial paralysis/swelling of face and lips (usually upper + development of folds and furrows on tongue).
- Cause: Unknown, genetic predisposition.
- Symptoms overlap with other conditions such as sarcoidosis and Crohn's.
- Management: Symptomatic—NSAIDs, steroids—pressure on nerve by tissues, antihistamines, immnosuppressants.

6.72 Angioedema

Hereditary

- Deficiency in C1-esterase inhibition—leads to uncontrolled complement activation.
- Decreased C4—diagnostic test.
- Minor trauma—oedema to head and neck.

Drugs

- ACE inhibitors and calcium channel blockers—cause similar angioedema.
- Allergic—IgE mediated acute hypersensitivity reaction (uncommon).
- Gross swelling lip/face/neck—increased IgE/history of allergy—0.5–1.0 ml of 1: 1,000 adrenalines by intramuscular injection, repeat every 15 min.

Chapter 7
Trauma

Abstract An overview of facial trauma including its assessment, imaging, management and surgical techniques.

Key points

- ATLS and initial management.
- Initial assessment of maxillofacial trauma.
- Imaging.
- Management of bleeding.
- Ocular injuries.
- Management of retrobulbar haemorrhage.
- Tetanus.
- Examination of maxillofacial trauma.
- Soft tissue management.
- Nerve injury.
- Eyelid injury.
- Ear injury.
- Approaches to the facial skeleton including periorbital and coronal.
- Management of mandibular fractures.
- Management of zygomatic complex fractures.
- Management of orbital fractures.
- Management of frontal fractures.
- Management of nasal and nasoorbitoethmoidal fractures.
- Management of midface fractures.
- Management of panfacial fractures.
- Cerebrospinal fluid leaks
- Dentoalveolar injuries.
- Paediatric trauma notes.

ATLS

- Systemic approach: Primary survey
  - Airway and cervical spine control.
  - Breathing.

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Table 7.1 Shock classification

	Class I	Class II	Class III	Class IV
Blood loss (ml)	Up to 750	750–1300	1500–2000	>2000
Blood loss (%)	15	15–30	0.75	>40
Pulse	<100	100–120	120–140	>140
Blood pressure	N	N	Decrease	Decrease
Respiratory	14–20	20–30	30–40	>35
U/O	>30	20–30	5–15	Negligible
CNS	Slight anxious	Mild anxious	Confused/anxious	Confused/lethargy
Fluids	Crystalloid	Crystalloid	Crystalloid/blood	Crystalloid/blood

  - Circulation with control of external haemorrhage
  - Disability: brief neurological exam.
  - Exposure/environment of patient, prevent hypothermia.

- Secondary survey.
- Indications for indefinite airway placement in the trauma patient:
- Unconscious, severe maxillofacial trauma, risk of aspiration (bleeding/vomiting/risk of obstruction—neck haematoma/laryngeal/stridor/BURNS).

Benefit of ET Tube

- Difficult airway, support ventilation, means of providing pulmonary care, prevention of aspiration.
- Trauma series x-rays, C-spine/AP chest/AP pelvis—portable.

Surgical Airway

- Adult 12–14: 30–45 min build-up CO_2 = 1 s on 4 s off-passive expiration.
- Child: 16–18.

Shock

- CO = HR x SV.
- Intravenous fluid Ringer's lactate/normal saline. Bolus 1–2 L for adults, 20 ml/kg for paediatrics.
- Urine output: 0.5 ml/kg/h—adults; 1 ml/kg/h—child >1 year.
- Inadequate organ perfusion and tissue oxygenation—hypovolemia is commonest cause of shock in trauma patients.
- Classification.
- Cardiogenic shock (Table 7.1)

  - Defect in cardiac function (i.e. Acute MI), sympathetic response.

Neurogenic Shock

- Loss of descending sympathetic control on smooth muscle in vessel walls: severe CNS/spinal cord injury.
- Decrease peripheral vascular resistance, decrease blood pressure, CVP monitoring to avoid fluid overload.
- Atropine/vasopressors used.

Chest

- Tension pnesumothorax: air enters pleural space, flap-valve mechanism prevents its escape, total lung collapse, shifts mediastinum to opposite side, decreased venous return, full cardiac output.
- Immediate decompression: 2nd intercostal space/mid-clavicular line.
- Open pneumothorax: need occlusive dressing, taped 3 sides only—let air escape during exhalation.
- Flail chest: monitor O_2/may relieve with intubation.
- Cardiac tamponade—Beck's triad
  - Muffled heart sounds.
  - Increased venous pressure: distended jugular veins.
  - Hypotension—decreased arterial pressure (FAST SCAN)—focused assessment sonograph.

GCS Score

- Eye opening
  - 4: Spontaneous.
  - 3: To speech.
  - 2: To pain.
  - 1: No response.
- Verbal
  - 5: Orientated.
  - 4: Confused conversation.
  - 3: Inappropriate words.
  - 2: Incomprehensible sounds.
  - 1: No response.
- Motor
  - 6: Obeys commands.
  - 5: Localises to pain.
  - 4: Withdraws from pain.
  - 3: Flexion to pain.
  - 2: Extension to pain.
  - 1: No response.

- Severe head injury: GCS < 8—ET tube.
- Moderate head injury: GCS 9–13.
- Mild head injury: GCS 14–15.
- Epidural 0.5%—biconvex herniation outside dura, arterial (middle meningeal artery).
- Subdural 30%—tearing small surface vessels

  – Guidance on head injury.

- Urgent CT scan if

  (a) GCS < 13 when first assessed.
  (b) GCS < 15 2 h after injury.
  (c) Suspected open/depressed skull fracture.
  (d) Sign of skull base fracture (hemotympanum, panda eyes, CSF leak from nose/ear, Battle's sign).
  (e) Post-traumatic seizure.
  (f) Focal neurological deficit.
  (g) >1 episode of vomiting.
  (h) Amnesia of event >30 min before impact.

- If amnesia/LOC since injury, and have other factors

  (a) Age >65.
  (b) Coagulopathy: bleeding/clotting/warfarin.
  (c) Dangerous mechanism of injury (cyclist/ejected from car/stairs >1 m/5 steps)
    i. Request CT scan, can admit and do CT scan.
    ii. Monitor GCS every 30 min until the following morning.

- Children

  – LOC >5 min witnessed—amnesia (ortho/retro), lashing >5 min.
  – Abnormal drowsiness, 3 or more episodes of vomiting, clinical suspicion NAI.
  – Post-traumatic seizure (no history of epilepsy/suspicion of depressed fracture of tense fontanelle.
  – Signs of skull fracture/focal neurological deficit/dangerous mechanism.

Medical Therapies

- Intravenous fluids: maintain normovolemia, not glucose—normal saline/Hartmann's—monitor increased sodium (damage).
- Hyperventilation: decreased pCO_2, cerebral vasoconstriction, only in moderation for brief periods.
- Mannitol: Decreases ICP, 20% solution 0.25–1 g/kg I.V. bolus.
- Steroids: not recommended.
- Anticonvulsants: phenytoin loading dose 1 g I.V. 50 mg/min—maintenance 100 mg/8 h.

(1) Corticospinal tract

- Posterior-lateral segment of core, controls motor power of ipsilateral side of body.

(2) Spinothalamic tract

- Anterolateral part of cord: temperature/pain from contralateral.

(3) Posterior columns

- Proprioception/vibration/light touch from ipsilateral side.

- Spinal shock—loss of reflexes and fluidity.
- Classic syndromes
  - Central cord syndrome: greater loss motor in arms/compared to legs.
  - Anterior cord syndrome: paraplegia and loss temperature/pain sensation.
- C1 burst fracture: Jefferson fracture.
- C2 fracture: Hangman's (bilateral fracture in the pars interarticularis of the C2).
- C2: odontoid peg.
- C-spine: lateral C7 and T1 needs to be seen, open mouth odontoid view.

Burns: Thermal

- 1st degree: Erythema, no blisters.
- 2nd degree: Partial thickness burn.
  - Mottled/erythematous: swelling/blisters—painful.
- 3rd degree: Full thickness burn.
  - Skin dark/mottled/surface does not blanch: Dry/painless.
- Rule of 9.

Paediatric Trauma

- Hypoxia is the most common cause of cardiac arrest in the paediatric patient.
- Anatomy: tongue/tonsils relatively large in oral cavity—visualisation more difficult.
- ET tube best method of airway, if not possible or bag mask not working then consider needle cricothyroidotomy. Not surgical cricothyroidotomy for small children but >12 years could consider surgical.
- Shock not as easy to pick up in children.
- Fluid resuscitation—20 ml/kg/bolus; urine output 1–2 ml/kg/h.
- Normal fluid requirement—Estimated weight = 2 × (age +4) (Table 7.2).

Table 7.2 Paediatric fluid requirements

Body weight	Fluid in 24 h
1st 10 kg	100 ml/kg/24 h
2nd 10 kg	50 ml/kg/24 h
For rest kg	20 ml/kg/24 h
25 kg baby	1.6 L in 24 h (1000 + 500 + 100 ml)

7.1 Maxillofacial Trauma

Initial Assessment

- Medical history/social history.

Injury Factors

- Sharp/blunt.
- Energy transfer of trauma—RTA/kinetic energy.
- Contamination of wounds.

ATLS

- Airway
  - Debris obstructing airway: blood/teeth fragments.
  - Oedema—pharyngeal tissues.
  - Grossly displaced mandibular fracture: lack of tongue support—obstruction of airway.
- Circulation
  - Bleeding—maxillary artery/pterygoid plexus (venous—displaced maxillary fracture).
  - Penetrating neck injury: carotid.
- Head injury
  - Cerebral injury frequently related to head injury—GCS/alcohol = CT scan.
- Sight
  - Orbital trauma: ocular injury—VA assessment.
  - Reduced vision: intraocular injury, primary optic injury, increased pressure in orbit (retrobulbar haemorrhage).

Post-trauma Priority

- Life or sight—treat immediately.
- Facial lacerations/unstable mandibular fracture within 24 h.
- Others on delayed basis: facial oedema—treatment 24–48 h or after.

Imaging

- Often immobilised.
- OM views/OPG/PA—need patient to be upright, consider CT 0.5 mm slices.
- Facial bleeding.
- Sitting up to decrease venous pressure and reduce bleeding.
- Most facial bleeding controlled by direct pressure.
- Torrential haemorrhage from midface fracture
  - Anterior/posterior: nasal padding.
  - Prop impacting maxilla against skull base—compress (wired to impact maxilla).

Bleeding from Neck

- Zone 3: angle of mandible to base of skull—difficult to access surgically.
- Zone 2: Mid portion, cricoid cartilage to angle mandible.
- Zone 1: Thoracic inlet to cricoid cartilage (highest mortality).

External Haemorrhage

- If haemorrhage external then local temporary measures, such as isolating blood vessel wall.
- Bleeding from major vessels needs prompt surgical exploration.

Concealed Haemorrhage

- Penetrating trauma sharp implements—knives can cause internal bleeding from damage without sigs of external haemorrhage = neck swelling (fatal).
- Patient with signs of neck swelling or hemodynamically unstable: urgent neck imaging, patient with penetrating injuries to upper/and lower third of neck should have urgent imaging.
- Consider embolisation of branches: rich vascular supply. Ligation of external carotid does not always lead to stopping the bleeding.

Management Summary

- **Zone 3—**CT head, CT angiogram, direct laryngoscopy.
- **Zone 2**—Selective intervention if hard signs (shock, expanding haematoma, airway compromise, stridor, extensive oedema), or CT, OGD, bronchoscopy.
- **Zone 1**—CXR, CT angiogram, OGD, bronchoscopy.

Ocular Injuries

- High proportion with orbital injuries will have ocular injuries—essential to have basic assessment.
- Conscious patient: VA with Snellen chart, count fingers, see hand movements.

RAPD

- Relative apparent pupillary defect: indication of damage to visual system if patient unconscious.

- Swinging light test: swing torch from normal eye then to affected eye results in bilateral pupillary dilation (Marcus Gunn pupil) = damage to retina/optic nerve.

Direct Examination

- Hyphema: blood in the anterior chamber.
- Irregular pupil: sign underlying ocular injury.
- Constricted pupil: opiates (check drug history).
- Dilated pupil: optic nerve/retinal/cocaine abuse.

Retrobulbar Haemorrhage

- Acute orbital compression syndrome as a result of an intraconal bleed.
- Bleeding can be from infraorbital arteries or one of its branches, commonest cause is orbital trauma or complications of orbital surgery (0.3% following ZMC surgeries).
- Haemorrhage causes increase in pressure within the orbit: decreased blood flow to retinal artery causes irreversible vascular changes within optic nerve and leads to reduced vision.
- The optic nerve does provide some protection to the central retinal artery. Other vessels do not have protection = posterior ciliary arteries, prepapillary choroid arteries.

Clinical Features

- Severe pain not resolved by analgesia = earliest sign.
- Reduce visual acuity: red vision goes first, red spot to check.
- Proptosis tense/hard eye.
- Ophthalmoplegia: no eye movement.
- Retinal signs: papilloedema, cherry red macula, pale optic disc.
- Loss of direct light reflex, but preservation of consensual RAPD.
- If in doubt globe tension, measured by Tonopen.
- Post-operative observations to check
  - Pupil size/light reflex/pain/visual acuity.
  - Every 15 min for 2 h.
  - Every 30 min for 4 h.

Referral Based on the BAD ACT Criteria

- B = Blindness.
- A = Amnesia.
- D = Diplopia.
- A = Acuity reduced or impaired vision at any stage of treatment.
- CT = Comminuted trauma in the midfacial region.
- Scoring system with referral advised based on score and incorporating vision, zygomatic fracture type, motility, amnesia.

Management

- Medical to decrease intraocular pressure
  - Mannitol 20% 2 g/kg intravenously over 5 min—osmotic diuretic.
  - Dexamethasone 8 mg intravenously (alternatively methylprednisolone 100–1000 mg).
  - Acetazolamide 500 mg intravenous bolus, 250 mg QDS/6 h—carbonic anhydrase inhibitor.

Surgical Decompression

- Lateral canthotomy and cantholysis
  - Clean the area with sterile saline, inject local anesthesia into the lateral canthus.
  - Insert straight artery forceps from commissure of lateral lids to orbital rim and compress one minute to decrease bleeding.
  - Toothed Adson forceps to pick up commissure of lateral lid and insert sharp iris or scissors in horizontally to depth of lateral orbital rim and cut.
  - Identify lateral canthus with forceps and reorient scissors in vertical plane—cut canthal ligament so globe herniates out.
  - The superior limb may also need to be cut.
- Return to theatre—definitive decompression/haemostasias—also allow evacuation of haematoma, as well as perform a decompressive orbitotomy in anterior orbital floor.
- Canthotomy: Down middle cut lateral tendon, cantholysis (only lower limb release), may need decompression of the eye (conal).

Antibiotics/Tetanus

- Prophylactic, used in:
  - Contaminated soft tissue injury.
  - Mandible fracture compound to oral cavity.
  - Surgical emphysema.
  - Bite injuries: co-amoxiclav.
  - Prompt surgical debridement very important.
- Antibiotics not routinely given for CSF leaks.
- Tetanus.
- Absorbed tetanus vaccine
  - Given 0.5 ml intramuscular/deep subcutaneous = full course 3 doses 0.5 ml at intervals >4 weeks.
  - Not given if have received reinforcing dose in the preceding year.
  - Patient with impaired immunity may not respond—require anti-tetanus immunoglobulin.

Table 7.3 Tetanus immunisation assessment

Immunisation status	Wound type	
	Clean wound Clean incised wound, superficial graze	Tetanus prone/high risk Wound >6 h old, contact with soil/punctured wound, infected wound, devitalised tissue, animal human bites
Last of 3 dose with reinforcing dose with last 10 years	Nil	Nil
Last of 3 dose course/reinforcing dose more than 10 years	Reinforcing dose of absorbed tetanus vaccine	Reinforcing dose + Human Tetanus Ig (Consider—Human tetanus Ig if high contamination e.g. manure)
Not immunised or unknown	Full course of absorbed tetanus	Full course + human tetanus Ig

- Human tetanus immunoglobulins (Table 7.3).
  - Given 250 iu in 1 ml—if more than 24 h/heavy contamination then administer 500 iu.
  - Treatment of tetanus: metronidazole.
  - *Clostridium tetani*: gram positive anaerobic—tetanospasmin → neurotoxin, blocks skeletal muscle contraction (chest/neck/back).

Examination/Definitive Treatment

- Soft tissues: laceration—blunt trauma, incised wound by sharp object.
- Document: contamination, condition of margins/size/depth/nerve damage.

Bruising

- Mastoid: Battle's sign—skull base fracture.
- Periorbital: Raccoon eyes—base of skull/NOE/Le Fort II/III.
- NOE: Telecanthus, depression of bridge and elevation of tip (ICD-30–32 mm), interpupillary 60–65 mm.
- Vertical dystopia: Zygomatic/orbital floor fracture.

Ear

- Pinna: Laceration/haematoma, cauliflower (need drainage).
- Hemotympanum (skull base).
- CSF otorrhea.
- Bleeding from EAM can be caused by
  - Base of skull: Battle's sign co-exist.
  - Laceration of cartilaginous meatus—condylar fracture.
  - Bleeding from tympanic membrane—rupture.

Nose

- Internal/external.
- Internal
  - Septal haematoma: immediate drainage.
  - Septal deviation.

CSF Rhinorrhoea: Clear Fluid from Nose

- Beta-2-transferrin assay.
- Seen tramlining.
- Glucose test (not sensitive/specific >0.4 g/dL).
- Cribriform fracture = other liquids with CSF/retinal/anosmia.

Intraoral

- Bruising: sublingual haematoma (Coleman sign) = suggest fracture.
- Gingival lacerations: upper jaw—segmental fracture.
- Palatal bruising: split palate/Le Fort I.
- Occlusion: AOB—bilateral condyle, posteriorly displaced maxilla (Le Fort I).
- Lateral open bite
  - Condylar fracture on contralateral side to open bite.
  - Unable to protrude mandible with condylar fracture.

Sensory Changes

- Supraorbital/supratrochlear → rarely injured → neuropraxia.
- Infraorbital: orbital floor/ZMC fracture.
- Mental/inferior alveolar nerve: mandibular/parasymphysial fracture.

Trigeminal

- Von Frey—light touch.
- Wisp cotton from sides—corneal reflex.
- Jaw jerk reflex.

Soft Tissue Management

- Repair as soon as possible after injury, contaminated wounds need debridement within 24 h.
- Small wounds under local anesthesia.
- Larger wound under general anesthesia, especially for contaminated/deep wounds.

Foreign Bodies

- Contamination needs addressing gun powder close range—particles into dermis.
- Abrasions on road surfaces.
- Tattooing effect, if inadequate primary debridement, it would be impossible to correct after.

- Inspect for glass debris: weigh risks and benefits, removal or leave it if embedded deep.
- Wood can cause fistulas.
- Metal detectors for metal.

Parotid Duct

- Line from tragus: midpoint of upper lip and alar base, lies middle 1/3.
- Closely associated with buccal branch of facial nerve, often injured.
- Any deep facial wound crossing area, explore under general anesthesia ± magnification.
- Inspect wound for leak, retrograde insertion methylene blue, cannulate from mouth, proximal end more difficult, milk gland for assessment.
- Duct repaired over stent (passed via mouth—plastic intravenous cannula, leave inside for 7 days).

Facial Nerve

- Intratemporal course: intrabony injuries.
- Extratemporal course: penetrating neck/facial injuries.

House-Brackmann (Table 7.4) ***and Seddon and Sunderland*** (Table 7.5)

- Allodynia: pain in response to a normally non-painful stimulus.
- Analgesia: Not painful.
- Anaesthesia: No sensation.
- Causalgia: Spontaneous pain/burning sensation.
- Dysaesthesia: unpleasant abnormal sensation.
- Hyperaesthsia: increased sensitivity.
- Neuralgia: pain in the distribution of nerve.
- Hyperalgesia: increased pain in response to a normally painful stimulus.

Table 7.4 House-Brackmann grading system

I	Normal symmetrical function in all areas
II	Slight weakness noticeable only on close inspection Complete eye closure with minimal effort Slight asymmetry of smile with maximum effort Synkinesis barely noticeable, contracture, or spasm absent
III	Obvious weakness, but not disfiguring May not be able to lift eyebrow Complete eye closure and strong but asymmetrical mouth movement with maximum effort Obvious, but not disfiguring synkinese, mass movement or spasm
IV	Obvious disfiguring weakness Inability to lift brow Incomplete eye closure and asymmetry of mouth with maximal effort Severe synkinesis, and spasm usually absent
V	No movement, loss of tone/no synkinesis, contracture, or spasm

Table 7.5 Seddon and Sunderland nerve injury scoring

Sunderland	Seddon	Injury	Recovery potential
I	Neuropraxia	Ionic block; Possible segmental demyelination	Full
II	Axonotmesis	Axon severed: Endoneurial tube intact	Full
III		Endoneurial tube torn	Slow; Incomplete
IV		Only epineurium intact	Neuroma in-continuity
V	Neurotmesis	Loss of continuity	None
VI		Combination of above	Unpredictable

– Not all facial nerves require repair. If obvious muscle weakness and nerve injury proximal to a line down vertically from lateral can thus, primary nerve injury can be delayed since it may take several months after original injury for the motor end plates to atrophy.
– Nerve repair: Epineural, and perineural repair can yield good results, if segment missing then need nerve graft.
– Nerves: Four layers (Fig. 7.1)
  (1) Mesoneurium: CT sheath.
  (2) Epineurium: CT sheath, protects from mechanical stress.
  (3) Perineurium: Continuation of pia-arachnoid mater of CNS—diffusion barrier.
  (4) Endoneurium: Individual nerve fibre and Schwann cells.
  (5) Nerve fibres: Functional component—transmission across axon/Schwann cells with myelin sheath.

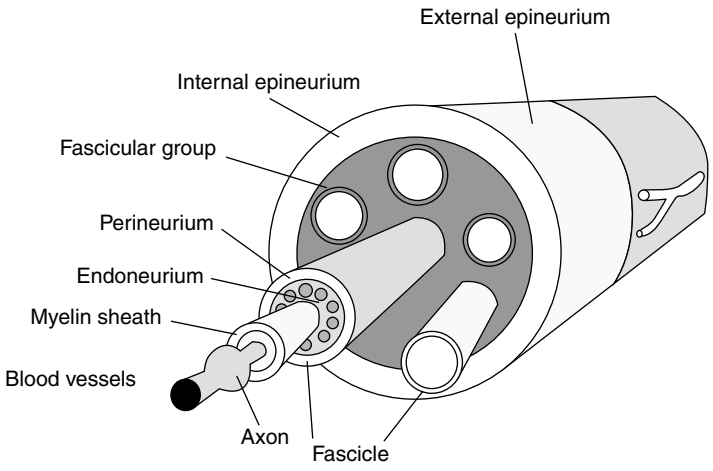

Fig. 7.1 Nerve anatomy

Response to Injury

(a) Proximal nerve trunk: back to last node of Ranvier.
(b) Distal nerve trunk: Wallerian degeneration, entire distal trunk shrinks, in time this becomes irreversible.
(c) Rate of axon advancement: 1–3 mm/day—regeneration is active invasion and displacement with axonal sprouting.

Other Tests

(d) Light touch—Alpha/beta fibres.
(e) 2-point discrimination: Alpha fibers (blunt)/Beta fibers (sharp).
(f) Thermal discrimination.
(g) Diagnostic nerve block.
(h) Facial nerve tests.
(i) Facial expression.
(j) Hyperacusis.
(k) Laceration.

Eyelids

- Marker for underlying injuries to globe, orbit, brain, local structures that can be damaged.
- Three areas managed with ophthalmic surgeon's input

  (1) Lacrimal apparatus: laceration medial canthal area

      – Preserve integrity of the lacrimal apparatus by cannulating a divided system with fine polyethylene catheter, or suture and repair with fine sutures.
      – Silicone tube: punctum of lacrimal sac → nasolacrimal duct → inferior meatus.
      – Silastic stent.
      – Monocanalicular injury: mini-Monoka stent
        Bicanalicular injury: Crawford stent

  (2) Levator apparatus within upper eyelid—check ptosis.
  (3) Corneal injury: frequently found in association with lid lacerations

      – Management of tissues loss depends on quantity of tissue loss, degree of contamination, site, damage to underlying structures, follow reconstructive ladder.

A. Primary injury

   - Dressings/healing with secondary intention.
   - Primary closure, delayed closure, skin grafts.

B. Secondary reconstruction

   - Tissue expansion.

- Local flaps.
- Distant flaps.
- Free flaps.

- Small areas: Allow to granulate, wound healing in 3 stages

  (1) Inflammatory.
  (2) Fibroplasia.
  (3) Remodelling in 3 weeks—maturation of fibroblasts and contraction of wound

    – Maturation of skin scars: up to 2 years, consider secondary scar revision.
    – Hypertrophic scars apparent within 2 months, massage, silicone sheet/gels may decrease scarring.

Ears

- Ear laceration/cut

  – Delayed/primary closure.
  – Rib graft, buried-2 stage pedicle.

- Auricular haematoma:

  – Mainly blunt trauma (cauliflower ear); extravascular blood replaced by fibrous tissue or new cartilage, also heal by secondary intention.
  – Management—pressure bandage/bolster after treatment to prevent reformation
    (1) Evacuation.
    (2) Needle aspiration.
    (3) Incision + drainage.

Ear Injury Classification

A. Laceration: Primary closure.
B. Avulsive

  (1) Avulsion of skin addressed by pedicle posterior auricular flap.
  (2) Full thickness tissue loss—primary closure with helical advancements, reduces auricular size.

Total Avulsion

- Simple attachment as composite graft.
- Sutured, small incision in skin to decrease venous congestion.
- Heparin to decrease blood viscosity.
- Success within 4 h post-injury

  – Difficult in adults to model cartilage framework/need more skin for tissue expansion/consider implant-based prosthesis.

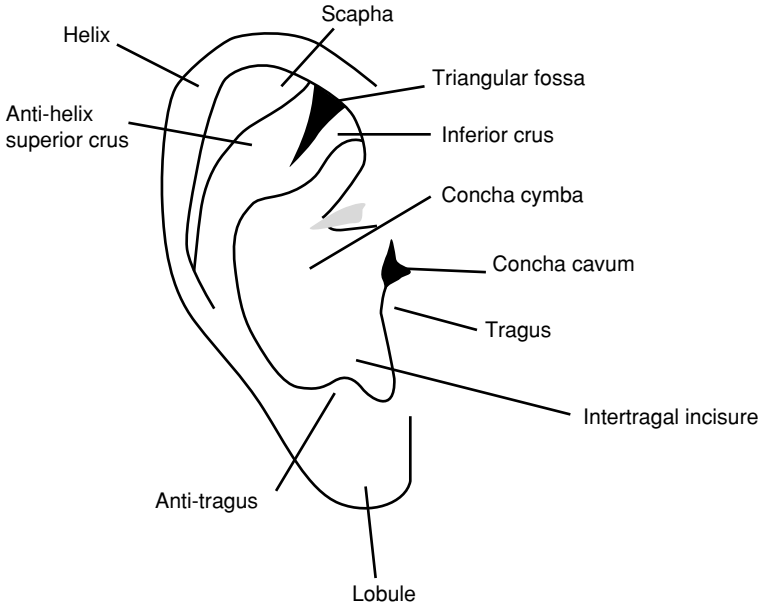

Fig. 7.2 Ear surface anantomy

External conchae blood supply (Fig. 7.2)
Superficial temporal

A. Superior auricular—anterior helix.
B. Medial auricular—tragus.
C. Inferior auricular—lobule.

Posterior auricular—cranial aspect of ear (superior/medial/inferior)

Blood supply.
Anterior: aurulotemporal, Arnold's nerve, greater auricular.
Posterior: Lesser occipital nerve.

- Lymphatic
  - Preaurciular.
  - Mastoid.
  - Parotid.

Approaches: Facial Skeleton

- Periorbital approaches.

(1) Transcutaneous
   - Lower eye lid

  - Subciliary.
  - Subtarsal (midlid).
  - Infraorbital.

- Upper eye lid
  - Blepharoplasty.

(2) Transconjunctival

  - With lateral canthotomy.
  - With transcaruncular extension.

(3) Coronal flap
(4) Endoscopic via Caldwell-luc (floor) or transnasal (medial wall)

Subciliary—Just Below Eyelid (1–2 mm)—3 Types of Flaps:

(1) Skin flap

- Dissect thin eyelid from level of incision to infraorbital rim.
- Orbicularis oculus and periosteum incision just below the rim.

(2) Skin muscle flap

- Incise skin and pretarsal orbicularis.
- Dissection superficial and tarsal plate, and orbital septum to rim.
- Orbicularis oculus and periosteum incised just below the rim.

(3) Step dissection

- Incision—subcutaneous dissection few mm (skin tent with skin hooks), then dissect through orbicularis oculus to periosteum—leaves same pretarsal muscle attached to skin and tarsal plate.

Complications

- Skin/septal buttonholes/darkening skin/ectropion/entropion.

Midlid/Subtarsal

- Scar more noticeable than subciliary but decreased incidence of scleral show and ectropion.
- Skin incision through skin and muscle in natural skin to depth of orbital septum.
- Tenotomy scissors used to dissect between orbital septum and orbicularis oculus (skin hooks to provide tension).
- Develop submuscular plane to rim through periosteum below infraorbital rim.

Transconjunctival (Fig. 7.3)

- Incision line 2–3 mm inferior to tarsus.
- Retract lower eyelid and incise with monopolar diathermy through conjunctiva.
- Preseptal approach used to avoid fat herniation into the operative field.

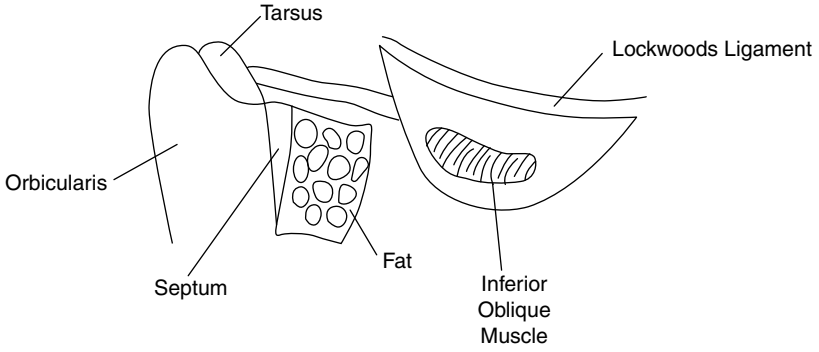

Fig. 7.3 Lower eyelid anatomy

(1) Lateral canthotomy

- Corneal shield—tip of iris scissors inserted into palpebral tissue to orbital rim.
- This transects the orbicularis oculus, orbital septum, lateral canthal tendon, and conjunctiva.

(2) Transcaruncular approach

- Assess medial wall, incision through conjunctiva anterior to semilunar line, and extend medially and superiorly just posterior to the caruncle.
- A retractor is placed to move globe laterally and one placed immediately posterior to the lacrimal crest.
- The medial wall periosteum is incised—subperiosteal dissection to expose medial wall.
- Care with anterior ethmoidal artery.

Advantages

- Hidden scar.

Disadvantage

- Risk of entropion.
- PRS 2001—Ridgeway: meta-analysis—incidence of lower eyelid malpositioned after facial fracture repair.
- Subciliary (ectropion 14%).
- Transconjunctival (entropion 1.5%—lid oedema).
- Subtarsal (hypertrophic scarring 3.4%).

- Suggested lateral canthotomy may reduce entropion as less lid traction: entropion required surgical intervention.
- Upper eyelid blepharoplasty approach.
- Excellent cosmetic/access = at least 10 mm above the upper eyelid, and 6 mm above lateral canthus.
- Incision through skin/orbicular oculus preseptal plane overlying FZ = cut through periosteum.
- Close in 3 layers, cover muscle sutures.

Coronal Flap

- Access to nasoethmoidal/medial wall fracture.
- Originates in hairline to mask scar, may be CI in men as prone to male pattern baldness.
- Cut straight/zig-zag/W-shaped across vertex of scalp
  - Subgaleal approach initially—through loose areolar tissue.
  - In the temporal region—the superficial layer of the superficial temporal fascia is split to 2 cm above the zygomatic arch (identified by interpositional fat that is found between the layers (protect branch of facial nerve).
  - Anteriorly pericranium incised (at level of hairline) and dissection (muscle deep to this).
  - This will maintain the integrity of sensation to the forehead.
  - At superior orbital margin—notch (may need osteotome).
  - Aid reflection of flap, a median cut out of the pericranium may be performed over nasal bridge.
- Surgical anatomy—coronal flap (SCALP).

(a) Galea: dense fibrous tissue (0.5 mm thick).
(b) Temporoparietal fascia
  - Arises from superior temporal line where it fuses with pericranium.
  - Fascia splits at level of superior rim—superficial layer to lateral border of zygomatic arch.
  - Deep layer of medial surface of zygomatic arch.
  - Superficial temporal fat pad in between.

(c) Temporal branch facial nerve
  - Innervates.

- Frontalis/corrugators/procerus.
- Crosses zygomatic arch 2 cm anterior to the anterior concavity of EAC (0.8–3.5 cm).
  - Enters frontalis no more than 2 cm above the level of supraorbital rim.
  - Usually has 4 terminal branches.
  - Pitanguy's line (Fig. 7.4).

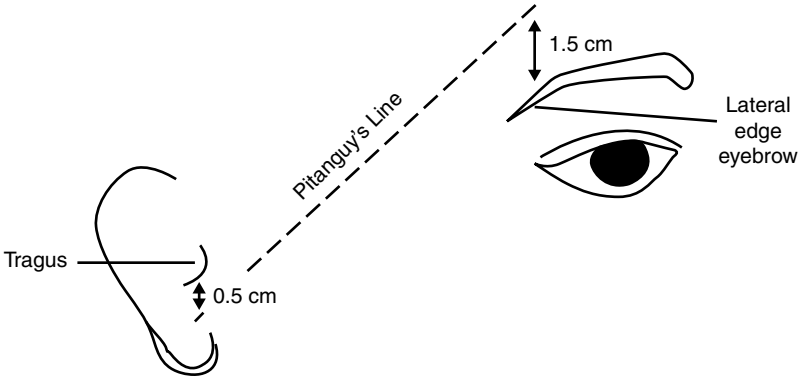

Fig. 7.4 Pitanguy's line

- Closure
  - Resuspension of the temporal fascia.
  - Close periosteum.
  - Raney clips.
  - Join with nylon/staples—suction drains.

- Complications
  - Bleeding, infection.
  - Temporal hollowing—atrophy fat pad, not resuspending, loss of blood supply to muscle.
  - Facial nerve injury, ptosis of brow due to inadequate resuspension.
  - Scar, alopecia, supraorbital/supratrochlear anaesthesia.

Tips: Tumescent solution, cutting diathermy, dry as you go, zig zag access, inferior extension

Mandibular Fracture

- Bone healing needs—reduction, fixation, immobilisation, if lack of any—fibrous union/non-union.
- Phases of bone healing

(1) Haematoma and acute inflammation.
(2) Osteolytic phase: Eburnation of bone ends and formation of granulation tissue.
(3) Osteogenic phase: Early callus and lamellar bone formation.
(4) Remodelling.

- Endochondral bone formation
  - Laying down of osteoid to fill gaps between bone fragments which undergo cartilaginous change, then replaced by bone.
- Membranous bone formation
  - Adequate mobility: Osteoid direct without cartilaginous precursor.
  - The use of rigid fixation to reduce amount of initial callus formation, allows osteoid to mineralise via membranous means.
  - More rapid formation of lamellar bone.
  - Lack of movement—not to disrupt growth of blood vessels.
  - Shield stressing: Bone does not remodel to full extent as not exposed to usual lines of force.
- Bone can heal via callus formation or by primary union.
- Primary union absolute rigid fixation across fracture with close approximation.

(a) Bone health via Haversian remodelling.
(b) Osteoclasts cross fracture.
(c) Osteoblasts follow and produce osteons—microscopic bony union.
(d) Further remodelling

- Compression across fracture site—one way of ensuring direct healing.
- Compression osteosynthesis—only use if absolute rigidity across fracture i.e. Lag screws in mandible.
- Mandibular fracture management has evolved with understanding of functional stress (Champy) and achieve best return to form and function with load bearing and load sharing principle (AO/SORG).

- Biomechanical principle (in craniomaxillofacial fractures management)

(1) Functionally stable occlusion.
(2) Atraumatic surgical technique.
(3) Accurate anatomic reduction.
(4) Early mobilisation

- Mandible: dense bone in the human body u-shaped with TMJ.

Classification

- Pattern of fracture—comminuted versus simple.
- Anatomical location: angle/parasymphysis/body.
- Degree of displacement/dislocation.

Management

(a) Goals are to restore function/aesthetics and prevent growth disturbance.
(b) Options

- Observation: Stable/non-mobile/non-displaced with compliant patient.
- Open reduction internal fixation: displaced/unstable fracture.

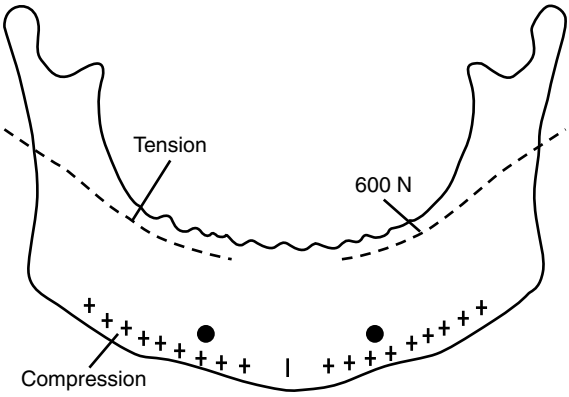

Fig. 7.5
Tension/compression lines mandible

Surgical

- Reduction/MMF.
- Fixation:
  - Type: Load bearing/load sharing.
  - Location: plates.
- Monocortical 2.0 diameter screw through outer cortex (avoid damage to roots, minimum 2 screws either side).
- All plates must be pre-bent to sit passively.
- Plates 5 mm apart.

Champy/Michelet 1973 (Fig. 7.5)

- Finite analysis of mandibles (see diagram).
- 1000 N canine/600 N molars.
- Line of maximal tension is more superior, focused around oblique ridge of mandible.
- e.g. Place 2 plates anterior—torsional forces, ideal line for osteosynthesis.

- Champy: Recommended to put patients in IMF post-operatively for 6 weeks, not done currently. Load sharing osteogenesis, relies on optimal fracture healing/conditions/bony contact, not recommended if:
  (a) Comminuted fracture.
  (b) Body of mandible fracture.
  (c) Multiple segments.
  (d) Multiple carious teeth.
  (e) Infected fracture (MRONJ/pathological).
  (f) Poor patient compliance/immunosuppression.

- Consider load bearing (AO) principle—different:
  - Rigid fixation with thick profile plate, along lower border to give maximal mucosal coverage/minimise risk of inferior alveolar nerve damage.
  - Bicortical fixation usually >2.0 mm screw, 3 screws each side.
  - Modern locking system.

(a) Eliminates micromovement between plate and screw—causes metal work loosening/failure.
(b) Requires less plate adaptation
  - Usually need transcutaneous approach.
  - Screw diameter 2.0 mm/drill is 1.5 mm diameter.

- Management of teeth in line with fracture, remove if
  - Pericoronal pathology.
  - Caries.
  - PDL.
  - Mobility.
  - Fractured roots.
  - Tooth preventing fracture reduction.
  - Delayed presentation.
- Fracture pattern
  - Angle.
  - Condyle.
  - Parasymphysis.
  - Symphysis.
  - 43% have associated other injuries: C-spine injury 11% (thorough history and examination to rule out).
- Non-union
  - Failure of healing between bony ends, causes:

    AVN—blood supply interruption.
    Unopposed fracture fragments.
    Infection.
    Mobile fragments.
    Soft tissue imposition (bone ends not in contact).

Malunion

- Healed but not in perfect/optimal position.
- Management: Osteotomy.
- **Edentulous/Atrophic**
- Atrophic less than 20 mm height.

- Blood supply: Endosteal/periosteal.
- Classification Luhr (1998)
  - Class I: 16–20 mm.
  - Class II: 11–15 mm.
  - Class III: <10 mm.

Challenges

- Reduce mandibular height.
- Fractured body: muscle pull greatest displacing fracture.
- Potential vascular compromise with significant periosteal stripping: reduced inferior dental artery contribution.
- Patient co-morbidities.

Treatment Options AO Principle

- Observation/soft diet/unfit for general anaesthesia.
- Closed reduction
  - Gunning splints with post-operative MMF using circummandibular wiring.
  - Patient's own denture—soft lining (gap at front for eating).

(1) Open reduction internal fixation

- Access—transoral/transcutaneous.
- Dissection—supra-endosteal versus supra-periosteal.
- Rigid fixation ± bone graft.

(2) External fixation: Pins

- A/O: external approach.
- Load bearing 2.4 mm locking plate if bilateral angle fractures.
- Can use 2.0 locking system if 2.4 too large.

(a) Plate needs limited adaptation.
(b) No pressure on bone.
(c) Reduce risk of screw loosening.

(a) Use template for plate bending.
(b) Reduce fracture with adaption plates to stabilise fracture before recon plate on.
(c) Bicoronal screw fixation.
(d) Can use autogenous bone to facilitate union (rib/tibia)

- Ellis (2008): 32 patients—Extraoral approach with locking system ± bone graft, no complications.
- Cochrane review (2008): Management of edentulous/atrophic mandible, no RCT found and could not give any recommendation.

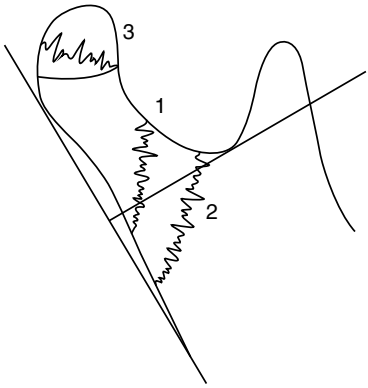

Fig. 7.6 Condylar fractures classification SORG

Condyle

- Classification: SORG/Loukota 2009 (Fig. 7.6)

  (1) High/condylar neck.
  (2) Low/condylar base.
  (3) Dicapitular fracture.

- Classification: Spiessl and Schroll (1972)

  – Type I: Undisplaced condylar neck.
  – Type II: Displaced low condylar neck.
  – Type III: Displaced high condylar neck.
  – Type IV: Fracture/dislocation—low neck.
  – Type V: Fracture/dislocation—high neck.
  – Type VI: Intracapsular fracture.

- Classification: Lindahl

  – Level of fracture (head/neck/subcondylar).
  – Displacement/angulation.
  – Relationship condylar head (displacement vs. dislocation).

- Zide and Kent: Indications for open reduction

  (A) Absolute

  – Inability to obtain adequate occlusion with closed.
  – Displacement condyle into middle cranial fossa.
  – Severe angulations of condyle, lateral extracapsular displacement.
  – Removal of fracture bone in joint (gunshot).

  (B) Relative

  – Bilateral fracture with comminuted midface fracture.

  - Bilateral fracture in edentulous patients when splint unavailable or impossible because of ridge atrophy.
  - Displaced fracture in medically compromised patients (e.g. seizure, psychotic, evidence of open bite or retrusion).

- Loukota: Current day practice fracture with deviation >10°, shortening of the ascending ramus >2 mm should be treated with open reduction. Open reduction provides better functional reconstruction than closed.
- Management

  (1) Conservative

  - Intracapsular fracture (early mobilisation to prevent ankylosis).
  - Most fractures in children (often intracapsular).
  - Undisplaced fracture or minimally displaced fracture with no malocclusion.
  - Edentulous patients.

  (2) Closed technique

  - Undisplaced/minimally displaced with malocclusion.
  - High condylar fracture where proximal segment too small to place meaningful internal fixation.
  - Displaced fracture in children.

- Arch bars/elastic traction (wire traction if bilateral)—2 weeks, 2 weeks light elastics.
- Disadvantages:

  - Reliant on good patient compliance.
  - Arch bars PDL damage.
  - Limitation of mouth opening (takes months to recover).
  - Requires secondary procedure to remove arch bars.

- Complications:

  - Occlusal abnormality.
  - Reduced range of mandibular movement.
  - Damage to teeth/PDL.
  - Risk of caries.

IMF

- Arch bars—Erich/preformed.
- IMF screws.
- Leonard buttons.
- Eyelet wires.
- Orthodontic brackets.
- Open reduction

  - Absolute and relative indications as before.
  - Three benefits: Direct visualisation/reduction (healing)/early mobilisation (normal functional).

Surgical Access

- Retromandibular approach (2 cm posterior ramus—most anterior just below earlobe), with transparotid access (most frequently used)—Hinds.
- Skin crease overlying posterior border of mandible.
- 0.5 cm below earlobe (incision 2–3 cm long), sharp dissection through platysma/SMAS/parotid capsule.
- Blunt dissection through parotid (parallel to direction of MM nerve) until mandible is visible.
- Dissect through pterygomasseteric sling onto mandible.
- Subperiosteal dissection to isolate fracture—channel retractor/sigmoid notch retractors.
- Fixation with 4-hole mini-plates, condylar plates, bicortical/lag screws.
- Closure in layers, can place suction drain.

Submandibular Approach

- 1.5–2.0 cm inferior to mandible, skin/platysma.
- Expose deep cervical/fascia and gland.
- Dissect deep cervical fascia (facial vessels).
- Dissection in this plane to lower border of mandible.

Pre-auricular Approach: At Junction of Facial Skin with Helix of Ear in Natural Skin Crease

- Anterior hockey stick may be used.
- Incision with scalpel to level of temporal fascia.
- Blunt dissection the area above the zygomatic arch and retract flap anteriorly (1.5–2.0 cm) at level of temporal fascia (outer layer, retract temporal vessels/auriculotemporal nerve).
- Develop flap in this plane inferiorly staying close to ear cartilage.
- Strip periosteum of arch.
- Continue at this level 1 cm below the arch—sharp dissection onto joint.

Other Approaches: Transmeatal Through EAM, Intraoral, Intra-Endoscopic

- Complications
  - Facial nerve injury: most studies reveal low rate—temporary/rare—permanently.
  - Salivary fistula, Frey's syndrome.
  - Risk of bleeding—maxillary arteries.
  - Loss of blood supply—avascular necrosis.
- Intracapsular fracture, avascular necrosis has been described.
- If occlusal discrepancies, would treat with elastic guidance.

- Blood supply
  - Transverse facial.
  - Superficial temporal.
  - Posterior tympanic.
  - Deep temporal (along LP).
- Literature Ellis (1998–2004) RCT: Patient with ORIF
  - Had greater mobility.
  - Less malocclusion.
  - Less shortening of posterior ramus length than closed.
  - Risk of cranial nerve VIII damage (17% for 6 weeks—0% at 6 months).
  - Hypertrophic scar 7%.
  - Salivary fistulas 2%.
- Oral Maxillofacial Surgery 2009—meta-analysis = no difference in open/closed.
- Children
  - <Age 12: few would operate, excellent potential for remodeling, 83% success with ORIF < age 14 (Lander et al. 2008).
  - Age 12–18: still have potential for growth, significant displaced fracture with medial displacement, consider surgery and need long-term follow-up.
  - Most condyles with minimal displacement = active exercise programmes.
  - Initial malocclusion: muscle spasm, usually settles.

Zygomatic Complex

Anatomy *(*Fig. 7.7*)*

(1) Frontozygomatic suture.
(2) Zygomaticotemporal suture.
(3) Zygomaticomaxillary buttress.
(4) Zygomaticosphenoidal suture
    - Goes into inferior orbital tissues.
    - Lateral orbital wall the most sensitive wall.
    - The most sensitive in detecting rotations
      - Zygomatic major/minor (muscle).
(5) Whitnall's tubercle
    - 11 mm below FZ suture.
    - Check ligament of lateral rectus muscle.
    - Lateral/canthal ligament.
    - Lockwood suspensory ligament.
    - Levator palpebrae superioris muscle.

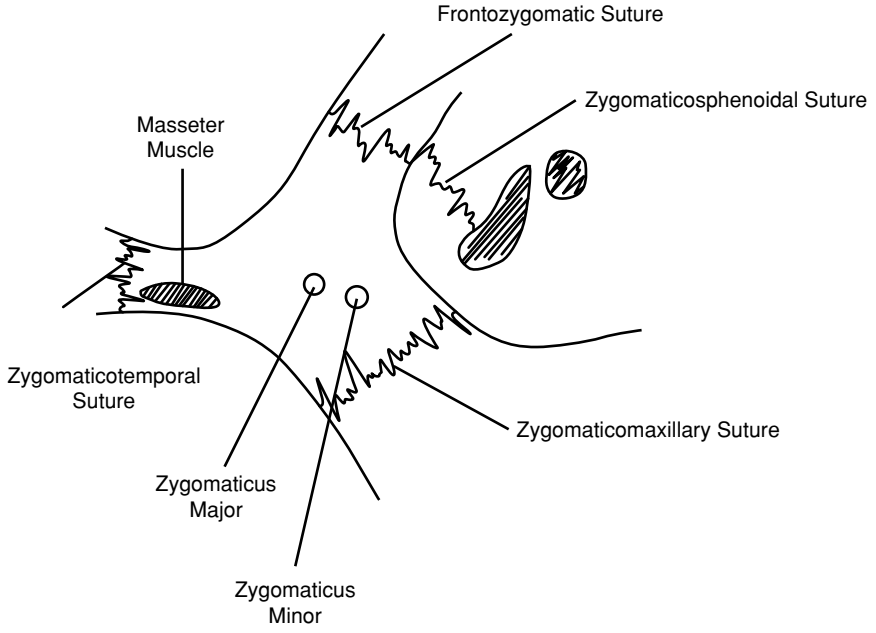

Fig. 7.7 Zygomatic complex

- Quadripod with lateral orbital wall the base and articulates with 4 bones—2 buttresses
  - Henderson's Classification:

    Undisplaced fracture at any site.
    Zygomatic arch fracture only.
    Tripod fracture—no destruction of zygomatic-frontal suture.
    Tripod fracture—distraction of ZF suture.
    Pure blowout fracture of orbital floor.
    Isolated orbital rim fracture.
    Comminuted fracture or other than any of the above.

- Clinical features or zygoma fracture
  - Pain.
  - Periorbital swelling.
  - Limitation eye movement.
  - Infraorbital nerve—paraesthesia.
  - Restricted jaw movement.
  - Flattened malar region.
  - Step rim of orbit.
  - Epistaxis.
  - Eno-/exophthalmos.
  - Hypoglobus (vertical ocular dystopia).

OM Radiographs

- Campbell's lines—4 lines of assessment of an occipitomental 10° view
  - Passes across frontozygomatic sutures, the superior margins of the orbits and the frontal sinuses.
  - Passes along the zygomatic arches, the zygomatic body, the inferior orbital margin and nasal bones.
  - Crosses the condyles, the coronoid processes, and the maxillary sinuses.
  - Across the mandibular ramus and bite line.
  - Trapnell's line along the inferior border of the mandible from angle to angle.
- Indication for surgery
  - Asymmetry of malar prominence—enophthalmos/exophthalmos/hypoglobus.
  - Unresolving diplopia.
  - Orbital deformity—underestimated: CT/ocular dystopia.
  - Restricted mouth opening due to impingement of coronoid process.
  - No firm evidence that reduction improves infraorbital nerve sensation.
- Treatment options—zygomatic arch fracture

  (1) Gillies approach (1923): Temporal incision (behind hairline in line with hair follicles)
    - Dissection to glistening deep temporal fascia, incise to explore temporalis.
    - Instrument: Rowe's elevator = elevate/rotate ZMC/reduce fractured arch.
    - Extraoral with J-hook/Poswillo hook—stab incision to articular eminence, tip hook under displaced arch and elevate.

  (2) Open reduction internal fixation
    - Standard of care last 25 years.
    - 1st stage: Reduce fracture—Gillies, intraoral Keen approach → upper buccal vestibular incision with subperiosteal dissection to beginning of arch, elevator used to elevate ZMC.
    - 2nd stage: 1, 2, 3-point fixation.
- Zygomaticomaxillary buttress—intraoral.
- Infraorbital rim via

  (a) Transcutaneous: subciliary, blepharoplasty, midtarsal, infraorbital.
  (b) Transconjunctival.
- FZ suture: upper lateral blepharoplasty, lateral eyebrow, lower lid transconjuntival, + lateral canthotomy.
- Complex/comminuted fracture: coronal incision—explore all buttresses.
- Single point fixation in selected cases, 2-point fixation suffice in most cases depending on the stability of the fracture.
- Complications: retrobulbar haemorrhage (RBH)/inability to reduce.
- Post-operative complications:

- Early: Reduced VA, restricted ocular mobility, RBH, trismus.
- Late: Malunion/non-union, enophthalmos, infraorbital nerve dysaesthesia.

Sequence

(a) Address FZ—re-establishment vertical height.
(b) Next if required repair arch.
(c) Repair lateral buttress—intraoral buccal approach.
(d) Assess rim with forced duction test.

Arch Isolated Fracture

(e) Suture external splint.
(f) Balloon inflation with Foley catheter.
(g) ORIF—periauricular.

Orbital Fracture

- Any part of the orbit both internal and external can be fractured. The commonest areas are the zygoma and inferomedial orbital wall.
- Isolated orbital injuries are termed blowout and blow-in fractures.
- Theories:
  - Hydraulic theory—Regan and Smith (90–95% present damage infraorbital nerve).
  - Buckling theory or ripple theory—Fujino.
  - Direct contact from globe.
  - Pure fracture of the floor

    Muscle entrapment—diplopia looking up.
    Muscle dysfunction—diplopia looking down.

- Impure fracture—thick orbital rim fracture.
- Investigation
  - Plain films/CT—1 mm axial cuts with coronal/sagittal reformat.
  - Orthoptic/ophthalmology assessment: Computer assisted pre-operative planning/navigation—especially post-traumatic enophthalmos.
  - Hertel exophthalmometer—measure globe position.
  - Hess Chart: pictorial record of ocular movement (for each eye/2 visual fields compared).
  - Patient wears red/green glasses in dark room, 50 cm—nine circles.
  - Patient asked to put green light in each of the points on the red light as illuminated.

- Indications for treatment.

(1) Enophthalmos

- Decreased orbital volume (normal orbital volume = 35 ml) of which globe occupies 6.5 ml.

- Degree of orbital volume change is most marked in area behind the globe = inferomedial portion, 1 cm^3 volume expansion correlates to 0.8 mm enophthalmos.
- True extent of enophthalmos revealed 2–4 weeks after injury, once initial swelling resolved.
- Clinically obvious >2 mm.

(2) Non-resolving diplopia

- Secondary to entrapment of muscles, diplopia is most common due to muscle contusion.
- Entrapment more common which results in diplopia on upward gaze.
- Muscle dysfunction usually results in diplopia looking downwards—gets better with time and hence delay repair.
- Orthoptic assessment very useful in distinguishing.

Diplopia

- Monocular: lens dislocation, retinal detachment, macular haemorrhage.
- Binocular: neurogenic, myogenic, mechanical.

Position of the Globe

- Enophthalmos: posterior position of globe.
- Hypoglobus: vertical position of the globe.
- Exorbitism: Anterior posterior globe = shallow orbit.

Surgical Tips (Fig. 7.8)

- Anatomical landmarks 'confluence of the orbit' Evans 2007.
- Infraorbital nerve, orbital floor, greater wing of sphenoid.
- Orbital plate of the palatine bone is rarely fractured, and solid bone usually encountered before reaching optic foramen.
- Optic canal: 40–45 mm posterior to normally positioned inferior orbital rim.

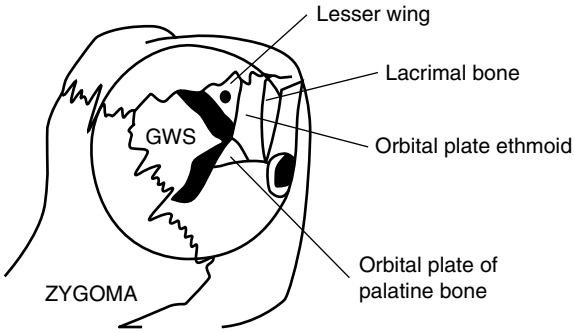

Fig. 7.8 Orbital anatomy

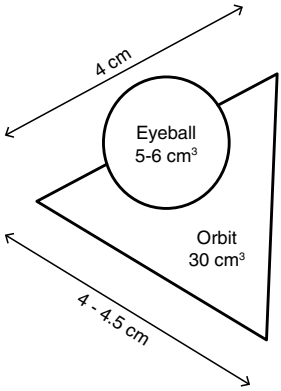

Fig. 7.9 Orbital distances

Orbital distances: anatomy (Fig. 7.9)

- Inferior rim to optic canal—48 mm.
- Inferior rim to inferior orbital fissure—12 mm.
- Posterior ethmoidal artery to optic ring—8 mm.
- Posterior lacrimal crest to anterior ethmoidal artery—20–25 mm.
- Anterior–posterior ethmoidal artery 11–15.

Evans concept of deep orbit

- Deep orbits start on anterior limit of inferior orbital nerve.
- Infraorbital nerve: runs parallel to medial wall and bisects inferior orbital fissure—following the nerve—takes towards orbital apex.
- Infraorbital fissure—contents safely divided.
- Just above and medial to the infraorbital nerve at its junction with the infraorbital fissure—orbital process of palatal bone.
- Not extend beyond this point, constant landmark.
- No reliable landmarks medial orbit—variety and position of ethmoidal arteries.
- Endoscopy assisted—has been described.

Reconstruction Materials

- Autogenous
  - Anterior maxillary wall, outer table calvarium, iliac crest.
  - Well tolerated, ultimately integrate.
  - Problems
    - Difficult to mold desired shape—3 dimensions—prone to resorption.
    - Increased length, operational donor site morbidity.
- Cartilage: Helix/nasal septum.

Alloplastic

- Non-resorbable—titanium.
- Various mesh/biocompatible/bend/shape 3D.
- Rarely requires removal.
- Polyethylene (Medpor).

Resorbable

- Polydioxanone (PDS) sheet: hydrolysis, can elicit inflammatory response.
- Complications—do forced duction test after operation
  - Loss of vision (0.03%)—haematoma/retrobulbar haemorrhage.
  - Worsening diplopia due to iatrogenic damage to the extraocular muscles.
  - Lid problems—oedema, ptosis, ectropion.
  - Enophthalmos, infections, change in vision.
  - Silicone—can migrate even with suture anchorage.

Paediatric Orbital Fractures

- White-eyed blowout fracture = main concern.
- Linear trapdoor orbital fracture causing impingement of periorbital/ocular muscles leading to extraocular mobility restriction ± oculocardiac reflex (hypotension ± vomiting).
- Rare, more common in patients age <18.
- Clinical features
  - Periocular trauma.
  - Minimal periorbital ecchymosis/oedema.
  - Marked restriction in ocular mobility.
  - ± Dizziness/nausea/vomiting/bradycardia/syncope

    Investigation: CT scan—fracture with entrapped muscle or periocular fat/soft tissue.
    Management: Urgent exploration, freeing the entrapped tissue and reconstruction to minimise muscle/fat necrosis.

- Orbital roof fracture
  - Often associated with other fracture—frontal bone/frontal sinus/ ± duct tear/CSF.
  - Tend to be associated with exophthalmos due to reduced orbital volume.
  - Trochlea of the superior oblique often damaged—diplopia.
  - Can get pulsatile exophthalmos due to brain vascularity (especially in children—indication for repair).
  - May require intracranial approach.

- Superior orbital fissure syndrome
  - Aetiology: Trauma (blunt)/infection/neoplasia/iatrogenic (good prognosis if treat conservatively, most cases recover in 3 months).

- Clinical

  V1: Anesthesia of the cornea, upper eyelid, nasal bridge, forehead.
  III, IV, VI—ptosis, ophthalmoplegia, fixed dilated pupil, consensual reflex acceptable.
  Difference between SOF and cavernous sinus thrombosis is loss of V2 sensation in CST.

- Management: intravenous steroids—identify and treat cause/ophthalmology. When displaced bony fragment is impinging on the SOF/orbital apex decompression should be considered.

Orbital Apex Syndrome

- SOF + visual loss due to damage to optic nerve.
- Optic nerve injuries—poor prognosis.

Frontal Bone Fracture

- Frontal sinus—only sinus not present at birth, appears at the 2nd year.
- 2 sinuses: equal and separated by bony septum.
- Pneumatic expansion at age 7 and completed age 18–20.
- Important relationship with anterior cranial fossa and orbit.
- Drains through an ostium at its lower medial corner (nasofrontal duct).
- Blood supply
  - Supratrochlear.
  - Supraorbital.
  - Anterior ethmoidal arteries.

Nerve Supply

- Supraorbital.
- Supratrochlear nerve.

Classification

- Anterior wall.
- Posterior wall.
- Floor.
- Combination.

Manolidis Classification (5 Types)

- Type 1: Anterior.
- Type 2: Anterior comminuted.
- Type 3: Anterior and posterior (not displaced).
- Type 4: Anterior/posterior—comminuted.
- Type 5: Severe comminuted.

Anterior Wall Defects

- Depends on the deformity or displacement, may be treated with open reduction internal fixation.
- Coronal flap: fragments are reduced (on/off table)—fixation with <1.5 mm screw.

Posterior Wall Injuries

- Associated with brain injury, CSF leaks, can have long-term risk of meningitis.
- All posterior fractures—discuss with neurosurgeon.
- Conservative: most CSF leaks stop with 7–10 days, if persists—consider operation.
- Open:
- Cranialisation—posterior sinus removed, brain allowed to herniate = pericranial flap.
- Can be interposed between brain/cranial vault, to reduce risk of infection, also repair dural continuity.
- Morbidity: craniotomy and neurosurgical input, can risk infection/recurrence/persistent CSF leaks. Superior to conservative management?
  - PRS—10-year follow-up frontal sinus fracture 2012—however only one year follow -up.
  - So, if chief indication is CSF leak, not more effective than conservative management.

Rodriguez* et al. *(PRS-2008)

- 857 patients/26 years—chief determinant of mucocoele formation was the status of the nasofrontal outflow (not posterior table displacement).
- Pre-operative imaging or intra-operative evaluation should decide if there is a need to obliterate sinus and prevent mucocoele.

Sinus Obliteration

- Mucosa removed and curettage from fractures lines and foramina.
- Duct plugged, sinus obliterated with fat/muscle/bone/cartilage/alloplastic (Tisseel) or undergo spontaneous osteogenesis.
- Separate nasal cavity to anterior cranial fossa.
- Evaluation NFD—fluorescence/methylene blue.

Floor Injuries

- Obliteration now largely superceded by functional endiscopic sinus surgery.
- Most say assess then obliterate.
- Duct damaged in NOE fracture: not lead to problem with sinus drainage—immense spontaneous reformation, argue not to attempt repair frontonasal duct.

Complications

- Intra-operative: neurological/bleeding/sagittal sinus.

- Post-operative.
- Early: infection (skin/sinus/meningitis/brain/neurological/CSF leak/ocular/neurosensory).
- Late: mucocoele/abscess/osteomyelitis/chronic pain/anosmia.

CSF Leaks (Post-operative)

- Lumbar drain, acetazolamide.
- Reoperate if >10 days.
- Chronic frontal sinusitis.
- Brain abscess—spread in foramen Breschet.

PRS—CHOI (2012)

- 10-year follow-up frontal sinus—vast majority managed conservatively without cranialisation—not address posterior wall (CSF resolved).
- CSF leak.
- Communication.
- Displacement
- Followed patients who met criteria, would do cranialisation for open injuries/grossly comminuted.

Nasal Fracture

- Common, treat acute if no swelling, or 10 days after.
- Radiology no role in assessment.
- Indications for treatment: Nasal deviation, cosmetic, nasal blockages (septal).

Surgery

- Closed reduction.
- If septum, then primary open septoplasty via intranasal incision.

Reduction

- Often impacted—may require disimpaction.
- Fixation via temporary/nasal splint.
- Complications—inadequate reduction secondary rhinoplasty (14–50%).
- Children—closed reduction until child completes growth—growth disturbance by disruption of septum and ANS.
- Septal haematomas—need acute treatment = drainage/nasal packing and antibiotics.

NOE Fracture

- Ethmoidal sinus—lies between orbit and nose, variable ethmoidal air cells (3–18 each with 1/5 own ostium).
- Lateral wall is paper thin, lamina papyracea.
- Anterior/middle ethmoidal cells: drain middle meatus.
- Posterior ethmoidal cells: drain to superior meatus.
- Fractures caused by high energy transfer, rare, difficult to treat.

Blood Supply (ICA/ECA)

- Supraorbital.
- Anterior/posterior ethmoidal.
- Sphenopalatine.

Nerve Supply

- Supraorbital.
- Anterior ethmoidal.
- Lateral posterior superior nasal.
- Orbital.
- Post-ethmoidal.

Clinical Findings

- Depression of nasal bone.
- Upturned nasal tip.
- Nasal fracture with telecanthus (ICD/IPD > 50%, suspect >35 mm, >40 high probability).
- Normal ICD—29–32 mm.
- Epistaxis/CSF/rhinorrhea/orbital rim steps.

Classification

- Markowitz
  - Central fragment intact.
  - Communication of major fragments to a fragment + ligaments attached to bone, large enough to stabilise.
  - As (2) but ligaments not attached.

Ayliffe Added I + S

- I: undisplaced.
- S: Requires grafting.

Grass

- Naso-orbital.
- Naso-orbital + central maxilla.
- Naso-orbital + Le Fort II, III.
- Naso-orbital + Dystopia.
- Naso-orbital + loss of bone.

Indications

- Significant nasal deformity or telecanthus, should be treated in a primary setting as secondary correction of facial deformity in this area is notoriously difficult.

Management

- Restore function and aesthetics via reconstruction of skeletal framework of the NOE unit stabilisation of IC width, and establish of nasal support.
- Consent pre-operatively should include grafting.
- Surgical access: usually coronal, or rim incisions.

  Type I/II: 3-point stabilisation with fixation at NP junction, infraorbital rim and piriform rim.
  Type III: Combination of microplates for large bony segments, wires for smaller segments and MCL suspension.

- MCL repositioning—precision: posterior superior to posterior lacrimal crest
  - Options: Transnasal wire, anchoring screws, cantilevered miniplates.
  - 2 drill hole (4–5 mm apart) at anterior position of canthal tendon insertion.
  - Ends of wire put in twisted—radius/stabilises fracture.
  - The NOE is then secured at superior orbital rim with miniplates.
  - Grafting of nasal dorsal (Type II/III—iliac crest/mandible.
  - If tendons partially avulsed,—reinforce with sutures to medial wall bone fragment.
- Outer table calvarial bone graft (4 $\times$ 1 cm)—contoured/rounded to support medial/distal third of nose.

Complications

- Inadequate repositioning of medial canthal ligament.
- Residual nasal deformity.
- Infection.
- NFD injury—most resolve.

- Medial Canthus
  - Anterior—stronger: inserts anterior lacrimal crest of frontal (process/maxilla) and posterior lacrimal crest of lacrimal bone.
  - Canthopexy wires.
- Ellis E. JOMFS 1993—Sequencing in NOE fractures
  - Exposure—usually a coronal flap unless contraindicated.
  - Identify of the tendon bearing fragment (either through the flap/transcutaneously).
  - Reduce/reconstruct medial orbital rims.
  - Reconstruct medial orbital wall.
  - Reduce septal fracture and displacements.
  - Transnasal canthopexy if required.
  - Dorsal nasal augmentation.
  - Soft tissue adaptation.

Midface Fracture

- Le Fort fracture levels: Classically described—used to describe the highest level fracture.

- Le Fort I
  - Pterygoid plates—anterior wall of antrum—piriform aperture of nose.
- Le Fort II
  - Pterygoid plates—anterior superior through infraorbital rim.
  - Into inferomedial orbital floor and runs across bridge of nose.
- Le Fort III
  - Craniofacial dysfunction.
  - Whole of midface separated from skull base.
  - Superior aspect of pterygoid plates, through lateral wall orbit.
  - Cut into FZ sutures and zygomatic arch as well as nasal bridge in the middle.

Indications for Treatment

- Occlusal abnormality: Lack of facial protection/width of face.
- Mobility of maxilla.

Surgeon's Access

- Upper buccal sulcus.
- Upper buccal sulcus—infraorbital—transcutaneous/transconjunctival.
- Multiple cutaneous or coronal—full exposure arch essential in presence of increase facial width/and lack of facial protection.

Management

- Fixation (miniplates) sequence.
- Zygomatic arch (width/facial projection).
- Stabilisation FZ suture—restore orbital height.
- Stabilisation inferior orbital rims.
- Reconstruction of the vertical buttress of maxilla.
- Anterior buttress—lateral piriform aperture.
- Posterior buttress—zygomatic buttress.

Complications

- Dish face deformity (inadequate reduction).
- Infection.
- Occlusal changes.
- Gruss and Mackinnon (1980)—vertical support pillars.
- Frontonasal.
- Zygomaticomaxillary.

- Pterygoid process/maxillary.
- Ramus + condyle.

Horizontal Facial Buttress

- Frontal bone.
- Zygomatic arch/body zygoma/infraorbital rim.
- Maxillary alveolus and palatal process.
- Mandible.

Panfacial Sequence ABC

- Tracheostomy/submental.
- Early.
- Fix mandible.
- Study models—impressions.
- Soft tissues—address primary/wash or glue.
- 10 days later—incisions (intraoral/transconjunctival/coronal).
- Arch bars.
- Top/bottom.

– Sequencing of Maxillofacial trauma management—Gruss et al. 1991.

(1) Restore the airway.
(2) Expose all fracture sites by planned incisions.
(3) Connect all incisions and lacerations by subperiosteal tunnelling.
(4) Reposition and stabilise the facial skeleton
    - Reconstruct projection of the face working anteriorly from the zygomatic arches.
    - Reconstruct the width by fixing to a stable area in the frontal region (ZF suture).
    - Recreate the NOE areas including orbital floors, medial canthi and nasal bridge.
(5) Restore the posterior vertical height by fixing condylar ramus fractures.
(6) Restore the occlusion via IMF if possible.
(7) Reposition and stabilise the mandibular fractures.
(8) Reposition and stabilise at the le fort I level.
(9) Soft tissue management, including defects and resuspension.
(10) Proper drainage (gaps greater than 5 mm should be primarily bone grafted).

Panfacial Fracture

- Fracture involving upper, middle and lower thirds of face.
- Facial buttress and key landmarks
  - 4 vertical: Nasomaxillary, zygomaticomaxillary, pterygomaxillary, ramus/condyle.
  - 4 horizontals: Frontal, zygomatic, maxillary, mandibular.

- Clinical assessment
  - ABCDE.
  - Facial fracture delayed often due to:
  - Life threatening injuries need management first.
  - Medically unstable for long operation.
  - Specifics: may need staged approach—secure airway.
- Management
  - Restore facial aesthetics/function (width/height/projection).
  - Pre-operative: impression/acrylic splint/arch bars.
  - Sequencing 'known to unknown'—bottom up/top down.
- Munson 1989—Bottom up/inside out
  - Tracheostomy.
  - Repair palatal fracture.
  - MMF: mandibular fracture, including condyles.
  - ZMC including arches, front sinus, NOE, maxilla, orbital reconstruction.
- Grass 1992—Top down (outside in)
  - Tracheostomy, frontal sinus, ZMC (including arch), NOE, maxillary (including palatal split), MMF condylar fracture, after mandibular fracture then orbital reconstruction.
- Complications
  - Post-operative:
  - Early: Blindness/CSF leak/bleeding/infection/telecanthus/facial asymmetry/enophthalmos/AOB/malocclusion.
  - Neurological, soft tissue
- Late: TMJ/ocular/nasal
  - CSF leaks.
  - CPP = MAP-ICP (5–15)—increase ICP to reduce cerebral perfusion.
  - 20-22 ml/h = 500 ml/day.
  - CSF rhinorrhoea/otorrhoea = communication between intracranial content and nasal cavity/Eustachian tube.
- Clinical evaluation
  - Direct result of compromise in the integrity of the dura that encloses CNS.
  - Beta-2 transferrin only found in CSF.
  - Headaches—alleviation of ICP.
  - CT.

- Neurosurgical involvement
  - Conservative—Bed Rest, no coughing/nose blowing/sneezing/straining.
  - Clemenza et al. JOMFS 1995—no benefit in prophylactic treatment.
  - Some believe antibiotics would increase risk of meningitis, alter normal flora.
  - Mincy (1996): 85% traumatic CSF resolved spontaneously, if conservative not working for 72 h, lumbar drain allows inflammatory response to repair leak.
- Complications—CSF fistulas, which may lead to meningitis
  - Repair.
  - Intracranial—direct visualisation, risk of invertebrate bleeding/brain oedema, repair with galea.
  - Extracranial—less morbidity: transphenoidal but intranasal route—temporalis fascia graft with fibrin glue.
  - Endoscopically repair—many reported successes, especially if defect <1 cm, address most orbital roof, cribiform plate, sphenoid defect.
- CSF
  - Beta-2 transferrin.
  - False positive beta-transferrin in perilymph and vitreous/aqueous humour.
  - Beta-trace protein.
  - Investigate with fluorescein and CT + cisternography.
  - Longer it persists more likely to need correction.
  - 35% resolve in 24 h, 85% in 7–10 days. If still leaking significant risk of meningitis.

Dental Injuries

- Ellis: Injury to dental tissues.
- Enamel.
- Enamel + dentine.
- Enamel + dentine + pulp.
- Root fracture.

- Injury to PDL tissues
  - Concussion.
  - Subluxation.
  - Luxation (lateral/extrusive/intrusive).
- Avulsed tooth
  - Deciduous: no treatment, leave it and monitor.
  - Permanent: Common in age 6–7 group, it is best to reimplant back into the socket immediately within 60 min, complications increased after 24 h

After one hour, most PDL cells non-viable.
Not possible to reimplant, then must store in milk to preserve the PDL for up to 3 h.
Gentle rinse with saline/saliva.
Flexible splint for two weeks with antibiotics.
Most would need RCT, exception in wide open apex.

- Complications
  - Pulp necrosis.
  - External root resorption.
  - Ankylosis.
- Dentoalveolar fracture
  - Fractured segment of alveolus.
  - Reduce/fixation with arch bars to correct occlusion for 4–6 weeks, may need endodontic treatment in future.
- Paediatric trauma notes.

- Epidemiology: Falls/different mechanism.
- Facial morphology: bigger cranium (less facial fracture).
- Consent issues.
- Anatomy: Airway (tongue)/physiology.
- Facial skeleton:
  - Elastic (greenstick fracture).
  - No frontal sinus (age 2).
  - Maxillary sinus small.
  - Mandible—mixed dentition.
  - Orbit.
  - Growth potential: condyle.

Chapter 8
Miscellaneous Topics

Abstract An overview of miscellaneous topics including research, audit and clinical governance, consenting patients, medical management of infections of the head and neck, anaesthesia and sedation, and airway management.

Key Points

- Critical appraisal of papers.
- Evidence-based medicine.
- Audit and research.
- Revalidation.
- Consent.
- Healthcare-associated infections.
- Use of antibiotics, antivirals and antifungals.
- Local anaesthetic, sedation and general anaesthetic.
- Surgical and non-surgical airway management.

8.1 How to Read a Paper

Evidence Based Medicine

- EBM use of mathematical estimates of the risk or benefit and harm, derived from high quality research on population samples, to inform clinical decision-making in the diagnosis, investigation or management of patients.
- Figures from population—individuals.

Websites

- Oxford Centre for Evidence Based Medicine—www.cebm.net.
- Clinical evidence: 6 monthly handbooks—best evidence for clinical decisions—BMJ.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_8

Searching

- Raw data bases—Medline.
- Pre-approved articles—Cochrane clinical trials register.

Levels of Evidence

- Ia: Systematic review or meta-analysis of RCT.
- Ib: At least one RCT.
- IIa: At least 1 well designed controlled study without randomisation.
- IIb: At least 1 well designed experimental study i.e. cohort Study.
- III: Well-designed, non-experimental descriptive studies, comparative, correlation, case control studies, or case series.
- IV: Case report.
- V: Expert/committee opinions, clinical experience.

Three Questions to Ask

(a) What was the research question and why was the study needed?
(b) What was the research design?

- Primary: Lab, clinical trial, survey, organisation, case study.
- Secondary: Systematic review, review meta-analysis (integrate numerical data from more than 1 study), economic analysis.

(c) Was the research design appropriate to answer the question?

Trial Designs

- RCT (random allocation): treatment arm versus placebo.

Cohort Study

- Two or more groups selected on basis of difference in exposure to particular agent and followed up.

  Usually for several years i.e. whether smokers/non-smokers develop lung cancer.

Case Control

- Patients with a particular disease are identified and matched with control—data collected via medical records/history on past exposure to a passive causal agent used for aetiology of disease, especially rare conditions—cannot demonstrate causality.

Cross-Sectional Study

- Representative sample recruited and then interviewed/examined
  - Example What is the normal height of a 3-year-old?.

Case Reports

What are the Steps Involved in Developing EBM?

- Identifying answer and clinical question.
- Assessing information which will help to answer question.
- Appraising the validity and relevance of the information available.
- Harnessing the information and applying it to everyday clinical practice.

Advantages of EBM

- Objective method of determining and maintaining high quality and safety standards.
- Facilitates transfer of clinical research into practice.
- Potentially reduces health crisis by improving utilisation of resources.
- Provides framework for problem solving and training.
- Improves computer library/data appraisal.

Research Ethics Service (RES)

- Each individual NHS Trust will have ethics committee that will consider in detail any proposal of research involving patients and healthy volunteers.

Medical Statistics

- Mean: [Sum of values/number of values].
- Median: average—point at which 1/2 values below, 1/2 above.
- Mode: Most frequent occurring event.
- Standard deviation: only for normally distributed data—indicates how much values vary from average.
- Confidence interval: Range at which we are confident true value lies
  - Example—Meta-analysis brings together results from similar studies to give average result.
- P-value
  - Null hypothesis (NH)—there is no difference between 2 tests.
  - P = probability that null hypothesis is true.
  - P = 0.05 means 5% chance NH is true or that 1 in 20 possibilities that result occurred by chance.
- T-test—parametric test
  - Compares two samples—tests probability that they both came from a population within the same mean value.
  - Chi-squared allows hypothesis of categorical data (e.g. no mean) as opposed to continuous data.

Table 8.1 Sensitivity and specificity

Disease test	Present	Absent
+ve	A	B-false + ve
−ve	C False −ve	D

- Risk ratio
  - Relative risk: used in prospective cohort studies only, over time, to investigate effect of treatment of risk factors.
  - Risk: probability an event will happen calculated by [number events /number of people at risk].
  - RR = 1—no difference.
  - RR > 1—risk of event increased compared to control.
- Odds ratio
  - Way of comparing patients with condition (cases) with those that do not (controls).
  - Calculated [number of times event happens/number of times it does not].
  - OR = 1—no difference.
  - OR > 1—risk of event increased compared to controls.
- Risk reduction and NNT (Table 8.1)
  - NNT—Number of treatments to get 1 benefit

    Sensitivity: Identifying those who have disease A/A + C.
    Specificity: Identifying those who do not have disease D/D + B.

- PPV—Positive predictive value: [A/(A + B)] if positive what is the chance that patient will have condition.
- NPV—Negative predictive value [D/(D + C)] if negative what is the chance that patient will have condition.
- True positive: those with a positive test have disease.
- True negative: those with negative test do not have disease.
- False positive: those with positive test result do not have disease.
- False negative: those with negative test who do have disease.
- Sensitivity test: have few negative, a specific test will have few positives.
- MRI

(a) Sensitivity 75–85% not very accurate—get false negatives (i.e. micro-metastases do not show).
(b) Specificity: 90–100%—few false positive.

- Incidence: Number of new cases over a given time.
- Prevalence: Existing number of cases of condition at a single point in time.
- Power: The probability that it would detect a statistically significant difference. If expected difference small, it would need larger study (power calculation).

  – Interpreting P leads to two types of errors

    Type 1 error: Rejecting a true null hypothesis (P-value problems).
    Type 2 error: Accepting a false null hypothesis—i.e. accepting no difference when they do actually exist = power of study.

8.2 Clinical Governance/Audit/Management

What is Clinical Governance?

A system in which NHS organisations are accountable for continuously improving the quality of these services and safeguarding a high standard of care, by creating an environment in which clinical excellence will flourish.

7. Pillars of Clinical Governance

(1) Clinical audit

- Quality improvement process that seeks to improve patient care and outcomes through systematic review of care against explicit criteria and implementation of change.
- Where individual changes are implemented and further monitoring is used to confirm improvement.

(2) Patient/public involvement.
(3) Clinical risk management.

- Systemic approach to improving the safety quality of healthcare.
- Critical incident reporting—near misses
  i. Go to quality assurance committee—determine one-off/or any that can be learnt.
  ii. Identify any pattern of behaviour that needs looking at in detail.
- Screen/auditing patient medical records.
- i.e. Reduce hospital acquired infections (NICE guidelines) HCAI—MRSA/C. diff).
- National Patient Safety Agent NPSA—Aim to put patient safety—top agendas, includes patient safety division which collects and analyses adverse incidents in NHS.

(4) Clinical effectiveness

- Extent to which specific clinical interventions do what they intend to do—"right thing, right way, at right time".

(5) Staffing and staff management.
(6) Education and training e.g. CPD.
(7) Information use to facilitate governance process.

National Institute for Health and Care Excellence (NICE)

- Independent organisation responsible for developing national guidance on the promotion of good health and preventative treatment.
- NHS—legally obliged to provide funding/resource for medicines and treatments recommended by NICE.

Revalidation

- Responsible officer: responsible to GMC, engaged in the process.
- Acting on behalf of designated body (organisation with duty to provide appraisal and support revalidation).
- SpR: Dean is responsible officer.
- Responsibility of Doctor—collect evidence. Whole practice including private work.
  - Audit.
  - CPD.
  - Appraisal.
  - MSF.
  - Patient surveys.
- NHS—Main base hospital provides responsible officer. Can only have one responsible officer.
- National clinical advisory service (NCAS), cannot suspend consultant until goes via NCAS.
- 18% new consultants:
  - Complaints (PALS)—response given.
  - Not resolved informally—investigate.
  - Still not satisfied—arrange review of complaint.
  - If still not happy with response—Health Service Ombudsman.
- NHS complaints (1st April 2009): 12 months from date of event or when becomes aware, limit can be waivered by manager, acknowledge within 3 days, resolve within 25 days.

- Negligence
  - Duty of care.
  - Causality.
  - Harm has happened.
  - Burden proof with claimant (standard of proof/balance of probability).

8.3 Consent

Recent Development

- Ms. B versus NHS Hospital (2002): Accident—tetraplegic/on ventilator—asked ventilator to be switched off, had capacity even if consequence was that lead to her death.
- Glass versus UK Government (2004): withheld decision that health professionals to override wishes of mother of severely ill child.
- Chester versus Afsar (2004): House of Lords—Failure to warn patient of risk of injury in surgery, however small the probability (tetraplegia). Advised all health professional to provide information about all significant risks, even if reasonable body would not.

Valid Consent

- Patients who may lack capacity
  - Age <18.
  - Lasting power of attorney.
  - Court appointed deputy.
- Assessment
  - Understand proposed treatment, including the risk(s) and benefit(s).
  - Able to weigh the proposed risk and benefit.
  - Able to retain information, communicate the decision (verbal/non-verbal).
  - Patient can have capacity for one treatment but not another.
- Who should seek consent?
  - Clinician—can be delegated: sufficient knowledge of investigation, treatment and risks involved.
  - When and before pre-medication, advised to complete it well in advance, also check before provider that patient has been consented appropriately.
  - If patient has capacity but unable to read and/or write, put mark on consent form (good to get witness, chaperone, also must record on case notes).
  - Validity of consent does not depend on the consent form.
  - Lack of completed form, not bar to treatment (but evidence that something took place).

- Duration of consent
  - Could be indefinite, unless withdraw/new intervention or method becomes available or if condition changes.
  - It is good practice to reconfirm the consent.
- Refused
  - Have to accept even death unborn child.
  - Advanced decision to refuse treatment: Lasting will/advanced directive, if life and death, must be with witness and start clearly if life at risk.
  - Must follow if valid and applicable, if unsure, referred to court of protection.
  - Adult without capacity—no one can get consent = consider previous wishes/consult family members—lasting power attorney/court appointed deputy—consent 4.
  - Children:
  - Age 16–17: Able to consent but can be overridden by parents/court, not need parents' consent although good practice, unless patient specifically excludes them.
  - Age <16: Gillick competent—sufficient understanding or intelligence.
  - If a young person refuses treatment, which may lead to their death or a severe permanent injury, their decision can be overruled by the Court of Protection.
  - Child lacking capacity: Parents can consent for 'best interest of child'—good to involve child, courts can override parent's decision.
  - In an emergency: justify treating a child who lacks capacity without consent of parents if treatment vital.
  - Court of Protection—get decision 24/7—children, Jehovah's witness.

8.4 Nice—Guidance Healthcare Associated Infections

Infections

- MRSA, C. diff, E. coli—direct results of healthcare.

Surgical Site Infection (Nice)

- Pre-operative
  - Not routinely use hair removal, if must—use electric clips single use head on day of surgery, avoid using razors and increasing risk of infection.
  - Antibiotic prophylaxis (before)
    (a) Clean surgery if placing prosthesis.
    (b) Clean contaminated—not for clean surgery.
    (c) Contaminated.

- Intra-operative
  - Prepare surgical site with antiseptic, allow for drying before diathermy avoid pooling
    (d) Povidone-iodine.
    (e) Chlorhexidine.
    (f) ChloraPrep 0.5%.
    (g) Chlorhexidine + alcohol = 70% isopropyl alcohol.
  - Cover surgical wounds with dressing.
- Post-operative
  - Refer to tissue viability, review management of surgical wounds that are healing by secondary intention.
- Sutures
  - Vicryl (Polyglactin 910): absorbable braided (polymer) synthetic.
  - Ethilon (Nylon): non-absorbable, monofilament, synthetic.
  - Prolene (Polypropylene): non-absorbable monofilament, synthetic.
  - PDS (p-dioxanone): absorbable, monofilament, synthetic—hydrolysis 180 days.
  - Silk: non-absorbable, braided, natural material.
  - Dexon (Polyglycolic acid): absorbable, braided, synthetic.
- Alvogyl: Butyl 4-aminobenzoate, eugenol, iodoform.
- Whitehead's Varnish
  - Benzoin 10 g/Iodoform 10 g/Balsam of Tolu 5 g/Storax 7.5 g/Solvent ether 100 ml.
- BIPP—Bismuth Iodoform Paraffin Paste: astringent/antiseptic.

Diathermy

- Use of high frequency electric current to produce heat.
- Bipolar (2 electrodes combined)—current passes between them to patient.
- Monopolar diathermy:
  - AC current 400 kHz–10 MHz (current though forceps)—local heating effect 1000 °C through patient plate (at least 70 cm^2).
  - Can use in pacemaker: short burst use bipolar—burns full thickness.
- Bipolar
  - Coagulation: Interrupted pulses (50–100/s)—square wave form.
  - Cutting: Continuous current, sinus wave form.

Medpor

- High density polyethylene—pore size 100–250 μm.

- Allow stabilisation by bony and soft tissue ingrowth.
- Foreign body reaction is minimal, only thin fibrous capsule and very few giant cells—low infection/extrusion rates.
- Available in sheet/block form—becomes adherent to surrounding bone.

Leech

- Hirudo medicinalis—they have vasodilator/anticoagulant properties (secrete hirudin) as sucking blood 50 ml/48 h.
- Must give antibiotics, infection with Aeromonas hydrophila.

PEEK

- Polyether ether ketone.
- Radiolucent, bone like stiffness/strength.
- Engineered from connective tissue—patient specific.

Pharmacology: Antibiotic Guidance

Oral Infections—Require Antibiotic Treatment

- Acute periapical/periodontal abscess, cellulitis/acute oroantral communication/severe pericoronitis/localised osteitis/ANUG.
- Certain cases: bacterial sialadenitis, osteomyelitis, actinomycosis, infections involving fascial spaces—Ludwig's Angina.
- Prevent spreading infection:
  - Fascial spaces: airway compromise.
  - Blood stream: cavernous sinus thrombosis.
- If infection fails to respond in 48 h—change on basis of bacteriological investigations. Reasons for failure:
  - Incorrect treatment.
  - Inadequate treatment.
  - Poor host response.
  - Poor patient compliance.
- Combination of penicillin (erythromycin) with metronidazole could be useful in some cases.

Superinfection

- Broad spectrum i.e. cephalosporins are more likely associated with adverse reactions.
- Example Fungal/C. diff.

Pericoronitis

- Metronidazole/amoxicillin.
- Only if systemic feature of infection/or trismus.
- Treat for 3 days or until symptoms resolve.

ANUG

- Metronidazole/amoxicillin.
- Periapical/periodontal abscess.
- Amoxicillin/metronidazole
  - Only if severe disease with cellulitis or systemic features.

Throat Infections

- Phenoxymethylpenicillin (or erythromycin if penicillin allergy)—most throat infections are viral.
- Avoid amoxicillin if possible (glandular fever) severe—Benzylpenicillin.

Sinusitis

- Amoxicillin or doxycycline or erythromycin—only if purulent discharge lasting 7 days or if severe symptoms.

Skin

- Impetigo
  - Fusidic acid—topical (mupirocin if MRSA).
  - Flucloxacillin/erythromycin.
- Erysipelas
  - Phenoxymethylpenicillin/erythromycin if penicillin allergy.
- Cellulitis
  - Benzylpenicillin and flucloxacillin (or erythromycin alone if penicillin allergy).

Animal/Human Bites

- Co-amoxiclav or doxycycline + metronidazole if penicillin allergy.
- Wash/check tetanus status.
- Tetanus vaccine—cell-free purified toxin
  - Primary immunisation for children <10: 3 doses, 1 month between.
  - 2 boosters: 1 before school, 1 just before leaving school.
  - Tetanus immunoglobin.
  - Wounds—tetanus prone
    (a) 6 h before treatment.
    (b) Contaminated with soil/manure.
    (c) Devitalised tissues/septic.
    (d) Foreign body.

Prevention of Endocarditis

- NICE 2008—antibiotics and chlorhexidine mouthwash not recommended—cause bacteraemia (no clear association).
- Joint prosthesis/dental treatment—no prophylactic cover needed.

Penicillin

- Bactericidal—interfere with bacterial cell wall synthesis, excreted in urine.
- Issues

  (1) Hypersensitivity

  – Rashes/anaphylaxis (1–10% of risk).
  – Urticaria/rash immediately after administration = risk of hypersensitivity.
  – If minor rash (no itching/small area of body or >72 h later) not allergy, but are intolerant—cephalosporins.

  (2) Encephalopathy

  – Cerebral irritation—high dose, or patients with renal failure.

  (3) Diarrhoea

- Benzylpenicillin (Pen G): inactivated by bacterial beta-lactamase, absorption low = intravenous.
- Phenoxymethylpenicillin (Pen V): similar to benzyl, gastric state = oral absorption unpredictable.
- Most Staphlococcal resistance to Benzylpenicillin—produce penicillinase. Flucloxacillin not inactivated by these enzymes—effective against Staphylococci.
- MRSA

  – Skin/soft tissue

  Tetracycline ± rifampicin and sodium fusidate.
  Clindamycin.

  – Severe infections

  Glycopeptide—vancomycin.
  If not suitable then linezolid (expert advice: not active against gram −ve).

  – Last resort

  Streptogramin antibiotics (quinupristin/dalfopristin).
  Tigecycline/daptomycin licensed.

  – Prophylaxis with vancomycin or teicoplanin (alone/combination) if patient has history MRSA = not eradicated/high risk MRSA).
  – Elimination nasal carriage

  Naseptin (chlorhexidine/neomycin).
  Mupirocin in resistant cases—three times daily for 5 days, sample 2 days after to confirm elimination.

Flucloxacillin

- Infections due to beta-lactamase-producing Staphylococci.
- Can survive cholestatic jaundice/hepatitis—after several weeks following treatment.
- Avoid in patients with hepatic dysfunction.

Broad Spectrum Penicillin

- Amoxcillin: amphotericin derivative—active against gram +ve and gram −ve but inactivated by beta-lactams, excreted in bile and urine.
- Maculopapular rash—glandular fever (also in patient with leukemia/CMV infections).
- Well-absorbed orally.
- Co-amoxiclav = amoxicillin + clavulanic acid (inactivate beta-lactamases).

Amoxicillin Cautions

- Renal impairment.
- Rashes (CMV/glandular fever).
- Side effects = nausea and vomiting, diarrhoea.

Co-Amoxiclav Cautions

- Cause cholestatic jaundice either during/or shortly after use.
- Increase in elderly usually self-limiting no treatment required >14 days.
- Side effects: same as amoxicillin, toxic epidermal necrosis, superficial staining of teeth with syrup.

Cephalosporins

- Broad spectrum, similar to penicillin, excreted by kidneys.
- Hypersensitivity (0.5–6.5% of penicillin allergy will have cephalosporin allergy (If immediate sensitivity to penicillin then avoid).
- Cefuroxime: second generation—less susceptive to beta-lactamase
- Cefotaxime/ceftriaxone: this generation—greater acting against gram −ve and less active against gram +ve (Staphylococci).
- Offer little advantage over penicillin in oral infections.
- Orally: Cefalexin 500 mg BD
  - Side effects: Diarrhoea, antibiotic-associated colitis, nausea and vomiting, abdominal discomfort, headache, rash, Stevens-Johnson Syndrome, liver enzyme disturbance—jaundice.

Tetracycline

- Broad spectrum, increased resistance.
- Treatment of Lyme disease, destructive periodontal condition ± MRSA.

- Oral—Streptococcal resistance.
- Doxycycline: Longer duration, take one daily—broad spectrum
  - Can use topically for oral ulceration.

Cautions

- Hepatic impairment.
- Increases muscles weakness—myeloma patients.
- Exacerbates lupus erythematous.
- Contraindications: Deposition in growing teeth—staining/dental hypoplasia: do not prescribe children age <12/pregnant woman/breast feeding.
- Side effects: Nausea and vomiting, dysphagia, oesophageal irritation.

Carbapenems

- Beta-lactam antibiotics with broad spectrum, not active against MRSA.
- Used for hospital acquired infections: septicaemia/skin infections/hospital acquired pneumonia.

Meropenem/Ertapenem

- Caution: Avoid if sensitive to beta-lactam antimicrobials, hepatic (monitor LFT), renal and pregnant women.
- Side effect: Nausea and vomiting, abdominal pain, disturbance in LFT, headache, reduced platelets.

Aminoglycosides

- Gentamicin, neomycin, streptomycin (TB)—bactericidal and active against gram +ve/−ve; pseudomonas but poor against anaerobes.
- Not absorbed from gut, intravenous is recommended, excretion by kidney, accumulation if renal problems.
- Side effects: dose related—nephrotoxicity, ototoxicity.
- It also affects neuromuscular transmission, avoid prescribing to myasthenia gravis.
- Once daily dosage: measure serum concentration after 3–4 doses
  - Peak—one-hour after.
  - Trough—just before next dose.

Gentamicin

- Inactive against anaerobes—for blind therapy continue with penicillin or metronidazole.
- Poor against hemolytic streptococci.
- Loading/maintenance—dose dependent 3–5 mg/kg/day.

Macrolides

- Erythromycin—like penicillin: give to penicillin allergy.
- Inhibit 50S subunit or bacteria ribosomal subunit—bacteriostatic.
- Causes nausea/vomiting/diarrhoea—reduce dose 250 mg/QDS.

- Clarithromycin—slightly greater activity—given BD.
- Oral infections—many organisms now resistant—use limited/short courses, use metronidazole instead of penicillin.

Cautions

- Can cause QT interval prolongation.
- Ok in Pregnancy, found small amount in milk.
- Avoid in patient with hepatic/renal impairment.

Side Effects

- Nausea and vomiting, abdominal pain (antibiotic-associated colitis), rashes, allergy, reversible hearing loss in large doses, cardiac effects (chest pain, arrhythmia), Stevens-Johnson Syndrome.

Clarithromycin

- Can cause tooth/tongue discoloration, smell and taste disturbance, glossitis, stomatitis, arthralgia, myalgia.
- 250–500 mg BD.

Clindamycin

- Active against gram +ve cocci = streptococci/penicillin resistant staphylococci/anaerobes (Bacteroides fragilis).
- Inhibits 50S subunit or bacteria ribosomal subunit—bacteriostatic.
- Well-concentrated in bone.
- Excreted in urine.

Recommended

- Staphylococcal joint infections/osteomyelitis.
- Erysipelas/cellulitis (penicillin allergy).
- MRSA (bone/joint/skin/soft tissue infections).
- Antibiotic-associated colitis—common = stop immediately if diarrhoea develops.
- Oral—not routinely, use only if not responded to penicillin or metronidazole.

Side Effects:

- Discontinue immediately if diarrhoea/monitor LFT/U&E if long-term.
- Oesophagitis/oesophageal ulcers/taste disturbance/nausea + vomiting/jaundice/leukopenia/rash/Stevens-Johnson Syndrome.
- Dose: 150–300 mg QDS.

Metronidazole

- Highly active against anaerobic bacteria and protozoa.
- Inactive against aerobes.
- Given rectally as effective as intravenous if not able to give oral.
- Intravenous—tetanus.
- Topical—rosacea.

- Oral—C. diff.
- Oral—alternative penicillin: drug of choice for ANUG 200 mg TDS/pericoronitis.

Cautions

- Disulfiram reaction—alcohol.
- Avoid: hepatic impairment, pregnancy/breast feeding/warfarin.

Side Effects

- GIT (nausea and vomiting), taste disturbance, furred tongue, oral mucositis.

Glycopeptides

- Vancomycin/teicoplanin—bactericidal: anaerobic/aerobic.
- Vancomycin: Oral—C. diff.
- Teicoplanin: OD, IM/I.V. not by mouth.

Vancomycin—Cautions

- Blood monitoring—FBC/renal, monitor auditory function in elderly.

Side Effects

- Nephrotoxicity, ototoxicity—stop if tinnitus, neutropenia, agranulocytosis, flushing upper body (Red man syndrome).

Linezolid

- Oxazolidinone antibacterial used in MRSA/vancomycin resistant Enterococci.
- Option if not using vancomycin, not active against gram −ve.

Cautions

- Monitor FBC frequently, avoid in uncontrolled hypertension as it could cause blood disorders/optic neuropathy (long courses).

Quinolones

- Bond bacterial DNA prevent replication—bactericidal.

Ciprofloxacin

- Gram +ve/gram −ve bacteria.
- Most anaerobes not susceptible.
- Good for bone/joint infections/septicaemia.

Cautions

- History of epilepsy/seizures/myasthenia/pregnancy/breastfeeding/children/young adults (not recommended): tendon damage.
- Induce convulsions if taking NSAIDs at the same time.

Side Effects

- Nausea and vomiting, abdominal pain, diarrhoea (rarely colitis), headaches, rash, Stevens-Johnson Syndrome.
- Ciprofloxacin dose: 500–750 mg BD.

Antifungals

(1) Polyene

- Amphotericin (I.V. systemic infection)/nystatin (candida)—not absorbed by mouth = topical.

(2) Imidazole

- Metronidazole.
- Miconazole—local oral infections, systemic absorption may result in significant drug interactions.

(3) Triazole

- Fluconazole.

Viral

- Herpes/Varicella zoster (VZ).
- VZ to treat within 24 h reduce duration/severity. Shingles—start within 72 h for 10 days (prevents post-herpetic neuralgia).
- Acyclovir—cautions
  - Adequate hydration—renal problems.
  - Side effects: Nausea and vomiting, diarrhoea, fatigue, rash, photosensitivity, very rarely hepatitis.
- Dosage
  - 200 mg 5 × daily.
  - VZ: 40 mg/kg/QDS in divided doses.
  - CMV infection—ganciclovir—profound in myelosuppression.

Local Anesthetic

- An aromatic ring to amide groups, links ether amide/ester.
- Amide: lidocaine, mepivacaine, articaine, prilocaine.
- Ester: procaine, benzocaine, tetracaine.
- Mainly metabolised by liver (microsomal enzymes)—amides.
- Ester types by plasma (pseudocholinesterase).
- Mechanism: reversible block to conduction—blocks sodium channel = stop action potential.
- Toxicity
  - Elevated plasma levels via vascular injection/or too much, classic signs—circumoral numbness, light headedness, twitching.

  - CVS: Increase in heart rate and blood pressure.
  - CNS: Drowsiness, confusion, tinnitus, metallic taste.
  - Progressive: Tremors, hallucinations, decrease in blood pressure/HR/CO = unconsciousness/seizure/ventricular dysrhythmias.
  - Finally—respiratory/cardiac arrest.

- Do not inject in inflamed/infected tissue—increase systemic absorption = side effects (also affected by local pH).
- Vasoconstrictors
  - Local anaesthetic causes dilation of blood vessels.
  - Vasoconstrictor added epinephrine, reduces local blood flow, slows rate of absorption/prolongs effect, used in low concentration—not exceeding 500 μg.
  - Lidocaine 2% with 1 in 80,000 (12–15 μg/ml)—severe hypertension/cardiac use procaine with/without felypressin = no evidence it is any safer, felypressin can cause coronary vasoconstriction
    (1) Lidocaine 7 mg/kg with adrenaline; 3.5 mg/kg without
        - 1 ml of 2% contains 20 mg.
        - Max 1 cartridge to 1 stone.
        - 42 mg of lidocaine = max 70 kg = 490 mg (11 cartridges).
    (2) Prilocaine: 4 mg/kg with octapressin
        - 6 mg/kg.
        - 9 mg/kg without adrenaline.
    (3) Articaine 4% in 1:100,000 Adrenaline—risk of sensory impairment.
    (4) Bupivicaine 0.25 2.5 mg/ml or 0.5% 5 mg/ml ± adrenaline
        - Maximum 2 mg/kg (70 kg = 140 mg).
    (5) Cocaine solution (4% 40 mg/ml–10% 100 mg/ml) = max 1.5 mg/kg = 100 mg in adult max.
- Adverse effects with vasoconstrictors
  - Tricyclic antidepressants—increase vascular supply of endogenous adrenaline—increase HR/BP.
  - Beta blockers—adrenaline counteracts.

Local Anesthetic Contents

- Adrenaline.
- Lidocaine.
- Saline.
- Preservatives.
- Thymol—antifungal.
- Sodium bisulphate (stabiliser).
- Sodium chloride.
- Distilled water.

Sedation

- Relative analgesia—mixture of nitrous oxide and oxygen with patient management techniques.
- Useful in children, quick onset/recovery, contraindicated in pregnancy.
- Oral: unpredictable in effects, consent before giving
  - Temazepam 10–30 mg 1-h pre-operative.
  - Diazepam 5–15 mg.
- Intravenous
  - Benzodiazepines cause hypnosis/sedation/amnesia.
  - Be careful using in elderly (sensitive)/children may become hyperstimulated.
  - Single drug safe Poswillo recommendation.
- Midazolam
  - Water soluble, rapid onset, short duration action with half-time 1–4 h.
  - Titrate to patient 2 × 5 ml ampoules = 10 mg.
  - Start with 1–2.5 mg and titrate.
  - Pulse oximeter/blood pressure/trained staff/suitable resuscitation facilities must be available.
  - Antagonist = Flumazenil—very short half-life (may need more than one dose); 200 μg with 100 μg/min until reversed.

General Anesthesia

ASA

- 1-Normal.
- 2-Mild systemic disease.
- 3-Severe systemic disease.
- 4-Severe systemic disease with constant threat to life.
- 5-Morbidity not expected to survive without surgery.
- 6-Brain dead—organ retrieval.

Principle—Combination

- Analgesia.
- Hypnosis.
- Muscle relaxation.
- Induction/maintenance performed by.

(1) Intravenous

(a) Propofol

- Induction/maintenance, rapid induction 30 s (2–2.5 mg/kg).
- Recovery 4–8 min with rapid elimination ± 30 min to 1 h.

- Hepatic metabolism, redistribution maintain with infusion (0.1–0.2 mg/kg).
- Causes 20–30% reduction in HR/BP due to rapid arterial/venous dilation.
- Low incidence nausea and vomiting, quick recovery.

(b) Etomidate

- Rapid induction—lasts for 3–12 min.
- Hydrolysis and redistribution, no cardiovascular changes.
- Use in patient with low cardiac reserve, reduced cerebral blood flow and ICP.
- Use in trauma as does not reduce BP.
- Side effects: Nausea and vomiting, involuntary skeletal muscle movement.

(c) Thiopentone

- Ketamine: highly lipid soluble—dissociate analgesia (CNS depression/amnesia).
- Increases HP/BP/CO, cerebral vasodilation—increases ICP.
- Used in emergency settings.

(2) Inhalational agents (vaporiser for inhalational administration)

- Sevoflurane.
- Isoflurane.
- Nitrous oxide.

Malignant Hyperthermia

- Hypermetabolic state: AD/FH, increased CK (incidence 0.5%, 40% mortality).
- Isoflurane, sevoflurane, halothane, suxamethonium.
- Acidosis, rigidity, fever, hyperventilation, myoglobinuria.
- Increased HR/BP/cyanosis/cardiac dysrhythmia—SR—increased Ca^{2+}.
- Management: stop Anaesthetic/hyperventilate with oxygen + I.V. dantrolene (blocks Ca^{2+} release)/fluids (not Ringer's lactate), cool patient.
- Muscle relaxants
  - Use to aid intubation and mechanical ventilation.
  - Skeletal muscle relaxation—2 groups
    (1) Depolarising
    - Mimics action of succinylcholine by depolarising post-synaptic membrane at junction.
    - Suxamethonium: short acting/rapid—used in rapid induction lasts 5-10 min, respiration returns if struggle to ventilate.

(2) Non-depolarising

- Complete blockage of post-synaptic membrane so acetylcholine blocked from receptors.
- Atracurium: Spontaneous breakdown (Hoffman elimination)—hepatic/renal problems.
- Rocuronium: biliary excretion/liver—liver dysfunction.

- Reversible: Acetylcholinesterase inhibitors = neostigmine.
- Analgesics
  - Supplement anaesthesia: e.g. remifentanil (continuous infusion)/alfentanil (aid induction).
  - Analgesia—morphine/fentanyl.
- Hypotensive anesthesia
  - Remifentanil infusion or vasodilator agents such as labetalol.
  - Commonly used in orthognathic = reduce morbidity.
  - Less profound in head and neck patients.
  - Inform anaesthetists when start wound closure—increase BP, ensure adequate haemostatasis.
  - Post-operative rebound hypertension and bleeding.

Other Airways

Laryngeal Mask Airway (LMA)

- Supraglottic airway device.
- Can be used as rescue device in failed intubation, not if you cannot intubate/ventilate, recommended surgical cricothyroid instead.
- Can be used in cardiac arrest.

Needle Cricothyroidotomy

- Temporary: 12–14 gauge with 5 ml syringe.
- Identify the cricothyoid membrane, between cricoid cartilage and thyroid cartilage.
- Stabilise trachea with thumb and fingers (prevent lateral movement—puncture skin).
- Direct 45° angle inferiorly and apply negative pressure to syringe—aspiration air.
- 1 s/off 4 s.

Surgical Cricothyoidoctomy

- Define airway—incise through membrane and use scalpel handle 90° rotation to open the airway.
- Insert tube—cuffed ETT or tracheostomy.

Tracheostomy

Head and Neck Cases—Elective Tracheostomy: Varies

(a) Conservative

- Elective tracheostomy only—bilateral necks, posterior tumours, mandibulectomy cases, chest disease.
- If not, remains intubated overnight, some may need tracheostomy due to failure in extubating.

(b) Aggressive

- High rate of elective tracheostomy.
- Return to high dependence area of head and neck ward.
- No adequate RCT compare approaches.

Indications

- Upper airway obstruction.
- Pulmonary.
- OSA.
- Prolonged ventilation.
- Head and neck patients.

Contraindications

- Infection or tumour at tracheostomy site.
- Neck fasciitis in the overlying skin.
- Adjustable tube for obese necks.

Advantages Over ETT

- Improved respiratory mechanism.
- Less work of breathing.
- Reduced laryngeal irritation/ulceration.
- Increased speech/mobility/nutrition.
- Patient can be nursed outside intensive care/high dependency unit.

Anatomy

- 18–22 C-shaped rings: soft membranous.
- Cricoid cartilage: carina 10–13 cm, 2–3 cm wide.
- Sternohyoid/sternothyroid—meet midline neck and used together by avascular fluid = linea alba.
- Thyroid isthmus: level 2nd/3rd tracheal rings.

Technique

- GA/head extended/supine/LA/tube check.
- Transverse incision (1/2 way between cricoid cartilage—suprasternal notch).
- Divide anterior jugular veins, if necessary, separate strap muscles.

- Divide thyroid isthmus with transfixation suture and reflect thyroid gland off tracheal wall.
- Tracheostomy: Horizontal incision, make window/cut window, stay sutures.
- ETT slowly withdrawn—insertion tube/haemostasis/secure and tie tape (3-0 silk, pressing foam).

Shiley Tube

- Size 6: Female (cuff/uncuffed).
- Size 7-8: Male—fenestrated with inner cannula.

Post-operative

- Humidification.
- Cuff pressure measured and calibrated—deflate ASAP.
- Suction, tube change—generally at 7 days.
- Cuff pressure: increases tracheal stenosis.
- If need high pressure to avoid leak—faulty cuff or small tube (change) = 10–25 cm/H_2O.

Humidification

- Increase moisture content in inspired air (as normally—nose/URT: filters/warm/via ciliated epithelium).
- Water humidifiers.
- Speaking valve: humidification via tracheostomy mask.

Complications

- Airway obstruction, apnoea, bleeding, recurrent laryngeal nerve injury, pneumothorax, subcutaneous emphysema, tracheomalacia, subglottic stenosis, dislodgement of tube, infection, granulation, aspiration.

Early Complications

- Pneumomediastinum.
- Pneumothorax: Lung apex caught as lung apex comes to root of neck.
- Acute haemorrhage: technical/thyroid isthmus/innominate vein (erosion).
- Accidental decannulation: properly secure with tube.
- Tubal obstruction: meticulous care, high humidity with air.
- Local infection: rare, peristomal cellulitis (systemic antibiotics).

Late Complications

(1) Tracheobranchial/periosteal granuloma

- 90% have granulomas.
- Aetiology
  - Trauma at stomal site.
  - Excessive suctioning.
  - Vascular anatomy at tip.

- Management
  - Prevention: tip well above carina.
  - Peristomal: silver nitrate, cup forceps, sphenoid punch or bronchoscope external deliver and electrocautery removal.

(2) Subglottic stenosis

- Generally caused by tracheostomy too high and rough healing (1–2%).

(3) Suprastomal collapse

- Most common in young patients with prolonged intubation.
- Causes

  Pressure on the 1st/2nd tracheal rings causing local chondritis and weakening.
  Upward pressure from undivided thyroid isthmus.
  Pooled secretions.

- Management

  Small segments excised allowed to heal by secondary intention.
  Large: tracheal reconstruction with cartilage grafts.

(4) Persistent tracheocutaneous fistulae

- More common in long-term cannulation of young patients.
- Management
  - Ensure adequate airway, excision, multilayer closure, drainage.
  - Patient may need post-operative intubation for 24–48 h.

(5) Tracheoinnominate fistula (<0–2%):

- Direct pressure of cannula against artery—overall survival 25%.

(6) Tracheo-oesophageal fissure (<1%):

- If tip irritating posterior wall, especially if have NGT.

(7) Aphonia

- Speech development addressed in child—if long-term planned.

Decannulation

- Check ABG, adequate co-operation, cough, and swallow, normal flexible nasendoscopy, haemodynamically stable.
- Pre-oxygenate → Suction → Breathe out → Remove → Suction if changing tube.

Chapter 9
Head and Neck Part 1

Abstract Assessment, investigation and multidisciplinary management of head and neck cancer.

Key Points

- Evaluation of patients with oral cancer.
- Key anatomy.
- Risk factors for oral cancer.
- Tumour assessment and staging.
- Imaging for cancer.
- Histological assessment.
- Pre-operative assessment and management of comorbidities.
- Nutrition.
- Management of chyle leak.
- Oral rehabilitation.
- Quality of life.
- Prognostic markers.
- Neck disease and complications of neck dissection.
- Other surgical complications.

Clinical Evaluation of Patients with OSCC

A. **Initial evaluations**.

(a) PC, HPC, systemic symptoms/signs.
(b) Risk factors likelihood 1° cancer and recurrence:

- Alcohol, smoking.
- Age at which patient began smoking.
- Occupational exposure.
- Heavy metals.
- Radiation.
- Betel nut/slaked lime.
- PMH.
- Previous Surgery.
- MI, DM.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
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- CVA/PVD—intermittent claudication.
  - Synchronous tumour 10%.
  - Medication.
  - Social history: living arrangements, social support, psychosocial assessment.
  - Examination (general physical exam, fitness for management):
- Detailed head and neck:
  - Skin/scalp/oral cavity/lymph nodes.
  - Cranial nerves (esp V, VIII, XI, XII).
- May require FNE, EUA, panendoscopy, laryngoscopy.
- Histopathological tissue diagnosis, endoscopy.
- EUA for base of tongue.
  - Radiographic evaluation.

B. **Radiographic**

- Extent of tumour, depth, dimensions, bony invasion, masticator space, skull base, proximity to vital structures.
- Regional lymph node assessment, metastatic assessment.
- Dental assessment: Clinical/OPG.
- CT/MRI/USS/FDG-PET—Usually axial/coronal CT (H&N) + chest, CXR, US neck, OPG.

Epidemiology

- 7500 cases of all H&N types in UK/annum. 6th commonest.
- 2600 oral cavity, 900 oropharynx, in India 40% of all cancers.
- 2–5x > incidence in men, commonest 6/7th decade, increase of incidence in young adults.
- 90% SCC, sarcoma, minor salivary gland tumour, melanoma, metastatic.
- 50% mortality, significant geographic variation, increased incidence Scotland.
- Long-term survival rates only slightly improving.

A. ***Anatomy***

- Oral cavity: Vermillion border of the lips to imaginary coronal plane at junction of soft/hard palate and circumvallate papillae.
- Tissues have very rich lymphatic drainage, various subsites.
- Oropharynx: Soft/hard palate—epiglottis—tonsils.
- Anatomical subsites:

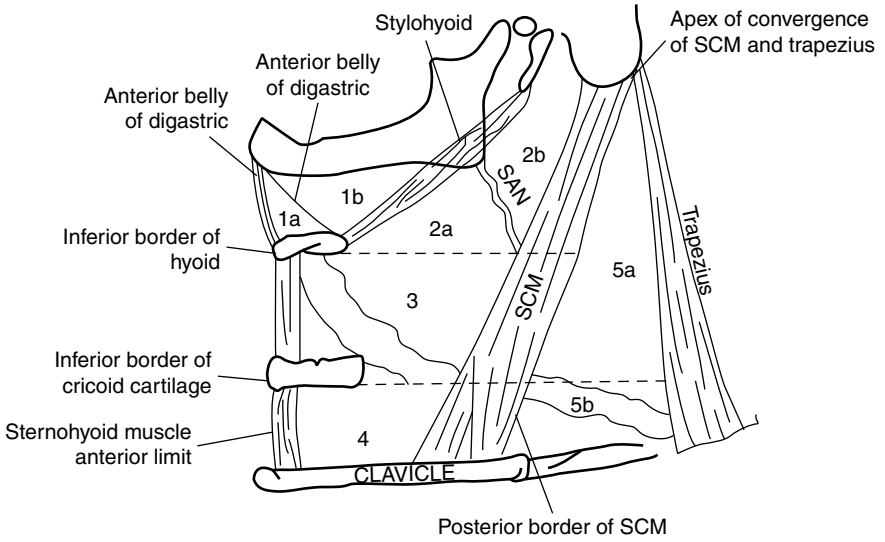


Fig. 9.1 Neck dissection levels

- Lips.
- Buccal mucosa—all mucous membranes lateral to alveolar sulcus—retromolar trigone and pterygomandibular raphe posteriorly—drain level I–II.
- Alveolar ridge—alveolar process and mucous membranes—drain I–II, lingual/palatal may drain to retropharyngeal nodes.
- FOM—mandibular alveolar ridge to ventral surface tongue.
- RMT—triangular region overlying ascending ramus, base is formed by last tooth and apex of maxillary tuberosity, anterior tonsillar pillar and buccal mucosa—drains level II ± periparotid/retropharyngeal nodes.
- Hard palate—maxillary alveolar ridge to palatine nodes—drains level II.
- Tongue—anterior 2/3 (circumvallate papillae)—is oral SCC lymph node extension to levels I, II, III and communication across midline exists.

Lymph Nodes (Fig. 9.1).

- Level I—submental and submandibular.
- Level II—upper jugular- A inferomedial, B superomedial (accessory).
- Level III—middle jugular (carotid bifurcation)- omohyoid, sternohyoid to SCM.
- Level IV—lower jugular to omohyoid- clavicle, SH to SCM.
- Level V—posterior triangle—trapezius—SCM, omohyoid divides 5A\5B.

Risk Factors

- Extrinsic:
- Smoking (2–12 × RR).
- 6–8 × more likely than non-smokers.
- Women × 2.
- Pipe and cigar increased risk.
- Smokeless tobacco increases risk especially lingual/buccal.
- >300 chemicals contribute to carcinogens.
- Smoking cessation reduces risk does not eliminate. 30% risk reduction 1–9 yrs. Cessation; 50% risk reduction >9 yrs.

Alcohol

- High intake.
- With smoking synergistic.
- Depends on sites—FOM/lower buccal vestibule.
- Low BMI—increased oropharynx tumour.
- Betel nut.
- HPV 16, 18.
  - HPV associated cancers of the tonsil and tongue base has increased by 5% per year.
  - Occur in younger, healthier individuals with minimum or little history of tobacco use.
  - Highly responsive to treatment and has an excellent prognosis.

Tumour Assessment/staging

- Objectives staging.
- Aid treatment planning.
- Indication prognosis.
- Evaluate results of treatment.
- Research.
- Mainly retrospective/observational—level 3.
- Panels experts using this data—level 4.

When all data collected stage tumour: According to AJCC TNM 8th edition (Tables 9.1 and 9.2).
TNM—all cases confirmed microscopy—2 classifications, cTNM, pTNM. If doubt, then lower TNM chosen—patient put in stage group.
Histopathological grading—estimation by pathologists expected biological behaviour—inter-/intra-observer error. TNM node provide biological characteristics.

(1) **Broders (1920)**.

- Graded on differentiation/keratinisation of cells.
- Grade 1 well differentiated, grade 2 moderately differentiated, grade 3 poorly differentiated, grade 4 anaplastic.

Table 9.1 TNM staging for oral cavity tumours

Lip and oral cavity	Seventh edition	Eighth edition
T1	Tumour <2 cm	Tumour ≤ 2 cm, ≤5 mm depth of invasion
T2	Tumour 2–4 cm	Tumour ≤ 2 cm, ≤ 5 mm and ≥ 10 mm depth of invasion or tumour >2 cm but ≤ 4 cm and depth of invasion ≤ 10 mm
T3	Tumour >4 cm	Tumour >4 cm or depth of invasion >10 mm, but ≤20 mm
T4a	Moderately advance local disease: (Lip) tumour invades through cortical bone or involved inferior alveolar nerve, floor of mouth, or skin of face (oral cavity) tumour involved adjacent structures such as cortical bone of maxilla or mandible, maxillary sinus or skin of face, extrinsic muscles of tongue	Extrinsic muscles of tongue removed, included extensive tumours with bilateral tongue involvement and/or DOI >20 mm
T4b	Very advanced local disease; tumour invades masticator space, pterygoid plates, skull base, and/or encases the internal carotid artery	No Change
N1	Metastases to single lymph node, 3 cm or less in greatest diameter	Same, expect node must be extranodal extension negative
N2a	Metastases to single ipsilateral node >3 cm but not >6 cm	Same, expect node must be extranodal extension negative or single ipsilateral or node 3 cm or smaller with extranodal extension
N2b	Metastases to multiple ipsilateral node >3 cm but not >6 cm	Same, expect nodes must be extranodal extension negative
N2c	Metastases to bilateral nodes or contralateral nodes none >6 cm	Same, expect nodes must be extranodal extension negative
N3	Metastases to nodes >6 cm	Subdivided into 3a: same as N3 before, but extranodal extension negative 3b: single ipsilateral node >3 cm in greatest dimension with extranodal extension Or multiple ipsilateral, contralateral, or bilateral nodes, any with extranodal extension Or single contralateral node 3 cm or smaller with extranodal extension

Table 9.2 Staging for oral cavity tumours

Staging grouping			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IVA	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
Stage IVB	Any T	N3	M0
	T4b	Any N	M0
Stage IVC	Any T	Any N	M1

- Widespread use—limited prognostic due to relative heterogeneity of the cell population present in tumours.

(2) **Anneroth's (1987) multifunctional grading system**.

- Keratinisation.
- Nuclear pleomorphism.
- Mitosis/pattern.
- Stage.
- Lymphoplasmacytic infiltration.
- Given points for each section.

(3) **Byrne et al. (1992)**

- Deep invasive cell grading system.
- Number mitosis/stage invasion—omitted, other 4 parameters in the deepest invasive margins only.
- Given scores.

9.1 Tumour Assessment/Staging-Radiology

Dosages X-rays—MSV = Background Radiation

- Periapical: 2 mSv = 8 h.
- OPG: 14–24 mSv = 52–100 h.
- Cephalogram: 6 mSv = 22 h.
- CT: 534–860 mSv = 89–143 days.
- CBCT (small value) 11–77 mSv = 2–3 days.

(1) ***OPG:***

- Bony pathology demonstrates where significant decalcification has occurred as result of pressure of direct invasion.
- Occasionally useful: rare osteosarcomas—sunray appearance.
- Bony invasion: loss cortical outline/irregular areas of lucency/rarely root resorption.
- Main value: dental assessment/planning osteotomy.
- Post-operative check: reduction/condyle placement/bony contour/placement of screws.

(2) ***CXR***

- Can supplement clinical assessment (respiratory/cardiac function).
- Baseline pre-treatment: developing clinical role—not used for follow-ups.

Computerised Tomography (CT)

- Rotating x-ray source in gantry captured by detector: images displayed by shades of grey that are based on attenuation of the x-ray beam through area of interest.
- Now spiral helical CT with multi slices (multi-detectors)—16:32 slice scanner.
- CT contrast:
  - Ionised contrast: Some tumours due to abnormal vascularity easiest to see (U/E required).
  - 70 s delay: Allow opacification of arterial/venous studies, improves lymph node detection. Delay allows optimal opacification of mucosal surfaces for assessing aerodigestive tract.

Advantages

- Widely available.
- Low cost.
- Cross sectional—ability to reconstruct in multiple planes.
- Very good bone anatomy, better for cortical bone involvement than MRI.
- Fast acquisition time.

Disadvantages

- Distortion of image: amalgam artefact.
- Ionising radiation
  - Lymph nodes—neck criteria.

    Increased size (short axis >10 mm).
    Morphology (normal ovoid, pathological rounded).
    Fatty hilum (loss in pathological infiltration).
    Central necrosis and rim contrast enhancement.
    Extracapsular extension and adjacent fat infiltration.

Cone Beam CT (CBCT)

- Same principle except delivers a cone-shaped beam, resulting in exposure of the region of interest (dose reduction).

Advantages

- Lower radiation.
- Cross sectional.
- Data manipulation is possible.

Disadvantages

- Poor tissue contrast.
- Lower resolution.

MRI

- Radiofrequency (RF) pulses delivered to region of interest and relaxation of hydrogen atoms in water results in emission of signal.
- When RF pulse switched off: H + returns to original alignment—free induction decay.
- Image weighting achieved by alteration of time of repetition and time of echo.
- T1: water (black)/fat (bright)—good for anatomy.
- T2: Water + fat both bright—good for inflammation/pathology and tumour signal (intermediate T2 signal).
- STIR (Short-TI Inversion Recovery): normal fat suppressed—highly sensitive to fluid (inflammation) and therefore pathology.
- Gadolinium:
  - Paramagnetic material = shortens T1/T2 relaxation times.
  - Tissue upload related to perfusion (more information on vascular/neural structures).

Advantages

- No ionising radiation.
- Excellent soft tissue.

- Reduced metallic artifact.
- Cross-sectional imaging.

Disadvantages

- Contraindicated with some metals (ferrous)/pacemakers.
- 40 min = claustrophobic.
- Fine bone detail not seen, good for marrow invasion.

Radionucleotide

- Functional study based on increase uptake of radioactive substance in metabolically active bone.
- Usually Tc-99—injected intravenously = gamma camera records activity.
- Uses: Condylar growth, osteomyelitis, mandibular invasion.
- 99Tcm (colloid)—sentinel node biopsy with gamma camera.

SPECT—Magnetic Field (MRI)

- Hydrogen ions: line up in the direction of the magnetic field.
- MRI machine applies radiofrequency pulse—specific to H + ions = aimed at tissue of interest.
- Turn off RF pulse = H + return to normal alignment with magnetic field and release absorbed energy form RF pulse.
- This signal picked up—free induction decay = computer adjusts.

Position Emission Tomography (PET)

- Functional study based on the uptake of 18 FDG (fluorodeoxyglucose—H12—109.7 min) in cells proportional to their rate of glycolysis—can be combined with CT to provide anatomic correlation.
- FDG given intravenously—trapped in cells as FDG-6-phosphate = unstable—release photons 180° to each other (localisation).
- Useful:
  - Unknown primary, tumour recurrence (distinguish active tumour form post-operative/radiotherapy changes), useful to have baseline.
  - False negatives 1–3 months following radiotherapy.

    Residual inflammatory reaction post PORT.
    Tumour must be >1 cm to be seen by PET.

- Low specificity
  - Infection/inflammation.
  - Expensive.
  - Sensitivity lymph nodes = 78%.
  - Can detect synchronous tumours and distant metastasis = 9–21% of cases.
  - Recommended for all T4 tumours to look for distant metastasis prior to any major treatment decisions.
  - Becoming more widely used.

Ultrasound

- Emits sound waves which rebound and are detected by probe.
- Hypoechoic versus hyperechoic.
- Doppler used for flow measurement.
- Accurate determination of site/morphology of neck cases with FNA.
- Simple (cheap/no radiation/highly operator dependent/unable to see deep structures).
- Sensitivity 89–95%, detect lymph nodes.
- Specificity 80–95%, increase with FNA.
- Real time—aid.
- FNAC—high false positive/negative = cytology.
- Head and neck (5–10 MHz)—acoustic coupling (gel) required to eliminate air/skin gap.
- Doppler = assess shift in frequency—show blood flow (colour map).
- Lymph nodes = 300 in neck vary in size 2–25 mm.
- Ultrasound.
- Normal doppler flow: position of vascularity seen at hilum
  - Tumour: peripheral nodal vascularity.
  - Pathological assessment:
  - Frozen section: assessment of surgical excision margins-not as good as paraffin—important info missed. No evidence improves prognosis.
  - Operative specimen: 10% neutral buffered formalin ($\times 3$ volume of specimen). Bone takes several days to decalcify depending on density.
  - Immunomolecular/immunocytological markers used to determine rare/subtypes during analysis. Molecular profiling not recommended.

Pre-treatment clinical assessment—evidence extrapolated from studies on patients undergoing general/orthopaedics

Comorbidity

- Presence of illness unrelated to the tumour. It significantly affects prognosis in H&N. Contributed by tobacco, alcohol, substance misuse.
- Adult comorbidity evaluation (ACE27)/Charlson Comorbidity Index/WHO/ECOG/Karnofsky (0-100) commonly used indices to quantify comorbidity.
- The effects of increased pre-treatment comorbid burden include:
  - Increased mortality in H&N patients.
  - Adverse influence on disease specific survival (probably due to advanced stage at presentation).
  - Increased incidence/severity of complications.
  - Adverse impact on QoL/increased cost of treatment.
  - Comorbidity data important—analysis survival. QoL/functional outcomes after treatment.

- Comorbidity data is important for surgical analysis—QOL/functional outcomes after treatment.

Pre-assessment:

- Avoid delay for surgery.
- Avoid unnecessary duplicate investigations.
- Role of anaesthetist in pre-assessment:
  - Identify difficult airway

    Mallampati (unreliable).
    Review anaesthetic records.
    Previous problems.
    Previous surgery/radiotherapy.
  - Risk stratification.
  - Optimisation of comorbidities.
  - Plan for perioperative care.

Risk stratification/optimisation:

Poor functional status is the most important predictor of peri-operative mortality. Assessed by:

- Use of metabolic equivalents (METs):
  - MET1 resting state.
  - MET4 climbing 2 flights of stairs uninterrupted.
- Lack of independence.
- Living in sheltered accommodation
  - Combination of subjective/objective-cardiopulmonary exercise testing/CPX.
  - Tools are used but the relative low mortality in H&N surgery suggest that formal assessment tools may overestimate the risk of surgery.

Risk factors predicting post-operative morbidity:

- CVS:
  - Heart failure—assessed with poor functional status.
  - Unstable coronary disease—coronary event in last 6 months.
  - Severe valvular disease.
  - Pulmonary hypertension.
  - Poorly controlled AF.
  - Cardiology referral.
- RS:
  - COPD.
  - Long operation.

RX Heart Failure:

- ACE/B-blockers—continue post-operatively.
- Stenting—following angiography—bare metal stents preferred—only 6 weeks of clopidogrel—increased risk of bleeding. Rather than drug eluting stents which require 1 year of antiplatelet therapy.

Recommendations:

- Clopidogrel—discontinued 5 days pre-operatively, aspirin continued.
- Warfarin for uncomplicated AF—discontinued 5 days before surgery.
- When warfarin for artificial valve/PE, require heparin pre-operatively.
- Neuroendocrine control of hypertension takes months to achieve and probably does not contribute to peri-operative mortality.
- Statin therapy should be continued
- COPD:
- Quantified with spirometry—FEV1 estimation helps prognostically:
  - Optimise bronchodilator therapy.
  - Trial of steroidal response in moderate/severe.
  - Smoking cessation.
  - Pre-operative nebuliser therapy.
  - Treat chest infection even if delays surgery.
  - Sputum sampling.

Recommendations:

- Severe hypertension (>180/110 mmHg) or hypertension in presence of AF, cardiac failure, LVH.
- An FEV1 <25% with hypoxia/increased CO_2/cor pulmonale—indicates increased need for post-operative ventilatory support.
- Endocrine:
  - Diabetes—tight control, oral hypoglycaemic omitted on day of surgery, restart when normal diet.
  - Patients >10 mg prednisolone—Risk of post-operative adrenal failure. Intravenous supplementation on induction of 50–100 mg hydrocortisone with additional post-operative (50–250 mg/daily in divided doses).
  - Also increased gastric ulceration—antacid therapy.
- Neurological:
  - Head positioning—RA—cervical spine stability assessed radiologically.
- Haematological:
  - Iron deficiency anaemia.
  - Aim Hb >10 g/dL—transfusion carried out at least 2 h pre-operatively to maximise O_2 carrying capacity of the blood.

- Alcohol:
  - Alcohol withdrawal syndrome—perioperative mortality up to 10%.
  - Risk prediction systems (e.g. CAGE)—identify/drug treatment recommend 48 h pre-operatively as an inpatient.
  - Benzodiazepine/chlordiazepoxide—alcohol abuse—assessed with cardiomyopathy, AF, hypophosphatemia, hypomagnesaemia.

9.2 Nutrition

Specialist Dietitian—Part of MDT

- Early identification/intervention—part of planning.
- Include quality of life, address psychosocial rehabilitation.
- Malnourished patients perform less well.
- Screening identifying tool
  (1) Subjective Global Assessment (SGA)—based on history and examination.
  (2) Patient-Generated Subjective Global Assessment (PG-SGA).
  (3) The Malnutrition Screening Tool (MST).
  (4) Malnutrition Universal Screening Tool (MUST)
    - Repeat weekly for inpatient.
    - Loss >2kgs within 2 weeks—report to dietitian.

  (5) BMI.
  (6) Unexplained weight loss for 3–6 months.
  (7) Acute disease effect scale.
  (8) $1 + 2 + 3 = 4$ Overall risk of malnutrition.
  (9) Management guidelines—high risk/dietitian review.

Caloric Needs

- Most patients 2000–2500 kcal + 7–14 g nitrogen every 24 h.
- Over 50% head and neck cancer patient are malnourished due to
  - Pain/reduced appetite/dysphagia.
  - Pre-morbidity lifestyle: alcohol/smoking/poor diet.
- Multifactorial.
- Worse after treatment—radiating mucositis.
- Reduced nutrition → weakened immune system → increased risk of infection.
- Dietitian/SALT
  - Infection.
  - Reduced wound healing.
  - Impaired cardiac/respiratory function.
  - Muscle weakness.

- Depression.
- Poor quality of life.
- Increased morbidity.
- Reduced response to chemo-/radiotherapy treatments.

Assessment

- Clinical: chew/swallow/medical history/weight loss.
- History: fluid intake.
- Calculation requirement
  - Energy 30–35 kcal/kg/day—increase if major complication.
  - Protein 0.8–2.0 g/kg/day.
  - Fluid 30–35 ml/kg/day—increase infections.
  - Height/weight

    BMI = weight (kg)/height $(m)^2$.
    Normal 20–25 kg/m^2.
    <18.5 kg/m^2 = underweight.

  - Triceps skin fold thickness (TST).
  - Mid arm muscle circumference (MAMC—lean tissue mass).
  - Hand grip.
- Biochemistry
  - Urea and electrolytes, albumin (half-life = 17–20 days)—not good indication and affected by sepsis/stress.
  - Pre-albumin level (half-life—2–3 days) but still affected by sepsis/stress.
  - CRP.
  - Transferrin—affected by inflammation/infection.
  - Total lymphocyte count—affected by infection.
- Social information
  - Alcohol.
  - Smoking.
  - Financial.
  - Patient's perception.

Cancer Cachexia

- Syndrome: reduced appetite/weight loss/metabolic alteration.
- Pro-inflammatory process.
- Insulin resistance.
- Increased loss body fat/muscle mass.
- Increase acute phase proteins.
- Cytokines induce metabolic alterations.
- Prevent patient from regaining body cell mass during nutritional support.
- Cancer patients are mildly hypermetabolic—increased energy expenditure.

Refeeding Syndrome

- Metabolic disturbance as result of reintroduction of nutrition to patient starved/malnourished.
- Reduction in phosphate ± abnormal sodium/glucose/protein and fat metabolism.
- Thiamine deficiency, reduced potassium and magnesium levels.
- Incidence is uncommon, but is defined as serum phosphate <0.4 mmol/L
  - 37.5% at risk.
- Patient at risk of developing refeeding syndrome

  (a) One or more of
  - BMI <16 kg/m^2.
  - Weight loss >15% in last 3–6 months.
  - Little or no nutrition >10 days.
  - Low potassium/phosphate/magnesium.

  (b) Or patient has 2 or more of
  - BMI <18.5 kg/m^2.
  - Weight loss 10% in last 3–6 months.
  - Little/no nutrition last 5 days.
  - History of drugs/alcohol.
  - Drugs: insulin/chemotherapy/antacid.

Management

(1) Patient at risk.
(2) Check K^+/PO^{4-}/Mg^{2+}.
(3) Before feed give
   - Thiamine 200–300 mg orally daily.
   - Vitamin B 1–2 tablets daily.

(4) Multivitamins.
(5) Start feeding
   - Slow increase 4–7 days.

(6) Rehydrate slowly—supplement/correct = potassium (2-4 mmol/kg/day); phosphate (0.3-0.6 mmol/kg/day); Calcium; magnesium.
(7) Monitor—amend in first 2 weeks.

Nutritional Support

- Aim to improve quality of life, reduce side effects of therapies/prevent/treat.
- Consider if BMI <18.5 kg/m^2, loss >10% in past 3–6 months, minimal intake >5 days.
- Increase nutritional intake—catabolism.

Types of Nutrition Support

(1) Oral

- Meal fortification.
- Supplements: liquid/powder—high energy/protein/vitamins = adjuncts
  - Bland.
  - Monitoring.
  - Result in taste fatigue.
- Try oral feed.

(2) Enteral

- NG/Nasojejunal/tracheo-oesophageal fistula tubes/orogastric/gastrostomy/gastrojejunostomy—all <4 weeks.
- No nationally agreed criteria for gastrostomy in head and neck cancer patients.
- NICE suggests if enteral longer than 4 weeks.
- Consider timing/method/screening/assessment.

NGT

- Fine bone/short term.
- Complications
  - Incorrect placement.
  - Risk of aspiration.
  - Tube blockage.
  - Infection.

Gastrostomy

- Abdominal surface into stomach/longer term/reduced displacement/blockage compared to NGT.
- Hide underneath clothes.

Methods

- Endoscopically/surgically/radiologically
  - Surgical: if previous abdominal surgeries.
  - Radiologically: not possible to reach stomach.
- Commonest—Pull method.
- Mouth—use this cord (to place PEG)—back through mouth → Stomach → Abdomen.
- Intubation—endoscopy/lie supine.
- Transillumination: confirm location in stomach, Direct contact with abdominal wall no intervening tissue.

- Pulling gastrostomy: Tape PEG—pull up to cannula → Cannula tube pulled out/internal bumper stops it from coming out.
- Post-operative: Nil by mouth for 12 h, sterile water for 12 h/check for bowel sounds before feed/regular antibiotics.

Complications

- Procedure related
  - Drug reaction: local anesthesia/sedation.
  - Hypoxia: monitor oxygen saturation.
  - Aspiration pneumonia as patient supine.
- Early complications
  - Local discomfort—gastric necrosis: loosen traction.
  - Resistant leakage: Reflects poor wound healing, loosen traction.
  - Bleeding wound: Tighten.
  - Wound infection: Antibiotics (pre-procedure).
  - Ileus: Resolves spontaneously.
  - Accidental removal.
  - Peritonitis.
- Late complication—tube blockage
  - Tube blockage
    (a) Avoid flushing.
    (b) Treat: dilute vinegar.
    (c) Pancreatic enzymes.
- Peristomal leakage.
- Buried bumper syndrome: excessive traction between inner and outer barrier (ulceration/necrosis).
- Hypergranulation: prevent turning external part daily/treat with nitrate.
- Accidental removal: can close within hours even after epithelialised—therefore place urinary catheter until replacement.
- Necrotising fasciitis: antibiotics/high traction/small abdominal wound.
- Gastrocolic fistula—often present diarrhoea.

Relative contraindications

- GORD.
- Gastric ulcer.
- Gastric rashes.
- Partial hypertension.
- Previous GIT surgery.
- Intestinal obstruction.
- Late pregnancy.
- Obesity.

RIG

- Stomach inserted with NG tube/external puncture with aid of radiological imaging.
- Avoid disturbing oropharyngeal region.

Placement of NG tube

- Explain to patient.
- Aseptic.
- Position patient to 5–60° neck neutral.
- Topical within nose.
- Measure—xiphsternum to tragus → across cheek-alar rim.
- Pass with lubricant—floor of nose/nasopharyngeal wall (resistance).
- Ask patient to swallow—gentle pressure → push to pharynx.
- Avoid allowing patient to tilt head back. If required, provide patient a glass of water as helps with peristalsis.
- Secure with tape.
- Check position
  - Leave guide wire—tip below diaphragm (XR chest—check if safe to use).
  - Litmus paper—check pH level.
- If patient complains of sharp pain withdraw immediately as it may have reached the trachea.
- Side effects
  - Nausea and vomiting/osmotic diarrhoea/tube blockage/displacement.
  - Feed should be started with water—gradual build up/pump.

Immune Enhanced Nutrition

- Immuno-nutrition are seeds with amino acids/nucleotides/lipid suggested reducing post-operatively.
- Infection—not proven, may reduce hospital stay.

Nutrition During Surgical Treatment

- Inadequate intake for >14 days—increased mortality—patients with severe risk should get support 10–14 days prior to major surgery.
- Indications: BMI <18.5 kg/m^2, weight loss <1 0–15% over 6 months, albumin <30, unable to maintain intake.

Management of Chyle Leak

- Rare incidence of 1–4% in neck dissections, when central lymphatic system damaged during surgery, caused by damage to thoracic duct (left), lymphatic duct (drains to right subclavian vein).
- Diagnosed during surgery—pooling/discharge in neck or excess fluid in drain.
- Clear (patient starved), milky (feeding due to fat content)—biochemistry reveals triglycerides/chylomicrons.

- Intra-operative identification can be aided by placing the patient in the Trendelenburg position or adopting a forced Valsalva manoeuvre. Post-operative leaks are usually identified when feeding is commenced. Multiple approaches to the treatment of an established leak have emerged including nutritional, surgical and pharmacological therapy.
- Fibrin sealant and a pectoralis myofascial flap—intra-operativeley.
- Patient can be fed a rich milk or cream product pre-operatively in an attempt to increase chyle production and aid intra-operative identification of a leak.
- Transthoracic ligation of the thoracic duct is an alternative to the above and has been found to be effective. The current trend is to do this via a thoracoscopic approach.
- In the neck, the thoracic duct raises 3–5 cm above the clavicle before arching back toward the junction of the left subclavian vein with the left internal jugular vein. Intra-operatively, the thoracic duct can be identified as it courses anterior to the vertebral and thoracocervical vessels between the internal jugular vein and the anterior scalene muscle.

Management:

Aim to maintain nutritional status, adequate fluid balance and electrolyte balance

(1) Dietary manipulation.
(2) Further surgery.
(3) Conservative.

A. High output

- >1000 ml/24 h.
- Surgical exploration—oversewing/tissue glue/pressure dressings.

B. Low output

- Supraclavicular pressure dressings.
- Fluid/leakage balance.
- Fat free or high medium chain triglycerides (MCT) product or TPN.
- MCT: directly absorbed into portal system—reduces chyle production. If unsuccessful, parenteral nutrition.
- Video Assisted Thorascopic Surgery (VATS)—used to repair defect (ligation thoracic duct).
- 80% in the left neck.
- Flow chyle 2–4 L/day: fat/protein/electrolytes/lymphocytes—production depends on diet
  - Thoracic duct: anatomical variation—3–5 cm above clavicle, towards junction of subclavian vein with IJV.

9.3 Restorative/Oral Rehabilitation

Important to include restorative consultant dentist/oral rehabilitation for head and neck cancer patients.

Assessment prior to treatment—aims:

- Pre-prosthetic planning/treatment—implants/obturators.
- Extraction of teeth—doubtful prognosis.
- Planning for restoration/advise treatment.

Treatment side effects

- Treatment surgery/chemo-/radiotherapy—short/long-term oral side effects

  A. Short term

  - Mucositis/infection—chemo-induced neutropenia—bacteria/viral/fungal (oral candidiasis is commonest).
  - Xerostomia: dry mouth with reduced salivary flow.

  B. Long term

  - Altered anatomy/dental caries: reduced salivary flow and radiogenic damage to amelodentinal junction.
  - Trismus—surgical scarring/radiotherapy induced fibrosis.
  - ORN/xerostomia—IMRT to reduce risk of xerostomia.

Management

- Prevention

  – Oral hygiene/diet to reduce caries/daily topical fluoride (custom made trays or brush).
  – Saliva replacement.

(a) Oral mucositis

- Chinese medicine/hydrolytic enzymes/ice chips/benzydamine hydrochloride/manuka honey/zinc sulphite/calcium phosphate/etoposide bolus.
- Specific patient responses.

(1) Candida

- Anti-fungal drugs prevent nystatin. Chlorhexidine has anti-fungal/anti-bacterial/.

(2) Xerostomia

- Sipping sugar-free fluids/sugar-free gum/lozenges.
- Carboxymethylcellulose saliva substitute as mouthwash.
- Oral gel.

- Pilocarpine: 5–10 mg/day—improves radiation induced xerostomia in patients.

(3) Trismus

- Therabite—prior/during radiotherapy.

(4) Implants

- Primary: Maxillary/rhinectomy/orbital.
- Jaws: Prosthodontic planning/risk factors/oral hygiene.

Psychological

- Communication of diagnosis and treatment.
- Psychological distress: HADS scale/anxiety/depression/fear of recurrence.
- Specialist mental health intervention.

9.4 Quality of Life

- Evaluation integral to optimal: body image/facial disfigurement.
- WHO: "Individual perception of their position in life in the context of culture/value systems in which they live and in relation to their goals, expectations, standards and concerns".
- Health related quality of life (HRQOL): More disease specific, allows assessment of it impact of disease and treat on physical/psychological/social aspects.
- Why?

  – HRQOL: indication how patient perceives impact of their cancer and treatment
    (a) Evaluate outcomes.
    (b) Define treatment protocols.
    (c) Individual decision making.

Problems

(1) Burden of admin/weighted to survivors/little evidence agreed.
(2) Lack of evidence has major role in treatment decisions.

How

– Patient self-completed questionnaire—no gold standard.
– Key issues: lack of long-term outcome/new treatment strategies not given enough time for HRQOL information.

Examples—HRQOL change practice.
Oral cavity

– Primary surgery/free tissue reconstruction.

HRQOL

- With technology easier.
- Assist decision making/delivering information/identifying problems.

University of Washington

- QOL questionnaire.
- Last 7 days.
- 12 parameters: pain/appearance/activity/recreation/swallow/chewing/speech/shoulder/taste/saliva/mood/anxiety.

Neck dissection impairment index—last 4 weeks

- Neck shoulder (discomfort).
- Neck shoulder activity.

  Limited to lift objects (light/heavy).
  Self-care/work.

9.5 Prognostic Markers

TNM

- Staging—good correlation with prognosis.
- Other standing systems
  - Bowel cancer—Dukes' Classification.
  - Hodgkin and non-Hodgkin lymphoma—Ann Arbor staging system.

Tumour Thickness

- Depth of invasion: correlated to prognosis and metastasis to the regional nodes.
- Obnan 2003: Prognosis changed significantly at 4 mm thickness for T1/T2 oral SCC all sites.
- D'Cruz-Elective vs. therapeutic neck dissection NEJM 2015—END higher overall and disease-free survival. Depth of primary tumour has relationship to regional spread (4 mm).

Nodal Classification

- Most important reliable prognostic factor in oral cancers.
- Preserve the cervical lymph nodes—reduce case by 50% (Shah).
- Woolgar 1995–5 year survival falls from 86 to 44% with nodal metastasis.
- Spiro T1–2: 5 year survival—N0 (90%), N1 (<50%).

Size and ECS

- Woolgar 1997: IJOMS poor prognosis of patients, entirely due to poor survival in ECS.
- Noguchi 1999: pN stage significantly influenced survival especially ECS.

Histological Factors

Broder: well-differentiated/moderate/undifferentiated—no correlation with survival.
In T1/T2.
Vascular and PNI signs of aggressive tumour, these values individually influence prognosis.

Surgical Margins

- Lack of consistency in the literature regarding the definition of "close resection margin".
- Several studies/papers—more than 1–2 mm not associated with significant local recurrence. Risk of local recurrence increased if less than 1 mm.

- ***No Clear Evidence of Margins >2 mm Shows as Good Local Control as 5 mm***.

Histological margins: The UK Royal College of Pathologists' guidelines for screening of margins propose: "Mucosal" and "deep—include submucosa, skeletal muscle, and bone".

Subdivided into clear, close, and involved.

- Clear/negative margin: Histological distance of >5 mm from the invasive carcinoma to surgical margins (SMs).
- Close margin: Histological distance of 1–5 mm from the invasive carcinoma to SMs.
- Involved/positive margin: Histological distance of <1 mm from the invasive carcinoma to SMs.
- 1 cm—histological.
- Dysplasia not a positive margin.
- 15–30% close margins in best hands especially large/posterior + LN. Indicates more aggressive biological disease.
- Close margin 2–6 × risk death despite PORT (Sutton 2003).

Batsakis

- Clear >5 mm from microscopic tumour margin.
- Close <5 mm.
- Involved at tumour margin <1 mm.
- Carcinoma in situ/dysplasia at margins not considered.

Tumour Markers

- HPV p16—oral SCC = better outcomes than tobacco mediated disease.
- 5 years surgical
  - p16 positive (57%).
  - P16 negative (26%).
- p16 important surrogate marker for HPV.
- P16 over expression related to HPV E7 protein.

  E7: binds/inactivates Rb—increases p16 due to loss of biofeedback.

9.6 Neck Disease

Presence/absence of level and size of metastatic neck is the most significant factor in head and neck SCC.

Crile: RND 1905

- LN status single most important prognostic factor (decrease survival by 50%).
- 300 LN in neck, Crile first to describe management of lymphatics in head and neck cancer.
- Hayes Martin 1951: Radical neck dissection.
- Osvaldo Suarez 1968: Functional neck dissection without compromising oncological principles.

Assessment

- Clinical palpation: sensitivity/specificity—70–80% inadequate.
- CT/MRI: sensitivity 81%
  - CT more specific.
  - PET-CT not shown distinct advantage.
- USS/FNAC:
  - High sensitivity 87% = expertise/experience/operator dependent.
  - Very high specificity 98% = No absolute US characteristiscs.
- Sentinel node
  - Sensitivity >90%.
  - False negative of 5% (skip lesion).

Treatment Options

Neck Dissection

- Comprehensive (I–V).
- Selective (<V)
  - Radical: I–V = IJV/sternomastoid/accessory.
  - Modified radical I–V preserving some structures.
  - Selective—preserve some levels.
  - Extended radical = additions VII/retropharyngeal/hypoglossal (Table 9.3).

Recommendations

- NICE recommends all oral cavity SCC neck dissection or sentinel node biopsy.
- Elective versus therapeutic neck dissection in node-negative oral cancer—A K. D'Cruz—Elective neck dissection results in higher rates of overall and disease-free survival vs. therapeutic neck dissection.
- Systematic review and meta-analysis: The findings confirmed that elective neck dissection in oral carcinoma with N0 neck can significantly reduce disease-specific death rate—A. J. Fasnula et al. Oral Oncology (2011).
- Sentinal node biopsy—Evidence that elective neck dissection (END) has 7% failure rate. Disease free survical with sentinel node is similar to END. But get cost savings, and bed days in UK healtcare system. Morbidity is less.

What are the predictors

- Only tumour thickness—relate with nodal metastasis = recommended 1.5–8 mm (huge variation).
- How it is measured—depth of invasion.
- Biopsies: incisional.
- Where is the deepest part?
- MRI/US guided.

Additional Notes

- High prevalence of micro-metastases (i.e. foci of tumour within lymph nodes smaller than 3 mm) when lymph nodes were serially sectioned, as opposed to the sampling technique of bisectioning lymph nodes that is used at most hospitals.
- The rates of these micro-metastases ranged from 16 to 25% in previously pathologically staged N0 necks.
- Although the prognostic significance of these micro-metastases is still unclear, at least some of these tumour cells may be ultimately responsible for neck recurrences seen even in pathologically N0 necks.
- Level of evidence is poor—lack of high quality of RCTs.

Table 9.3 Neck dissection levels

Level	Anatomic name	Key borders and landmarks	Primary drainage site
I	IA = Submental IB = Submandibular	Hyoid Bone Posterior margin of submandibular gland IA or IB—anterior belly of digastric muscle Inferior margin of hyoid	Anterior oral cavity, lip, sinonasal
II	Anterior cervical or upper jugular	Inferior margin of hyoid Posterior margin of submandibular gland Posterior margin of sternocleidomastoid IIA or IIB—posterior margin of internal jugular vein	Oropharynx, posterior oral cavity, supraglottic larynx, parotid gland
III	Middle jugular	Inferior margin of hyoid Inferior margin of cricoid	Glottic, subglottic, and hypopharyngeal regions
IV	Lower jugular	Inferior margin of cricoid Clavicle	Subglottic, thyroid, and cervical oesophagus
V	Posterior compartment or spinal accessory	Posterior border of sternocleidomastoid Clavicle VA or VB—inferior margin of cricoid	Nasopharynx; skin carcinomas of the neck or occipital scalp
VI	Visceral or central compartment	Medial margins of carotid arteries Inferior margin of hyoid Superior aspect of manubrium	Subglottic; thyroid and cervical oesophagus
VII	Superior mediastinal Retropharyngeal Parotid Supraclavicular fossa	Superior aspect of manubrium Innominate vein Medial margin of internal carotid arteries to the level of hyoid Within parotid gland On axial images at or below the clavicle, lateral to the medial edge of the common carotid artery, and medial to clavicle; includes some level IV and V nodes	Subglottic; thyroid and cervical oesophagus Nasopharynx, oral cavity, sinonasal, thyroid and pharyngeal and laryngeal tumours with posterior wall involvement Skin of scalp, orbit, nasopharynx Any head and neck cancer and cancers of the thorax and abdomen, including lung, breast, oesophageal, gastric, pancreatic, gynaecological, and prostate cancers

- Lymph node status—single most important prognostic factor.
- Prognosis
  - Affected by involved nodes, level in neck, tumour load, ECS, PNI, vascular invasion, previous treatment.
- Large number of malignant nodes that CT/MRI do not identify
  - <10 mm.
  - 2 mm can have ECS.

Management Strategies = N +

- Treatment of choice for N + : Guided by treatment to primary site.
- Salvage surgery considered for partial response failing chemo-radiotherapy.
- EORTC/RTO—RCT: Improved control with chemo-radiotherapy ECS/positive margin.
- Patients with >2 or more nodes but no ECS = No benefit from chemotherapy.

Management of recurrent disease

- Prior to salvage treatment—assess distant metastasis PET-CT.
- If recurrence after RT/CRT and resection = surgery should be offered.
- Higher complications.
- If unresectable, then re-irradiation with/without chemotherapy.

9.7 Complications of Neck Dissection

Pre-operative Evaluation

- Increase nutrition.
- Tobacco cessation (even 12–24 h).
- Decrease alcohol intake.
- Avoid delirium tremens.

Anaesthesia

- LMWH.
- Pneumatic compression.
- Elastic stockings.
- Avoid hypo-/hyperglycaemia (CVP/Lid-CO).
- Fluid optimisation.

Post-Operative

- Intra-operative lignocaine on carotid sinus—avoid arrhythmia.
- Carotid sinus sensitivity—carotid body—acute bradycardia—atropine.

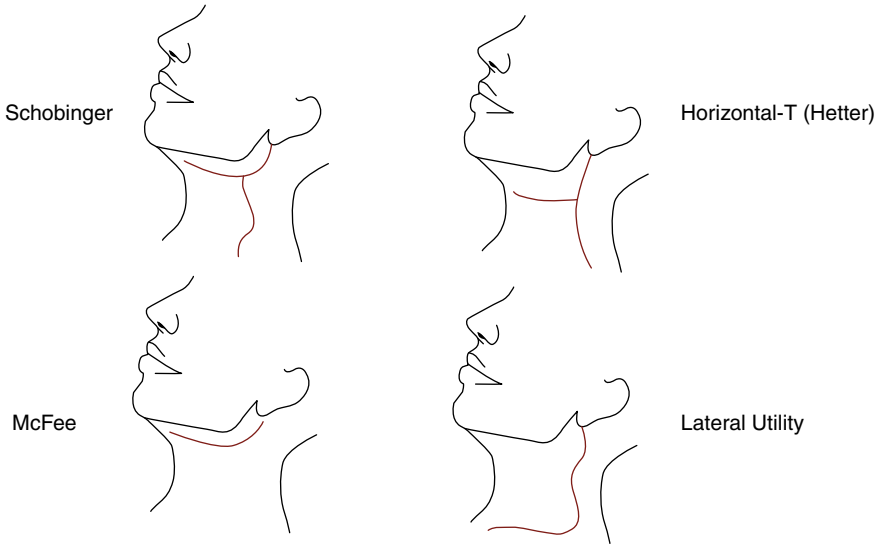


Fig. 9.2 Neck dissection skin incisons

- QT Prolongation = right sided neck = K^+ level.
- Aggressive respiratory support = clear secretion/early mobilisation.
- Shoulder improved with proactive physiotherapy.

Surgical Technique

- Asepsis: surgical preparation/removal of beard.
  - Incisions: apron/wine glass Cervical skin BS: external carotid/subclavian (Fig. 9.2).
  - Avoid incision parallel to carotid especially salvage.
  - MacFee.

Flap Elevation

- Subplatysmal—maximise blood supply, not dry out.
- Tracheostomy away from main surgery field.

Progression Neck

- SCM, omohyoid, IJV, digastric.

Air Embolus

- Rare, injury IJV; sudden fall in end tidal CO_2 and decrease in blood pressure.
- Patient placed in Trendelenburg position and rotated left.
- Severe: catheter to aspirate from right side heart; HBO is an effective treatment.

Pneumothorax

- Tears closed—Trendelenburg position.
- Head down: irrigate clear fluid—spot bubbles, may need CXR ± chest drain on tube.

9.8 Chyle Leak (See Nutrition Section for More Info)

Thoracic duct: cisterna chyli—between aorta/azygous vein

- Intra-operatively: Trendelenburg position/or forced valsava manoeuvre—post-operative feeding.
- Treatment: surgical/pharmacological—no definite evidence.
- Chyle leak—sclerosing agents, such as doxycycline or fibrin glue have been advocated. Oversewing the area and consideration of importation of vascularised tissue, such as a myofascial free or pedicled flap.

Neural

- Prevent neuroma: ligation/cautery/burying—no evidence proven value.
- SAN—Shoulder syndrome.
- Ansa cervicalis frequently sacrificed.
- Preserve vagus/hypoglossal/marginal mandibular.
- Hypoglossal
  - Crossing external carotid, emerges posterior belly of digastric on hypoglossus.
- MM
  - 1 cm below mandible, divide facial vessels—Hayes-Martin.
- Phrenic
  - Diaphragm, if pulmonary problems—prolonged ventilation.
- Carotid plexus
  - Sympathetic trunk—Horner's syndrome.
  - Horner's syndrome (i.e. ptosis, miosis and anhydrosis) results from injury to the cervical sympathetic trunk, which lies posterior to the carotid sheath vagus—unilateral VC paralysis—weak/breathy voice.
- Brachial plexus
  - Between scalenus anterior/medius muscles—crosses posterior triangle.
  - Vagus nerve: cardiac problems.

Drains

- Active—no evidence of affecting free flaps.

Extended RND

- Balloon occlusion test—ICA.
- Still develop neurological symptoms.

Bilateral Neck

- Increase rates of infection/fistulas.
- Facial oedema/swelling, especially IJV cut.
- Increased ICP—bilateral IJV ligation = secondary system hypertension (Cushing's).
- Even if IJV preserved—30% get thrombosis—keep wet/meticulous technique.
- If cut IJC = try and persevere external jugular vein.

Previously Irradiated Neck

- Fibrous, no tissues planes, vessels difficult to find.
- Higher rate of complications.

Carotid Blowout

- 50% mortality—emergency ligation vs. elective endovascular stenting.
- Muscle flap to protect vessels after neck dissection (pectoralis major).
- Fascia lata, dermal grafting.

Chapter 10
Head and Neck Cancer Part 2

Abstract A Summary of the assessment and management of cancers of the head and neck.

Key Points

- Oral cavity and lip.
- Mandible.
- Oral and pharyngeal tongue.
- Pharyngeal tongue.
- Alveolar ridge/retromolar triangle.
- Gingiva.
- Buccal mucosa.
- Maxilla.
- HPV and oropharyngeal cancer.
- Nasopharyngeal carcinoma.
- Thyroid cancer and thyroid disease.
- Cancer with unknown primary.
- Skull base tumours.
- Radiotherapy.

Oral Cavity and Lip

- Anterior 2/3 of tongue, FOM, buccal mucosa, retromolar triangle, hard palate gingiva
  - Mainly SCC.
  - Now increasing SCC cases found in salivary glands.
- Lip:
  - Most common malignant tumour affecting head and neck.
  - Similar to that of skin cancer.
  - 12/100,000 Europe.
  - Factors: solar radiation, tobacco smoking, viruses could be found 7% in upper lip/3% oral commissure, 90% lower lip.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_10

Pathology

- De-novo or from leukoplakia/erythroplakia
  - Carcinogens: tobacco/alcohol/local trauma.
  - Carcinogenesis: Multiple steps in process. Inactivation of tumour suppressing gene p53.

    HPV: Expression of p16 oncoprotein.
    EGF: unclear—overexpression related to poor prognosis, does not correlate with response to cetuximab.

Subtypes

- Verrucous (better prognosis).
- Basaloid (worse).

Classified According to Grade (Various)

- Limited prognostic value due to heterogeneity of tumour.

Prognostic Significance

- Tumour thickness: oral tumours SCC >4 mm significant risk of lymph node metastasis.
- ECS of nodal metastasis: poor prognosis—increased risk of locoregional recurrence (distant metastasis/reduced surgical rate).
- Pattern of invasion (non-cohesive/peri-neural): increase risk of locoregional relapse.

Management

- No RCT comparing the different treatment modalities.

Surgery

- Patient factors fitness for anaesthesia/treatment/resources.
- Curative surgery: Resection/reconstruction.
- Transoral route: Circumferential muscular sphincter maintained.
- Posterior—Lip split/mandibulotomy.

Methods

- Scalpel/laser/diathermy/Harmonic scalpel.
- Small/superficial
  - Laser vaporisation—do not get histological assessment.
  - Lasers/thermal technique reduces risk of bleeding—histological artefact, morphological distortion.

Ideal Margins

- 1 cm clinical, close pathological margins <5 mm.
- No clear evidence of margins. >2 mm shows as good local control as 5 mm.
- Frozen section: Controversial, helps in recurrent disease and complex cases.
- Toludine blue: High false positives in margins with limited values.
- Lugol's iodine: For dysplasia at margins.

Bony Resection

- Clinical/radiological.
- Intra—operative: Periosteal stripping—relative, frozen section of cancellous bone.

Planning Resection/Reconstruction/Both

(1) Transoral.
(2) Lip split/mandibulotomy.
(3) Visor, drop/down approach.

Lip Split/Mandibulotomy

- Lip split may be used to gain access to tumour of buccal mucosa.
- Incision through labial mucosa/gingiva/stepped, so not above osteotomy cut.
- Periosteal elevation/mentalis stripping/mucoperiosteal flaps pedicles on non-tumour side.
- No incision through lingual periosteum—pre—localise plates and screws.
- Straight osteotomy cut—following division.
- Mandible retracted laterally—lingual soft tissue stripped off mandible.
- Others: Angle osteotomy/double mandibular osteotomy—infratemporal fossa/pharyngeal spaces.
- Close of lips in 3 layers—attention to orbicularis oris/vermillion repair.

Pull Through Technique

- Large lesion anterior floor of mouth—transcervical pull through.
- Bilateral neck dissection usually performed, segmental osteotomy of lower border of mandible (not common practice).
- Cut a segment of bone containing the attachments of the anterior belly of digastric, geniohyoid, past of the attachment of genioglossus
  - Divide mylohyoid.
  - With tongue/FOM released from lingual surface of mandible—pull through the neck.
  - Accurate reattachment is important for swallowing function.

Resection of Tumours Involving Mandible

- Tumours of tongue/FOM/retromolar triangle invade the mandible.
- Treatment: Very important decision in the management of the mandible.

- Erosive not T4, only infiltrative T4.
- Alveolar not T4, only basal bone.
- CT—cortical bone erosion, MRI—marrow signal changes.
- Different types of invasion pattern described; a number of routes suspected
  - PD membrane.
  - Occlusal surface.
  - Mental foramen.
  - Cortical defects in the edentulous ridge.
  - Attached gingivae.
- Main paper (J Brown Head and Neck 2001).
  - Pattern of invasion and routes of tumour entry into the mandible by oral SCC—perspective.
  - Hypothesised major portal of entry—point of abutment, which is often at the junction of attached and unattached mucosa below the crest of the ridge (Sharpey's Fibres of the attached mucosa).
  - Rim resection angulated to the lingual aspect of the mandible.
- Two patterns of mandibular invasion described
  - Infiltrative (invasive)

    Islands and processes of tumour tissue inside the bone with little osteoclastic activity.
    More aggressive tumour biology and worse prognosis.
  - Erosive pattern
    - Interfering connective tissue later and active osteoclasts between the mandible and tumour.
    - i.e. Erosive: Osteoclasts break down alveolar bone—deeper bone invasive pattern (Slootweg and Müller 1989).
- No evidence indicates to resect IAN in resection of the mandible.

Surgical Management

- No resection.
- Marginal/rim—functional lower border, include lingual cortical plate.
- Segmental—disturbing continuity, increased morbidity, reduced quality of life.
- Posterior defects—cause only minor cosmetic/functional impact.
- Anterior defects—lip fixation/speech/deglutition.

Recommendations for Segmental

- Extensive invasion of mandible—radiology.
- Extensive and deep soft tissue infiltration close to mandible.
- Tumour seen to approximately involve whole height of mandible.

- Rim resection—insufficient residual mandible—risk of fracture if it is less than 10 mm.

Recommendations for RIM

- Depth of invasion <5 mm.
- Pre-operative scanning: no evidence mandibular invasion, but periosteal stripping at time of operation reveals mandibular invasion.

Investigations

- OPG, CT (specificity 88%), MRI (specificity 86%), SPECT (most sensitive).
- MRI demonstrates enhanced marrow signal when mandibular involvement and depth of tumour.

Tongue Cancer

- Oral tongue—anterior circumvallate papilla.
- Accounting for 40% of oral cancers, mostly found on

  (a) Lateral border.
  (b) Ventral surface.

- Usually present early as visible (compared to asymptomatic lesions on base of tongue).
- Clinical presentation

  (1) Stage I—37%.
  (2) Stage II—34%.
  (3) Stage III—21%.
  (4) Stage IV—8%.

    – Spread along mucosal surface—floor of mouth.
    – Deep invasion: muscle fibres provide pathway with no fascial barriers.
    – Sometimes difficult to palpate—imaging.
    – The midline raphe of the tongue does not provide substantial resistance, spread across midline easily.

- Clinically (Woolgar 1999)

  – 54% T2 with neck metastasis level I/II.
  – Embolus—jugulo-omohyoid at level IV skip metastasis (10%).

- Wide local excision with 1 cm normal tissue to ensure adequate margins.

Margins and Neck Management

1 cm clinical, close pathological margins <5 mm.

- No clear evidence of margins. >2 mm shows as good local control as 5 mm.
- Frozen section—controversial, helps in recurrent disease and complex cases.

- High incidence of occult metastasis >30% T1/T2 clinically N0 neck.
- Current evidence: recommend SND or SNB (recommendations following D'Cruz paper).
- 29.6% of patients had occult neck nodes identified by elective neck dissection.
- NICE recommends offering all patient SND (selective neck dissection) or SNB (sentinel node biopsy).
- Neck dissection could have staging/therapeutic benefits.
- Larger lesions requiring near total glossectomy—high morbidity and aspiration incidence
  - May ultimately require laryngectomy for aspiration control.
  - Treat with radiotherapy alone—unacceptable failure rate.
  - Chemo- and radiotherapy—surgery for salvage or residual/recurrent disease.
- Post-operative XRT
  - PNI (perineural invasion)/LVI (lymphovascular invasion).
  - Margins close <2 mm.
  - Multiple positive nodes or ECS.
- Prognosis
  - Poorer than other sites.
  - More aggressive in younger women.
  - 5 years prognosis = stage I + II (70%)/stage III + IV (50%) = overall 40–60%.
  - 30% developed secondary tumour (head and neck—primary site).

SCC Pharyngeal Tongue

- Posterior to circumvallate papillae.
- Grow silently/early lymph node metastasis.
- Frequent cause of unknown primary.
- Present with neck mass, often N3, primary tumour often asymptomatic.
- Can have pain/dysphagia/bleeding history.
- Direct laryngoscopy with random biopsies—pharynx/tongue base.
- Robotic mucosectomy.
- Small T1/T2—surgery = XRT.
- Large: Organ sparing approach (chemo-radiotherapy).
- Overall 5 year prognosis: 15–25%.
- Reconstruction
  - Speech and swallowing once tumour across midline or posterior tongue.
  - Presentation tip priority—tongue can go to root, enhance pronunciation/and swallowing.
  - Anterior tongue—RFFF/MSAP.
  - Posterior—ALT, pectoralis major.

Floor of Mouth

- 25% of cancers (common).
- Pooling saliva, usually extend to involve tongue/lingual gingiva
  - T1: 30%.
  - T2: 37%.
  - T3: 19%.
  - T4: 14%.
- 41% regional neck metastasis.
- 15% micrometastasis.

Treatment Failure

- 21% local.
- 37% neck recurrence.
- 21% Both sites.

Surgical

- Stage I: 88%.
- Stage II: 80%.
- Stage III: 66%.
- Stage IV: 32%.

- 30% of T2 present with occult neck metastasis.
- Assessing tumour thickness essential.
- Usually bilateral neck.
- SNB for small tumours.
- Occult nodes 22% with tumour >2% (anywhere)—low threshold for rim resection
  - Radiotherapy (brachytherapy): risk of osteoradionecrosis.

Alveolar Ridge/Retromolar Triangle

- Retromolar triangle (15% of all oral SCC)—very small area, usually affects adjacent areas
  - Oropharynx (soft palate/anterior tonsillar pillar), buccal mucosa, gingiva, tongue, mandible.
  - Difficult to assess

    Posterior/mucosa lingually/small area.
    Trismus—pterygoid involvement.
  - Difficult to treat—spread early to deep structures

    Ascending ramus.
    Pterygoid.

Masticatory space.
Skull base.
Foramen of IAN—trigeminal ganglion (CNV).

- Difficult surgical access.
- Usually recurrence—segmental resection: ramus/body.
- Overall 5 year survival: 40–60%.

Gingiva

- Attached gingiva only (<10% all intraoral carcinomas)—indolent, slow growing, low metastasis (local/neck metastasis).
- Smaller lesions: both buccal/lingual gingiva and alveolar bone resected to level of root apices.
- Larger lesions: partial maxillectomy/segmental mandibulectomy + END.
- Excellent 5 year prognosis >85–90%.

Buccal Mucosa

- 10% Intraoral, 40% nodal
  - Infiltrate masseter.
  - Parotid.
  - Alveolus inferiorly.
  - Pterygoid space posteriorly.
- More aggressive, 40% present stage III/IV occult node metastasis 25%.
- Low threshold lip/split.
- Most require reconstruction—trismus.
- Reconstruction:
  - Nil—secondary intention.
  - Primary closure.
  - SSG, buccal fat pad.
  - Free flap—radial.
- 5 year overall survival: 40–60%.

10.1 Maxillary

- Maxillary tumours: 39% of all head and neck tumours.
- 75% malignancies
  - 85% epithelial origin.
  - Remainder: salivary gland.

- 5 year survival: 30–50%—commonest 5th/6th decade.
- Pre-operative planning.
  - Most importantly—determine reconstruction (prosthetics/biological tissues).
  - Always have cover plate just in case.
  - Obturators

    Reduce surgical times, no donor site morbidity, returns ability to inspect cavity.
    Demands patient compliance.
    Need interim obturator changes under general anaesthesia.
    If obturation is to be performed a simultaneous coronoidectomy should be carried out.

  - Access

    Transoral: can be supplemented with midface degloving.
    Transcutaneous: Weber Ferguson can have lateral extension (Dieffenbach's)—skin flaps in submandibular plane to maintain blood supply and reduce risk of facial nerve damage.

Maxillary Approaches

- Weber Ferguson (1845—Weber)—tumours involving anterior and superior aspect of the maxilla (not involving retro-maxillary space).
- Maxillary Vestibular Approach.
- Midface degloving.
- Lip split mandibulotomy.
- Non lip split mandibulotomy.

HPV

- Overexpression of p16: A cell cycle checkpoint regulator—surrogate marker for productive HPV infection in OPSCC.
- Mechanism.
- High risk HPVs encodes two known viral oncogenes, E6 and E7. E6 protein inactivates tumour suppressor gene p53 mediated DNA damage and apoptosis pathway.
- E7 protein binds tumour suppressor pRb mediated cell cycle regulation pathway.

Oropharyngeal Cancer

- Increase in developing countries.
- HPV 16 predominant subtype responsible for the proportion with evidence of HPV infection, which is rising rapidly.
- Data from recently published studies: 55% of 654 cases reported oropharyngeal SCC cases were HPV +ve.

Clinical

- Painless neck lump, sore throat, tongue, otalgia, dysphagia, odynophagia.
- Voice changes (hot potato voice).

Examination

- FNE.

Imaging

- MRI—primary site, CT scan neck + chest.
- US FNA—neck.
- CT—primary site: assess bone invasion
  - Body mandible/skull base—tonsillar tumour.
  - Cervical spine—posterior pharyngeal wall.
- PET CT—staging, assess response after therapy and assessment for recurrence.

T Stage

- T4a: Tumour invades larynx, deep/extrinsic muscles tongue/medial pterygoid/hard palate/mandible (same as oral SCC).
- T4b: Skull base/internal carotid artery (encasement)/lateral pterygoid/pterygoid plates.

Pathology

- USA—HPV testing (PCR/ISH).
- HPV +ve has better prognosis, alcohol/smoking confounding factor—worsen survival rates.

Management

- T1/T2 N0
  - Primary surgery—81–100%.
  - Radiotherapy—77–89%.
- Surgery
  - Transoral: Lasers, TORS (transoral robotic surgery).
  - Open: mandibulectomy, reconstruct with flap—radial/ALT.
- TORS—Trials ongoing, margins.
- More patients may require post-operative radiotherapy/chemo-radiotherapy, results comparable to open surgery.
- About 10–31% clinically no occult nodal disease
  - All with primary surgery—neck dissection.
  - Treat contralateral if near midline: level I–IV, not needed level IIb (retrospective studies).

- Radiotherapy
  - 60 Gy in 30 fractions.
  - Or 4 weeks hypofractionated.
  - Techniques to avoid parotid—IMRT

    No evidence or benefit in swallowing function or trismus.
    IMRT reduces risk of ORN.

- T3/T4/N0-3 (advanced)
  - Review literature—Cochrane report 2009.
  - Only evidence of statistically significant benefit was addition of concomitant chemotherapy to post-operative radiotherapy.
  - Offer radiotherapy/chemo-radiotherapy—as 'organ preservation strategy' (not the same as larynx).
  - Surgery

    Overall surgical—best with radical surgery and post-operative radiotherapy.
    Functional results poor.
    TORS: base of tongue/pharyngeal wall/tonsil ± radiotherapy (margins ECS)—cure rate as good as chemo-radiotherapy, whilst preserving speech and swallowing.

  - If TORS not appropriate with large primary tumour: Transcervical with lip split/mandibulotomy and reconstruction, functional outcomes can be good. If soft palate involved—poor functional outcome.
  - Chemo-radiotherapy.
  - Organ preservation—effective treatment choice.
  - 70 Gy in 2 Gy fractions with concurrent cisplatin.
  - Advanced case stage IV—may warrant induction chemotherapy.

- HPV: profound influence on prognosis—de-escalation regimes.

Nasopharyngeal Carcinoma (NPC)

- SCC—Nasopharynx: common site fossa of Rosenmüller, just medial to medial crura of Eustachian tube.
- NPC—Frequently found in southern Chinese, North African, Alaskan.

Aetiology

- EBV.
- Salted fish: containing dimethylnitrosamine (DMN).
- Genetic: deletion of chromosomal reigns 1p/14q/16p application 4q12q.

Clinical

- Male:Female 3:1—Median age 50 years.
- Symptoms

  - Nasal obstruction.
  - Epistaxis.
  - Conductive hearing loss with OMS—Eustachian tube dysfunction.
  - Cranial nerves: III, IV, V, VI.
  - Neck lumps, often bilateral.

Pathology—NPC

- Non-keratinising carcinoma (commonest).
- Keratinising carcinoma.
- Basaloid squamous carcinoma.
- Immunochemistry: identifying keratin filament (AE1/AE3 and MNF116 antibodies).
- All non-keratinising harbor EBV
  - In situ hybridisation.
  - Serological markers for EBV—adjunct monitor disease progression and response to treatment.
- Detection of
  - Viral capsid antigen (VCA).
  - Early antigens (EA).

Imaging

- CT/MRI/US/FNA/PET-CT.

Staging

- T1: tumour confined to nasopharynx, or extends to oropharynx or nasal cavity.
- T2: tumour with parapharyngeal extensions.
- T3: tumour invades skull base/paranasal sinus.
- T4: tumours with extracranial extension/involvement cranial nerve, infratemporal fossa hypopharynx, orbit, masticator spaces.
- N1: Unilateral metastasis less than or equal 6 cm above supraclavicular fossa.
- N2: Bilateral metastasis less than or equal 6 cm.
- N3: Metastasis >6 cm or supraclavicular fossa.
- N3a: >6 cm.
- N3b: Supraclavicular fossa.

Management

- Radiotherapy mainstay for radical treatment for NPC—improved with use of chemotherapy better disease control/survival = IMRT.
- Concurrent chemo-radiotherapy offered significant improvement in overall surgical in stage III and IV disease.
- Primary surgery: Tissue diagnosis/treatment for otitis media.
- Stage I and II—RT.

- Stage III and IV—CRT.
- Follow-up/treatment response: 3 months for assessment for treatment.
- Residual and recurrent
  - Surgery: transnasal/transoral endoscopic resection.
  - Anterolateral approach with maxillary swing.
  - Local control (62%).
  - Palatal fistulae occur in 20–25% of patients.
- Neck: re-irradiation—high risk tissue necrosis/fibrosis.
- Salvage radial dissection—may need to excise involved skin and reconstruct with free flap.
- 5 years local control = 66%.

Thyroid Cancer

Differentiated Thyroid Cancer

- Male (1%), female (3%).
- Rare.
- Increased survival status—survival 80–98% with 10 years follow-up.

Factors Affecting Prognosis

- Gender.
- Age.
- Histology.
- Staging.

Several Guidelines—Thyroid MDT

- Surgeon.
- Endocrinologist.
- Oncologist.
- Nuclear medicine physician.

2-Week Referral

- Hoarseness/voice changes—associated with node/goitre.
- Cervical lymphadenopathy and thyroid nodes.

Clinical

- TSH.
- US/FNAC—documented cytological score (Table 10.1).
- Core biopsy—if suspect lymphoma.
- Pre-operative vocal cord check.

TNM

- T1: <2 cm.

Table 10.1 Cytology score for thyroid lesions

Scale	Diagnosis	Action
Thy 1	Non-diagnostic	Repeat FNA—describe as cyst if no epithelial cells present
Thy 2	A-C non-neoplastic	Colloid nodule/thyroiditis—repeat FNA if no surgery plan
Thy 3-3F	Follicular lesion	Suspected follicular neoplasm MDT: Diagnostic lobectomy/total thyroid if >4 cm
Thy 3A	Atypia present	MDT: Diagnostic lobectomy—large lesions increase risk of malignancy
Thy 4	Suspicious not diagnostic of thyroid cancer (papillary/nodular/anasplastic lymphoma)	MDTR; Diagram checking ± on table frozen section to proceed total thyroidectomy ± central node clearance
Thy 5	Thyroid cancer	MDT: radio/chemo/surgery—staging = total thyroidectomy

- T1a: less than or equal 1 cm—limited to thyroid.
- T1b: >1 cm—less than or equal 2 cm limited to thyroid.
- T2: >2 cm—less than or equal 4 cm.
- T3: >4 cm—limited to thyroid or minimal extrathyroid extension.
- T4a: Any size—invading capsule/subcutaneous tissue/larynx/trachea/oesophageal /recurrent laryngeal nerve.
- T4b: pre-vertebral fascia/encasing carotid.
- T4a: intraglandular anaplastic carcinoma.
- T5b: Anaplastic carcinoma with gross extraglandular extension.
- N1:
  - N1a—level VI.
  - N1b: level 1–V.
- Management
  - Surgery MDT—high risk have level VI

    Male.
    Age 40–50.
    Extracapsular.
    Extrathyroidal disease.
    60% affect neck nodes—90% 10 year survival

    (1) Papillary

    - <1 cm—lobectomy.
    - 1 cm—total thyroidectomy.
    - T3/T4 + N1: high risk—total thyroidectomy + level V.

(2) Follicular

- Age 50–60.
- Well-defined capsular spread via blood stream, 30% bone metastasis (worse prognosis).
- Usually diagnosed by chance as patients don't experience symptoms in early stage.

(a) High risk: total thyroidectomy.
(b) Low risk: thyroid lobectomy.

(3) Anaplastic

- Age >70, more common in females with rapid enlargement.
- Airway compromised/changes in voice/swallowing issue.
- 95% die within 1 year.
- Locally advanced disease: radiotherapy is often the main treatment.

- Post-operative
  - Total/near total.
  - Thyroid hormone replacement—thyroxine.
  - Check Ca^{2+}.
  - Tg (thyroglobulin) check after 6 weeks.

Radioiodine

- Give to tumour >4 cm/metastasis/extrathyroid.
- 10 nodes.
- 3 weeks with ECS.
- Follow-up.
- Post-operative VC check.
- Reduce Ca^{2+}—review with endocrine.
- Long-term suppression TSD <0.1.
- Monitoring thyroglobulin levels (Tg): Best following total near/total thyroidectomy and radioiodine. Important to detect recurrent/residual disease every 6–12 months.
- Iodine-131 whole body scanning (WBS/and USS at 6–9 months: Further WBS only necessary if increase Tg levels, USS useful in high-risk patients.
- Lifelong follow-up.
- Clinical examination.
- Serum Tg and TSH suppression.
- Graves' disease.
- Autoimmune hyperthyroidism.
- Eye signs.
- Treatment: Carbimazole.
- Hashimoto's thyroiditis.

- Autoimmune: Hypo-/hyperthyroidism.
- May need thyroxine.

10.2 Unknown Primary

SCC

- LN(s) with no primary index site in head and neck identified.
- Tests assisted through neck lump clinic—LN = US FNA as suspected SCC.

Clinical Presentation

- Solid/cystic lesion can be solitary or multiple lumps—usually level II/III.
- Bilateral involvement/pain/dysphagia <10%—usually N2a, N2b, N2c.
- Preservation of cystic malignant metastasis—hallmark of HPV.
- First echelon lymph nodes for various. sites
  - Oral Cavity—Levels I, II, III.
  - Larynx, Pharynx—Level II, III, IV.
  - Thyroid—Level IV, VI, superior mediastina.
  - Parotid—Levels II, III, Pre-auricular, Peri & intra parotid, Upper accessory chain.
  - Submandibular/sublingual gland—Level I, II, III.

Assessment

- Clinical examination.
- Check tongue base.
- Rigid endoscopy.
- Scalp, head and neck—cutaneous lesions.
- If no obvious lesion: clinically unknown primary = investigations.

Pathology of Lymph Nodes

- Core biopsy: Clearer histological picture compared to FNA.
- Possible to suggest index primary.

- Immunohistology
  - Lung, thyroid markers.
  - EBV: Nasopharyngeal.
  - HPV: Oropharyngeal.
- CT: skull base—diaphragm
  - Confirm extent lymphadenopathy.
  - Lung.

- If level II/III, then MRI—oropharynx
  - Tongue base/tonsils.
  - Some argue MRI from skull base to diaphragm.

PET-CT

- Helps to identify 1/2 cases—level 3 observational evidence.
- Significant false positive rate.

Panendoscopy

- Complete all imaging first.
- Scans may direct targeted biopsy.
- If nothing is found, age old practice of random biopsy
  - PNS, tongue base, pyriform fossa (common practice).
- If nothing is found = ipsilateral tonsillectomy
  - Consider bilateral tonsillectomy.
  - 10% risk of bilateral tonsils involved.

Robotic—Base of Tongue Mucosectomy

- Treatment.
- Rare 1–2% SCC = treat with intent curative.
- Management is controversial, based on individual treatment centres
  - Surgery.
  - Radiotherapy.

Total Mucosal Irradiation (TMI)

- If TMI advocated, use of IMRT recommended as side effects very high.

Chemotherapy

- No randomised data.

Follow-up

- Same as all patients, high risk relapse first two years.
- Every 4 weeks for first two years.
- 3–6 months thereafter.
- PET-CT follow-up: 3–4 months after completion—high negative predictive value.
- Biopsy if secondary FNA then do lump removal.

10.3 Skull Base

Anatomical Divisions (Fig. 10.1)

- Anterior: frontal bone—lesser wing of sphenoid.
- Middle: posterior margin CNS—petrous portion of temporal bone.
- Posterior: petrous temporal—occipital bone.
- Primary tumours rare in temporal bone/lateral skull.
- Advanced skin cancer (conchal bowl, pinna, periauricular skin): SCC, melanoma, BCC.
- Advanced parotid (ear/temporal bone): salivary gland/SCC from skin.
- TMJ/infratemporal fossa: sarcomas (chondrosarcoma/rhabdomyosarcoma /osteosarcoma).
- EAM/middle ear: SCC/BCC.
- Benign
  - Osteomas.
  - Neurofibroma.
  - Paraganglioma.
  - Adenoma.
  - Lipoma.

Malignant

- SCC/BCC.
- Adenocarcinoma.

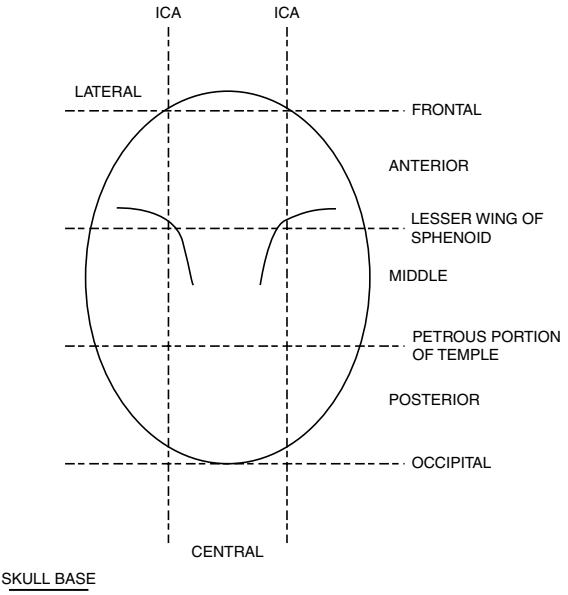


Fig. 10.1 Skull base divisions

- Adenoid cystic carcinoma (ACC), mucoepidermoid carcinoma (MEC).
- Melanoma.
- Osteosarcoma.
- Lymphoma.
- CNS malignancy.

Clinical Presentation

- Silent, due to compression.
- Orbit: proptosis, diplopia, epiphora.
- Malignant: headache, focal seizure, loss cranial nerve function.
- Check otalgia/blood otorrhoea/bleeding/facial swelling.
- Tumours infratemporal—TMJ pain.

Clinical Examination

- FNA/US.
- Sarcomas: need histopathology.
- History: TB/granulomatosis with polyangiitis (Wegener's).

Imaging

- CT: fine cut/high density—essential EAC erosion.
- MRI: dural/brain involvement.
- Carotid angiography/balloon occlusion—to assess ipsilateral carotid involvement.
- ICA resection—temporary balloon occlusion, if acceptable then permanent.
- Pre-operative coils performed 2 weeks prior to operation.
- No UICC/AJCC—modified Pittsburgh TNM staging.
- Minimum operation for cancers involving the temporal bone is lateral temporal bone resection.

Surgical Approaches 3

- Approaches

  (1) Transfrontal approach

    - Bicoronal flap/radix/upper orbit exposed—temporalis muscles reflected and bicoronal craniotomy done. Can remove supraorbital bar.
    - Expose anterior crania fossa/cribriform plate.
    - Watertight reconstruction—space nasopharynx (cranial fossa with galeal flap, cranial autografts/fibrin glue).
    - Can be done centrally/unilaterally.

  (2) Transfrontal level II approach

    - Coronal incision—remove canthal ligaments/upper lateral cartilages detached from nasal bones.
    - Nasolacrimal ducts exposed and presented.
    - Supraorbital bar and nasal orbit completely osteotomised.

(3) Transfrontal level III approach

- Level II ± orbitozygomatic osteotomy—based on tumour site.

(4) Transfrontal level IV approach

- Modified Weber Ferguson + Le Fort II osteotomy—rarely used.

(5) Transmaxillary approach

- Intraoral approach (Le Fort I)—midline palatal split, soft palate incised to one side of uvula.

(6) Transpalatal level VI

- Access via palate/nasal floor-endoscope.

Flap Reconstruction

- Local: pericranial/temporalis.
- Regional: pectoralis major/latissimus dorsi/trapezius.
- Distant: anterolateral thigh.

Complications

- Systemic.
- Wound infection.
- CSF leak.
- Malocclusion.
- Palatal fistula.

- The Hadad-Bassagasteguy nasoseptal flap (NSF) is the workhorse for endonasal skull base reconstruction. The pedicle of this flap is the posterior septal artery (PSA), a terminal branch of the sphenopalatine artery (SPA). If not available, the endoscopic pericranial flap and the transpterygoid temporoparietal fascia flap can be used. Although both flaps require scalp incisions, they can be transposed endonasally and placed onto the skull base defect without the use of a craniotomy.

10.4 Radiotherapy

Principle: Medical use of ionising radiation—works by damaging tumour cells via

(a) Direct: DNA damage.
(b) Indirect: Via ionisation of water—forming free radicals (hydroxyl radicals) = damage DNA

- Normal cells have ability to repair sublethal damage compared to tumour cells.

Physics: Linear acceleration in which electrons are accelerated by microwaves and then hit tungsten—producing high energy x-rays. Deposition maximum dose few cm below skin.

(1) External beam:

- Conventional.
- Stereotactic radiation (gamma knife).
- Interstitial (brachytherapy).

(2) Interstitial (brachytherapy)

- Radioactive seeds/needles/loops e.g. radium-226 or iridium-192 (+ I2–74 days).
- Emit high energy photons (gamma rays)—generate free radicals from water molecule.
- More damaging because directly on tumour, continuous energy emission (not fractionated).
- No time for normal tissues to recover.
- Increased risk of osteoradionecrosis (especially floor of mouth, damages lingual cortex of periosteum).

Cell Susceptibility Depends On

- Intrinsic cell radiosensitivity.
- Oxygenation—O_2 is a radiosensitiser.
- Position of cells in mitotic cycles.

Key Role in Management—Early-Stage Tumours Similar Cure Rates but Different Side Effect Profiles

- Radiotherapy kills cells via direct DNA damage or formation of free radicals.
- Kills small number of cells immediately—most survive but have internal damage especially.
- Vascular endothelial cells.
- Healing releases fibroblasts.
- Impaired, often not replaced by daughter cells.
- Tissue becomes less cellular/less vascular/less oxygenated over time
  - Hypocellular.
  - Hypovascular.
  - Hypoxic.

Tissue Sensitive to Radiation (Related to Cell Replication Rate)

(1) Most: germ cells.
(2) Intermediate: endothelial/fibroblasts.
(3) Little: muscle/nerves.

Principles of Radiotherapy

- 5 Rs of radiotherapy: 1. Repair, 2. Repopulation, 3. Redistribution, 4. Reoxygenation, 5. Radiosensitivity.
- Tumour growth/shrinkage—balance between cell proliferation and cell loss (hypoxia/shredding from surface/apoptosis).
- The observed rate of proliferation—end result of these factors.
- Potential doubling of head and neck cancer = 2–10 days if no cells loss, also tumour proliferation continues during radiotherapy.
- Intrinsic radiosensitivity of tumour—most significant factor.

Hypoxia

- As tumours grow = new vessels due to hypoxia—functional MRI shows most cells severely hypoxic.
- Increased hypoxia if tumour not stimulated by vascular growth.
- Lymph node metastasis usually more hypoxic than primary tumour—anoxic cells in-vitro may require twice the dose of radiation to achieve cell kill.

During Fractionated Radiotherapy

- Cell death, reduces oxygen use, previously hypoxic cells become sensitive to radiotherapy.

Radiotherapy

- Usually photons/electrons = 6–10MV/615 meV head and neck—linear accelerations (generate photons x-rays).
- X-rays interact with better—2° electrons, increased energy/increased electrons = cause ionizing effect.
- 6MV photons = maximum dose is at 1.5 cm depth below skin.
- Use of fields = deliver high dose to appropriate area/multi-leaf collimators to shape the beams.
- Same dose to normal tissue is produced by internal scatter (within patient) = external beam shielding does not provide total protection.
- Intensity-modulated radiotherapy is the most common technique for treating head and neck cancer.

Electrons

- Produced by linear acceleration: produces beams of different energies.
- 6 meV electron beam: skin tumours, 12 meV—neck nodes.

Proton Beam Therapy

- Type of radiation therapy.
- Uses focused beam of high energy protons.
- Reduced localised tissue damage/swelling.
- Useful when treating patients with malignant lesion close to vital structures.
- Used to treat brain tumours, and tumours in children.

- NHS England: not enough evidence to treat head and neck cancer in adults.
- Proton beam therapy not available through routine commissioning. Excludes skull base tumours.

Prognosis of Treatment

- Radical: cure.
- Palliative: relieving pain/bleeding/pressure symptoms.
- Radiotherapy commences 2–4 weeks post-operatively. No difference in locoregional failure rates if done within 6 weeks.
- Pre-operative/neoadjuvant radiotherapy: Gap leave 4–5 weeks for complete recovery of mucositis.

- Therapeutic ratio based on biological differences between normal and cancer cells
  - Normal cells greater ability to repair DNA damage and usually to resting phase of cell cycle compared to actively dividing tumour cells.

Treatment Failure Due To

- Tumour repopulation during treatment.
- Tumour hypoxia = keep Hb 12 g/dL.
- Intrinsic radio-resistance.
- Smoking = reduces response by 10–15%.

Radiotherapy Planning

CT Scan + Mask

- Polymer sheets, heated—soften.
- Moulded to patient's face—accuracy within 3 mm.

Fractionation

- Schedule on which radiation dose administered usually 2 Gy/day for 5 days.
- Weekends off for 6 weeks.

Hyperfractionated

- Delivery of a higher dose of radiation by 2–3 low dose fractions/day.
- Increased acute toxicity, does not increase late effects.

Accelerated

- Delivery over a shorter period of time Gy/fraction/day—6 weeks–12 days.
- Hypoxia/anaemia interferes with radiotherapy because lack of O_2 in tissues—correct EPO.
- Regimes have not shown consistent improvement in overall survival and not adopted widely worldwide.
- Radio-sensitisers: imidazoles—investigated.

Conformal Radiotherapy

- Reconstructed CT/MRI used during treatment planning.
- Dose distribution to target volume—shape/conform tightly to shape of tumour.
- Reduce volume of tissue irradiated by up to 50% and decrease late damage.
- Dose to tumour increased.

Intensity Modulated Radiotherapy—Most Advanced form of Conformal Radiotherapy

- Conventional radiation beams have uniform intensity across a field, In IMRT dose distribution is non-uniform across several radiation beams: produces optimum dose distribution, conforms closely to shape of target volume.
- Beam modulated by multi-leak collimators—major decrease xerostomia from 75 to 35%.
- Spare the contralateral parotid.
- Concomitant chemotherapy (with radiotherapy): improve locoregional control—Pignon JP meta-analysis (radiotherapy oncology 2009).
- Benefits largely if given chemotherapy together with radiotherapy.
- Combining chemo- + radiotherapy
  - Improves organ preservation.
  - Cisplatin most effective.
- Concurrent administration of cetuximab = increased survival + locoregional control.
- Zelefsky 1992 (MSK): Post-operative radiotherapy improves local control.
- Cosler 1992: Post-operative radiotherapy—after neck dissection reduces recurrence by 50%.

Indications for Adjuvant Radiotherapy

- Close <2 mm/involved margins at primary site if cannot excise again.
- Perineural/vascular spread, large tumour, involving >1 lymph nodes/extracapsular spread of lymph nodes.

Chapter 11
Head and Neck Reconstruction

Abstract Methods of surgical reconstruction of the head and neck.

Key Points

- Indications for reconstruction.
- Mandibular reconstruction.
- Maxillary reconstruction.
- Oropharyngeal reconstruction.
- Buccal fat pad.
- Nasolabial flap.
- Facial artery musculomucosal (FAMM).
- Masseter flap.
- Submental island flap.
- Temporalis flap.
- Temporoparietal fascial flap.
- Tongue flap.
- Sternomastoid flap.
- Skin grafts.
- Forehead flap.
- Free tissue transfer.
- Radial forearm free flap.
- Fibular free flap.
- Latissimus dorsi flap.
- Pectoralis major flap.
- Gracilis flap.
- Anterolateral thigh flap.
- Scapular flap.
- Deep circumflex iliac artery flap (DCIA).

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_11

11.1 Oral Soft Tissues

- Following excision of tumour.
- Fistulae.
- ORN.

Principles

- Restore oral cavity lining.
- Maintain oral competence.
- Maintain function—speech/swallowing.
- Aesthetic result.
- No RCT—mainly surgeon's preference tailored to each patient.

Soft Tissues

- Tongue/floor of mouth.
- Buccal mucosa.
- Tonsils.
- Retromolar triangles.

First Consideration

- RFFF.
- ALT.
- Latissimus dorsi.
- Rectus abdominus.
- Scapula.

Second Chance

- Pectoralis major.
- Deltopectoral.
- Local mucosal flaps.

11.2 Mandibular Reconstruction

Indications: Tumour/Infection/ORN/Congenital Deformities

Purpose

- Restore aesthetics.
- Restore function

(1) Speech.
(2) Mastication.
(3) Swallowing.
(4) Occlusion.

Planning Considerations

- Surgical defect: Determine size/location/composition of defect—assess quality and quantity of remaining tissue.
- Soft tissue: Recon plate/3D model (pre-bent/template: mirror).
- Patient: Co-morbidities/prognosis/patient's wishes.
- Timing of bony reconstruction

(a) ***Primary:***

- At time of procedure: Early return to form and function, no second procedure, early implant placement.
- Disadvantages: Increased operation time, flap failure, clearance of margins unknown, cost.

(b) ***Secondary:***

- Confirm clean margins—after one year when 70% recurrences will have occurred.
- Disadvantages: Form/function, definite recon plate—more complicated with suboptimal results due to excessive scarring/anatomical distortion, especially if radiotherapy administered.

Techinque

- Alloplastic—reconstruction plate
  - Poor prognosis and general health, secondary reconstruction planned.
  - CT modelling/pre-bent or bent recon plate.
  - Quick and easy.
  - Does not address all reconstruction goals—need to have adequate soft tissue coverage for plate (pectoralis major), risk of late hardware failure.
- Autogenous: Non-vascularised/vascularised (pedicle/free flap—fibular/DCIA/scapular).

Free Bone Graft

- Particulate cancellous bone marrow: from iliac crest $+/-$ PRP, BMP
  - Need trays/cribs to provide structural support.
  - Reduce morbidity harvest.
- Non-vascularised cortico-cancellous bone
  - Defect <6 cm with primary soft tissue closure is possible in non-invaded tissue (Foster and Pogrel 1999), non-cancer cases (post-operative radiotherapy).
- Optimum iliac crest/costochondral graft
  - Technically easier, quicker and no microscope required.

  - Disadvantages: Higher risk of non-union, if >6 cm, graft susceptible to fracture especially during loading, infection, high implant failure, donor site morbidity, limited use in radiotherapy patients.

- Vascularised—Pedicled

  - Temporoparietal osteofascial flap—calvarial bone pedicle of temporalis.
  - Osteo-musculocutaneous trapezius (scapula spine).
  - Disadvantages: Limited osseous segments, limited arc rotation.

- Free tissue transfer:

  - Fibular: Long/adequate height/good skin (lack of mobility).
  - DCIA: High bony segment, natural curve.
  - Scapular: Small amount of bone—large volume skin/muscle.
  - RFFF: Small volume/fracture radius.

Maxillary Reconstruction Classification

- Restore aesthetics: Support orbital content, cheek, lip, nose.
- Restore function: Speech, mastication, swallowing.
- Timing

  - The maxillectomy defect to detect early recurrence has to be considered against functional aesthetic advantages of maxillary reconstruction.

- Reconstructive options

  - Obturators (+/− implant retained).
  - Non-vascularised: soft tissue (SSG)/bone (NVBG).
  - Vascularised:

    Local (+/− bone graft)—buccal fat/FAMM/palatal mucoperiosteal.
    Regional (+/− bone)—temporalis, submental island, pectoralis major.

  - Free tissue: RFFF (DCIA/scapular).
  - Zygomatic implants—at time of surgery.

- Maxilla/midface (classification—Brown 2008) (Figs. 11.1 and 11.2).

  (1) Class I:

    - Alveolar bone/not resulting in OAF—left to granulate/local flap.
    - Junction hard/soft palate—need obturation/soft tissue flap (radial/ALT).

  (2) Class II

    - Standard hemi-maxillectomy: not involving orbital floor.
    - Obturation (very successful): orbit does not require support, if defect not too large—less problem with retention/stability.

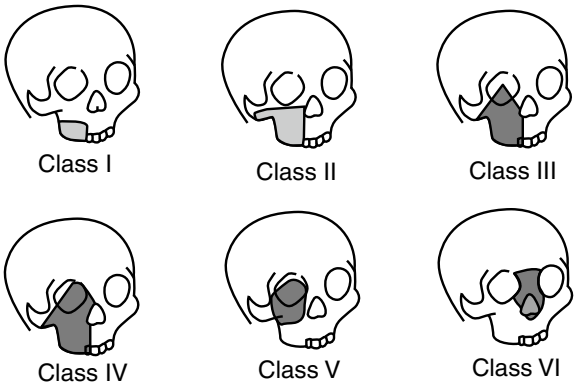


Fig. 11.1 Maxillary defect grading

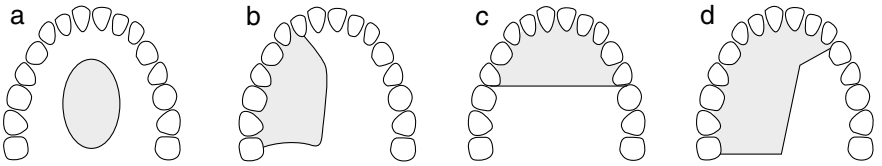


Fig. 11.2 Maxillary defect palate

- Class II c–d
  - Can get good outcomes, implant retained prosthesis.
  - Fibular flap: good outcomes.
  - Scapular—large pedicle (based on angular branch of the thoracodorsal artery).
  - DCIA.

(3) Class III

- Loss of orbital support +/− nasal bones.
- Evidence restoration of orbital support is essential to ensure healing and avoid epiphora and ectropion.
- DCIA.
- Scapular—best in children as DCIA is cartilage.
- Fibula: Difficult to adapt.
- Obturation: Facial collapse—increased risk of orbital dystopia and ectropion.

(4) Class IV

- DCIA.
- Scapular.

(5) Class V

- Orbitomaxillary defect—do not obturate orbital space with too much soft tissue to allow space for orbital prosthesis: temporalis, temporoparietal flap.
- If extensive—radial/ALT (thin patient).

(6) Class VI

- Loss skin and nasal bone.
- Composite radial
  - Fascia—line nasal cavity.
  - Skin restore face.
  - Radial as bony strut.
  - +/− Forehead/glabella.

11.3 Oropharyngeal Reconstruction

Maintain Function of Residual Tissues

- Soft palate
  - RFFF + local flap: Superior based pharyngeal/superior constrictor advancement.
  - RFFF Folded de-epithelialised (sutured to posterior pharyngeal wall).
- Pharyngeal wall + tonsillar region
  - Placing free tissue transfer—disrupts muscular tube and reduces function.
  - Role of transoral laser.
- Posterior tongue
  - Most difficult area: Allow normal movement of epiglottis and maintain swallow and speech function.
- Soft palate flaps.
  - Local

    Pharyngeal flap: superiorly based.
    Palatoplasty and lateral pharyngeal flaps.
    Palatal island—mucoperiosteal.
    Masseter/buccal mucosa transposition.
    Temporalis.
    Superior constriction advancement.

  - Pedicles

Galeal-pericranial.
Temporal.

– Free

RFFF (folded/lateral arm/jejenum/ALT).

Mathes and Nahai Classification

- Type I: 1 Vascular pedicle.
- Type II: 1 Dominant + 1 minor = plastysma/SCM/temporalis/trapezius.
- Type III: 2 Dominant = rectus abdominus.
- Type IV: Segmental = sartorius.
- Type V: 1 Dominant with secondary segment = pectoralis major/latissimus dorsi.

Flaps : Buccal Fat Pad

Uses

- Maxillectomy Class I and IIa defects maxilla <4 cm, contraindication for radiotherapy.
- Buccal mucosa/RMT/marginal mandibulectomy defect.
- OAF.

Anatomy

- 10 ml volume—6 mm thickness.
- 3 lobes (Anterior/intermediate/posterior) with 4 processes derived from posterior lobe.
- Constant throughout life—no direct relationship to total body fat.

Relationship

- Lies between buccinator and masseter.
- Facial nerve (buccal/zygomatic) lies lateral to the fat pad.
- Blood supply: Buccal/deep/temporal maxillary artery and branches of facial artery.

Function

- Provides separation allowing gliding motion between muscles and protects neuromuscular bundles from injury.

Surgical

- Fat pad with 7–9 cm which reproduces vascularity as long as tension free.
- Re-epithelialisation starts with initial phase of granulation—subsequent transformation to parakeratotic stratified squamous epithelium.
- Unilateral—causes minimal facial asymmetry.

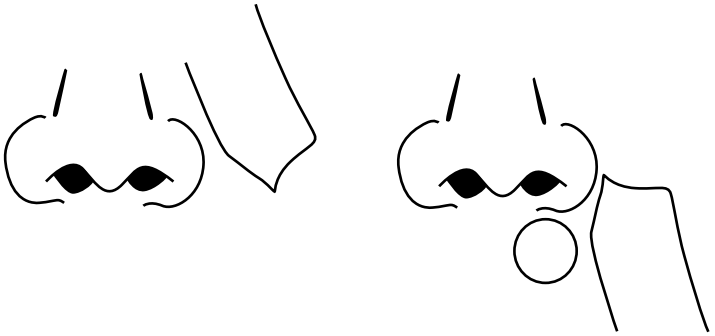


Fig. 11.3 Nasolabial flap (superior/inferior)

Technique

- Distobuccal depth of maxillary tuberosity. Blunt dissection with scissors—mobilise flap.

Post-Operative

- 24 h clear fluid.

Complication

- Partial necrosis, trismus from scarring, possible change in facial contour.

Nasolabial Flap

Axial Pattern Flap (Fig. 11.3)

- Type B: Based on angular artery and termination of facial artery which anastomoses with ophthalmic artery.
- Peri-oral defects: Upper lip/commissure/buccal mucosa.
- Floor of mouth.
- Nasal reconstruction (ala, comissure, infra-tip lobule).
- Need secondary procedure 2–3 weeks with closure of orocutaneous tunnel.

Facial Artery Musculomucosal Flap (FAMM)

- 1992 Pribaz—Axial musculomucosal flap based on facial artery as the main paddle.

Clinical Approach

- Superior: Anterior palatal maxillary vestibule/nasal floor/septum.
- Inferior: Posterior palate/tonsillar fossa/floor of mouth/lower lip.

Anatomy

- Facial artery—Hook round lower border: anterior edge of masseter.

- Upward/forwards—Lateral commissure of the mouth.
- FLAP.
- Mucosa/submucosa/small amount of buccinator muscle, deeper plane of dissection to facial artery and venous plexus.
- Buccinator has such rich blood supply—buccal artery/branches from facial artery.
- Venous drainage—posterior to venous plexus/anteriorly to facial vein.

Technique

- Course of Facial artery mapped with doppler—extend from RMT to level of ipsilateral alar margin.
- Width 1.5–2 cm—up to 8–9 cm long.
- Incision through mucosa/buccinators, FA identified—ligature and cut = dissection just deep to facial vessels.
- 2 layered closure of donor defect.

Disadvantages

- Can cause trismus.

Submental Island Flap

- Axial patter flap based on submental artery used for coverage of facial, perioral and intraoral defects.
- Provides excellent colour/texture to match the facial skin of the donor site that can be closed primarily.

Technique

- Supine with head extended.
- Flap elliptical according to defect.
- Dissection—ipsilateral or contralateral to vascular pedicle.
- Subplastysmal dissection ensures elevation of the skin island.
- Submandibular gland identified—retracted posteriorly.
- Submental artery between gland and mylohyoid.
- Some cases vessels run beneath anterior belly of digastric—suggest incorporating.
- It can be used as free flap.
- Donor site close primarily.
- Underlying mylohyoid muscle cut out or strip included with pedicle if the flap needs to be tunneled medial to mandible for intraoral reconstruction.

Applications

- Cutaneous facial defects: lower/midface.
- Intraoral (SCC—issues as neck dissection)—floor of mouth, buccal mucosa, tongue.

Temporalis Flap

- Temporalis muscle lies in the temporal fossa—arises from pericranium and deep fibres from the infratemporal fossa = 5–15 mm thick.

Fig. 11.4 Temporalis flap

- Temporalis fascia covers the temporalis muscle—inferior the fascia splits approx 2 cm superior to the zygomatic arch into superficial and deep layers.

Blood Supply—3 Arteries (Fig. 11.4)

- Anterior left and poster deep temporal arteries—maxillary artery.
- Middle temporal artery—superficial temporal artery.
- Venous drainage runs parallel to artery.

Surgically

- 1/3 anterior muscle.
- 2/3 posterior muscle.
- Rule: preserving both deep and superficial branches of the temporal artery to allow bilobed elevation.

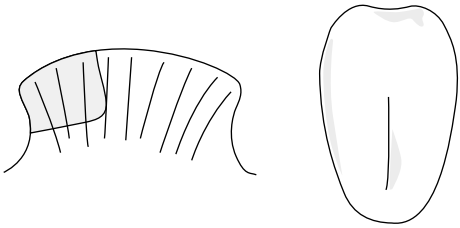
Nerve Supply

- Anterior/posterior deep temporal nerve.

Surgical Technique

- Hemicoronal: Deep temporal fascia—1.5–2 cm superior to zygomatic arch.
- Incise superficial layer—to reach fat pad between deep/superficial.
- At arch: Subperiosteal—expose muscle.
- Raise muscle from skull—vascular pedicle, on deep surface.
- Osteotomy of arch of underneath—avoid compression.
- May require intraoral coronoidectomy to increase movement.
- Disadvantages.
- Temporal hollowing, trismus, facial nerve damage.

Fig. 11.5 Tongue flap



Useful

- Orbital/palatal/buccal/retromolar areas.

Temporoparietal Fascial Flaps

- Component of SMAS—highly adjacent to the subcutaneous layer of overlying skin.
- Blood supply: Superior temporal artery.
- Venous drainage: Superior temporal vein.
- Indications
  - Malar augmentation, orbital defects after enucleation, skull base reconstruction, TMJ reconstruction.

Technique

- Hemicoronal incision.
- Superficial dissection just below hair follicles with minimal amount of subcutaneous fat left with overlying skin.
- STA identified above arch—2 cm anterior to external auditory meatus and posterior branches followed to the extent of desired length.
- Passage into the oral cavity is through a subcutaneous tunnel superficial to SMAS.

Tongue Flap (Fig. 11.5)

Useful For

- Oral nasal fistulae/OAC/lip reconstruction/buccal mucosa reconstruction.

Classification

- Random: Dorsal (anterior versus posterior), lateral, double-door, median transit.
- Axial: Sliding posterior flap.

Anatomy

- Lingual artery second branch ECA: Caudal to posterior belly of digastric—passes deep to hyoglossus.
- Lingual artery gives the dorsal lingual artery—supplies the dorsum of the tongue, valleculla, epiglottis, and adjacent soft palate.

Dorsal Tongue Flap

- 3–10 mm thick—2/3 tongue can be used.
- Anterior: Hard palate, anterior buccal mucosa, anterior floor of mouth.
- Posterior: Defects of soft palate, retromolar area, posterior buccal mucosa.
- Elevation posterior to circumvallate papillae avoided, 14–21 days before pedicle severed.

Advantages

- Abundant vascularity, low morbidity.

Disadvantages

- Speech impairment if tongue mobility compromised, loss of tongue sensation.

Complications

- Bleeding/loss of tongue sensation, speech impairment.

Sternocleidomastoid Flaps (Fig. 11.6)

- Superior versus inferior.
- Skin paddle marked on muscle.
- Dissect/suture skin paddles.
- Dissection of muscle down to clavicle—cut 1 cm above.
- Rotation of flap—if tension then blood supply to middle 1/3 sacrificed.

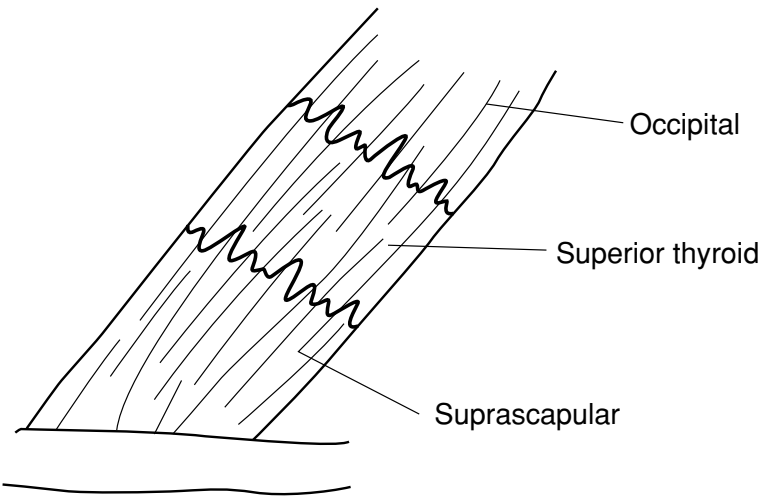


Fig. 11.6 SCM flap

Skin Grafts

Epidermis

- Stratum corneum, stratum lucidum, stratum spinosum, stratum basale.
- Papillary layer.

Dermis

- Papillary layer.
- Reticular layer.

Split Thickness

- Epidermis and portion of dermis.
- Thin: 0.005–0.012 inch.
- Medium: 0.012–0.018 inch.
- Thin/rapid vascularised can be expanded/less optional conditions, donor site heals with minimal scarring.

Full Thickness

- Epidermis and dermis.
- Better results, reduce contraction, donor site closed primarily.

Forehead Flap

- Supraorbital artery: Supraorbital foramen (midpoint of pupils)—palpate bony landmark.
- Supratrochlear artery 0.5–1.5 cm medial to the supraorbital.
- All vessels anastomose, can raise on any of the vessels.
- Planning—template defect (extra 15–20% in pedicle for pivot), if hairline—fit obliquely and lower pivot point.
- Raise flap—go subperiosteal 1 cm above rim to protect vessel, distal position with blood supply in the subdermal plexus, can be thinned but be careful if smaller.
- Inset—leave for 3–4 weeks.
- Donor site—primary closure (scoring galea—or undermine/H flap/SSG/leave to granulate).

Free Tissue Transfer Length of Ischaemia Time

- Anaerobic metabolism in the tissue during ischaemia increases production of superoxide radicals, following reperfusion free radial scavengers (superoxide dismutase), attacks radicals and injures cells.
- Increased endothelial swelling/fluid/leakage—causes narrowing vessels, and intravascular platelet aggregation can then occur.
- Ischaemia induced reperfusion injury gives rise to no flow effect.
- Clinically—flap perfusion tails off = irreversible flap ischaemia.

Skin/Subcutaneous

- 4–6 h warm.

- 12 h cold.

Muscle

- 2 h warm.
- 8 h cold.

Bone

- 3 h warm.
- 24 h cold.

Failure

- Check clamps, kinking, blood pressure.
- Check vein position, spasm—secondary to lidocaine (leave 5–10 min).
- Redo anastomosis.

Fluorescent Dye

- Intact circulation fluorescence with UV light.
- Implantable doppler.

Radial Forearm

- Most commonly used—fasciocutaneous flap—can be used as osteofasciocutaneous flap.
- 1981 Yang "Chinese Flap".
- Anatomy
  - Radial artery forms the deep palmar arch of the hand after tracing down the forearm between brachioradialis and flexor carpi radialis.
  - Ulnar artery forms the superficial palmar arch.
  - Anastomosis between deep and superficial arches allows the safe dissection of the radial artery without causing ischaemia and necrosis of the hand.
  - Venous return

    Superficial—cephalic vein.
    Deep—vena comitans (VC).

- Nerve supply: medial and lateral anterobrachial cutaneous nerves (sensate flap).

Pre-operative

- Allen's test $+/-$ doppler.

Surgical

- Non-dominant hand, tourniquet 250 mmHg.
- Size/shape outlines.
- Incision—fascial superficial dissection.
- Ulnar—radial.

- If bone is to be taken—preserve cuff of pronator quadratus and flexor pollicis longus (FPL).

Composite Flap

- Maximum 11–13 cm.
- Allow 2 cm from radial styloid—allow placement of bone plate.
- Retract flexor carpi radialis (median nerve closure).
- Exposes flexor palmaris longus and pronator quadratus—incised to reach (ulnar) medial border of radius.
- Preserve muscle cuff for periosteal perforating vessels going through muscle.

Bone Exposed and Osteotomy Performed

- 1/3–1/2 radial circumference.
- Using fissure bur/postage stamp method before sawing dissection radial aspect.
- Retract brachioradialis, dissect deep and identify the osteotomy site.
- Preserve intermuscular septum—lift flap superiorly by incising periosteum.
- Bone plate—3/5 plate dynamic compression plate on anteromedial surface—2 bicortical screws either site.
- Below elbow cast for 1 week and supporting cast for 6 weeks.

Vein

- Cephalic vein (empties axillary vein) → Superficial vein → Communicated with basilic vain via median cubital vein at the elbow.

Fibula Free Flap

Popularised by Hidalgo (1978) for head and neck reconstruction.

Up to 25 cm of bone preserving 6 cm at either end for stability of the joint.

Common peroneal nerve wrapping around the fibula head is the limiting factor.

Contraindications

- Long pedicle and long bony requirement may mandate vein grafting.
- Pre-operative vascular imaging may demonstrate reliance of the foot on the peroneal artery in which case the other side or an alternative flap should be chosen.

Positioning

- Supine, hip internally rotated and knee flexed. Tourniquet optional.
- Pre-operative assessment should include doppler pressures, angiography or MR angiography.
- USS doppler mapping may be useful.

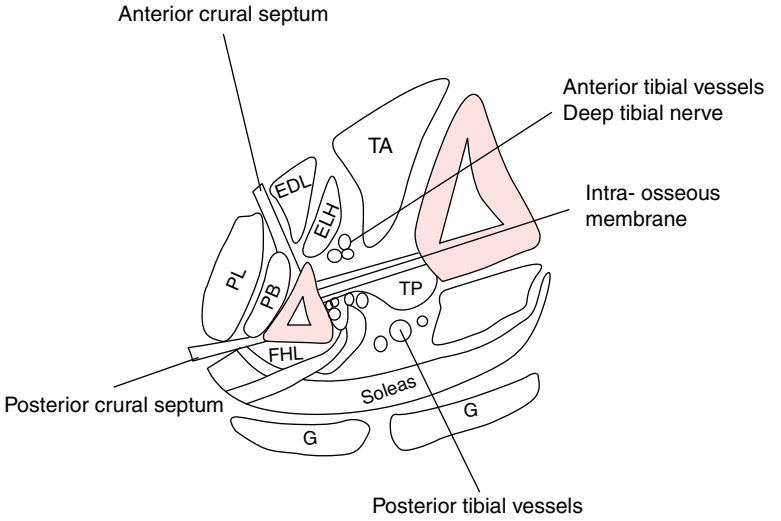


Fig. 11.7 Fibula flap (leg compartments)

Relevant Anatomy (Fig. 11.7)

- Based on the peroneal artery and two venae comitans which run under tibialis posterior and over flexor hallucis longus in the posterior compartment.
- Popliteal gives off the anterior tibial vessels which perforate the interosseous membrane and run in the anterior compartment.
- The common peroneal nerve divides into the deep peroneal nerve which supplies tibialis anterior, anterior compartment muscles and the superficial peroneal nerve which runs to supply the skin.
- The sciatic nerve turns into tibial nerve which runs with posterior tibial vessels and supplies all muscles in the posterior compartment.
- Cutaneous perforators supply the skin and may be septocutaneous or musculocutaneous. Pre-operative doppler will help in identifying perforators but the skin paddle should be centered around the junction of middle and distal 1/3.

Variations

- The fibula is the most commonly missing bone and is associated with bowing of the tibia.
- All vessels are usually present however anterior tibial may be significantly attenuated and is supplemented by a branch from the peroneal.
- In these cases, sacrifice of the peroneal may compromise the foot.
- Pre-operative assessment should reveal such potential problems.

Technique

- Leg is marked with intermuscular septum.
- Mark is 6 cm below proximal and distal ends of the fibula.
- Common peroneal nerve passes 1–2 cm below the fibula head.
- Skin paddle is centred on the middle and distal 1/3.
- Skin incision is made and septocutaneous perforators are identified if possible.
- Peroneus longus, peroneus brevis and extensor hallucis longus are elevated from the fibula revealing the intermuscular septum.
- Anterior tibial artery and nerve are revealed in the anterior compartment.
- Bone is sectioned superiorly and inferiorly in a subperiosteal plane. Rotated laterally with bone holding forceps. Intermuscular septum revealed and incised.
- Tibialis posterior is behind bipennate muscle. Tibialis posterior divided revealing pedicle. Trial clamp of distal end and foot circulation checked.
- Pedicle dissected out clearing tibial nerve and posterior tibial vessels away. Cuff of flexor hallucis longus may be taken with the bone to give added certainty regarding the skin paddle.
- Wash, haemostasis, drain, primary closure if possible—beware compartment syndrome.

Complications

- Partial or complete necrosis of skin paddle (~10%).
- Ischaemic complications regarding the foot are the most feared.
- Prevent with pre-operative assessment and trial ligation.
- Haematomas.
- Damage to common peroneal nerve leading to anaesthesia of anterior and lateral leg and dorsum of foot with foot drop.
- Gait disturbance secondary to muscle weakness (disruption of muscular origins or nerve supply).

Latissimus Dorsi

Relevant Anatomy

- Based on the thoracodorsal artery and vein with thoracodorsal nerve included. Type V muscle according to the Mathes and Nahai classification with one dominant vascular pedicle and series of smaller segmental pedicles (specifically the posterior intercostal perforators).
- The thoracodorsal artery arises from the subscapular artery which is a branch of the 3rd portion of the axillary artery. The subscapular artery bifurcates into the circumflex scapular and the thoracodorsal. The thoracodorsal descends and splits into the horizontal and vertical branches. It also gives off branches to serratus anterior.
- The latissimus dorsi arises from the tip of the scapula, lower six thoracic vertebrae, thoracolumbar fascia and iliac crest, and inserts into the tubercular groove on the medial aspect of the humerus. It medially rotates, adducts and extends the arm

(as in the freestyle stroke). Pedicle enters muscle approximately 8 cm below the tendinous insertion and approx 2.5 cm from lateral border.

Technique

- Pre-operative markings include midpoint of axilla, and midpoint between ASIS and PSIS with a line joining them which marks the anterior border of the latissimus dorsi.
- The patient may be marked pre-operatively by placing the hand on the hip and asking them to push down. The majority of perforators exit along the anterior border of the muscle.
- The skin paddle is outlined and the incision is made along or in front of the anterior aspect of the muscle to identify the anterior border of latissimus dorsi.
- The muscle is dissected laterally often exposing the branches to serratus anterior which may help identify the thoracodorsal artery and vein.
- The artery and vein should be seen on the underside of the muscle prior to entering it and dividing.
- The inferior and posterior incisions around the skin paddle can then be made and the flap is carefully elevated, ligating the angular branch (to tip of scapula) and the muscular branches.
- The tendinous insertion to the humerus must be divided to allow additional length for a pedicled flap and to release a free flap.
- The circumflex scapular artery is ligated and the subscapular artery and vein are divided from the axillary artery and vein. If using as a pedicled flap the tunnel is made between pectoralis major and minor.
- Drainage is used and wide undermining of the skin usually allows primary closure.

Contraindications

- Mastectomy and axillary dissection are a contraindication if pedicle is injured.

Clincal Indications

- Used to cover large soft tissue defects
  - Calvarium.
  - Orbit.
  - Maxillary when no bone required.
  - Neck (as free flap or pedicled flap).
- Perforating defects of cheek, nose and palate
  - Muscle with 2–3 skin islands used to separate oral cavity from nasal cavity as well as to reconstruct cheek.
  - Total glossectomies (re-innervate).

Complications

- Haematoma.
- Seroma.

- Wound dehiscence.
- Denervation of remaining muscle commonly occurs.

Advantages

- Reliable anatomy.
- Ease of dissection.
- Long pedicle, good diameter.
- Large flap, can be innervated, can be subdivided.
- Can be combined with scapular osteofasciocutaneous flap based on subscapular vascular system.

Disadvantages

- Repositioning.
- Bulky.
- Secondary defect not too bad but some scarring.
- Limited width if primary closure.

Complications

- Marginal flap necrosis may occur if excessively distal portions of muscle are taken.
- Care must be taken in positioning as injury to the brachial plexus may occur if the arm is elevated too much.
- Loss of muscle function is usually well accommodated; however, it may prove a problem in young people and if there is additional insult to the shoulder girdle i.e. if a radical neck dissection is undertaken on the same side.
- Care must be taken when transecting the tendon to avoid damage to the underlying pedicle.

Pectoralis Major

- First described—Ayrian 1979: myocutaneous or muscle only
  - Large/broad triangle shaped muscle.
- Major vascular supply is the pectoral branch of the thoracoacromial artery which comes off the subclavian/axillary artery and pierces the clavipectoral fascia between the pectoralis minor muscle and the subclavius muscle.
- Additional supply to the muscle comes from the lateral thoracic artery (which also arises from the axillary artery), and the parasternal perforators from the internal mammary arteries.
- Arises
  - Clavicular head: inserted medial 1/2 clavicle.
  - Sternocostal head: sternum, 6 upper costal cartilages and external oblique aponeurosis.

- Inserts
  - Lateral tip—bicipital groove of humerus.
  - Anterior tip of deltoid tuberosity.
- Nerve
  - Medial pectoral nerve.
  - Lateral pectoral nerve (C6,7,8).
- Action
  - Clavicular lead: flexes/adducts arms.
  - Sternal head: Adducts/medially rotates arm.
  - Accessory breathing.
- Muscle invests deep fascial layer, on the deep surface fascia distinct from underlying clavipectoral fascia which invests subclavias/pectoralis minor muscles.
- Inferiorly pectoral fascia is continuous with rectus fascia and fascia overlying external oblique.
- Blood supply
  - Branches subclavian—thoracodorsal and internal mammary artery perforators.
  - Thoracoacromial trunk divides.

    Deltoid: clavicular head

    (a) Pectoral: sternocostal head.
- Pedicle used for rotation of flap
  - Axial pattern pectoral dominant.
  - Skin reliability

    Total necrosis 3.2%.
    Partial necrosis 29%.

Indications

- Quicker then free tissue—not true.
- Previous surgery/radiotherapy/no vessels.
- Previous flap failure.
- Not useful for anterior defects.

Procedure—Abducted 60–90°

- Surface markings—line from acromion to xiphisternum—midpoint where expect vascular pedicle (Fig. 11.8).
- Skin paddle based on distal portion of line.
- Skin cut and expose lateral and inferior edge of muscle.

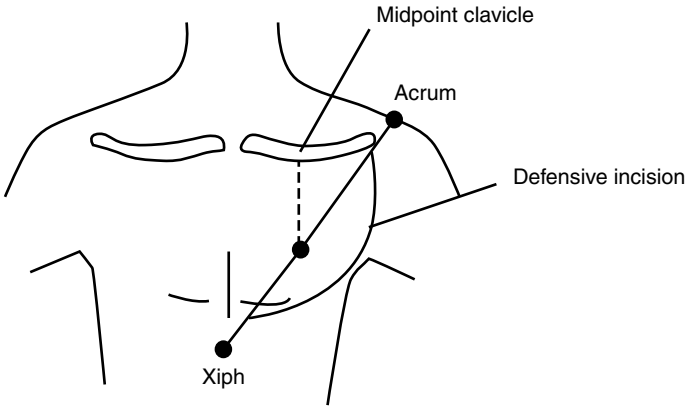


Fig. 11.8 Surface markings Pec Major flap

- Raise skin paddle in continuity with underlying pectoralis major.
- Go from inferior to superior until pedicle seen.
- Dissect the pedicle from clavicular head/separate clavicular head from clavicle near pivot point—need 4 fingers (safety) to get through.
- Remove sternomastoid—subplastysma skin tunnel.
- Muscle sutured along its length—resist gravitational pull.

Advantages

- Easy/constant anatomy.
- Reliable.
- Bulky.
- Minimal donor site morbidity.

Disadvantages

- Arc rotation.
- Bulky.
- Gravitational pull.
- Does not reach all sites.

Deltopectoral Flap (DP)

- Described by Aymard in 1917.
- Considered for head and neck reconstruction in 1960s.
- Contraindicated in patients who have had cardiac surgery where the internal mammary artery has been compromised.
- No muscle component.
- Raised as cutaneous or fasciocutaneous (to protect blood supply).
- Based on fasciocutaneous perforating branches of the internal mammary artery (IMA) (branch of the subclavian artery).

- Dominant blood supply is from the second and third perforating branches of the IMA.
- Venous drainage via VC which drains into internal mammary vein.
- Innovated by supraclavicular nerves C3/C4 and intercostal nerves.
- Modifications: DP island flap, split DP flap, free flap.

Advantages

- Thin and pliable.
- Good colour and texture match for head and neck region.
- Consistent anatomy.
- Easy to harvest and larger areas can be incorporated.
- Minimum donor site morbidity.
- Minimum functional deficit.
- Can be raised as island flap on the perforators.

Disadvantages

- Skin grafting may be required if large areas harvested (more than 8 cm of skin).
- Distal necrosis is possible if extended over the deltoid region.
- Possible alteration of breast symmetry/positioning of nipple if significant chest scarring.

Procedure

- Patient in supine position (Fig. 11.9).
- Mark the flap: Superior line parallel to the clavicle, centred over the 2nd and 3rd intercostal space to capture the perforators, extend towards the acromion.
- Can extend beyond the deltopectoral grove (8 cm) if length is required.
- Incision and subfascial dissection to preserve the perforating vessels.
- Elevated from a lateral to medial direction.
- Dissection terminated at least 2 cm from the lateral border of the sternum to protect the perforators.
- Several different designs have been introduced.

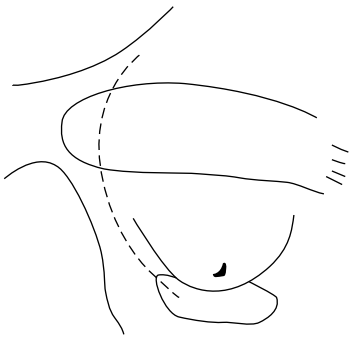


Fig. 11.9 Deltopectoral flap

Gracillis Flap

- Free neuromuscular tissue transfer—facial reanimation after long standing paralysis.
- Low profile muscle.
- Organisation is in small segments—good mimic muscles.

Anatomy

- Flat/thin adductor muscle.
- Origin: Ramus of pubic bone.
- Inserts: Medial tibial tuberosity—below the knee.
- Type II muscle (dominant vascular).
- Pedicle from adductors artery and vein, from profunda femoris (has 2 VC).

Motor

- Anterior branch of obturator nerve.
- Enters muscle approximately 2 cm proximal to vascular pedicle.

Position

- Leg flexed, abducted, rotate outward, knee flexed.
- Leg/pubic hair shave pre-operatively.
- Surgeon: Opposite side table.
- Incision 20 cm—down the fascia lata.
- Gracilis identified after going through fascia.
- Vascular pedicle—runs in intermuscular septum between adductor longus and adductor magnus before entering gracilis.
- After identification of artery + 2 VC—go proximal, nerve enters gracilis muscle approximately 2 mm proximal.
- Before entering gracilis—pedicle gives off branches to adductor longus—ligate.
- Reach profunda femoris—leave 5 mm to junction (pedicle length 6 cm with diameter 2–2.5 mm).
- Obturator nerve followed proximately until 8 cm length achieved
  - Nerve stimulation: define muscular units.
  - Nerve usually has 2 fascicles (can be up to 7).
  - Anterior 1/2 muscle belly commonly innervated by 1 fascicle.
  - Usually need 1/2 width muscle belly.

Post-operative

- Drain.
- Mobilise after 24 h.
- Low morbidity.

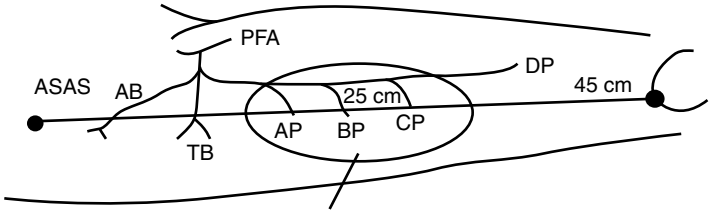


Fig. 11.10 ALT flap perforators

Anterolateral Thigh Flap (ALT)

Perforator Flap

- Artery 4 mm diameter—descending branch of the lateral circumflex artery (DBLCFA).
- Vein 6 mm diameter.
- Long pedicle: incorporate cutaneous nerve for motor/sensory nerve reconstruction.
- Avoid BMI > 30.

Useful

- Parotid resection/total glossectomy.
- Can thin flap by almost 50%.
- Artery.
- Descending branch of lateral circumflex femoral artery.

Pre-operative (Fig. 11.10)

- ASIS—lateral patellar.
- Midpoint—4 cm circle—doppler (10 MHz—search perforators).
- 96% usable perforators.
- Doppler 65% PPV.

Skin

- Incise along line—unless perforators—draw 1 cm away.
- If sensate flap—lateral femoral cutaneous nerve harvested, it runs along same line incision 45° laterally to protect perforator
  - Deepen to incise the deep fascia and expose rectus femoris and vastus lateralis.

Perforator Location

- Scissors—lateral/medial, nearly all run through vastus lateralis (>85%) (Fig. 11.11).
- If none found laterally, can look medial to septum (becomes anteromedial thigh flap—bulky).

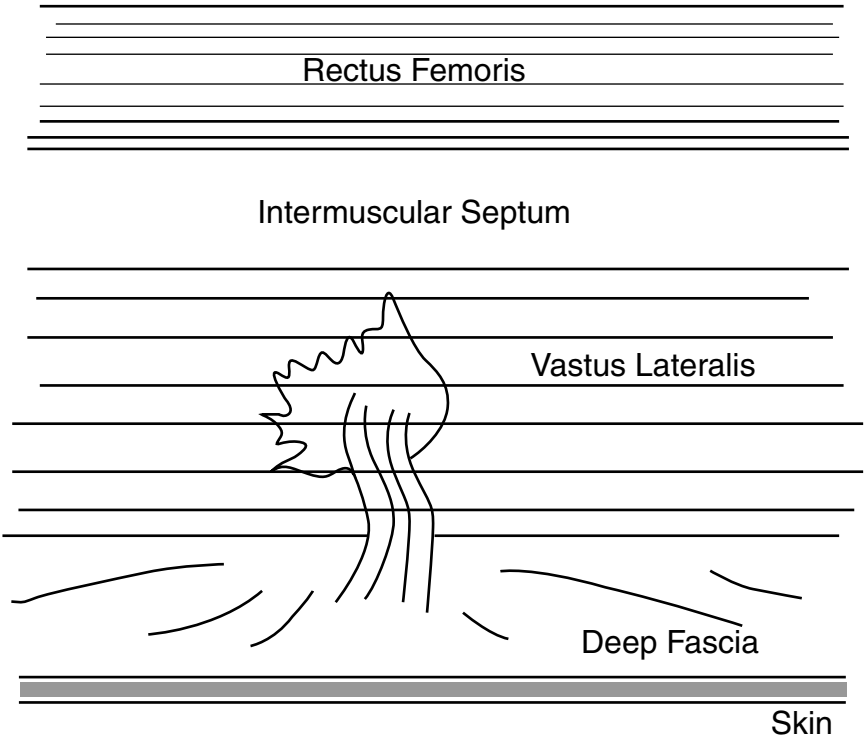


Fig. 11.11 ALT flap anatomy

- Perforators are followed though muscle and/or fascia until the main pedicle is reached between rectus femoris and vastus lateralis.

- DBLCFA with VC followed to the junction with profunda femoris (can use sutures to retract muscles).
- Once main pedicle and perforators dissected out—flap defined, and skin cuts made
  - Flap raised/isolated.
  - Observe flap undivided—bleeding.

Practical Tips

- Medial incision is the safest—go midline, risk of damaging on initial incision.
- Dissection perforation

  (a) Loops, micro dissecting forceps—can use topical lidocaine.
  (b) Safe to dissect at least 2 perforators until anatomy defined.

- Reduce risk of twisting: use 2 or more perforators.
- Mark perforators, so thin flap not damaged.

3 Types of Perforators

- Type I (90%)—from descending branches either septocutaneous or musculocutaneous (through vastus).
- Type II (6%)—single perforator from transverse branch of lateral circumflex artery (includes transverse branch).
- Type III (4%)—single perforator directly from profunda femoris, goes through rectus fermoris—abandon.

Flap Correlates With BMI

- Thinner in men.
- Peripheral vascular disease not a contraindication.
- Right side—right handed surgeons (easier).

Scapular Flap

- Description—dos Santos performed the first, popularised by Swartz.
- 10–14 cm of bone from 1 cm below the glenoid tubercle to the spine of the scapula (Fig. 11.12).
- Horizontal is called scapular flap (max dimensions 10 × 25 cm), vertical is called parascapular flap (15 × 30 cm) with primary closure.
- Area of supply may be much larger.
- Pedicle length is approximately 11–14 cm for soft tissue only and a little larger for bone as well.

Contraindications—Very Few

- Turning the patient may prolong the duration of the procedure and should not be used with insecure airway.
- Previous axillary surgery (e.g. breast cancer with lymphadenectomy).

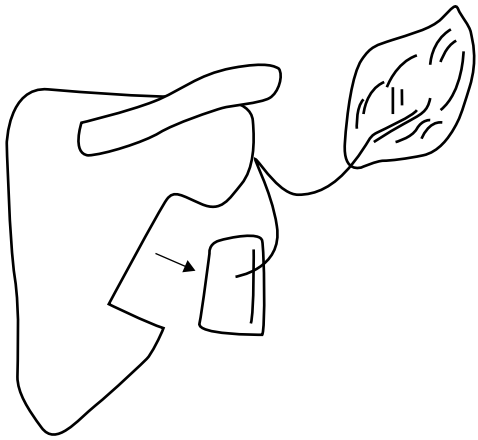


Fig. 11.12 Scapular flap

Positioning

- Decubitus position with arm extended and held with arm support. Often need a hip roll and back support for patient as well.
- Pre-operative marking with doppler of transverse circumflex scapular and descending circumflex scapular may be useful with marking of triangular space.
- Triangular space found 2/5 of the way down from the spine of the scapula to the tip.

Relevant Anatomy

- Based on the circumflex scapular artery which is one of the two branches of the subscapular artery, the other being the thoracodorsal artery.
- It passes through the triangular space, formed by teres minor superiorly, teres major inferiorly and long head of triceps laterally.
- It gives of branches to the bone and divides into the transverse branch (scapular flap) and descending branch (parascapular flap).
- Variations—Separate take off from artery (4%) or vein (12%) may occur from the axillary artery.

Technique Posterior Approach:

- Back is marked with skin paddle overlying transverse or descending vessels depending on preference with flap overlying triangular space.
- Urken describes suprafascial dissection, Boyd subfascial dissection towards lateral border of scapula.
- Teres major, minor and latissimus dorsi should be identified. Lower border of deltoid may impinge on the upper end of the dissection.
- Triangular space is outlined and pedicle followed to origin.
- If bone is taken, 2–3 cm of lateral border may be harvested preserving at least 1 cm below the glenoid fossa, and a cuff of teres major and subscapularis to preserve bony blood supply.
- Dissection may proceed through tunnel or a counter incision in the axilla may be made to allow dissection of the remaining pedicle whilst back is closed.
- Thoracodorsal artery will have to be ligated if extra length is required, however thoracodorsal nerve should be preserved.
- Teres major should be reattached to the lateral border of the scapula and the wound closed over drains with extensive undermining.

Complications

- Damage to the brachial plexus can occur from positioning, particularly if the operation takes too long. Some morbidity from separation and division of muscles is expected as is an unaesthetic scar.

Deep Circumflex Iliac Artery Free Flap

Introduction

- Osteomyocutaneous flap.
- Utilizes iliac crest bone with or without skin based on DCIA.
- Ipsilateral crest chosen for reconstruction.
- Crest forms lower border mandible.
- ASIS becomes angle of mandible.

Surgical Anatomy

- Based on deep circumflex iliac artery (DCIA: 2 mm diameter) and vein (DCIV: 2–4 mm diameter).
- DCIA originates lateral aspect external iliac artery immediately above inguinal ligament.
- Travels on deep surface inguinal ligament. gives off ascending branch (can mistake for DCIA).
- Continues along inside of iliac crest sending perforators into bone and others over crest into muscles attached.
- Major perforators to overlying skin come off 6–8 cm posterior to ASIS.
- Thus flap consists of bone, an obligatory muscle cuff (through which perforators transmitted) and overlying skin.

Positioning

- Supine, sandbag under hip may assist in access. Mark out the pubic tubercle, ASIS, iliac crest, inguinal ligament, iliac artery and vein.

Technique

- Hip is marked as previously described. Incision is made extending from the iliac artery laterally about 1–2 cm medial to the crest and following the curve posteriorly.
- An incision is made down to the external oblique which is incised in the line of the fibres along the length of the incision.
- The internal oblique is identified and cleared to allow a large proportion of the muscle to be displayed, and the dissection should proceed laterally to the iliac crest (preserving any perforators here if a skin paddle is to be taken).
- After the muscle is exposed the muscle cuff is identified and incised at the posterior superior aspect to the depth of the transversus muscle.
- The dissection then proceeds along the transversus muscle deep to the overlying fascia.
- The ascending branch should be identified on the posterior aspect of the internal oblique muscle.
- The dissection continues down and the ascending branch can be used to identify the DCIA. The DCIA is followed medially to the external iliac artery.
- Once this is complete the transversus is incised approximately 1–2 cm lateral to the crest and the preperitoneal fat is swept medially to expose the iliacus muscle.

- The DCIA may be seen at the posterior aspect.
- The iliacus is incised and the periosteum elevated to allow the bone cuts to occur.
- Dissection at the lateral aspect may also proceed at this point elevating the gluteal muscles and the TFL from the lateral aspect of the hip. The bone is marked and cut with protection of the pedicle if necessary, particularly at the medial aspect.
- The cuts are completed and the bone is delivered, severing the remaining attachments of iliacus.
- The pedicle is then followed all the way to the iliac artery removing its posterior attachments.
- The lateral femoral cutaneous nerve may run either superficial or deep to the pedicle.
- The VCs often join to form a common vein prior to entering the external iliac vein.
- Bleeding is stopped, the flap is replaced in the wound and rested to ensure vascular supply and to allow vasospasm to resolve.
- After disconnection, bone wax may be used to control bleeding.
- Transversus is stitched to the remnants of iliacus.
- Mesh may be used to replace the internal oblique, particularly over the site of the bone cut in the hip.
- A suction drain is placed, and the external oblique is closed, as are scarpa's fascia, the fat and skin.

Mandible

- Defects > 5 cm to hemimandible.
- Ameloblastoma, ORN, trauma.
- Anterior through and through defects.

Maxilla

- DCIA
  - Bone with internal oblique.
  - Unique in being able to provide height/depth of bone—restore contour of the face with adequate base for implants.
  - Internal oblique lines nasal and oral cavity.

Advantages

- Donor site—scar hidden.
- Skin paddle—discarded if use internal oblique.
- Bone—large corticocancellous 6–16 cm in length.

Disadvantages

- Bone not ideal for angle-to-angle bony defect—shaping difficult: bulky skin paddle.
- Skin paddle—colour match is poor (20% skin necrosis), closely attached to bone (not independent), lack independent movement like scapular/RFFF/fibular.
- Short vascular pedicle.

Complications

- Early post-operative pain.
- Sensory change due to transection/traction injuries of nerve crossing crest
  - Lateral cutaneous branch—subcostal nerve T1/iliohypogastric nerve L1.
  - Lateral femoral cutaneous nerve thigh (L2/L3).
- Contour irregularity, less large skin paddles.
- Poor scarring—gait disturbance—inflammation tensor fascia lata.
- Hernia—use mesh, close in layers.

Complications

- Integrity of the abdominal wall is the greatest concern as inadequate repair or poor quality tissues may lead to herniation of abdominal contents.
- Damage to the bowel and intra-abdominal contents may occur.
- Damage to the lateral femoral cutaneous nerve is possible and leads to numbness of the side of the thigh.
- Damage to the subcostal and iliohypogastric etc.are almost inevitable.
- Gait problems including limp and pain are possible, and are related to disruption of the muscular attachments of the gluteal musculature.

Chapter 12
Craniofacial Surgery

Abstract An overview of craniofacial abnormalities, their aetiology, assessment and management.

Key Points

- Development of the cranium, nasomaxilla and mandible.
- Growth evaluation.
- Craniosynostosis.
- Positional plagiocephaly.
- Frontal plagiocephaly.
- Brachycephaly.
- Metopic synostosis (trigonocephaly).
- Sagittal craniosynostosis (scaphocephaly).
- Unilateral lambdoid/occipital/posterior plagiocephaly.
- Lambdoid suture.
- Pansynostosis.
- Frontal orbital advancement.
- Monobloc/frontofacial advancement.
- Apert Syndrome.
- Pfeiffer Syndrome.
- Saethre-Chotzen Syndrome.
- Pierre Robin Sequence.
- Treacher Collins Syndrome.
- Orbital dystopia.
- Hemifacial microsomia.
- Distraction osteogenesis.

- At birth, cranial bones are separated by sutures with fontanelles—where the corners of the bones meet, permitting compression of the skull during birthing process.
- Post-natal growth results in narrowing of the sutures with all the fontanelles closing within first two years.
- As brain expands, the pressure creates tension across the sutures and compression against the cranial bones.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_12

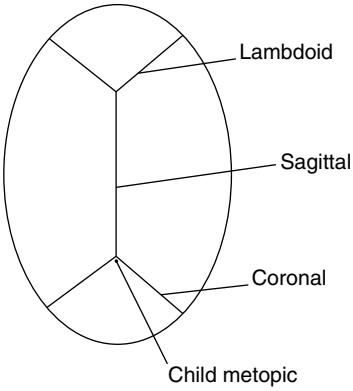


Fig. 12.1 Sutures

12.1 Cranial Base

- Growth cranial base genetically determined.
- Anterior cranial base matures before posterior.
- Spheno-occipital synchondrosis: Greatest contribution to cranial base growth post-natally delaying fusion until adolescence.
- Normal development—ossification sutures (Fig. 12.1)
  - Metopic 2 years.
  - Sagittal 22 years.
  - Coronal 24 years.
  - Lambdoid 26 years.
- Growth generally pre-determined
  - Dura induces bone formation.
- Skull base: Endochondral.
- Calvarium: Membranous.

12.2 Nasomaxilla

- Overall middle and lower third face develop primarily downwards and forward from cranial base due to brain development, maxillary/palate sutural growth and nasal septum growth.
- This results in downwards and forwards translation—adequate space for developing nasopharynx and growth of the posterior aspect of the maxilla to provide adequate space for development and eruption of maxillary molars.
- Minimal maxillary growth after age 10.

Table 12.1 Growth completion craniofacial structures

	1 y/o (%)	5 y/o (%)	Average age maturity
Cranium	85	92	Male 14/Female 16
Orbits	85	96	Male 15/Female 13
Zygoma	72	83	Male 15/Female 13
Maxilla	75	85	Male 15/Female 14
Mandible	65	80	Male 16/Female 14

12.3 Mandible

- Most postnatal development.
- R + L bodies still separate at birth: face at midline—mental symphysis at 1 year-old.
- Primary site of mandible post-natal growth
  - Endochondral apposition—condylar cartilages.
  - Intra-membranous opposition of posterior aspects of rami and alveolar ridges.
- Growth dependent on postnatal demands—LP/tongue growth + function/facial soft tissue.

12.4 Clinical Evaluation of Growth

- Anthropometrics measurements (clinical and on CT).
- Hand wrist X R/central spine maturation.
- Serial cephalometric.

12.5 Craniosynostosis

- Premature fusion of cranial sutures (Table 12.1).
- Classification
  - Non-syndromic/syndromic.
  - Single/multiple.

12.6 Aetiology/Pathogenesis

- Majority: Gene mutation in the fibroblast growth factor reception (FGFR) family—which control osteogenesis of the cranial sutures (David 2003).

- Virchow's law.
- Compensatory growth following premature fusion.
- Inhibition of normal suture growth perpendicular to the fused suture and compensatory growth in direction of affected suture.
- Moss Theory.
- Cranial base is the primary locus of abnormality in craniosynostosis. Altered cranial base transmitted through transmission of tensile forces through the dura leading to premature closure of sutures.

12.7 Average % Growth Completion

See Table 12.1.

12.8 Non-syndromic

- 1:2500.
- Sagittal suture (Scaphocephaly)—commonest (40–50%).
- Metopic synostosis (trigonocephaly)—2nd commonest (15%).
- Unilateral coronal (anterior plagiocephaly)—15–20%
- Bilateral coronal (brachycephaly).
- Unilateral Lambdoid (posterior plagocephaly)—<10%.
- Plagiocephaly—slanted head: Oxycephaly—Sagittal + both coronal.

Aetiology

- Primary.
- Secondary
  - Microcephaly.
  - Overshunting.
  - Bone dysplasia.
  - Medication.
- Multifactorial: Genetic/environment.
- If one child: increased risk, multifactorial—environmental
  - Pregnancy.
  - Birth—difficult with forceps.
  - Twins: compression/oligohydramnios.
  - Diabetes: Large babies.

Diagnosis

- Suspect any infant with abnormal head shape.
- Ask about sleeping positions, family history of abnormal head shape.

Clinically

- Palpation skull—movement, ridging, preserve anterior/posterior fontanelles.
- Relationship of supraorbital rims to cornea (should be infant).

Work-up: Imaging

- Plain skull x-ray: absence of radioluncent line in normal anatomic position of suture.
- Lateral cephalogram.
- Some units use ultrasound.
- 3D CT.
- Refer craniofacial center.
- some evidence single suture may get
  - Increased ICP (0–20% risk).
  - Deformity.
  - Developmental delay (No evidence).

12.9 Principles

- Craniofacial unit: 4 units in United Kingdom (less centralised in Europe)
  - GOSH.
  - Birmingham.
  - Oxford.
  - Alder Hey.

Surgical Goals

- Allow brain to grow, prevent visual complications.
- Optimise aesthetics, prevent long-term complications.
- Release of fused suture and calvarial ± orbital re-shaping.

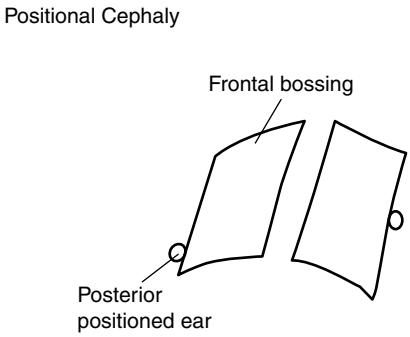


Fig. 12.2 Positional plagiocephaly

12.10 Positional Plagiocephaly

Moulding

- Differential diagnosis from craniosynostosis (more common).
- Associated with
  - Posture, cervical scoliosis, torticollis, squint, maternal smoking, sleeping position.

Clinical (Fig. 12.2)

- Asymmetrical head shaped: normal development/head size/sutures on imaging.

Management

- None/moulding devices/change position.
- Surgery only for severe secondary deformity.

12.11 Frontal Plagiocephaly (Unilateral Coronal)

- Produces helical twisting on face and cranial base.
- Facial scoliosis.
- FOA 6–12 months.
- Harlequin deformity of superiorly displaced lesser wing of sphenoid.
- 20% non-syndromic, most sporadic.

Clinically

- Rule out infant molding.
- Positional plagiocephaly: normal eyebrow and nose.

- Congenital torticollis shortening SCM, tilting of head, chin points up towards affected side.
- CT scan/clinical.
- X-ray: Harlequin deformity/sign.

Superior View

- Ipsilateral
  - Frontal bone flat, supraorbital and lateral orbital rim recessed.
  - Orbit shallow, anterior cranial base short in AP.
- Contralateral: Bossing forehead, and fronto-orbital rim.

Frontal View

- Root of nose constricted and deviated to affected side. Nasal tip points opposite side.
- Ipsilateral eyebrow raised with increased palpebral fissure.

Surgery

- Suture release and cranial vault and orbital osteotomies for reshaping and advancement.
- Patient on Mayfield head rest, coronal incision, subperiosteal flap along with temporalis bilaterally.
- Extend to periorbital/temporal regions, maintain attachment medial canthal tendons.
- Then fronto-orbital advancement at year one.

12.12 Brachycephaly (Bicoronal)

- Most common syndromic.
- 20% non-syndromic.
- Some FGFR3 point mutation, sporadic.

Clinically

- Anterior cranial base short AP dimension, wide transverse.
- Overlying cranial vault high in superior-inferior dimension—anterior bulging upper forehead resulting from compensatory growth metopic suture.
- Supraorbital rims/eyebrows—displaced superior/posterior.
- Orbits: Shallow (exorbitism), exophthalmos, hypertelorism, harlequin deformity of superiorly displaced lesser wing of sphenoid.

Surgery

- Fronto-orbital advancement—1 year.
- Cephalic index >80%.

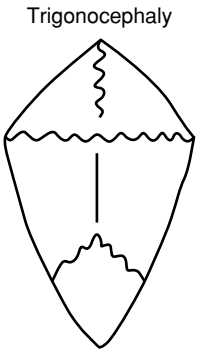


Fig. 12.3 Trigonocephaly

12.13 Metopic Synostosis/Trigonocephaly

- 10% non-syndromic cases.
- Metopic (single suture).
- Fuses 12–18 months.
- Produces keel shaped head = Trigonocephaly.
- FOA 12 months.

Clinically (Fig. 12.3)

- Triangular shaped head, rare to have raised ICP.
- Bitemporal narrowing, compensatory biparietal widening.

Frontal

- Orbital dystopia, including triangular orbits with hypotelorism.
- Forehead sloped posteriorly
  - 20–30% measure development defect.
- Operations: make significant difference (unsure).
- ACF—hypoplastic = affect frontal lobe.

Surgery

- Metopic sutures release, fronto-orbital advancement.
- Orbital hypotelorism—splitting supraorbital ridge: vertical in midline and placing autogenous cranial base grafts to increase intraorbital distance.

12.14 Sagittal Craniosynostosis (Scaphocephaly)

- 50% of all suture craniosynostosis.
- Commonest 1:4000.
- Keel (boat) shaped—long thin.

- Palpable ridge.
- Cephalic index—biparietal width/AP dimension = normal 70–75% (<75%).

Clinically

- Elongated AP dimension, narrow transverse
  - Frontal bossing
    1. Bitemporal hollowing.
    2. Occipital bullet.
- Anterior fusion—anterior bulge.
- Posterior fusion—posterior bulge.

Surgery

- Usually done at 3–6 months (strip/craniectomy).
- Exam of sutures.
- Calvarial remodelling.
- Allow brain to growth.
- Attach it with Tisseel/sutures as bone thin for plates.
- Some units: Total calvarial remodelling—do front and back.
- Some units: 1 front/1 side—baby can lay on head.

12.15 Unilateral Lambdoid/Occipital/Posterior Plagiocephaly

- Rare 3%—least common.
- Posterior flattening.
- Must distinguish from moulding (far more common and innocent).
- May need posterior release.

Clinically

- Rule out infant moulding/torticollis.
- Positional/deformational plagiocephaly.
- Ipsilateral ear and forehead are postured anteriorly.
- Ear not inferiorly placed.

Superior View

- Flatness affected ipsilateral parieto-occipital region.
- Ear canal and external ear more posterior and inferior on ipsilateral side.

Surgery

- Vault craniectomy and reshaping generally necessary.
- Some strip osteotomy—spring distraction.

12.16 Lambdoid Suture

- Contralateral frontal bossing.
- Ear: Ipsilateral (posterior and lower).
- Trapezoid skull.
- Facial scoliosis—orbital tilt.
- Management: Posterior release = distraction/spring.

12.17 Cloverleaf—Pan Synostosis

Copper Beaten Skull—Increased ICP

- ICP monitoring.
- Pressure transducer.
- Delayed childhood milestones.
- Fontanelles.
- Eyes—papilloedema.

12.18 Surgical Management Considerations

Functional Considerations

Neuro

- Increased ICP—pressure > 15 mm/Hg 1%—1 suture/42% > 1 sutures.
- Headache, poor feeding, difficulty sleeping, no evidence for developmental delay.
- XR: Fingerprinting/copper beaten skull.

Visual Impairment

- Untreated intracranial hypertension—papilloedema, eventually optic atrophy and develop visual loss.
- Posterior plagiocephaly—significant hemifield defect (visual asymmetry).
- Some involve orbital hypertelorism—compromise visual acuity/binocular vision.

Aesthetics

- Cosmetic—parents: integration into society.
- Why treat: Bertelum 1958.
- Increased deformity = get worse.
- 171 cases untreated
  - 26 papilloedema.
  - 33 optic nerve damaged.

- 29 epilepsy.
- 42 reduced IQ.

12.19 Timing Surgery

- Intracranial volume triples in the first years of life.
- Up to 18 months—sutural growth is very important, after that age brain growth velocity slows, appositional growth takes over.
- Posterior fontanelle closes 3–6 months, anterior fontanelle closes 9–12 months.
- A good indication of clinical growth is skull circumferential measurement.
- Children >1-year-old, thicker bone difficult to modify.

Timing Depends On

- Sutures involved.
- Size/health of child.
- Development of frontal sinus (2–9 years).
- Ability to form new bone.
- Repair 4–8 months.
- Others age 2.5–3 years.
- Early repair: allows rapid frontal lobe growth—supports forehead and supraorbital ridge advancement: cranium highly malleable, easier to contour.

12.20 Fronto-Orbital Advancement

- Coronal flap/extending lift off temporalis.
- Anterior extension—start of medial canthal tendons.
- Fronto-orbital bandeau/bar and bifrontal craniotomy.
- Barrel staving osteotomies for reshaping/moulding.
- Development supraorbital bandeau with minimum 1.5 cm height.
- Resorptive plate fixation (Figs. 12.4 and 12.5).

Complications

Peri-Operative

- Blood loss.
- Subdural haematoma.
- CSF loss.
- Anaesthetic risk.
- Periorbital injury.
- Death.

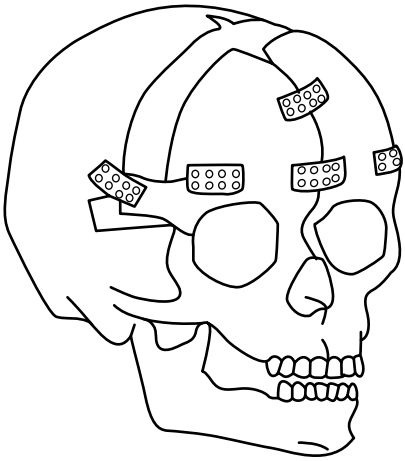


Fig. 12.4 Fronto-orbital advancement

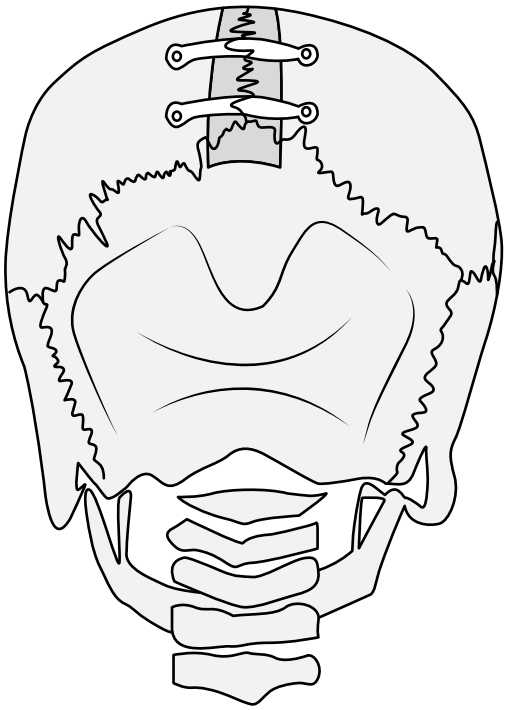


Fig. 12.5 Fronto-orbital advancement (posterior)

Post-operative

- Bleeding.
- Haematoma.
- Corneal abrasion.

- Stitch abscess
  - CSF leak.
  - SIADH.
  - Infection/meningitis.
  - Vision loss.
  - Airway obstruction/death.

12.21 Monobloc/Frontofacial Advancement

- Simultaneous advancement of forehead/orbits/midface = for correction of severe exorbitism (Fig. 12.6).
- Osteotomy cuts similar to Le Fort III/frontal bone advancement—except leave
  - Nasofrontal.
  - Frontozygomatic.
- Expansion of orbital volume
  - Massive morbidity: Cranial fossa opens to nose, dural tears, infections—use pericranial flaps.
  - May need repeating.
- Some units do posterior calvarial distraction instead of mono bloc = create volume.

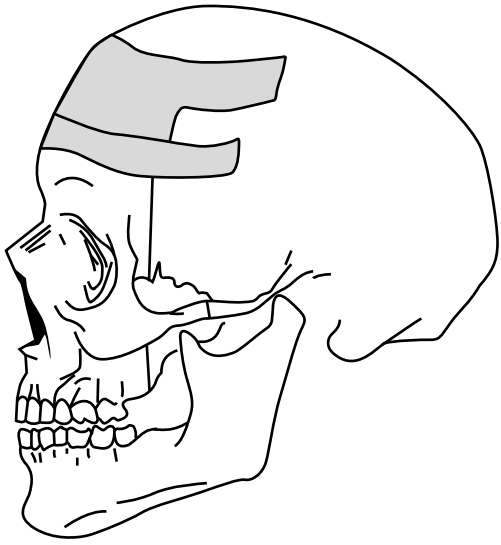


Fig. 12.6 Frontofacial advancement

- Le Fort III: Advancement for severe midface retraction
  - Bony framework nose divided at nasofrontal junction osteotomy carried backward across medial wall of orbit on each side, then downwards to floor of orbit.
  - Narrow osteotome used to section the delicate lamina papyracea of the ethmoid.
  - Transverse cut across orbital floor and joins the inferior orbital tissue to the lower end of the medial osteotomy.
  - Lateral orbital wall: cut at FZ suture or above, the arch is retracted.
  - Orbital contents restricted medially—lateral orbital wall is divided at its junction with cranium.
  - The lateral orbital wall osteotomy is continued inferiorly and posteriorly through pterygomaxillary fissure.
  - Can use red frame—for vector changes.
  - Distractors: RED (rigid external distractor).

12.22 Syndromic Craniosynostosis

300 Cases All Genetic, FGFR

- Apert FGFR2: 1:60,000 (10–20 children)—syndactyly/lower IQ.
- Crouzon FGFR2: 1:60,000.
- Muenke syndrome FGFR3: 1:30,000 commonest, p250 gene
  - Thumblike fingers.
  - Variable expression.
- Pfeiffer syndrome FGRR 1–2: 1:50,000 (big thumbs).
- Saethre-Chotzen syndrome—Twist gene: ptosis eyelids.

12.23 Clinical Examination (Top to Bottom)

- Skull: Synostosis.
- Eyes: Hyperplastic orbitis—Apert: Hypetelorsim and down-slanting due to facial bipartition.
- Nose: short nose/nose beaked.
- Midface: hyperplastic (all)/may have cleft palate.
- Mandible: relatively class III.
- Limbs: Apert Syndrome.
- Issues—must address
  - Airway: Lack of AP growth—pharyngeal airway problems.
  - Feeding: NG tube.

- Increase in ICP—increased vomiting/no treatment.
- Eye position: shallow orbits/prevent dislocation (tarsorhaphy/FOA).
- Growth.
- Sleep apnoea.
- Other issues: Limbs/cardiac/GIT.

12.24 Why Treat?

Early

- Airway: crisis.
- ICP.
- Eye protection—exorbitism.
- Feeding.
- Crisis intervention: straightaway.
- Or usually, 1–2 years old.
- Middle: Brain development deformity.

12.25 Operations

- Dura causes skull growth.
- Bone will grow with different timings
  - Age 1: Early: increase reoccurrence.
  - Late: Appearance/moulding of brain not as good.
- Can do in stages—6 months: posterior vault expansion, especially Apert's.

Midface

- Ideally age 18: orbits fully formed 6–8 years.
- Consider Le Fort III (earliest age 7–8): will need bimax later or wait until fully grown.
- Apert: Bipartition—stops slanting eyes, causes diastema in central incisors.

Monobloc

- Distraction.

LE Fort III

- Distraction.

Now

- Cranial vault expansion: posterior vault.
- And later monobloc distraction.

Apert Syndrome

- 1:60,000 (10–20 children in all).
- Autosomal dominant and sporadic.
- FGFR2 receptor gene mutation (cartilage dysplasia at cranial base).

Clinical

- Brachycephaly (±other sutures).
- Hypoplastic midface with depressed nasal bridge and parrot beak/beaked nose
  - High arched palate, Class III with AOB.
  - 30% Cleft.
- Orbital hypertelorism and proptosis/exorbitism (due to midface hypoplasia, downslanting palpebral fissure).
- Symmetrical syndactyly of hands and feet.
- 30% deafness, decrease IQ.
- Acne—thickened facial skin, increase sebaceous discharge.
- Similar to Crouzon except
  - Hand deformity, more severe (orbits—midface hyposplasia).
  - Greater antimongoloid slant.
- Management: Similar to Crouzon except facial bipartition often required.

(a) ***Early-infancy***

- Suture release and cranial vault reshaping, usually FOA.
- Consider infant destruction of mono block severe cases.

(b) ***Middle (6–12)***

- Midface advancement with extracranial Le Fort III or mono block advancement ± facial bipartition (depending on degree of hypertelorism—rare Crouzon).
- Le Fort III—DO = internal/external distraction depending on stability of zygomatico-maxillary junction.

(c) ***Late (>16 years)***

- Le Fort I: Advancement ± mandibular procedures ± genioplasty.
- Aesthetic: nasal augmentation, soft tissue correction.

12.26 Crouzon Syndrome

- 1:60,000.
- Autosomal dominant mutations FGFR2 receptor—most common form of craniosynostosis.
- Variably expressed, sporadic.

Clinical

- Normal extremities.
- Brachycephaly (±other sutures).
- Symmetrical hypoplasia of midface (cranial base + upper sutures involved).
- Class III, mandibular growth is normal.
- Shallow orbits, hypertelorism, proptosis, exorbitism = drying/eye ulceration.
- Conductive hearing loss—common EAC stenosis.
- Hydrocephalus (5–10% children).
- Occasional intellectual impairment.
- Management: Same as Apert.

12.27 Pfeiffer Syndrome

- Autosomal dominant. Complete penetrance, variable expression.
- Mutations FGFR 1 and 2.

Clinical

- Broad thumbs and great toes (spoon shaped).
- Craniosynostosis—often brachycephaly (cloverleaf skull—multiple synostosis).
- Midface hypoplasia with small nose/forehead looks big.
- Hypertelorism/proptosis—not as prominent as Apert/Crouzon.
- Conductive hearing loss, IQ usually normal.
- Subtypes by Cohen 1993—3 types.

    1. Normal life span, near normal IQ, variable midface involvement/hydrocephaly.
    2. Cloverleaf skull—severe midface, broad thumbs/toes—limited life span.
    3. Cloverleaf skull—very poor prognosis, neurologically impaired.

- Management: Same as Crouzon.

12.28 Saethre-Chotzen Syndrome

- Autosomal dominat—coronal synostosis.
- Mutation TWIST 1 gene.

- Low set frontal hairline, ptosis upper eyelids, no midface hypoplasia.
- Face asymmetrical with deviation of nasal septum and orbits at different levels, nose beaked with absence of fronto-nasal angle.
- Short digits and index/middle fingers and partial syndactaly.

12.29 Pierre Robin Sequence

- French stomatologist.
- 1:2000–30,000.
- Aetiology (3 theories).

Mechanical

- Most accepted: initial event—7th–11th neck gestation: posturing of head on chest wall = mandibular hypoplasia.
- This keeps tongue high in the oral cavity = causing cleft by preventing closure of palatal shelves.
- Explains classic U-shaped cleft/and absence of lip.
- Oligohydramnios could play a role—lack of amniotic fluid = cause formation of chin and subsequent impaction of tongue between palatal shelves.

Neurological Maturation Theory

- Delay in tongue musculature neurological maturation = noted on electrography.
- Spontaneous correction of majority of cases with age supports this theory.

Clinical (msg)

- Micrognathia—often severe.
- Cleft Soft Palate: Wide U-shaped + GORD—severe.
- Glossoptosis (downward displacement of tongue).
- Airway difficulties—frequent aspiration of food = NG tube feeding 6 months.
- Airway obstruction in severe cases—mandible retrognathic, genioglossus muscles are shortened = tongue falls back and occludes airway.

Ears

- Otitis media—80%, auricular anomalies in 75% cases.
- Hearing loss, mostly conductive (60%)/EAC atresia (5%).

Management

- Airway compromise.
- Monitor: Suture tongue to lip, tracheostomy, Distraction osteogenesis (DO).
- Position patient.
- Soft palate.
- Delay closure until mandible grows, early repair could cause obstruction by displacing tongue inferior and posterior.

Micrognathia

- Distraction osteogenesis—gradual lengthening, may be able to wean of tracheostomy.

12.30 Treacher Collins Syndrome

- Autosomal Dominant with variable expression—Treacle (TCOF1) gene—90–95% patients = genetic test available.
- Mandibulofacial dysostosis.
- Bilateral, symmetrical abnormalities within the 1st and 2nd branchial arches.

Clinical/Pathogenesis (3 Theories)

- Disordered development of 1st and 2nd branchial placodes.
- Failure of neural crest migration.
- Marx and Stern: Absence abnormal fetal stapedial artery. Artery supplies middle ear (maxilla/mandible).

Clinical

- 'Fish' and 'bird'; like features.
- Hypoplastic molars, retrognathia.
- Down sloping palpebral fissures.
- Convex facial profile due to prominent nasal dorsum/Class II AOB/retrognathic mandible.
- Antimongoloid slant = coloboma/hypoplasia of lower eyelid/lateral canthi.

Ears

- Middle ear abnormalities (hypoplastic/absent ossicles)
  - Uni/bilateral conductive hearing loss—learning difficulties (normal intelligence).
  - External ears malformed/malpositioned (anterior/posterior).

Midface

- Hypoplastic zygomas—cheek flattened
  - Maxillary/mastoid process—not pneumatised.
  - Maxila hypoplastic—may show (AOB/widely spaced teeth/high arched palatal vault).
  - Cleft palate ±lip (30%).
- Nose: Nasal dorsal prominent due to hypoplastic maxilla.
- Mandible: Grade as per Kaban's Classification.

- Occlusion/dental: Class II AOB with steep (occlusion plane, 60% tooth agenesis, ectopic eruption of first maxillary molars.
- Soft tissues deformities (4 regions)
  - External ears.
  - Eyelid.
  - Pre-auricular/cheek skin.
  - Temporal fossa (temporalis hypoplasia).
- Mental: Normal intelligence = learning delays due to hearing problems.

Diagnostic Tools

- Audiology/ophthalmology assessment/genetic testing/3D CT.

Differential Diagnosis

- Acrofacial dysostosis (Nager Syndrome (AR—very similar, except have hypoplastic/absent thumbs).

12.31 Orbital Dystopia

- Any type of abnormal displacement of the entire orbital cones—bones (occur in 3D).
- Horizontal: Hypertelorbitism.
- Vertical: Vertical orbital dystopia—depends on intercanthal distance.

Causes

- Facial trauma.
- Muscular torticollis.
- Facial skeletal tumours.
- Idiopathic.
- Iatrogenic.
- Facial clefts—0/14, unilateral coronal synostosis.

MDT Approach

- Orthodontist—occlusion, facial bipartition—oral splint especially if do Le Fort I carried out.
- Ophthalmology—visual impairments.
- Psychology.
- Craniofacial/neurosurgeon.

CT/3D Skull Planning

- Complex craniofacial deformity.
- Herniation of brain and meninges: pre-operative planning.

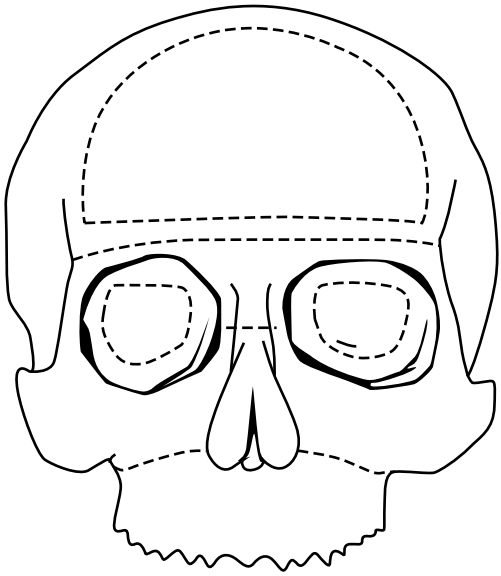


Fig. 12.7 Box osteotomy

Orbital Box Osteotomy

- Correction horizontal—orbital hypertelorbitism and also vertical orbital dystopia.
- Osteotomies (Fig. 12.7)
  - Frontal craniotomy.
  - Zygomatic/maxillary/orbital.
  - Central translation of orbits.

12.32 Hemifacial Microsomia

- Also known as craniofacial microsomia 1st/2nd branchial arch, Goldenhar syndrome.
- Canton (1861), Von Arlt (1881)—term HFM cured by Gorlin and Pinborg in 1964.
- Malformation affecting 1st/2nd branchial arch structures—orbit/maxilla/mandible/ear/cranial nerve/facial soft tissues.
- 10% CLP, some have other structures—Goldenhar (10%)
  - Epibulbar dermoid.
  - Rib/vertebral anomalies.
  - Usually bilateral.
  - Cardiac/renal abnormalities.

- 2nd most common after CLP.
- 1:5600 live birth.
- M = F.
- 5–15% bilateral hallmark asymmetry compared to Treacher Collins.

Aetiology

- Poorly understood, sporadic, can occur in families.
- Two theories
  - Stapedial artery haematoma (Poswillo 1973)—size correlates to extent.
  - Neurodermal—Johnson (1991) = interference neural crest migration.

Classification

- OMENS
  - O

    0: Normal orbit.
    1: Abnormal size.
    2: Abnormal position.
    3: Abnormal size + position.

  - M

    0: Normal mandible.
    1: Hypoplastic ramus.
    2: Hypoplastic + malformed ramus.
    3: Abnormal ramus + glenoid fossa.

  - E

    0: Normal ear.
    1: Auricular hypoplasia.
    2: Absence of EAC.
    3: Absence of auricle + malposition lobe.

  - N

    0: Normal facial nerve.
    1: Upper facial nerve involvement.
    2: Lower facial nerve involvement.
    3: All branches.

  - S

    0: No soft tissue deficiency.
    1–3 Minimal/moderate/severe soft tissue or muscle deficiencies.

  - **SAT**

    Skeletal 1–5.

Auricular deformities (0–3 Meurman) Ear deformities

- Hyperplasia—all present.
- Atresia EAC—hearing loss—(conductive/hypoplasia ossicles).
- Absent auricle/lobular remnant infection (hearing loss—conductive/hypoplasia ossicles).

Soft tissues (1–3 mild/moderate/severe).

Pruzansky (1969)

- Type I: All present hypoplastic.
- Type II: Small abnormal shaped ramus and under-developed displaced TMJ.
- Type IIa: Glenoid acceptable position (Kaban modification).
- Type IIb: Glenoid abnormal position.
- Type III Absent ramus and glenoid fossa.

Clinically—Assessment

- Clinical recognition.
- Imaging.
- Models.
- ECHO.
- Hearing assessment.

12.33 Management

- Management very similar to Treacher Collins—two theories

A. Interceptive progressive (Kaban)

  – Asymmetry progression.
  – Early correction mandible, maxilla less affected.

B. Leave/non-progressive (Poswillo)

  – Deformity changes in proportion to child—correct mandible in adolescence.

Early

- Pre-natal

  – Airway support ± crisis tracheotomy – if hypoplastic mandibular ± choanal atresia stenosis.
  – Nutritional support ± feeding tube (especially cleft).
  – Confirm diagnosis, exclude other abnormalities.

- Infant 0–3 months
  - CT assess skull/face/inner ears.
  - Ophthalmology—assess hearing, brainstem evoked potential.
- 3 months to 5 years
  - Monitor growth and development, ongoing support.
  - Consider mandibular surgery if persistent airway problem e.g. OSA.
  - Cleft as per protocol.
  - Surgery to improve hearing by preschool—bone anchored hearing aid.
- Middle (age 5–8)
  - Reconstruct orbits/zygomas using cranial bone grafting.
  - Lateral canthopexies to correct palpebral fissures.
  - Consider mandibular lengthening especially Kaban 2b+ and 3.
  - Ear reconstruction/prothesis.
  - Staged: sculpted rib cartilage around age 6–7.
  - Consider implant supported prosthesis of remnant.
- Late
  - Orthognathic

    Maxilla: limited by hypo plastic tissues.
    Mandible: VSSO, inverted with osteotomy ± genioplasty.
    TMJ: Reconstruction/costochondral.
  - Rhinoplasty.

Nose

- Rhinoplasty: dorsal hump reduction, septal grafting improves tip support.
- All after orthognathic surgery.

Temporal Fossa Hollowing

- Due to deficiency in skin/temporals.
- Often need free flaps—paracapsular.

Skin Tags

- Removed 1st year—have sinus tract.
- Macrostomia: Repair orbicularis oris muscles.

Skeletal

- Type IIa/b = Distraction osteogenesis/mandibular osteotomy.
- Type IIb/III = costochondral graft

  - Microvascular second metatarsophalangeal joint transfer—epiphyseal growth planes for centre growth.
  - Mild cases—functional orthodontics.

End stage

- Orthodontics, CF/BSSO.
- Soft tissue—fat infection (seal)/free flaps.

Facial Nerve

- Complete deficiency (rare)—protect eye
  - Eye patches/lubricants.
  - Complete—tarsorrhaphy.
- Facial nerve grafting
  - 2 stage from opposite side with sural nerve.
  - 2nd stage free tissue transfer.
- Static—slings fascia lata.

Mandible

- Uncomplicated leave full end growth—type 1 and 2a (early after skeletal maturity—BSSO, Le Fort I Maxilla).
- Type 2b: May be severe—airway compromised, may need ramus osteotomies ± Distraction osteogenesis with gradual lengthening.
- Type 3: Need to reconstruct congenital missing parts—1st stage costochondral graft at age 7–10.
- Severe missing tissues/skeletal + soft tissues = may need free flap (e.g. fibula).
- Final orthognathic surgery at skeletal maturity.

12.34 Distraction Osteogenesis

- Process of generating new bone between two bone segments in response to gradual stress across the bone gap (Fig. 12.8).
- Principle—tension stimulates histogenesis.
- Three phases
  - Latency: Perform osteotomy/corticotomy to provide time for reparative callus formation in the destruction zone in around 5–7 days, depends on age/site.
  - Activation: Gradual force to elongate the inter-segmenting callus under tension—new bone (1 mm/day—0.5 mm × 2 turns).
  - Consolidation: 8–12 weeks.

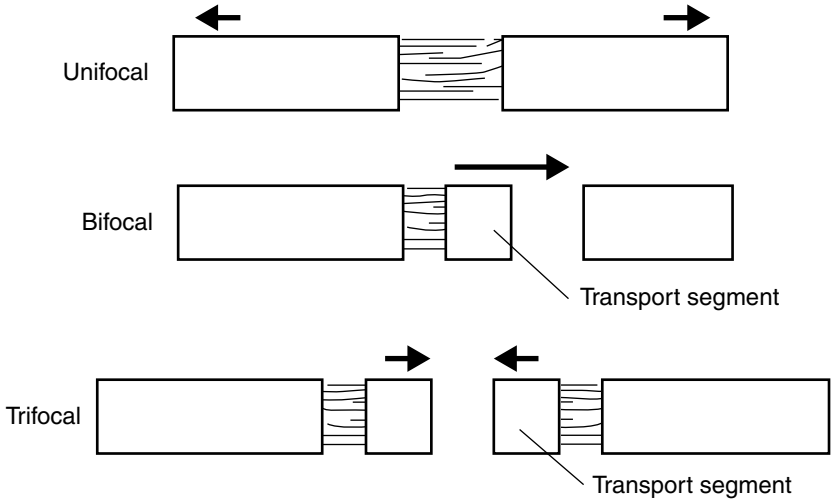


Fig. 12.8 Distraction osteogenesis

- Distraction histogenesis: Generation of membrane and lengthening/generation of new soft tissues.
- Theory.

Haematoma

- Inflammatory cells—fracture gap.

Angiogenesis

- Mesenchymal cells—increase blood flow.

Both Stages Above Activate

- Increase TGF beta 1 = collagen deposition—osteoid synthesis and mineralisation.
- External/internal distractors.
- <Age 2 = airway—distraction.
- Age 2–6 = Pruzanky 2b/3.
- Maxilla distraction = Le Fort I.
- Midface = Le Fort III.
- RED—Rigid External Distraction.
- External: RED—scars, psychological appearance, screws can become loose—tighten.
- Can cause Dural injury, may lead to infection.
- Advantages: Easily make changes with different vectors easily.

Chapter 13
Skin Surgery

Abstract An overview of the aetiology, incidence and types of skin cancers. Management and techniques of surgical reconstruction.

Key Points

- Benign skin lesions including Bowen's disease, keratoacanthoma, chondrodermatitis nodularis and Merkel cell carcinoma.
- Basal cell carcinoma.
- Squamous cell carcinoma.
- Flap design.
- Use of integra and skin grafts.
- Melanoma.
- Largest organ of the body.
- Made up of 3 layers.
- Epidermis: contains keratinocytes. Has 5 inner layers.
- Dermis: contains blood vessels, immune cells and fibroblasts. Mast cells in this layer release histamine which leads to hives in response to an allergic reaction.
- Subcutaneous layer.
- Fitzpatrick Skin Type.

13.1 Benign Skin Lesions

Solar Keratosis

- Very thin/scaly/erythematous.
- Slow-growing, scale, indistinct region, can be painful.
- Pathology: Atypia/dysplasia epidermis.
- 3% → SCC

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_13

  - When ulceration/induration noted.
  - Can regress spontaneously.

Prevention

- Sunscreen, factor 16.
- No treatment if small/thin.
- If close to eyes → cryotherapy, curettage or 5-fluorouracil (5FU).

Bowen's Disease

- Considered SCC in situ, or premalignant—sun, arsenic, HPV.

Clinical

- Slow growing.
- Well-defined borders unlike solar keratosis.
- Pink scaly plaque.
- Can have surrounding telangiectasia.

Pathology

- Atypia/dysplasia epidermis, mitosis.

Management

- Cryotherapy.
- Curettage.
- Excision.

Keratoacanthoma

- Contentious
  - Self-resolving tumour.
  - Variant SCC.
  - Rapidly proliferating polypoid lesion.
  - Central ulceration/horn.
  - Grows 2–3 weeks = static then resolves over 3 weeks.
- 1:1000—common.
- 2:1 Male:Female with age.

Aetiology

- Age over 50.
- Fair skin, light hair, or light eyes.
- Male.
- Chronic exposure to sunlight or other ultraviolet light.
- Exposure to certain chemicals, such as tar.
- Exposure to radiation.
- Longterm immune suppression: post transplant.
- Longterm presence of scars.

- Chronic ulcers.
- HPV.
- Previous skin cancer.

PRICTG

- P: Pre-malignant.
- R: Radiation.
- I: Immunodeficency/infection.
- C: Chronic wound.
- T: Toxins.
- G: Genetic.

Chondrodermatitis Nodularis

- Scaly ulcerated lesions of ears.
- Sharp pain.
- Actin damage, exposed cartilage.
- Risk of SCC.
- Management → Excision.

Merkel Cell Carcinoma

- Rare malignant tumour
  - Increase risk of recurrence.
  - Skin lesions.
  - Metastasis.
- Merkel cell → touch receptors
  - Lip/hard palate/neck.
- <1% skin cancers.

Aetiology

- Age >75 years.
- UV.
- Immunosuppression.
- Congenital ectodermal dysplasia.

Clinical Presentation

- Painless subcutaneous nodules.
- Slightly red/purple hue.
- 30% present with lymphadenopathy.
- Grows rapidly and metastasise.

Differential Diagnosis

- BCC, SCC, melanoma, cutaneous lymphoma, metastatic small cell cancer.

Management

- Excision—wide local excision ±RT/SNB.

13.2 Basal Cell Carcinoma

Definition

- Slow growing.
- Local invasive tumour.
- Metastasis → extremely rare.
- Local tissue destruction.
- Basal cell layer invaded.

Types

- Less aggressive
  - Nodular.
  - Superficial.
  - Pigmented.
- Aggressive → Can show perivascular/perineural invasion
  - Morphoeic.
  - Micro-nodular.
  - Infiltrative.
  - Basosquamous.

Incidence/aetiology

- 100 in 100,000 Common, increase incidence, no accurate figures for UK.
- Predicted to increase due to shift in population.

Aetiology

- Genetic—Basal Cell Naevus Syndrome (Gorlin-Goltz—KCOT).
- Sun exposure (especially in childhood).
- Age, skin types (I + II), immunosuppression, arsenic exposure, high dietary fat.

Diagnosis/Investigations

Clinical

- Dermatoscope.
- Biopsy → Clinical doubt (exfoliative cytology).
- CT/MRI → Bony involvement, invade nerve/orbit/parotid gland.

High Risk

- Site: central face/eyes/nose/lips.
- Size >2 cm.
- Histology—aggressive features (PNI/PVS).
- Recurrent tumours.
- Immunosuppression (increased risk of recurrence).

Treatment Decisions

- Age.
- Comorbidity.
- Local services.
- Experience.
- Poor general health.

Management

- Surgical
  - Excision.
  - Pre-determined margins—highly effective.

Nodular

- 3 mm → 85% chance clearance.
- 4 mm → 95% chance clearance.

Morphoeic

- 3 mm → 66% chance of complete excision.
- 5 mm → 82%.
- 13 mm → 95%.

Incomplete Excision

- 4–7%.
- Up to 30–40% recurrence.
- Risk of recurrence increases if lateral + deep margins involved.
- Lateral margins—17%.
- Deep margins—33%.

Evidence → Re-treatment Especially If Involved

- Critical sites (midface).
- Deep margins → RT may have role in preventing recurrence in incomplete excision tumours.
- Aggressive histology.

Recurrent Basal Cell Carcinoma

- More difficult to cure → need wider margins or Mohs (margins 5–10 mm suggested).

Mohs micrographic surgery (MMS)

- Indications.
  - Critical site (face/eye/nose/lips).
  - >2 cm in size.
  - Aggressive histology PNI/PVI.
  - Recurrent
    - Histology → Re-excision → Either via frozen sections (over hours)/formalin (takes days).
    - Often option is to do → Slow Mohs—staged excision + delayed reconstruction.

Destructive technique

(a) Curettage/cautery

- Small <4 mm.
- Non-critical sites.
- 5 year cure—92%.

(b) Cryosurgery

- Liquid nitrogen (−50 to 60 °C) → Deep destruction (multiple freeze/thaw cycles) heals with minimal scar.
- Higher recurrence rates then surgery.

Non-surgical

- RT/PDT/topical immunotherapy with imiquimod.
- Topical immunotherapy imiquimod → Immune response modifier → 5% IMQ cream
  - Enhance APC on skin.
  - Stimulates T cells.
  - Induces cell-mediated immune response cell.
  - Treatment for 6 weeks → No RCT/5 year (data).

PDT

- Topical.
- Can get pain.

Radiotherapy

- Treatment for high risk if patient do not want surgery/unable to tolerate surgery.
- Superficial RT.
- Electron beam therapy.
- Single fractions → Higher complications.

- Not to use if <50 years/previous radiotherapy or involve important structures such as eye.
- Complications
  - 5% telangiectasia.
  - High recurrence than surgical excision (10% vs. 2%).

MDT

- High risk/close excision margins.
- Recurrence.
- Patient suitable Mohs.
- Immunosuppressed.

Follow-Up

- Risk 2nd BCC → 33–77% → Completely excised (discharged with information).
- >2 BCC → 70% chance recurrence → Surveillance with dermatologist/GP for 3 years.
- Incomplete excision → Aim 95% clearance.
- Positive margin → Recurrent 20–40% cases → Further excision.
- Multiple positive margins/deep margins → Re-excision.

13.3 Squamous Cell Carcinoma

- >10% skin cancers (2nd commonest).
- Sun exposed head and neck regions.
- Locally invasive, may metastasis.

Risk factors

- Chronic UV, genetic predisposition, male, immunosuppression.
- Xeroderma pigmentosum, albinism, exposure to arsenic (rare).
- May arise in chronic wounds, scars, burns, sinus tracts.
- Can follow Bowen's disease, actinic keratosis.
- Tanning.

Presentation

- Ulcer, nodular, keratotic horn, often rapidly growing.

Staging

- Table 13.1 TNM8 (tumour–nodes–metastasis) classification for cutaneous squamous cell carcinoma (cSCC).

  Table 13.2 TNM8 (tumour–nodes–metastasis) stage groups for cSCC.

Table 13.1 TNM cutaneous SCC

T categories	
T1	≤2 cm in greatest dimension
T2	>2 to 4 cm in greatest dimension
T3	>4 cm in greatest dimension or minor bone erosion or specified perineural invasion (≥0.1 mm diameter and/or deeper than the dermis and/or a named nerve) or deep invasion (thickness >6 mm and/or beyond the subcutaneous fat)
T4a	Tumour with gross cortical bone/marrow invasion
T4b	Tumour with skull base or axial skeleton invasion including foraminal involvement and/or vertebral foramen involvement to the epidural space
N categories for non-head and neck	
N1	Metastasis in a single node ≤3 cm in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, >3 cm but ≤6 cm or in multiple ipsilateral nodes with none >6 cm in greatest dimension
N3	Metastasis in a lymph node >6 cm in greatest dimension
N categories head and neck region	
N1	Metastasis in a single ipsilateral lymph node ≤3 cm in greatest dimension without ENE[a]
N2a	Metastasis in a single ipsilateral lymph node >3 cm but <6 cm in greatest dimension without ENE
N2b	Metastasis in multiple ipsilateral lymph nodes, where none are >6 cm in greatest dimension without ENE
N2c	Metastasis in bilateral or contralateral lymph nodes, where none are >6 cm in greatest dimension without ENE
N3a	Metastasis in a single or multiple lymph nodes >6 cm in greatest dimension without ENE
N3b	Metastasis in a single or multiple lymph nodes with ENE
M categories	
M0	No distant metastasis
M1	Distant metastasis (including contralateral nodes in non-head and neck cSCC)

[a] Extranodal extension (ENE) can be clinical or pathological

Table 13.2 Staging cutaneous SCC

Stage	T	N	M
I	T1	N0	M0
II	T2	N0	M0
III	T3	N0	M0
	T1, T2, T3	N1	M0
IVA	T1, T2, T3	N2, N3	M0
	T4	Any N	M0
IVB	Any T	Any N	M1

High Risk SCC for Recurrence or Metastasis

- Site.
- Lip, ear, non-exposed sites, chronic wounds, after RT or from Bowen's disease.
- Size: >2 cm.
- Depth: >4 mm.
- Histology → Poorly differentiated, perineural infiltration.
- Immunosuppression → Post-transplant, haematological malignancy.

Treatment → Surgical Excision

- Low risk → Well-defined tumours—4 mm margins → 95% clearance.
- Curettage and cautery/cryotherapy for low risk.

High Risk (Flowchart 13.1)

- 6 mm margins (including lip).
- High risk lesions may recur as in-transit metastasis in surrounding skin (local metastasis).
- Consider Mohs surgery in high risk/recurrence.
- RT → Treatment of choice where the patient is unwilling/unable to tolerate surgery and in recurrent disease.
- ≥4 mm for a low-risk cSCC tumour.
- ≥6 mm for a high-risk cSCC tumour.
- ≥10 mm for a very high-risk cSCC tumour.

Neck management

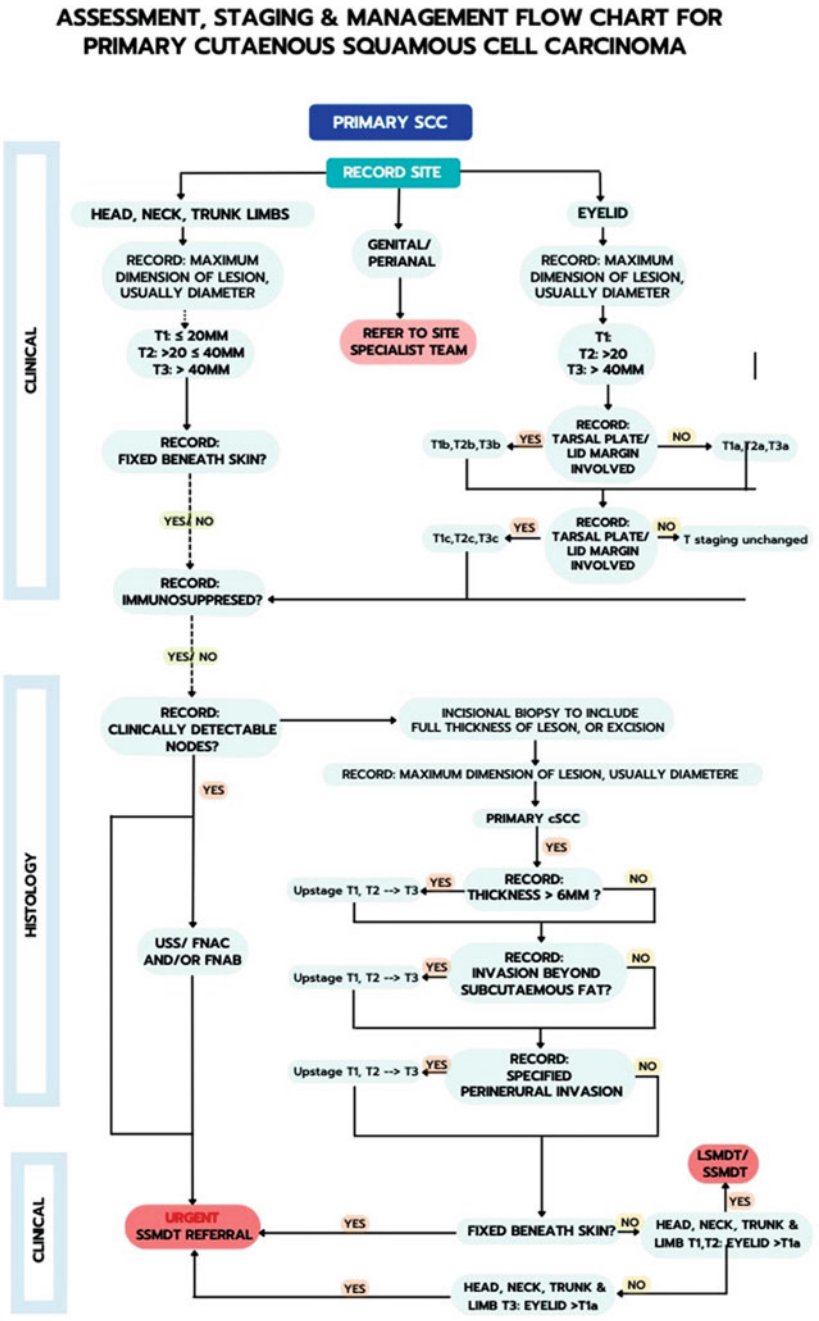
- The evidence of elective lymph node dissection in N0 neck.
- FNA enlarged nodes.
- Scalp/forehead → Primary lymphatic drainage is to the parotid.

Follow-up

- 95% of recurrence/metastasis → Occurs within 5 years in high-risk tumours.
- BAD recommended follow-up in those with high-risk tumours for 5 years → Every 3 months.

Incomplete Margins

- Discuss in MDT meeting.
- Rare recurrence → High in high-risk lesion/where deep margins involved → BCC.
- SCC.
- Re-excision if not able to have radiotherapy.
- Further surgery via Mohs.
- Closely excised → Consider adjuvant radiotherapy.



Flowchart 13.1 Management of cutaneous SCC

Metastasis

- SCC—5%: >4 mm thickness → 45%.
- 35% 5 year survival..
- Lymph nodes → Parotid/superficial lymphatics → Lack concordance with primary site.
- Anterior scalp/forehead/ears → Parotid 70% metastasis.

Lip Squamous Cell Carcinoma

Lips: 25% of Overall SCC

- 10% Lower lip—submental nodes.
- Upper lip—pre-auricular/peri-parotid nodes.
- 20% Commissure—submandibular nodes.

Management

- Surgical excision with 0.6 cm margin.

Lower Lip

- <1/2: Wedge.
- 1/2–2/3: Karapandzic/Abbe-Estlander.

Commissure Invaded Could Try

- Yes: Abbe-Estlander.
- No: Abbe Flap
  - 2/3: Bernard Burrow, Gillies Fan Flap, Webster flap.

Cheek Adequate

- Midline → Bernard Burrow advancement.
- Lateral—Nasolabial transposition.

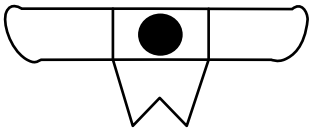
Cheek Inadequate

- Free flap.
- Upper lip
  - <1/2: Wedge.
  - 1/2–2/3: Peri-alar crescenteric/Abbe-Estlander/Reverse Karapandzic/Free flap.

Flap Designs—Reconstructions

- Wedge
  - V-plasty.
  - M-plasty (Fig. 13.1).
  - 3 layer closure (labial artery).

Fig. 13.1 Wedge excision



- Karapandzic Flap (Fig. 13.2)
  - Good functional lip → Preserve blood vessels/nerves via blunt dissection muscle, must also release intraoral mucosa → To allow flap to move medially and mobilise soft tissue.
  - Sensate with lip function.
  - Microstomia → May need secondary tension/prosthetic stretching.

Reverse Karpandzic

See Fig. 13.3.

Bernard Von Burrow Flap (Fig. 13.4)

- Midline defect/adequate cheek tissue → Raise full thickness flap.
- Brings some oral mucosa to advance and create new lower vermillion (Webster modifications).

Fig. 13.2 Karapandzic flap

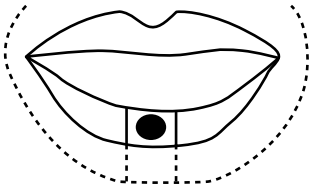


Fig. 13.3 Reverse Karapandzic

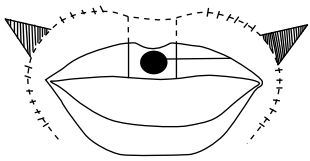
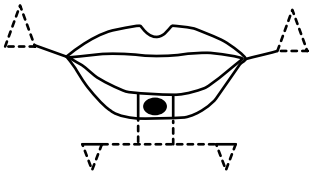


Fig. 13.4 Bernard von Burrow flap



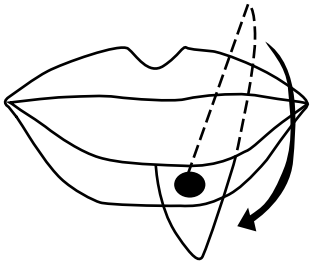


Fig. 13.5 Abbe flap

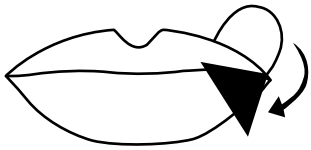


Fig. 13.6 Abbe-Estlander flap

Abbe Flap (Fig. 13.5)

- Based on labial artery, pedicle rotation flap → Divided at 10–14 days.
- Can be based on medial or lateral pedicle 180° rotation.

Abbe-Estlander Flap (Fig. 13.6)

- If involves commissure → Lip switch.
- Pedicle always medial to enable the commissure to be reconstructed, may need secondary revision.
- Flap—height 1–2 mm greater than defect.

Gillies Fan Flap (Fig. 13.7)

- Fan shaped rotational flap based on superior labial vessels → The flap disrupts oral commissure.
- Changes direction of pull of orbicularis oris → Denervates muscles/skin/mucosa → Reduce oral stoma.

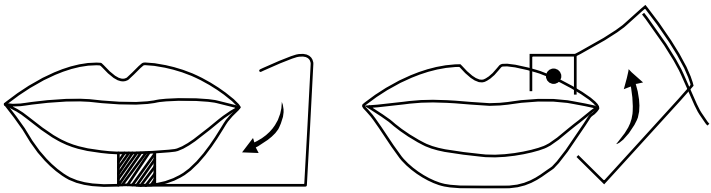


Fig. 13.7 Gilles fan flap

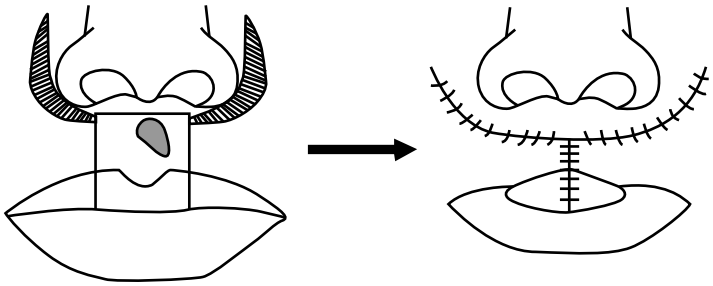


Fig. 13.8 Modified Burrow technique

Modified Burrow Technique (Fig. 13.8)

- Upper lip midline with adequate cheek tissue.

Reconstruction

- Reconstructive ladder.
- RTSL (Relaxed skin tension lines) → Related to skin lesion → Collage orientation to skin.
- FTSG

(1) **Scalp**

- Primary closure ± tissue expansion.
- Skin grafts.
- Rotational flaps.
- Purse string.
- If bone exposed → Integra (new connective tissues).

(2) **Forehead**

- Delayed healing.
- Primary closure.
- O to T.
- Skin graft.
- Advancement (H-plasty).

Bilobed Flap (Fig. 13.9)

- Double transposition flap secondary flap → Closed 1st defect → 1/2 size of primary flap and in area of skin laxity → Close primarily.

Rhomboid Flap (Fig. 13.10)

- Relaxed skin tension lines + maximum laxity.
- Design flap by first drawing the V-shape (2 flap sides) that will allow easy closure and result in scar in a RSTC.

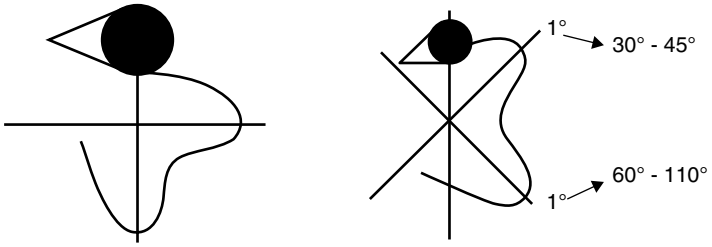


Fig. 13.9 Bilobed flap

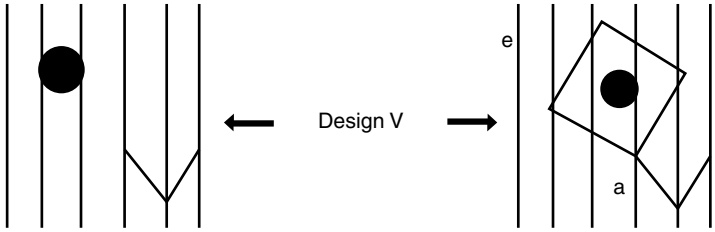


Fig. 13.10 Rhomboid flap

Dorsal Nasal Flap

- Whole dorsal nasal skin → Raised as a flap based on the angular artery.
- For top/and alar defects → 2 designs ipsilateral/contralateral.
- Secondary defect in glabella → Closed V-X fashion → usually for defects >2 cm.
- Random pattern.

Tissue Expander

- Large forehead defect → Staged reconstruction → Place expander in subgaleal pocket.
- Expansion for 2 weeks after placed → Usually increase until patient tolerates → Overlying skin blanches.
- Excise capsule formed around expander.
- Contraindications.
- Active infection.
  - Clinically persistent or recurrent cancer.
  - Poor vascularisation at the implanting.
  - History of compromised wound healing.
  - Compromised immune system.

"RULE OF 10" for Filling of the Tissue Expander

- 10% filling is performed intra-operatively.
- Post-operative filling is started at the 10th post-operative day.

- 1/10 of the total capacity is filled in each filling session.
- The entire filling process is completed over 10 weeks.
- 10% overfilling beyond the prescribed capacity is routinely performed.

13.4 Cutaneous Melanoma

- Neural crest derived cutaneous melanocytes.
- Increasing incidence.
- Improved prognosis → Earlier diagnosis → Treat at early stage.
- Rate 600/year → Legs (female); Back (male).
- 4% of all skin cancers.
- 80% skin cancer deaths.
- Common in Caucasian women 25–29 years.

Aetiology Complex

- UV in suspected individuals.
- Fitzpatrick type 1 skin has 3× increased risk.
- Shades → Intense burn/sun exposure → Increased risk.
- Increased risk in pilots/crew members.
- Xeroderma pigmentosum → Increased risk of all skin cancers.
- Giant congenital melanocytic naevus → Long-term follow-up.
- Family history → CDKN2A (genetic mutations 10–30% patients)—genetic.

Subtypes

- Superficial spreading—most frequent 70%.
- Nodular 2nd commonest 10–15%.
- Lentigo maligna melanoma—5% → Sun damaged skin head and neck.
- Pre-invasive in-situ phase → Lentigo maligna.
- Acral lentiginous—least common UK (palms/soles of feet/subungual).
- Desmoplastic neurotropic melanoma—Increased risk local recurrence → Perineural spread → Usually head and neck.
- Clinical assessment (7-point checklist)
  - Asymmetry.
  - Border irregular.
  - Colour—different colours/irregular pigments.
  - Diameter >6 mm/change in size.
  - Elevation—lesion.
    - Inflammation/itch/altered sensation/crusting.

Diagnosis

- Clinically difficult → Experience.
- Dermatoscopy → Only if trained → Dermatologist.

- Excisional biopsy
  - Clinical photographs first.
  - Aim 2–5 mm margins + subdermal fat.
- Allow assessment thickness.
- Depth prevention
  - Difficult eyelid/lip → Punch biopsy.
  - If very large >6 mm excison may be difficult—biopsy.

Imaging

- CT/MRI/Ultrasound.
- Stage I + II → Not routinely staged.
- Stage IIb + IV → CT head/chest/abdomen/pelvis.

Management

- Skin MDT.
- Staging.
- Investigations.
- Stage II → not local MDT.
- Trials: Stage IIc.
- Lymph nodes status.

Biopsy Lesion × Excision Margins

- In situ ×5 mm.
- <1 mm × 1 cm.
- 1–2 mm × 1–2 cm.
- 2.1–4 mm × 2–3 cm.
- >4 mm × 3 cm.

Anatomical Restrictions → Try to Keep Excision Margins Uniform

Mohs Micrographic Surgery

- Role in primary treatment especially face—need trials.
- Reconstruction → Immediate/delayed if unsure.

Regional

- Nodes—US/FNAC → Suspicious core/open.
- Staged CT.
- Consider dissection ± superficial parotidectomy considered if likely to pass parotid.

Occult Lymph Nodes

- Sentinel lymph node biopsy → Tc-99 m into site of excision biopsy.
- Pre-operative lymphoscintigraphy identifies approximate location.

- Intra-operative → Gamma camera probe/blue dye → Aid location.
- Clinically nodes negative → SNB can be considered for >1 mm/Stage 1b and upward.

Unknown Primary Source

(a) Dermatologist.
(b) Head and neck

- CT head/chest/abdomen/pelvis.

Metastasis

- Intra-abdominal/liver/lung/brain/bone.
- Increased lactate dehydrogenase → Poor result from treatment.
- C-kit mutation → Imatinib.
- Surgery → Improves median survival.

Non-surgical

- RT/chemo → Not proven → Stage IV/recurrent disease/unresectable disease.
- Immunotherapy → 1L–2/ipilimumab.
- Signal transduction inhibitions—Vemurafenib cancer growth blocker (if positive for BRAF V600 mutation).
- Chemotherapy.
- Distant metastasis → Palliative.

Mucosal Melanoma

- 1% head and neck cancer.
- Oral cavity.
- Nasal cavity—aggressive.

Management

- Surgery → 5 year survival very low.
- Adjuvant chemotherapy/biological therapy.

Follow-up

- In situ: No follow-up.
- Ia: 3–12 months.
- Ib-IIa: 3 months to 3–5 years.
- IIc → Trials

Clark Level—Melanoma

- I: Epidermoid.
- II: Papillary dermis

  – IIb+ → Investigations → CT chest/abdomen/pelvis/CDH.

- III: Junction of papilla/reticular.
- IV: Reticular dermis.
- V: Subcutaneous fat

Stages Versus Breslow

- IA—<1 mm mitosis <1 m if ulceration present.
- IB
  - <1 mm—ulceration mitosis 1 mm and more.
  - 1.01–3 mm—no ulceration.
- IIA
  - 1.01–3 mm—with ulceration.
  - 2.01–4 mm—no ulceration.
- IIB
  - 2.01–4 mm—with ulceration.
  - >4 mm—no ulceration.
- IIC
  - >4 mm—with ulceration.
- IIIA
  - any Breslow, no ulceration → 1–3 nodes.
- IIIB
  - any Breslow, with ulceration → 1–3 nodes.
- IIIC
  - any Breslow, with ulceration → up to 3 palpable lymph nodes.
- IV → M1.

Lentigo Maligna

- Melanoma in situ → Risk of melanoma → 5% like (depends on site).
- Excise with 1 mm margin.

PDT—Photodynamic Therapy

- "Oxygen dependent reaction between drug and light with tissues."
- Generate O_2 species → Cell killing/vascular shut down.
- Non thermal destruction → Collagen/connective tissue (undamaged).

Method

- Apply drug—systemic/topical.
- Selectively retained by tumours.
- After period of time—target illuminated.

Drugs

- Photofrin.
- Foscan
  - Only systemic drug licensed for head and neck.
  - Issues: Systemic photosensitisation → 2–3 weeks—very sensitive to light.
- Aminolevulinic acid (5-ALA).

BCC

- 5-ALA cream (occlusive dressing)—4–5 h.
- Illuminate 635 nm
  - Laser light → Blisters heal over time.
  - LA → Max depth 1–2 mm.

(a) Surface Illumination

- Good for field change.

(b) Interstitial Illumination

- Using bare light fibres.
- Need CT/US/MRI.

Post-operative

- If airway oedematous → Excessive swelling → Need tracheostomy.
- Steroids.

VAC—Vacuum Assisted Closure

- Promotes wound healing.
- Sponge material.
- Occlusive dressing.
- Vacuum 75–125 mmHg.

Works Via

- Removal of interstitial fluid.
- Decreased bacterial colonisation.
- Increased wound vascularity.

Can Use to Stent a Skin Graft → to Improve Take by Faster Revascularisation

Integra

- Dermal substitute.

Skin Graft

1. Dermatome
   - No. 15 blade = 0.025 mm/0.015 inch.
   - Intermediate thickness → Lubricate skin → 30–45° angle to skin.
   - Uniform thickness.
2. Split (STSG)
   - Epidermis + portion of dermis.
3. Full thickness (FTSG)
   - Entire skin (epidermis + dermis).
   - 3 phases for taking
     - Serum imbibition

       Plasmatic circulation.
       First 48 h.
       Allows graft to survive.
       Plasma exudate from host bed → Capillaries provide serum → Nourish graft.

     - Revascularisation

       After 48 h → Growth blood vessels into graft from host bed.

     - Organisation

       Within fibrin clot.
       Day 7 → Infiltrated fibroblasts.
       Day 9 → Secured to bed within vascular supply.
       Failure → Haematoma/infection/shearing.
       Meshing → Cutting slits → Expand.

       - Increase surface area.

Chapter 14
Oral Surgery

Abstract An overview of oral surgical procedures, complications, and surgical management of medically compromised patients.

Key Points

- Management of medically compromised patients including steroids, anticoagulants, antiplatelets and bleeding disorders.
- Third molar extractions.
- Nerve damage.
- Dentoalveolar surgery for orthodontics.
- Oroantral communication.
- Apicectomy.
- Orofacial infections.
- Medication-related osteonecrosis of the jaw (MRONJ).
- Dental implants.

14.1 Management Medically Compromised Patients

Steroids

- Steroid: Exogenous steroids, patients develop hypothalamic-adrenocortical suppression.
- Increased stress—adrenal medulla: Secrete catecholamines, bind vascular alpha/beta adrenoreceptors to direct blood flow (fight/flight).
- In the absence of steroids—net result of catecholamines surge leads to decrease in blood pressure = adrenal crisis.

Management

- LA: 7.5 mg prednisolone/day − no cover = If higher close double dose on the day of surgery only.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_14

GA

- Minor: 100 mg hydrocortisone I.V. pre-operative.
- Major: 100 mg hydrocortisone I.V. pre-operative—50 mg/TDS for 48 h.

Warfarin

- Inhibits Vitamin K dependent factors II, VII, IX, X.
- Check INR: assess control and target, <4.0 safe dentoalveolar surgery.
- Major surgery
  - Emergency: Consider prothrombin complex concentrate ± Vitamin K—haematology advice.
  - Elective major: Stop warfarin 3 days, if prosthetic heart valve − LMWH: Restart warfarin on day of surgery, continue LMWH in target range.

Drugs Affecting Warfarin

- Metronidazole: Avoid as increases INR.
- NSAIDs: Caution advise vigilance.

Aspirin

- Irreversible inhibits the thromboxane A2 stage of the COX pathway response for aggregation of platelets, does not usually require stopping, stopped for face lifts/facial aesthetic surgery to prevent platelet loss, increased bleeding at home.
- Permanent effect: Lasts for lifetime of platelet (3–7 days).

Clopidogrel

- Irreversibly blocks $P2Y_{12}$ADP receptors on platelets inhibiting aggregation has synergy with aspirin.
- Platelet half-life 5–7 days TDS, takes 7–10 days for adequate number of functional platelets to reach circulation.
- BNF: Discontinue for 7 days before major surgery, continue for dentoalveolar surgery, carboxymethylcellulose pads/suturing.

Haemophillia/Von Willebrand's Disease

- Factor VIII/IX Haemophilia = lower blood clotting factors, bleed for long time

  (a) Haemophilia

  - Mild/moderate: factor level 2–40 iu/dl: liaise with haematology, patient may be given own cover = desmopressin.
  - Severe (<1 iu/dl: recombinant factor: given pre-operatively and post-operative (tranexamic acid).

  (b) Von Willebrand's

  - Type 1: Desmopressin − pre-operative (I.V./nasal spray) = post-operative tranexamic acid/no block injections.

Table 14.1 Antiplatelet agents

Generic name	Brand name	Mechanism	Recommended drug-free period prior to elective surgery
Antiplatelet agents: mechanism of action and recommended duration of discontinuation prior to elective surgery			
Clopidogrel	Plavix	Irreversible platelet binding	5–10 days
Prasugrel	Efient		7–10 days
Tricagrelor	Brilique	Reversible platelet binding	5–7 days

Generic name	Brand name	Dosing regime	Target
Non-vitamin K oral anti-coagulation (NOACs): dosing regime and mechanism of action			
Dabigatran	Pradaxa	BD	Thrombin inhibitor
Edoxcaban	Lixiana	OD	Factor Xa inhibitor
Apixaban	Eliquis	BD	
Rivaroxaban	Xarelto	OD and BD depending on indication	

  - Type 2: Desmopressin or Humate-P (post-operative tranexamic acid/no blocks).
  - Type 2/3: Humate-P (purified produce with VWF and Factor VIII).

Platelets

- 1 unit of platelet increases by 50–80—transfused intravenously.
- Normal 140–440 (varies)
  - <50: Minor bleeding/risk of major bleeding increases.
  - 20–50: Predictable bleed with minor trauma.
  - <20: Spontaneous bleeding may occur.

Post-operative Bleeding (Table 14.1)

- Primary: Immediate at time of surgery.
- Reactionary: Occurs with 48 h of surgery due to opening of small blood vessels which contracted, disturbance to blood clot due to wearing off vasoconstriction.
- Secondary: Occurs after 48 h/often 1 week post-operatively, usually result of infection which destroys clot or damages blood vessels at base of wound.

Tranexamic Acid

- Inhibits fibrinolysis, S.E: Nausea/vomiting, diarrhoea, disturbances in colour vision.
- PO 1–1.5 g/TDS (15–25 mg/kgs), or slow I.V. −0.5 to 1 g TDS.
- Desmopressin: Boost factor VIII in mild/moderate haemophilia, and VW disease (intra-nasal/subcutaneous.

Carboxymethylcellulose

- Surgical: Haemostatic agent—oxidised cellulose polymer.

Directoral Anticoagulants (DOAC) (Table 14.1)

- Apixaban: Inhibits factor Xa. half-life 12 h.
- Rivaroxaban: Inhibits factor Xa-half-life 11–13 h.
- Many more (edoxaban, dabigatran).
- Complex surgery miss morning dose day of surgery and take evening dose.

14.2 Third Molars

3rd Molars

- Commonest missing tooth 25%.
- Eruption at age 16–25.
- May remain unerupted.
- Impaction
  - Vertical—38%.
  - Mesio-angular 49%.
  - Disto-angular 9%
  - Horizontal 4%.
- Still top 15 NHS surgical procedures.

Nice Guidance

- Based on the evidence, NICE has recommended
  a. Impacted wisdom teeth that are free from disease (healthy) should not be operated on. There are two reasons for this:
- No reliable research to suggest that this practice benefits patients.
- Exposure to risks following surgery:
  - Nerve damage.
  - Damage to other teeth.
  - Post-operative infection.
  - Haematoma, swelling.
  - Reduced mouth opening.
- Patients who have asymptomatic impacted wisdom teeth can visit the dentist for regular review.
- Only diseased, symptomatic wisdom teeth need to be removed:
  - Abscesses.
  - Cysts or tumours.
  - Disease of the tissues around the tooth.
  - If the tooth is in the way of other surgery.

Possible Consequences of Retention

- Removal when older—increased surgical complications.
- Decay—distal of lower 7 s and periodontal disease.
- No evidence of lower incisor crowding.

Classification

- Nature of overlying tissues: Soft tissue, hard tissues (bony impaction), partial bony.
- Winter's classification
  - Based on inclination of the impacted wisdom tooth to the long axis of 2nd molars.
  - Vertical/mesioangular/distoangular/buccal oblique/lingual oblique.
- Indices for detecting difficult molars.

Winter's Lines (WAR) (Fig. 14.1 and Table 14.2)

- White line: Occlusal surface.
- Amber line: Height of bone.
- Red line: Imaginary line—perpendicular from amber to application point.
- Red line indicates the amount of the bone removal.
- Red line increase by 1 mm = 3 × more difficult − Red line <5 mm − possible LA.

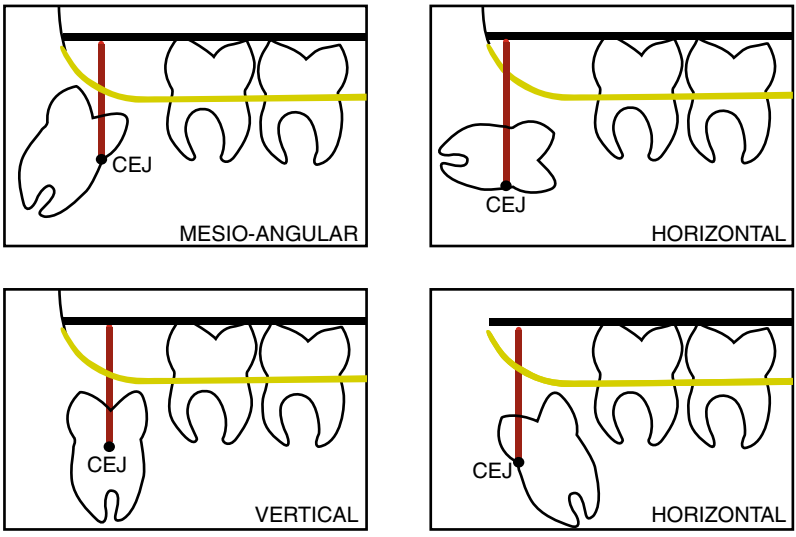


Fig. 14.1 Wisdom teeth impaction patterns

Table 14.2 Winters lines

White line	– Corresponding to occlusal plane of molar teeth – Indicates the **difference in occlusal level** of second and third molar
Amber line	– Represents the bone level – Denotes the **alveolar bone covering the impacted tooth** and the portion of tooth not covered by the bone
Red line	– The red line is an imaginary line drawn perpendicularly from the amber line to an imaginary point of application of elevator – Represents **depth of the tooth in bone** and the difficulty encountered in removing the tooth. Indicated the amount of bone that has to be removed before elevation – If the length of red line is **more than 5 mm then extraction is difficult** – **For every additional 1 mm difficulty increases three times (3X)**

14.3 Winter's Lines (War Lines)

Many Others (Not Used)—Factors that Make Surgery More Difficult

- Distoangular.
- Long thin root (especially older patients).
- Divergent curved roots.
- Dense bone (especially elderly).
- Close to IAN/complete bony impaction.

Operculectomy

- Sometimes considered if the upper opposing teeth is traumatising the operculum.

Complications of Tooth Extraction

- Haemorrhage, swelling, infection, dry socket (up to 20% especially smokers), osteomyelitis, fractured roots, wound breakdown, damage to adjacent teeth, OAF, fracture of mandibles.
- Displacement upper 8 s into maxillary sinus/infra-temporal fossa.
- Displacement of lower 8 s into sublingual/submandibular spaces.
- Nerve damage
  - Lingual nerve: 1–3% temporary, 0.3% permanent/increased incidence with lingual retraction (up to 9%).
  - IAN: temporary up to less than 8%/less than 3.6% permanent/1–2% temporary with 70% recovery.

Radiographic Nerve Assessment

- Radiolucency across the roots of the 3rd molars.
- Deviation of canal.
- Interruption of white lines.
- Deflection of 3rd molar roots by canal—clinically important.

- Narrowing of 3rd molar root
  - Not reliable.
  - CBCT is recommended.

Renton (2005) BJOMS—RCT Coronectomy

- Wind instrument players/actors/singers—risk to nerve can be life changing.
- 196 3rd molars.
- Coronectomy = transection tooth 3–4 mm between the enamel of the crown intro dentine/3–4 mm below crest.
- Follow-up in 25 months—decreased risk of IAN damage, no change in morbidity.
- 19% had sensory disturbance, only 2 patients permanent.
- 0% damage versus 19% high risk, but 38% coronectomies failed including root dislodge (thin roots).
- Follow-up only 2 years—fate of retained roots?

Dry Socket

- Chlorhexidine (CHX) mouthwash pre-operatively + 7 days post-operative would have significant effect.
- Antibiotics do not prevent dry socket occurrence.
- Application of Alvogyl (Alveogyl) recommended
  - Contains: Butamben (butylparaminobenzoate), iodoform (triiodomethane) and eugenol.

14.4 Oral Surgery—Nerve Damage

Nerve Damage

- Hypoaesthesia: Decrease in sensation.
- Anaesthesia: No sensation.
- Paraesthesia: Abnormal sensation.
- Dysaesthesia: Unpleasant abnormal sensation
  - Permanent: 0.2–1.0%.
  - Temporary: less than 8%.
- Simple sensitivity testing: mapping with photographic area
  - Light touch with wisp of cotton wool.
  - Von Frey Filaments.
- Pin pinch sensation: probe/needles.
- Two-point discrimination.
- Taste stimulation: Cotton pledgets in saline/vinegar/sugar.

Risk Factors

Inferior Alveolar Nerve

- Age of the patient.
- Surgeon's experience.
- Radiographic findings.
- Lingual split.
- Lingual retractor.

Lingual Nerve

- Surgeon's experience.
- Lingual plate perforation.
- Lingual split.
- Lingual retractor.

Anatomy

- 3 layers: Epineurium/perineurium/endoneurium.
- Fibres: A-alpha (fine touch)/A-beta (proprioception)/A-delta (superficial pain/temperature)/C (deep pain/temperature).

Seddon Classification (Table 14.3)

Neuropraxia

- Physiological disruption.
- Anatomy intact.

Axonotmesis

- Axons disrupted.
- Epineurium/perineurium preserved.

Table 14.3 Nerve injuries

Birch & Bonney	Non-degenerative			Degenerative			
Lundborg 1988	Physiological conduction block		Myelin damage	Axonal damage	Axon + endoneurium damage	Axon + endoneurium + perineurium damage	Axon + endoneurium + perineurium + epineurium damage
	Type A	Type B					
Sunderland 1951	I			II	III	IV	V
Seddon 1942	Neurapraxia (transient block)			Axonotmesis (lesion in continuity)	Neurotmesis (division of a nerve)		

Neurotmesis

- Severed nerve.

14.5 Nerve Injuries Occur on Continuum of Severity

Nerve Response to Injury

- Proximal axons suffer retrograde degeneration—may extend as far as cell body (trigeminal ganglion), next node of Ranvier.
- The distal axonal segments undergo Wallerien degeneration with loss of the myelin sheath.
- Regeneration (2 mm/day).

Surgical Risk Factors

- Divisions of lingual nerve at time of surgery
  - Immediate repair.
- Division of IAN nerve at time of surgery
  - Immediate repair if severed ends do not remain apposed.
- Post-operative review (IAN)
  - Paraesthesia.
  - Anaesthesia—radiograph check bony fragments disrupting bony canal.
  - 3 month review = surgery only if dysaesthesia, discontinue ID canal visible.
- Post-operative review (lingual)
  - Paraesthesia—recovery likely.
  - Anaesthesia—may need surgery.
  - 3 month review no evidence of recovery = refer/exploration.

Contraindications for Repair

- Central neuropathic pain.
- Improving sensory function.
- Hypoaesthesia—acceptable to patients
  - 1 year Since Injury.

Surgical Procedures

- Expose/haemostats, removal of scar tissues, nerve prepared with anastomosis without tension.
- Nerve grafts if required.

IAN

- 'Decompression if obvious disruption of canal.'
- Intraoral approach, buccal plate removed.
- Any lateral neuroma excised ± neurolysis, approximation with 8-0 nylon epineural sutures.
- Results varied/sometimes disappointing.

Lingual

- Intraoral: Excise scar tissues (neurolysis/if neuroma (expansion/excise damaged segment repair with epineural sutures).
- Use sulcular lingual extension as per 3rd molar access.
- External neurolysis—micro-dissection to identify injured section.
- Internal neurolysis—longitudinal incisions through epineurium to release scar tissue.
- Gap closure of less than 10 mm = Neurorrhaphy ± nerve graft—GAN, sural (S1, S2).

14.6 Dentoalveolar Surgery of Orthodontics

Upper Labial Frenectomy

- No adequate RCT comparing intervention with conservative management.
- Upper midline diastema common in mixed dentition—often closes with eruption of canines.
- Excision of frenum must include the part which passes between upper incisors.

Impacted Canines

- 2nd most common impacted 0.8–2.8%, of which 85% palatal, more common in females.
- Aetiology: longest eruption patch, crowding, pathology, genetic/trauma.
- Clinical: usually palpable in buccal sulcus around age 10–11 = OPG at age 11 if no sign of eruption with anterior occlusal maxilla view.

Management

- Keep deciduous—leave permanent, or remove (external root resorption/cyst formation).
- Remove both with space closure/implant.
- Expose and bond canine.
- Transplantation—poor success as patient not willing to wear orthodontic appliances, may need RCT within 10 days (Hussain 2010—RCS guidelines).
- Cochrane review 2008: No evidence to support any surgical techniques.

Interceptive Treatment

- Extraction of deciduous canines at age 10–13 = alignment in 75% of ectopic canines (give 12 months to align).

Supernumerary 0.5–3.9%

- Most common between upper central incisors (mesiodens) or premolars (paramolars—80–90% maxilla).
- Can delay or prevent eruption = cause resorption/cyst formation.
- Parallax
  - 1× OPG.
  - Anterior occlusal maxilla view.
  - More distant object appears to travel in same direction.

Oroantral Communication

- Maxillary sinus to oral cavity (5% incidence posterior maxillary molars).

Aetiology:

- Iatrogenic—extractions/preprosthetic surgery/tumour.
- Secondary pathology—tumour/MRONJ/ORN.
- Regurgitation of liquids from mouth into nose.
- Air escapes/nasal resonance.
- Sinusitis: Discharge from nose, foul taste.

Diagnosis

- Inspection, Valsalva (whistling deflection of cotton wool).
- Radiographs.

Management

- Closed with 24–48 h.

Medical: Antibiotics, Decongestants, Saline Irrigation

- Epinephrine nasal drops.
- Steam inhalation—menthol with eucalyptus.
- Surgery: Fistulectomy with repair.

Surgical Options

- Autogenous—soft tissues
  - Buccal advancement flap, buccal fat pad, palatal rotational advancement flap.
  - Others: Split thickness palatal, palatal pedicle island flap.
  - Tongue flap.
- Allogeneic: Fibrin glue/Dura.
- Xenograft: Collagen, foam/bioguide/bio-oss.
- Synthetic: Cover plate.

Apicectomy

Indications

- Failure of RCT.
- Post-crown on tooth with apical pathology—good coronal seal where removal will risk root fracture.
- Calcified/curved roots/cast posts.
- Fractured apical third of root/perforation.
- Biopsy required.

Evans/Bishop 2012—RCS Guidelines

- 3 mm—deep.
- Whole pulp.
- Using ultrasonic tip.
- Post-operative radiography before wound closure.

Procedure

- 90° cut—3 mm apex removed.
- MTA: Mineral trioxide aggregate: no root end preparation required—stimulates hard tissues deposition.
- Modified zinc oxide eugenol.
- IRM—Poly-reinforced zinc oxide eugenol in powder form which is formed by zinc oxide.
- >80% success.
- Osteoinductive material.

Orofacial Infections

- Odontogenic infections by endogenous bacteria—mostly gram −ve rods (bacteroides/fusobacterial).
- Some gram +ve cocci (streptococcus, Peptostreptococcus—most caused by multiple types.
- Fusobacterium associated with severe infections.
- Staphylococci
  - Epidermis/saprophyticus/aureus.
  - Staphylococcus aureus coagulate ±ve—coagulates bacteria with fibrin.

Aerobes

- Gram +ve cocci
  (a) Streptococci
    - Alpha-hemolytic—Streptococci mutans, milleri.
    - Beta hemolytic—Streptococcus pyogenes.

(b) Staphylococci

- Gram −ve cocci: Neisseria.
- Gram +ve rods: Corynebacterium, Diptheroids.
- Gram −ve rods: Haemophilus influenzae, Eikenella, Pseudomonas.

Anaerobes

- Gram +ve cocci: Streptococcus/Peptostreptococcus.
- Gram −ve cocci: Veillonella.
- Gram +ve rods: Actinomyces, Clostridia.
- Gram −ve rods: Bacteroides/Fusobacterium.

Surgical Anatomy

- Blood supply rich.
- Venous Drainage—jugular venous system.
- Connection intracranial/extracranial venous system.
- Face–brain–cavernous sinus thrombosis/cerebral abscess.

Primary Fascial Spaces (Fig. 14.2)

- Adjacent to odontogenic infection.
- Buccal/canine/submandibular/sublingual/submental/vestibular.

Secondary Fascial Spaces

- Invading spread from primary spaces.
- Mesenteric/infratemporal spaces/masticator/pterygomandibular/superficial & deep temporal/retropharyngeal/prevertebral.

14.7 Fascial Spaces

- Fascial–plasma–SMAS.
- Deep cervical fascia
  - Superficial.
  - Middle (visceral/muscular).
  - Posterior.
- Superficial investing layer
  - Clavicle to FOM, hyoid, FOM—submandibular gland, splits to encase SCM.
- Middle

(a) Visceral

- Hyoid/thyroid/oesophagus/larynx/pharynx/also forms medial carotid fascia.
- Fuses with pericardium.

(b) Muscular

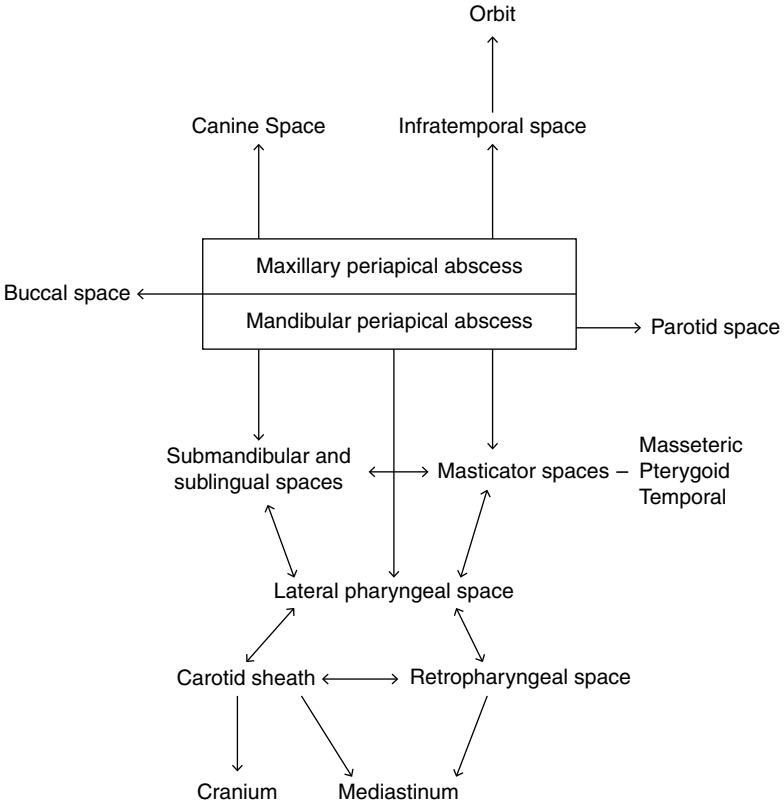


Fig. 14.2 Fascial spaces

  - Strap muscles (hyoid/suprahyoid/sternohyoid).

- Posterior (deep layer of investing fascia)

  (a) Anterior

    - Alar fossa (deep part of carotid).

  (b) Posterior

    - Prevertebral fascia.

  Main principle of treatment: Surgical (dependent) drainage of pus with antibiotic therapy.

  Important role of anaerobic bacteria: Clindamycin substitute for penicillin or add metronidazole in severe infection.

Ludwig's Angina

- Severe form cervicofascial infection (usually from molars/wisdom teeth).
- 90% dental/fracture/submandibular sialadenitis.
- Bilateral board like induration submandibular/sublingual/submental space.
- Cause airway obstruction.
- Management: Bilateral exploration/surgical drainage/antibiotics.
- Mortality: 5%.

Necrotising Fasciitis

- Severe bacterial soft tissue infection, characterised by progressive necrosis of fascia in skin and subcutaneous tissue with high morbidity and mortality.
- Poly-microbial—both aerobic/anaerobic organisms.
- Exception is Clostridium perfringens (gas gangrene) single organism.

Clinical Features

- Necrosis of the fascia with subcutaneous tissue.
- Skin: Pale/mottled/dusty purple color/crepitus.
- Systemic: Fever/tachycardia/decrease in blood pressure/gas on soft tissue radiograph/CT.

Investigations

- Haematology/blood cultures/OPG/CT.
- Most accurate diagnostic test: Fresh frozen section biopsy.

Management

- Early recognition/prompt resuscitation.
- Early aggressive surgical intervention—medical management ICU.

Surgical

- Wide surgical debridement, removal of all non-vital tissues, back to bleeding tissues.
- 2nd local surgery in 24 h—repeated debridement may be necessary.
- Later reconstruction.
- 10–40% mortality.
- HBO—no RCT.

Osteomyelitis

- Inflammation of the bone marrow which can extend through the cortex and into periosteal tissues.
- Classification (Hudson 1993)
  - Acute <1 month: contiguous focus, progressive, haematogenous.
  - Chronic >1 month: recurrent multifocal/Garres OM, suppurative/non-suppurative, chronic sclerosis.

Aetiology

- Often polymicrobial—Streptococci, anaerobic bacteria (bacteroides/Peptostreptococci), usual pathogens, immunosuppressed.
- Primary form contiguous spread from odontogenic infection/trauma.
- Increased intramedullary pressure (accumulation of pus) decreased blood supply.
- Can cause cortical destruction and exert pus into soft tissue (intra/extraoral fistulas).

Risk Factors

- Decreased host defense with increased age.
- Systemic: DM/AIDS/malnutrition/medication: steroids, chemotherapy, bisphosphonates.
- Local: XRT, osteoporosis.

Clinical

- Mandibular > maxilla (more dense/less vascularised).
- Signs of inflammation—swelling/erythema/fever/trismus/pain
  - Pain: deep/boring pain, out of proportion with clinical pictures.
  - Paraesthesia of mental/IAN.
  - Fistulas with chronic OM.

Investigations (Exclude Malignancy)

- OPG: chronic OM—'moth eaten' bone or sequestration.
- MRI: loss marrow signal, CT.
- Tc-99 + gallium: specifically binds white cells sharing site with increased bone turnover.
- MC + S/biopsy/bloods.

Management

- Removal of focus of infection with debridement of necrotic tissue to improve blood supply.
- Sequestrectomy, decortication—explore medullary space to overlying periosteal surface.
- If mandibular paraesthesia/chronic—consider resection/reconstruction.

Medical

- Antibiotics 6 weeks.
- Hyperbaric oxygen controversial.

Complications

- Chronic refractory/diffuse sclerosis—reduced vascularity.
- Can be due to Actinomyces/Eikenella.
- Needs long-term I.V. antibiotics with surgical debridement ± Hyberbaric oxygen.
- Can cause severe nerve injury—decompress nerves in bony canal.

14.8 Medication-Related Osteonecrosis of The Jaw (MRONJ)

MRONJ

- Exposed, necrotic bone in jaws for >8 weeks in patient with current/previous medical treatment (including bisphosphonates) with no history of XRT.
- 1% of cancer patients and 0.1% in patients with metabolic disease.

Bone Modifying Agents (BMAS)

- Used for breast cancer, metabolic bone disease, myeloma, hypercalcaemia of malignancy.

Types of BMAS

- I.V.—zoledronate, pamidronate.
- Oral bisphosphonates—clodronate/ibandronate.
- Anti-RANKL antibody—denosumab.

Medciations Associated with MRONJ

- Bisphosphonates.
- Tyrosine kinase inhibitors.
- Immunosuppressants.
- Monoclonal antibodies.
- SERMs—selective estrogen-receptor modulators.
- Many others.

Incidence

- I.V. Bisphosphonates: 1–10% (1/100 to 1/10).
- Oral bisphosphonates: 0.01–0.001% (1 in 10,000—1 in 100,000).

Pre-treatment

Dental Review

- Extractions of teeth with poor prognosis.
- Dental treatment.
- Treatment of periodontal disease.
- Improve/replace dentures.
- Oral hygiene review.

Wait miniumum of 4 weeks or until all healed:

Most common cause of MRONJ is dental extractions, risk increases with cumulative doses particularly bisphosphonates (have long half-life).

Table 14.4 MRONJ staging

0	No evidence of exposed/necrotic bone Clinical or radiographic features suggestive of MRONJ	Systemic management including the use of pain medication and antibiotics
1	Asymptomatic exposed and necrotic dento-alveolar bone with no associated infection	Antibacterial mouth rinse Clinical follow-up on a quarterly basis Patient education and review of indications for continued BMA or AA therapy
2	Symptomatic exposed and necrotic dento-alveolar bone with associated infection	Symptomatic treatment with oral antibiotics Oral antibacterial mouth rinse Pain control Debridement to relieve soft tissue irritation and infection control
3	Exposed and necrotic bone leading to invasion of vital structures, fractured jaw or extraoral fistula	Antibacterial mouth rinse Antibiotic therapy and pain control Surgical debridement/resection for longer term palliation or infection and pain

Other Risk Factors

- Mandible > Maxilla 2:1.
- DM, poor oral hygiene.

Clinical

- ONJ: presents—non-healing socket/exposed and/or necrotic bone.
- Rarely pathological fracture/fistula.
- Pain/tooth mobility/mucosal erythema.

Staging AAOMS 2014 (Table 14.4)

- Insufficient evidence to discontinue medications.
- Implants:
  - No CI for non-cancer patients.
  - ADA 2012 Grant et al.—No MRONJ 115 cases.

14.9 Implants

Osseointegration

- Direct structural and functional connection between living ordered bone and surface of load bearing implant.

Materials

- Titanium materials of choice.

- Forms oxide surface on exposure to air.
- Oxide layer > inert/practically insoluble/resistant to body fluids.

Assessment of Integration

Clinical

- Absence of signs of inflammation (e.g. pain/pus/mobility).
- Tests: Torque test (20Ncm, Periotest, resonance frequency).

Types of Implants

- Many designs tapered/cylindrical.
- 6 mm–20 mm length/2.25–6 mm width.
- Endosteal implant with roughened surface for faster integration
  - Grit.
  - Acid Etch.
  - Anodisation—increased oxide layer.
  - Combination.
  - Supraperiosteal/submucosal/transossesous.

Clinical Approach

- History: Functional versus aesthetics.
- Past medical history—cause tooth loss/risk factors
  - Osteoporos.
  - Uncontrolled diabetes.
  - Smoking—increase failure.
  - Uncontrolled periodontal disease.
  - Bisphosphonates—I.V. absolute contraindication.
  - Psychiatric disorders.
  - Radiotherapy—increased risk of failure/consider hyperbaric oxygen therapy.
  - Erosive lichen planus/vesiculbullous disease- Poor wound healing.

Examination

- Face: Facial aesthetics—lip line/smile line.
- Soft tissues: Biotype, quantity, quality, fraenum attachment.
- Hard tissues: Bone/teeth.
- Occlusion.
- Investigations: Periapical radiographs/OPG, CBCT, study models.

Assessment of Ridge Morphology and Bone Quality—Lekholm and Zarb

Ridge Morphology

- Most of alveolar ridge present.
- Moderate residual ridge resorption.
- Only basal bone remains.

- Some basal bone resorption has taken place.
- Extreme resorption of the basal bone.

Quality

- Homogenous compact bone.
- Thick cortex surrounds a cord of dense trabecular bone.
- Thin layer of cortical bone surrounds a cord of trabecular bone of low density.

Cawood and Howell

- Dentate.
- Immediate extraction.
- Ridge rounded with moderate width with height.
- Knife edge ridge with moderate height but no width.
- Basal bone only.
- Resorption of basal bone.

General Rules of Placement

- 1 mm of buccal and lingual side bone on each implant.
- 2 mm from adjacent teeth and vital structure (2-4 mm from inferior alveolar nerve).
- 3 mm space between implants.

Consent

- Leaflets/further maintenance requirements—oral hygiene instructions.

Surgical Procedure

- Local anaesthesia/sedation/general anaesthesia: Minimal soft tissue trauma.
- Drilling but avoid temperature rise above 47 °C (slow speed).
- Use surgical guide (stent)
  - Position/spacing/angulation.
  - CBCT—implant planning programmes: Simplant/NobelGuide.

Flaps

- Flap/flapless.
- Preparation of gingiva—free grafts/pedicle grafts
  - Keratinised graft from palate.
  - Tissue expansion.
  - GTR—guided tissue regeneration.

Stages: No Evidence of Improved Outcomes—Single Versus 2 Stages

- Single stage: Convenient/decreased treatment time—use healing cap.
- 2 stage: Buried then exposed 4 months later—consider if

- Temporary prothesis.
- Bone augmentation carried out.
- Poor initial stability of implant.

Healing Times

- Original: 4–6 months mandible/6 months maxilla.
- Not with microtopograhic implants—earlier osteointegration (3 months mandible/4 months maxilla).
- Immediate implants—into tooth socket
  - Healing bone, no periodontal infection, not suitable in posterior maxilla.
  - Indications: avulsed teeth.
  - Contraindications: multiple wall defects/compromised aesthetics.

Classification of Extraction Sockets

- Based on number of walls that make up extraction socket
  - 5 wall: immediate implants.
  - 4 wall: immediate implant (possible), may need grafting.
  - 3 wall: requires grafting.
  - 2 wall: requires significant grafting.

Immediate Loading

- Temporary crown/prosthesis (top of implant).
- Central occlusal loading.
- Evidence shows success in anterior mandible.

Clinical Success of Implant

- Clinically immobile.
- Vertical bone supports <0.2 mm annually after first year of placement.
- Absence of pain/infections/neuropathy/paraesthesia or violation of the mandibular canal.
- No peri-implant radioluncency.

Interim Appliances

- Bridge/crown: can improve soft tissues.

Fixed Bridge

- Maxilla: 6 implants.
- Mandible: 4 implants.

Over-Denture: Retained by Bar/Clip

- Maxilla: 4 implants.
- Mandible: 2 implants.

Screw/Cemented

- Screw: may have visible screw.
- Cement allows for angulation changes/compensate for poor implant alignment—magnets.

Implants

- Do not move in function.
- Teeth move 30-120 microns in function.

Complications

- 2–4 mm from nerves.
- Primary stability, movement failure.
- Cement failure.
- Minimum bone loss: 0–2 mm/year (acceptable range)—cause:
  - Peri-implantitis.
  - Pocketing.
  - Recession.

Care

- Occlusal overload.
- Localised infection: implant failure.
- Need good scaling/oral hygiene instruction.

Implant Salvage

- Bone loss around implant: surgical salvage—implant exposed, surface smoothed.
- Surface disinfected with topical chlorhexidine or citric acid, then GTR or soft tissue suture around implant.
- If extensive: implant removal, using trephines
  - If long implant: cut to level of bone.

Peri-Implant Mucositis

- Gingival inflammation
  - Oral hygiene instruction.
  - Mouthwash.
  - Clinical history.
  - Social history.

Peri-Implantitis

- Inflammatory process affecting the soft and hard tissues surrounding an implant.
- Complex aetiology

  - Patient factors: Systemic factors—diabetes/osteoporosis/periodontal.
  - Social factors: Smoking/poor oral hygiene.
  - Mechanical: Extreme loading/parafunctional habit.
- Mechanical debridement
  - CHX/local antibiotic placement.
- Antibiotics.

Management

- Remove abutment.
- Healing screw.
- Etch surface: Place bone graft around defect and membrane—collagen membrane/Bioguide = GBR guided bone regeneration.

Placement of Implants in Fibula

Primary

- Advantages: balance height, number of operations.
- Disadvantages: increase radiotherapy dose around implants.

Secondary

- Advantages: planning/soft tissue healing.
- Disadvantage: radiation.

Craniofacial Implants

- 50–70% success.

Magnets

- Impressions of face.
- Photographs.
- Usually small implants—special craniofacial.

Case for Primary Placement

- If limited bone available.
- Influence placement of bone flap.
- Easier surgical access.
- Undergo radiotherapy without osseointegration problems.
- Earlier rehabilitation.

Case for Secondary Placement

- Await histology bony margins.
- End of long case.
- Await stent (long case).

- Effect of implant on radiotherapy. Implant has shielding effect—1 mm around implant.
- Long-term survival of patient.

Zygomatic Implant

- Allows for establishing posterior maxillary support by using limited crestal alveolar bone in the pre-molar region and the zygomatic bone for establishing stability of the implant.
- 96% at 5 years Branemark 2004.
- Highly predictive implant: Allows establishing post-maxillary support for reconstruction with a fixed prosthesis without need for bone grafting.

Indications

- Atrophic maxilla/maxillectomy.
- Contraindications: Limited interarch distance, limited access/mouth opening, sufficient bone for routine implants, sinus pathology.

Clinical Assessment

- OPG, CBCT (evaluate/height zygomatic body).

Anatomy

- Engagement of 4 cortices
  - Crestal: Lingual cortex maxillary alveolus, cortical floor of sinus.
  - Apical: Inferior and lateral portions of the zygomatic bone.

Operate

- Crestal incision, vertical window made on lateral wall of maxilla.
- Can immediately load, minimum 40 Ncm of insertion torque.

Advantages

- Minimum of 2 premaxillary implants (preferably 4).

Disadvantages

- Technique sensitive, risk of antral injury, post-sinusitis.

Extraoral Implants:

Why Not Use Autogenous

- Vascular compliance.
- Morbidity of patient.
- Difficult to reconstruct—auricular/orbit/ear.

Retention

- Mechanical.

- Adhesives—medical grade.
- Implants.

Issues: Limited Bone

- Temporal bone.
- Supraorbital rim: 2.5–5.0 mm only

1. Impression/CT/Laser scanning.
2. Patient must be able to look after/frequent visits/sufficient dexterity to place implants, patient should be able to clean area, also needs periodic replacements as loses colour/fades.
3. Place implants after radiation—do not cause shielding (wait for 12 months).
4. Template of prosthetic to decide where to place implants—surgical stent

   – Healing—cover screw: allow osseointegration (6–12 month).
   – Especially for orbits/midface as increased risk of failure.
   – Magnets/clip bars systems.

Ear

- Implants in mastoid—use CT scan to avoid facial nerve.
- 3–4 mm length—temporal/mastoid bone.
- 2/3 implants: place 1 cm behind EAM in semilunar fashion.
- Use CT/impression/mirror image/models.

Orbital

- Evisceration
  - Removal of contends of globe—leave sclera/extra-ocular muscles.
- Enucleation
  - Removal of eye from orbit—leave all other orbital structure.
- Exenteration
  - Radical—remove eye/adnexia and part of bony orbit.
- Supraorbital/infraorbital—bone thin: CT evaluation—usually orbital rim
  - Ideally preserve eyebrow.
- Usually—3 implants—using surgical stents
  - Leave to heal for 3–5 months.
  - Past success rates some as low as 35%.
  - Higher success rate in lateral orbital rim versus supraorbital rim (poor blood supply).
  - Difficult to keep clean with patient with one eye, also orbital rim has remodeling potential.
  - Need multiple impressions to get all abutments.
  - Bar + clip/magnets (easier to clean)/combination.

Nasal

- CT—check bone levels
  - Usually 2 implants.
  - Floor of nose/piriform aperture for nasal prosthesis.
  - Can place up to 10 mm without damaging teeth.
- Magnets
  - Easier to handle/clean.
  - Leave implants free standing.
- Hygiene
  - Remove debris with cotton wool.
  - Infection—treatment.
- Peri-implantitis
  - Major cause of decreased long-term survival.
  - Difficult to keep adequate tissues.
  - Follow-up required.
  - May need: split thickness graft around implant.

Chapter 15
TMJ Surgery

Abstract An overview of disorders of the TMJ including assessment, non-surgical and surgical management.

Key Points

- TMJ anatomy.
- TMJ disorders: Articular and muscle disorders.
- Wilkes classification.
- Degenerative disease and osteoarthritis of the TMJ.
- Non-surgical management of TMJ disorders.
- Surgical management of TMJ disorders.
- TMJ reconstruction.
- TMJ ankylosis.
- TMJ dislocation.

15.1 TMJ Anatomy

- Classified as a "ginglymoarthrodial" joint.
- Between condyle and temporal bone with intervening articular disc.

Anatomy

- Condyle is olive shaped and sits at 15° to the frontal plane.
- Fibrocartilaginous disc with thick posterior band.
- Small amount of synovial fluid.

Ligaments

- Lateral temporomandibular ligament—thickened lateral capsule.
- Stylomandibular ligament.
- Sphenomandibular ligament—medially.

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Nerves

- Auriculotemporal nerve.
- Branches of V3 via auriculotemporal, masseteric, and posterior deep temporal nerves.
- Articular cartilage/central disc with no nerves.
- Capsule/disc periphery/bilaminar zone → Contain pain and proprioception fibres.

Blood Supply

- Deep to joint → maxillary artery, pterygoid plexus.
- Superficial temporal artery, inferior maxillary artery, masseteric artery anteriorly.
- Maxillary artery.
- Veins—Facial vein/pterygoid plexus.

Movement

- Both translation (AP and MC) and hinge movement.
- Free and load-bearing.

Function → Muscles of Mastication—V3

- Elevators.
- Temporalis.
- Masseter.
- Medial pterygoid.

Depressors

- Infra/suprahyoid.
- Digastric/mylohyoid/geniohyoid.
- Lateral pterygoid.

Protrusion

- Lateral pterygoid.

Lateral Excursion

- Contralateral pterygoids.
- Ipsilateral temporalis.

Range of Movement

- >40 mm opening.
- >7 mm lateral excursion.
- 6 mm protrusion.

15.2 TMJ Disorders

Articular Disorders

- Congenital (1st and 2nd branchial arch, hemifacial microsomia, Treacher Collins Syndrome).
- Acquired.
- Developmental (condylar hyperplasia).
- Internal derangement.
- Degenerative.
- Non-inflammatory.
- Inflammatory → Rheumatoid arthritis/gout/psoriasis.
- Trauma, infection, neoplasia, hyper/hypomobility.

Muscle Disorders

- Myositis, myospasm.
- Neoplasia.

TMJD

Assessment

(1) Joint line tenderness → Pain over joint → Relieved by local anaesthesia over joint.
(2) Masticatory myofascial pain
    - Trigger spots in muscle/muscle spasm.
    - Botulinum toxin into areas muscle spasm → 1/3 get long-term benefit.
    - Pain team → Tricyclic antidepressant.
    - ** Both have restriction in mouth opening.
(3) Myofascial pain (80%).

Aetiology

- More common in females.
- Depression.
- Anxiety.
- Linked with IBS/Fibromyalgia.
- Worse in the morning → parafunctional habit i.e. bruxism/clenching.
- Neck ache/headaches.

Treatment

- Patient education → Reassurance/leaflet → RCT effective intervention.
- Medications.
- Topical NSAIDs.
- Splint → RCT effective.
- TCA → Amitriptyline/nortriptyline/gabapentin.

Table 15.1 Wilkes staging TMJ

I	Painless clicking No locking No restricted motion	Slight anterior disc displacement that reduces on opening Normal osseous contours	Normal disc form Slight anterior disc displacement
II	Occasional painful clicking Intermittent locking Headaches	Slight anterior disc displacement that reduces on opening Early disc deformity Normal osseous contours	Thickened disc Anterior disc displacement
III	Frequent pain Joint tenderness Headaches Locking Restricted motion	Anterior disc displacement that does not reduce on opening Moderate disc deformity Normal osseous contours	Disc deformed and displaced Variable adhesions No bone changes
IV	Chronic pain Headaches Restricted motion with crepitus	Anterior disc displacement that does not recapture on opening Marked disc deformity Degenerative osseous changes	Disc perforations, displacement, and adhesions Degenerative changes in condyle and/or fossa
V	Variable pain Joint crepitus	Anterior disc displacement that does not recapture on opening Marked disc deformity Degenerative osseous changes	Disc perforation, displacement, and adhesions Degenerative changes in condyle and/or fossa

- Cognitive Behavioural Therapy T → RCT effective.
- 30 years → 85% get better.

Internal Derangement (10%)

- Joint tenderness.
- Reduction in mouth opening.
- Clicking indicates → Head of the condyle impinges on an anteriorly displaced disc. Disc may perforate → associated with joint crepitus and development of osteoarthritis.
- Bite on stick
  - Ipsilateral hard → Hurts.
  - Contralateral → Still hurts the opposite joint.
- Wilkes Classification (Table 15.1)

Wilkes 3 → Arthrocentesis/Arthroscopy

MRI

- 35% have disc displacement with no symptoms.
- 93% with symptoms have disc displacement.

Degenerative Disease/Osteoarthritis (10%)

- Displaced disc → Degenerate joint disc
  - Condyle has regenerative capacity.

OA

- Balance → Loading versus remodeling.
- Disease modifying drugs:
  - Topical NSAIDs.
  - Offload joint → mouthguard.
  - Intra-articular steroid → can worsen symptoms.

Investigations

- OPG → Eliminate any gross bony abnormalities.
- Osteoarthritis → Narrowing of joint space, cyst formation, remodeling, erosions, osteophytes formation.

MRI

- T1—TMJ anatomy.
- T2—allows visualisation of joint effusion → significant number have TMJ anterior displacement with no symptoms.

CT (good bone details)

15.3 Non-surgical Options

Splint Therapy

- Exact mechanism of action not well understood.
- Reduces bruxism/creates gap between the condyle and fossa to free disc.
- Placebo effect.
- Earlier firing of mechanoreceptors—further inhibition of jaw muscles → allows relaxation.

Types

- Stabilisation/reposition.
- Variety of materials/design/for either jaw.

Occlusal Rehabilitation

- Cochrane review → no evidence of benefit.
- Only if new fillings/crowns caused problem.

Pharmacotherapy—Central/Adjunct Role

- NSAIDs.
- Corticosteroids (intra-articular vs. systemic).
- Analgesics.
- Muscle relaxants—baclofen/botulinum toxin.
- Anti-depressants → TCA, MAOI, SSRI—no RCT showed efficacy.
- Anxiolytics → Benzodiazepines, buspirone.

Physical Therapy

- Exercise.
- Massage (ultrasound/thermal therapy, electric stimulation—TENS).

Sidebottom (2018 BJOMS)

- 80% → conservative management.
- Rest, pain in joint → Inflammation → rest.

Reassurance

- NSAID → Topical QDS.
- Bite splints.
- Retrusive jaw exercises—only useful for internal derangement
  - Acute closed lock → arthrocentesis/arthroscopy.
  - Physiotherapy (not harmful) but no evidence of benefit.

15.4 Surgery: Goals/Criteria for Surgery

- Surgery is reserved for patients whose TMJ pain with dysfunction is refractory to non-surgical modalities, and when symptoms result in significant impairment.

Criteria for Success: Goss 1993

- Improvement in pre-treatment pain-based on Visual analogue scale.
- ROM (vertical >35 mm).
- Lateral excursion/protrusive >6 mm, ability to eat.
- Absence of significant complications.
- Radiographic evidence of stabilisation of degenerative changes.

Management

- MDT (physiotherapy/GP/psychology/GDP).
- Disease therapy and pain—psychosocial management.
- Explanation and reassurance, education regarding parafunctional habits.

1995

- De Leeuw et al. (1958–1962) → 99 patients treated conservatively without surgery.
- Reassessed 30 years alter → Assessed TMJ → 90% chew foods.
- Conclusion: TMJD—self-limiting long term.

15.5 Indications for TMJ Surgery

- TMJD refractory to appropriate non-surgical therapies, causing significant impairment to patients' daily activities

  (1) Arthrocentesis and manipulation.
  (2) To treat displaced/closed locks unresponsive to conservative measures

    - 90% success rate, local anaesthesia/general anaesthesia.
    - Joint washout and adhesion freed.
    - Washout debris/inflammatory mediators, decrease intracapsular pressure in inflamed joint.

  (3) TMJ arthroscopy

    - Only access upper joint space for diagnosis and treatment under general anaesthesia.
    - Sidebottom BJOMS—7 years prospective → 86% success—inspect/ washout joint/breakdown adhesions.
    - 1.9 mm—fibreoptic/glass rod, 1.3 mm disposable scopes.
    - After 6 months of non-conservative therapy → 30° scope.
    - Fixation of retrodiscal tissues to eliminate anterior dislocation.
    - Meniscopexy (fixing disc into posterior position).

Complications

- Increased risk compared to arthrocentesis → Perforation of middle cranial fossa, EAM, tympanic membrane.
- Bleeding into EAM/hemarthrosis.
- Auriculotemporal and facial nerve injuries/AV fistulas/broken instruments

  – Studies comparing arthrocentesis and arthroscopy → No significant statistical difference.
  – Arthrocentesis → Less invasive.

15.6 Open Joint

- Eminectomy → No evidence of improvement.
- Condylar shave
  - Condylar hyperplasia.
  - Reduce osteophytes and removal joint surface → no evidence prevents progression.
- Discectomy → Grossly deranged disc → Debate whether to replace disc
  - Interposition temporalis flap to prevent further damage to surface. Risk of flap necrosis and reduced mouth opening.
  - Leave → Get fibrosis/scarring → No evidence any other better options.
- Condylar neck osteotomy → Pain relief from internal derangement → Need IMF (risk malocclusion, condylar sag).

15.7 TMJ Reconstruction

Goals of TMJ Reconstructions

- Improvement in function and aesthetics.
- Reduction pain/disability.
- Prevention further morbidity.

Indications

- Joint ankylosis.
- End stage degenerative disease/rheumatoid arthritis.
- Neoplastic.
- Revision procedures (previous failed reconstructions).
- Avascular necrosis.

Contraindications

- Acute infection.
- Known allergy to material—metal.
- Inability to undergo rehabilitation.
- Patient still growing.

Reconstructive Options

(1) Autogenous

- Bone—non-vascularised (costochondral, clavicle).
- Bone—vascularised (fibula, rib, Iliac crest, metatarsal).

- Soft tissues—non-vascularised (dermal fat, fascial lata, auricular cartilage, masseter).
- Soft tissues—vascularised (temporalis).

(2) Alloplastic

- TMJ concepts.
- Biomet.

(3) Bone

- Distraction osteogenesis.
- Tissue engineering.

Autogenous

Costochondral Graft

- Harvest 6/7th rib—5 cm bone/10 mm cartilage—preserve perichondrium/periosteum at bone cartilage junction → soft tissue graft—perichondrium between glenoid and rib.
- 4–6 screws to hold in place.
- Immediate post-operative physio and soft diet ±IMF.

Complications

- Intra-operative: Bleeding, facial nerve damage, pneumothorax.
- Post-operative
  - Early: Infection, sialocele, fistula, pneumonia.
  - Late: Re-ankylosis, variable growth/resorption, fracture graft/resorption Frey's syndrome.

Advantages

- Biologically compatible/growth potential/infections rare.

Disadvantages

- Morbidity donor site, fracture/ankylosis, overgrowth/undergrowth.

Total Joint Replacement

- NICE Guidelines—indication for joint replacement
  - Total prosthetic replacement of the temporomandibular joint is considered when alternative treatments have failed.
  - Involves replacing both the skull base component (the fossa or socket) and the condyle with prostheses.
  - The aims of the procedure are to re-establish function of the temporomandibular joint and to relieve pain.

Nice Guidelines

- Team with regular practice and expertise → audit dates, explain to patient about safety/efficacy no long-term data.
- Key outcomes → pain relief, correction of bite /improvement in mouth opening, ability to eat normal diet.
- Side effects: facial nerve damage, bite disturbance, dislocation, bleeding, allergy, ankylosis, ongoing pain, infection.

Allergy Testing

- Nickel.
- Cobalt.
- Chromium.
- Molybdenum.
- Stock versus custom options available.

Need to patch test/exclude nickel allergy first.
Two main companies currently approved for use by FDA others are historic:

- Zimmer Biomet—prosthesis is a stock prosthesis with a fossa component of ultra-high weight polyethylene, condylar prosthesis made of cobalt-chrome alloy. Both offered in three sizes (small, medium and large).
- Stryker (TMJ concepts) offers custom prosthesis only. The fossa component is a titanium backed Ultra-High molecular Weight Polyethylene (UHMWPE), condylar prosthesis made of cobalt-chromium alloy.
- These design differences in the fossa are apparent on radiograph.
- Main advantages of custom:
  - Less on table adjustment of ramus or condyle required especially if previous surgeries, better adaptation and seating.
  - Custom can be integrated into virtual surgical planning design for more complex deformity corrections (combined bimaxillary oestetomy/oncology resection etc.).
  - Can have all titanium condylar components for those with nickel allergy.
  - Extended designs to replace longer spans.
  - Expected life span of TMJ replacement around 15–25 years based on other joint replacements and experience.
  - Mouth opening 3–3.5 cm (usually nearer 3 cm)—more than enough for function, but patients lose their lateral excursion.
  - Access incisions—Modified Al Kayat-Bramley and Risden incisions.
  - Time to produce is 6–8 weeks once design finalised.

15.8 TMJ Ankylosis

- Fusion of joint with reduced/absent movement.
- Trismus (muscle spasm)
  - Trauma, infection, neoplasia, TMJD, drugs (phenothiazines).
- Pseudoankylosis
  - Extracapsular mechanical intervention → trauma (depressed malar fracture), coronoid hyperplasia/neoplasms.
  - Scarring of masticatory muscles (previous radiotherapy, submucous fibrosis).
- False Ankylosis—Extracapsular
  - Trauma (periauricular fibrosis), infection (chronic peri-auricular suppuration).
  - Radiation (periauricular fibrosis/osteoradionecrosis).
- True Ankylosis
  - Trauma: Birth, intracapsular fracture.
  - Infection: Osteomyelitis, mastoiditis, iatrogenic (post-TMJ surgery).
  - Neoplasia: Primary/secondary.
- Autoimmune: Juvenile idiopathic arthritis, ankylosing spondylitis, rheumatoid arthritis.

Classification

- Location: Extra-/intracapsular.
- Type of tissue: Bony/fibrous/mixed.
- Extent of ankylosis: Complete/incomplete.

Topazian's Staging true ankylosis

- I. Ankylotic bone limited to condylar process.
- II. Extending to sigmoid notch.
- III. Extending to coronoid process
  - Type I: fibrous adhesions in or around joint.
  - Type II: Formation bony bridge between condyle and glenoid.
- Condylar neck is ankylosed.

Sawhney (1986)

- Type 1: Extensive fibrous adhesions around the joint.
- Type II: More bony fusion at the outer edge of the articular surface, but no fusion within the medial area of the joint.
- Type III: Bony bridge between the mandible and the temporal bone.
- Type IV: The joint is replaced by a mass of bone.

Clinical Assessment

- CT ± modelling—important to measure the medial extent, relation of coronoid process to zygomatic arch and distance of maxillary artery.
- Relationship of ankylotic mass is important anatomic structure especially skull base, pterygoid plates, carotid canal, jugular foramen, and foramen spinosum.

Clinically

- Affects growth, reduces posterior facial height—influences maxillary growth.
- Affects joint tissues—pterygomasseteric sling.
- During growth
  - Deviation of the chin/mandible to affected side.
  - Reduced vertical height—ipsilateral, retrognathic mandible (short ramus/body).
  - Retruded mandible—facial profile = convex.
  - Occlusal cant/class II/posterior cross bites.
- OSA/airway problems.

Management

- Restore mouth opening, joint function.
- Allow condylar growth.
- Relieve upper airway problems.

Kaban (1990) Management Protocol

- Wide intra-operative exposure.
- Aggressive total excision of ankylotic segment (especially medially).
- Ipsilateral coronoidectomy, contralateral if mouth opening <35 mm via intraoral approach.
- Lining of the TMJ with native disc if salvaged or temporalis fossa.
- Reconstruction with distraction/costochondral graft/joint replacement.
- Active jaw exercise immediately after operation.

Surgical Options

- Gap arthroplasty: False joint at glenoid fossa (10–20 mm).
- Interposition arthroplasty: Autogenous, allogenic, alloplastic, xenograft (temporalis fascia, fascia lata, skin, fat).

Joint Reconstruction

- Autogenous: Costochondral, sternoclavicular, fibula.
- Alloplastic TMJ reconstruction.

15.9 TMJ Dislocation

- Condyle displaced anteriorly beyond eminence.
- Subluxation—soft reducing displacement.
- Acute versus chronic (>1 month).

Stability of TMJ Governed By

- Shape of glenoid fossa/condylar head.
- Ligament of joint—lateral capsule/ligament, sphenomandibular and stylomandibular muscles.
- Occlusion or lack of it (loss of dentition).
- Hyperfunction of protractor muscles.

Recurrent Dislocations

- Laxity of ligaments following dislocation due to trauma with tearing of ligaments/capsule.
- Hyperactivity of the mandibular depressor muscles—suprahyoid/lateral pterygoid by conditions such as epilepsy, cerebral palsy, medications (anti-psychotics), Parkinson's disease.
- Psychiatric disease.
- Systemic disease—arthritis, Ehler's Danlos syndrome.
- Loss of dentition—overclosure and joint laxity.
- Degeneration of ligaments secondary to disease (e.g., rheumatoid arthritis).

Management

- Acute.
- Radiographs to exclude fractures.
- Stimulation of gag reflex (not predictable).
- Manipulation—sustained pressure on posterior teeth to overcome muscle spasm (LA/GA/I.V. sedation).
- Consider limiting opening for 2 weeks—barrel bandage/MMF.

Chronic

- Non-invasive
  - Reconstruction with/without (sedation/muscle relaxation/GA)—barrel bandage to limit mouth opening/MMF.
  - Physiotherapy to improve muscle function/awareness.
  - Patient education—avoiding wide opening, self-reduction.
- Dentures to prevent overclosure.

Invasive

- Allteration of ligaments
  - Lateral capsule tarsorrhaphy.

  - Injection of sclerosing agent into joint space (blood/sodium tetradecyl sulphate).
  - Reinforcement procedure—Mitek anchor suture from condyle to zygomatic arch/or temporal fossa.

Relaxation of Muscles

- Botulinum toxin injection to lateral pterygoid.
- Temporalis—scarification.
- Detachment of lateral pterygoid muscle.

Alteration of Bony Architect

- Dautrey's procedure (down fracture zygomatic arch onto coronoid).
- Eminectomy.
- Placement of mechanical stops—plates/screws at level of eminence.
- Bone graft to augment eminence.

Chapter 16
Clinical Examinations

Abstract A summary of clinical examinations of the head and neck.

Key Points

- Clinical examination for orthognathic surgery.
- Head and neck examination.
- Examination of cutaneous lesions.
- Examination of salivary glands.
- Examination for facial pain including TMJD.
- Trauma exam.

16.1 Orthognathic

- Position: Clinical Frankfort Plane horizontal to floor/lips at rest.

Lateral View

- Facial 1/3.
- Frontal bossing, nasal shape, nasofrontal, nasolabial, labiomental, cervicofascial.
- Dorsum/tip of nose.
- Maxilla concave/convex.
- Paranasal hollowing.
- Gross skeletal pattern.

Frontal View

- Facial 1/3: Medial canthus to alar base.
- Facial 1/3: Upper face height/lower face height.
- Brow/tip: Aesthetic lip.
- Contributes—nasal/dental upper and lower/chin point = feel TMJ/check for deviations—asymmetry.
- Upper lip length.
- Incisors show at rest/dynamic.

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- Dental centrelines.
- Intraoral examination: OH/incisors (overjet/overbite/openbite), molars, missing/crowding, wisdom teeth.

16.2 Head and Neck Exam

- Firstly, check the primary site, FNE where appropriate.
- FNE
  - Head level.
  - Spray: Lidocaine 5%/Phenylephrine 0.5% Nasal Spray.
  - Focus and white balance.
  - Nasal floor.
  - Soft palate/Eustachian tube.
  - Fossa of Rosenmuller.
  - Posterior wall.
  - Vallecula, vocal cords

    Lip compensatory and drooling.
    Tongue mobility, speech, swallowing, mastication.
    Oral health and radiation caries/mucositis.
    Dental status—e.g. problems with denture/obturators.
    Mouth opening: Measure with Willis gauge.
    Speech intelligibile—understood over phone.
    Drooling.
    Diet—PEG/RIG.
    Appearance—facial nerve (MMN).
    Sensation—Von Frey filament (nylon).
    Weight—malnutrition.

Neck Exam—Primary Drainage Areas

- Under lower border.
- Facial lymph nodes.
- Sternomastoid—fingers.

Shoulder

- Stand behind—abduct to 90°.
- Can they take bra off.
- Comb back of head.
- Hand—radial site.
- Scar—contraction/hypertrophic.
- Skin graft.
- Tendon exposure.

- Loss of sensation over first dorsal interosseous.
- Function: grip/pain.
- Median/ulnar nerve.

Scapula

- Shoulder function.

DCIA

- Sensation cutaneous nerve.

Fibular

- Wound healing.
- Hammer knee—flexor hallucis.
- Sensation—lateral sural cutaneous nerve: posterior/lateral leg gait.
- Foot drop—common peroneal nerve: wasting of anterior tibial and peroneus muscles.
- Pulses—dorsalis pedis.

Look

- Muscle wasting.
- Hair.
- Scar.
- Foot drop.
- Skin graft.

Feel

- Swelling.
- Warmth.
- Tenderness.
- Sensation.
- Pulses—posterior tibial.
- Tendon expose.

Move

- Knee: Joint—flexion/extension.
- Ankle: Plantarflexion/eversion/dorsiflexion.
- Gait: Foot drop.
- Toe: Hammer toe.

New Oral SCC

- Introduction.
- Patient demographics.
- History of lesion—duration/growth/change of colour or pigmentation/bleeding/pain/previous surgery.

QOL Issues

- Speech, swallowing, taste, checking change in nerve sensation—trigeminal.
- Any other lesions of face/head/neck.

Miscellaneous History

- Industrial chemicals.
- Haematological conditions.
- Immunosuppression.
- Head and neck irradiation.

Past Medical History

- Especially with regards to intended surgery.
- Previous surgery—bleeding.
- Pulmonary, cardiovascular fitness, exercise tolerance (claudication/vascular disease).

Social

- Smoking/occupation/alcohol/living circumstances/support.

Clinical Examination—Inspect with Features

(a) Appearance, location, texture, size (with ruler), proximity to surrounding structures, thickness.
(b) Trismus—whole oral cavity.
(c) Head and neck exam/neck nodes.
(d) FNE.
(e) Examination around possible donor site.

16.3 Cutaneous Lesion

Introduction

- Patient demographics.
- Age/occupation.
- History—lesion

  (a) Duration, growth, change in colour/pigmentation, bleeding, previous surgery on lesion.
  (b) Any other lesions—head/face/neck/body.
  (c) Any other lumps/bumps.

History Of

- Sun exposure/sunscreen usage/hat-wearing.

Miscellaneous History

- Related to aetiology—industrial chemicals, hereditary conditions, burns, ulcers, scars, immunosuppression.

PMH

- Previous surgery/bleeding, also with regards to intended surgery.
- Medication: Aspirin/warfarin.

Social History

- Smoking (lip), alcohol, occupation (sun exposure/industrial chemicals), living circumstances, social support.

Family History

- Melanomas, non-melanoma skin cancer, immunosuppression, other cancers in family.

Examination—Wash Hands/Gloves

- Inspection.
- Palpation—determine attachment to underlying structures.
- Examine nerve/vital structures affected—e.g. trigeminal/facial nerve.
- Measure—lesion using ruler.
- Examination.
- Surrounding skin for laxity.
- Head and neck for other lesions.
- Regional nodes.
- Neck/pre- and post-auricular—donor site.

16.4 Salivary Gland

- Patient demographics.
- History of lesion
  - Duration, growth, previous surgery on lesion, pain, infections, swellings with food, previous stone history.
  - Change in sensation—trigeminal nerve.
  - Change in facial movements—facial nerve.
  - Any other lesions in other salivary glands.
- History/PMH/medication, especially xerostomia
  - Antihistamines.
  - Antidepressants.
  - Antihypertensives.

- Social history
  - Smoking/alcohol.
  - Social circumstances.
  - Support.
- Examination
  - Inspection of lesion with description of features

    Appearance.
    Location.
    Texture.
    Size measured with a ruler.
    Proximity to surrounding structures.
    Palpable thickness.

  - Trismus.
  - Comprehensive examination of oral cavity.
  - Bimanual palpation.
  - Examination of nerves or other vital structures that may be affected—e.g. trigmenial, facial, hypoglossal, lingual.
  - Examination of head and neck for other lesions.
  - Examination of regional nodes—systematic examination from level 1–5 and include post auricular nodes.
  - Any masses should be characterized by size, shape, mobility, consistency and proximity to adjacent structures.
  - Include thyroid and parotid glands.

16.5 TMJ/Facial Pain

- Nature of patient's complaints
  - Nature, intensity, location, duration, onset. Aggravating or relieving factors.
  - Other symptoms/parafunction.
  - Headaches, neuralgia, bruxism, clenching, clicks, crepitus, locking, dislocation.
- PMH
  - Arthritis, joint surgery, other chronic pain, facial skeletal surgery, bone pain, IBS etc.
- Medication
  - Pain killers (history of analgesics, antidepressants/benzodiazepines).

- Family/social history
  - Family—children, stress, anxiety, depression.
- Examination
  (a) Inspection: Abnormal asymmetry.
  (b) Jaw opening: Maximal (measure), protrusion, lateral excursion.
  (c) Palpate TMJ: Feel crepitus/clicking.
  (d) Palpate muscles: Lateral and medial pterygoids, masseters, temporalis, suprahyoid—test power, check accessory muscles (scalp/neck/back).
  (e) Examine dentition: Occlusion/centric relationship—check canine guidance.
  (f) Cranial nerves: Auriculotemporal, facial nerves.
  (g) Otoscopy/auscultation of ear and joints.

Trauma Examination

- Extraoral examination.
- Intraoral examination.

Extraoral examination: Remove and clear debris, photographs, if open fractures cover with betadine soaked dressing, systematic examination.

Systematic Examination:

Skull base and cranial vault: Laceration, contusions, Battle's sign (ecchymosis post auricular/astoid region), open/depressed skull fractures.

Midface

- Eyes: Pupil (size, reaction), visual acuity, eye movement, circumorbital oedema, subconjunctival haemorrhage, proptosis, dystopia/globe position (Hertel exophthalmometer), laceration of eye lid, corneal abrasions.
- Ear: Laceration, bleeding, otorrhoea, haematoma of auricular cartilage.
- Pain/swelling medial canthal region: check for depression, intercanthal distance, check for NOE fracture.
- Bony deformity: Zygomatic arch/body, infraorbital rim, infraorbital hypoaesthesia, check for Le fort fractures.
- Nose: Shape, nose bleed, rhinorrhoea (halo sign/beta-transferrin), septal haematoma, air entry.

Mandible: Look for asymmetry, deviation, dislocation, trismus, reduced jaw movement, ecchymosis, laceration. Palpate over TMJ region.

Dental alveolar and intraoral examination: Malocclusion, loss of dentition, laceration, ecchymosis, mobile mandibular and maxillary segments, position of the tongue.

Le Fort Fractures:

- Le Fort I: Only maxillary movement.
- Le Fort II: Movement maxilla & base of nose.
- Le Fort III: Movement of midface.

Chapter 17
Neck Lumps

Abstract An overview of the assessment and management of congenital and acquired neck lumps.

Key Points

- Aetiology of neck lumps.
- Assessment of neck lumps.
- Branchial cleft cysts.
- Thyroglossal duct cysts.
- Dermoid cysts.
- Epidermoid cysts.
- Teratomas.
- Laryngoceles.
- Carotid body tumours.

Congenital

- Midline (2TRED): Thyroglossal duct, Teratoma, ranula, epidermoid/dermoid cyst.
- Lateral (LVTBF): laryngocele, vascular malformation, thymic cyst, branchial cleft cyst, fibromatosis, lymphangioma, haemangioma.

Acquired

- Infectious: Lymphadenitis, abscess, TB, cat scratch disease.
- Inflammatory.
- Neoplastic
  - Benign: Thyroid goitre, lipoma, salivary gland tumour, paragangliomas.
  - Malignant: Salivary gland, thyroid, metastatic, sarcoma, lymphoma.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_17

Table 17.1 Branchial clefts

	Nerve	Arteries	Muscle	Cartilage/bone
I	V3	None	Tensor tympani/palatini, masticator mm, mylohyoid. ABD	Mandible, malleus, incus
II	VII	Stapedial	Platysma, facial mm, PBD, stylohyoid	Hyoid, stapes, styloid process
III	IX	CCA/ICA	Stylopharyngeus, sup/mid constrictors	Hyoid, stapes, styloid process
IV	X sup. Larynx	Aorta/prox SC	Inf. Constrictor, cricopharyngeus, cricothyroid	Thyroid cartilage
V	X rec larynx	Ductus art	Intrinsic laryngeal muscle (exc cricothyroid)	Cricoid, trachea, arytenoid

Clinical Approach

History

- Growth/pain, associated symptoms, referred pain, voice changes, dysphagia, epistaxis, weight loss, night sweats.

Examination

- Head and neck, oral cavity, cranial nerves, FNE.

Investigation

- FNE, imaging—CT/MRI/US.
- Path—FNA, core biopsy, external biopsy.
- Bloods: TFT/FBC/ESR/CRP.

Branchial Cleft Cysts (Table 17.1)

Aetiology

- 5 branchial arches of mesodermal origin, between each arch is groove or cleft arising from ectoderm. Formation of branchial cleft cyst is thought to be attributed to incomplete obliteration of the branchial cleft tracts.
- Abnormalities of the branchial cleft are usually cysts/sinuses/fistulas.

Clinical Features

- May be associated with other defects including PDA, tear duct atresia, hearing abnormalities and malformed auricles.
- Investigations: Children (USS)/Adult (MRI).

1st Branchial Cleft Cyst (5%)

- Occur parotid gland lympho-epithelial cyst

  - Type I: ectodermal, course parallel to EAC.
  - Type II: 1st and 2nd mesodermal elements—contains cartilage + adnexal structures.

- Radical excision ± parotidectomy

  - 2nd branchial cleft cyst (95%).

- Painless, fluctuant assess—lateral portion of neck = adjacent anterior/medial border SCM.
- Majority: Fistulas/sinus (3–10% bilateral).
- Can present with recurrent infections, sore throat/dysphagia.

Bailey Type I–IV

- Type I: Just deep to platysma.
- Type II: Commonest in lateral carotid space.
- Type III: Below ICA + ECA.
- Type IV: Pharyngeal mucosal space.

Tract

- Courses superficial to XII and XI between ICA and ECA and then pierces middle constrictors and opens to tonsillar fossa.

3rd Branchial Cleft Cyst (Rare)

- Presents anterior to SCM near superior pole of thyroid.

4th Branchial Cleft Cyst (Rare)

- Prevents neck mass—near the inferior lobe of thyroid.

Definitive Diagnosis

- Imperative to exclude malignant pathology.
- Cystic metastasis/lymphatic malformation.

Management

- Cyst infection—antibiotics/incision + drainage.
- Complete surgical excision.

Thyroglossal Duct Cyst

- 70% congenital neck mass—failure of obliteration.
- <1% risk of malignancy.
- Painless cystic mass in midline of the neck (or just off midline <2 cm) of the neck.
- 2/3 adjacent hyoid—located anywhere from tongue—pyramidal lobe of thyroid (rarely mass FOM).
- Move with deglutition and protrusion of tongue (unlike dermoid).

Investigations

- US/CT/MRI.
- Need TFT ± radionuclide scan to exclude median ectopic thyroid.

Management

- Surgical excision—Sistrunk procedure = en bloc excision of the entire TGD tract including 1–2 cm of the central hyoid bone to foramen cecum.
- Recurrence 4–6%.

Dermoid

- Germ cell tumour from ectoderm and mesoderm.
- Has skin appendages/sebaceous glands/hair follicles = 5% risk of SCC transformation.
- Totipotent cells: undergo disorganised growth vs. implantation vs. failure of complete embryonic fusion.

Clinical History

- Potential regions/nasal/scalp/neck.
- FOM: 10–12 cm interfering with speech/mastication.
- Lateral neck.

Investigation

- CT/US/MRI/FNA.

Management

- Excision.
- Above mylohyoid transoral. If below mylohyoid transcervical.

Epidermoid

- Germ cell tumour from ectodermal origin—usually adolescent (associated with Gardner Syndrome).
- Investigations: CT/US.
- Management: Excision.

Teratoma

- Germ cell tumour from 3 germ cell layers (ectoderm/mesoderm/endoderm).
- 10% in head and neck—neck (nasopharynx/oropharynx/oral cavity).
- Neonates airway obstruction.
- Management: Excision.

Laryngocele

- Abnormal saccular dilations of the laryngeal ventricles of Morgagni.
- Internal = no penetration thyroid membrane versus external (perforates).
- Mostly asymptomatic—may have hoarseness/cough/dyspnoea.

- Management: Excision, recurrence is rare (4%).

Carotid Body Tumour

- Benign extra-adrenal paraganglioma located at the carotid bifurcation (chemoreceptor cells).
- Very rare, most common in age 40–60.
- Painless, slow growing, pulsatile mass, bruit, 2% bilateral.

Differential Diagnosis

- Aneurysm.
- Lymphadenopathy.
- Branchial cyst.
- Schwannoma (mobile in one direction).

Management

- Wide access/total excision—peel off carotid, or resect + graft.
- ICA balloon test.
- Will need bypass shunt—internal carotid repaired with vein graft.
- Conservative/radiotherapy/surgical.
- 75% cure rate.
- Can be moved side to side by not up and down.
- Carotid body tumour can metastasise.
- Check for catecholamine breakdown products.

Shamblin Stage

- Minimal carotid.
- Partial encroachment.
- Encroaches carotid—bypass grafting required.

Chapter 18
Applied Head and Neck Anatomy

Abstract An overview of head and neck anatomy relevant to Oral and Maxillofacial Surgery.

Key Points

- Cranial nerves: ocular signs.
- Muscles of mastication and facial muscles.
- Orbital anatomy.
- Pterygopalatine fossa.

Second Cranial Nerve-Optic Nerve

Causes of Small Pupil (Miosis)

- Horner's syndrome.
- Argyll Robertson pupil: do not react to light but normal accommodation.
- Senile.
- Drugs: Opiates.

Causes of Large Pupil (Mydriasis)

- Holmes-Adie pupil—idiopathic (reacts poorly to light).
- CNIII palsy.
- Drugs: anti-depressants/ atropine.
- Trauma.

Horner's Syndrome

- Clinical.
- Miosis.
- Enophthalmos.
- Ptosis.
- Anhidrosis.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_18

Caused by Lesion Throughout Course of Sympathetic Trunk

- Brain stem/spinal cord.
- Vascular.
- Trauma.
- Neoplasia.
- Demyelination.
- Syringomyelia
  - Pre-ganglionic.

Chest

- Apical tumour/Pancoast tumour.
- Cervical rib.
- Mediastinal mass.

Cervical

- Lymphadenopathy.
- Neck surgery.
- Trauma.
- Thyroid neoplasia.

Post-Ganglionic

- Internal carotid dissection.
- Cavernous sinus lesion.
- Orbital apex disease.

18.1 Third Cranial Nerve–Oculomotor (CNIII) Palsy

Clinical

- Ptosis–drooping eyelid.
- Unable to move eye: superiorly/ medially/inferiorly.
- Eye: down and out.
- Pupil fixed/dilated.

Causes

- Vascular/diabetes/demyelination/ trauma.
- PCA aneurysms.
- Cavernous sinus thrombosis.
- Orbital trauma.
- Thyroid eye disease.

Pupils—RAPD

- Light shines on good eye—both pupils constricted.
- Light shine on bad eye—both pupils dilate.
- Optic nerve damage—damage beyond lateral geniculate body does not cause RAPD.

Optic Nerve CN II Damage

- Shining light causes non pupillary non- constriction and non-consensual reflex as no light reaches brain.
- If light shines on normal eye, get consensual reflex as 3rd nerve intact.

Oculomotor CN III Damage

- Shining light causes no pupillary non-constriction but get consensual reflex as light can transmit signal to brain.
- No consensual reflex on the bad eye.

Fourth Cranial Nerve—Trochlear (CNIV) (SO4)

- Clinical—superior oblique muscles.
- Exits dorsal aspect of brainstem.
- Diplopia when descending stairs/reading.

Causes

- Vascular/diabetes/demyelination/trauma.
- Congenital.
- Cavernous sinus thrombosis.
- Orbital apex syndrome
  - Sixth cranial nerve—Abducens (CN VI) palsy.

Clinical—Lateral Rectus

- Diplopia on lateral gaze (affected side).
- Affected eye elevates medially.

Causes

- Vascular/ diabetes/demyelination/trauma.
- Cavernous sinus thrombosis.
- Orbital apex syndrome.
- Increase in ICP—false localising sign
  - Trigeminal nerve.

Trigeminal Nerve—Pathway (Figs. 18.1,18.2,18.3)

- Sensory root of trigeminal IV (mesencephalic/ chief sensory/ spinal).
- Emerges ventral surface of the upper pons to enter middle cranial fossa.

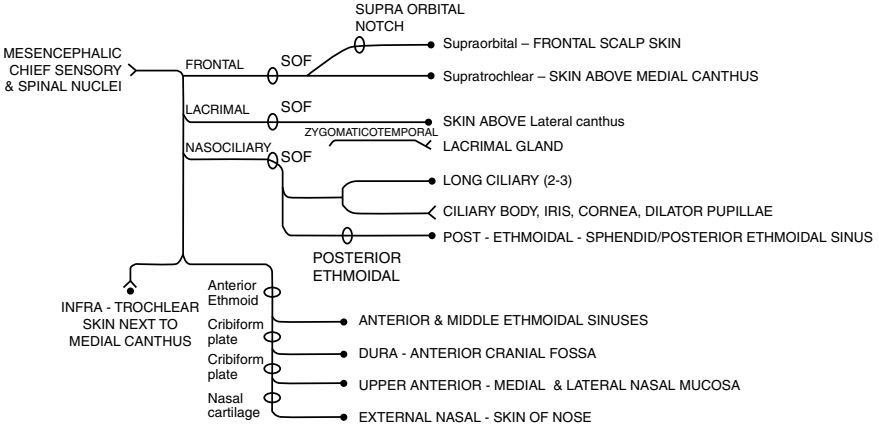


Fig. 18.1 Trigeminal nerve Va

- Passes to trigeminal ganglion which lies in Meckel's cave (prolongation of dura at the apex of the petrous/ temporal bone).
- Nerves leave ganglion—run in lateral wall cavernous sinus and then exit via:
- V1—superior orbital fissure.
- V2—foramen rotundum = crosses posterior aspect of palatine bone through inferior orbital fissure infraorbital foramen.
- V3—foramen ovale = passes into infra-temporal fossa between tensor veil palatine with CP divided into divisions:

1. Anterior: Muscles.
2. Posterior
   (a) Auriculotemporal: Ear.
   (b) Lingual: Goes between MP and mandible through floor of mouth, first lateral then inferior/ medial to submandibular duct (joined by chorda tympani).
   (c) Inferior alveolar: Passes deep to lateral pterygoid and lies between sphenomandibular ligament and ramus, enters mandible inferior alveolar foramen (mylohyoid branch before enters).

Masseter (VC)

- Anterior 2/3 zygomatic arch and zygomatic process of maxilla.
- Lateral surface of angle and lower ramus of the mandible.
- Elevates mandible.

Medial Pterygoid (VC) (Fig. 18.4)

- Deep head: Medial side of lateral pterygoid plate and fossa between medial/ lateral plates.
- Superficial head: Tuberosity of the maxilla and pyramidal process of the palatal bone.

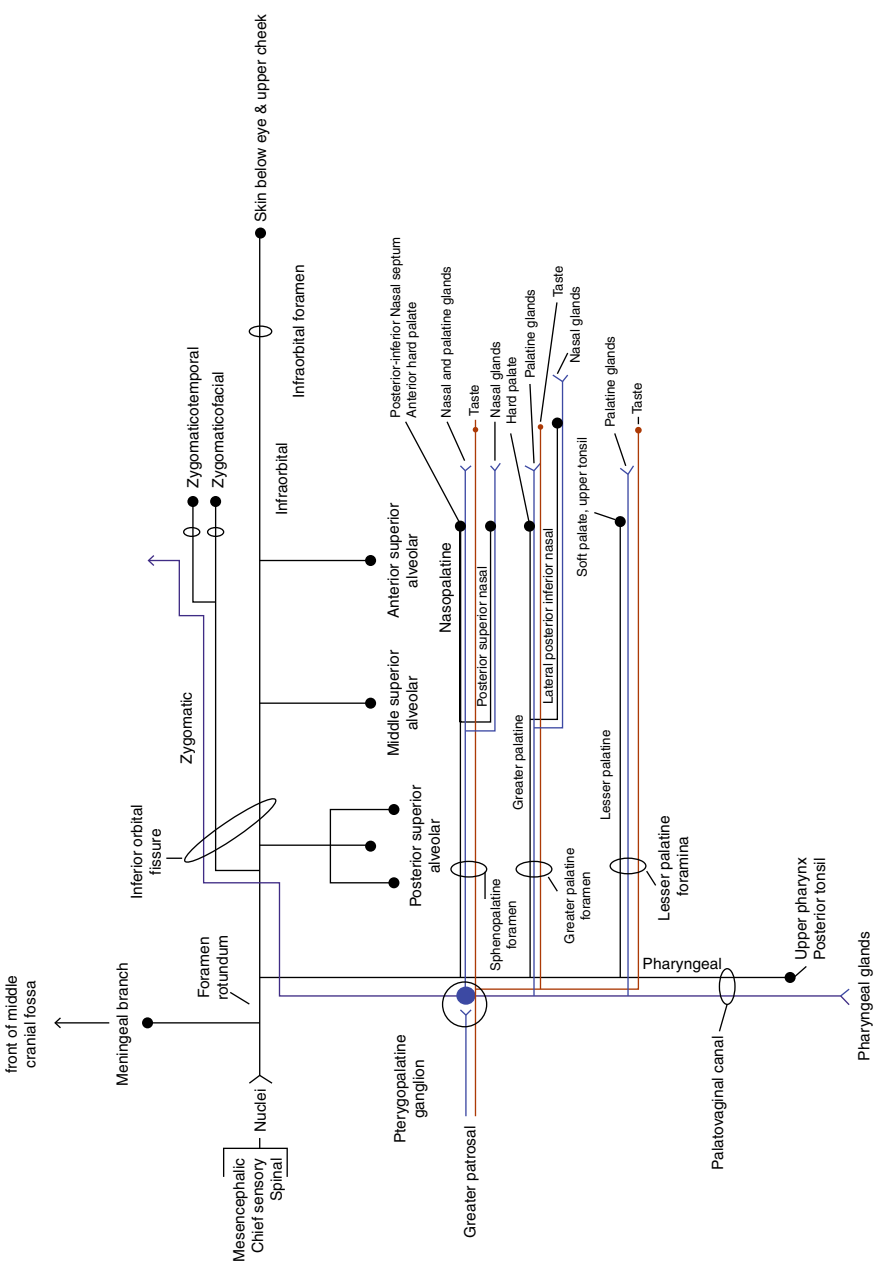


Fig. 18.2 Trigiminal nerve Vb

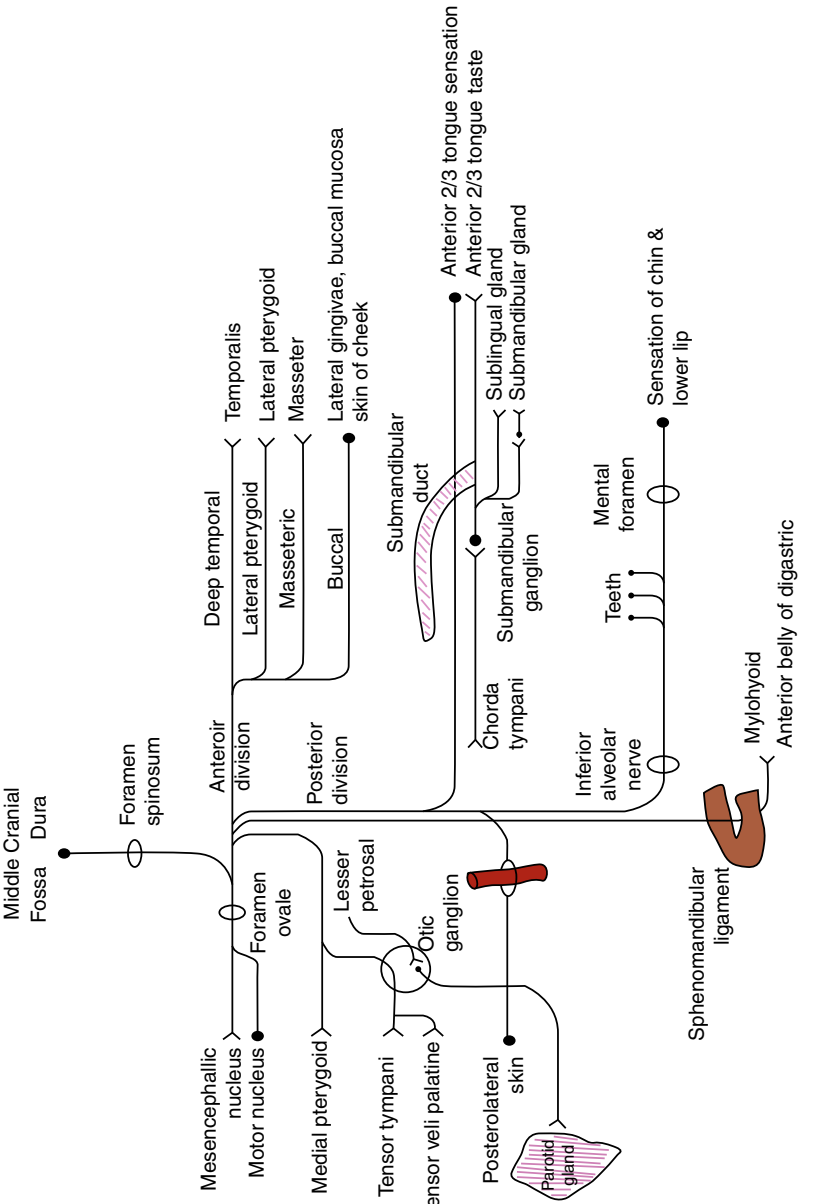


Fig. 18.3 Trigiminal nerve Vc

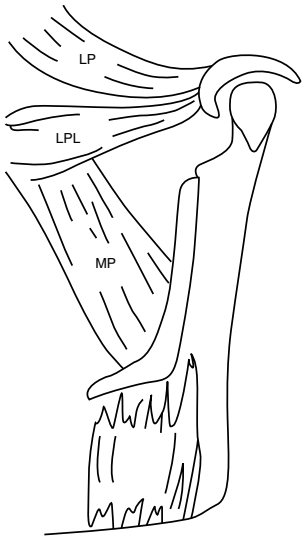


Fig. 18.4 Muscles of mastication

- Inserts: Medial aspect of the angle of the mandible.
- It acts as an elevator: Protracts and laterally displaces to opposite side for chewing.

Lateral Pterygoid (VC) (Fig. 18.4)

- Superior upper head: Infratemporal surface of sphenoid bone—anterior margins of the articular disc.
- Inferior lower head: Lateral surface of lateral pterygoid plate—fovea of condylar neck.
- Depresses and protracts mandible to open mouth.
- Action: Pulls cartilage of the joint during mouth opening.

Temporalis (VC)

- Arises: Temporal fossa/ infra-temporal line and infra-temporal crest.
- Insertion: Medial and anterior of coronoid process.
- It acts as an elevator: Elevates mandible/ posterior fibres retract.

Tensor Tympani (VC) (Fig. 18.5)

- Arises: Cartilaginous and bony margins of auditory tube.
- Insertion: Handle of malleus.
- Action: Protects and critically damps vibration in the ossicular chain.

Tensor Veli Palatini (VC)

- Arises: Scaphoid fossa and medial aspect of spine of sphenoid bone.
- Insertion: Palatine aponeurosis (via pulley of pterygoid hamulus).
- Action: Tenses soft palate to elevate.

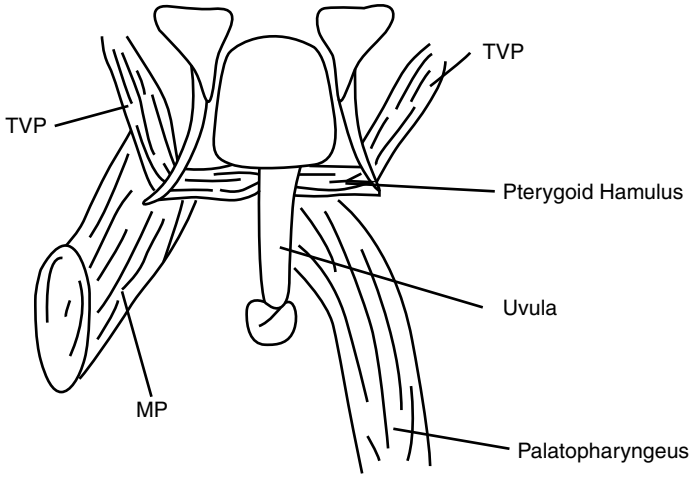


Fig. 18.5 Muscles of the pharynx

Mylohyoid (Fig. 18.6)

- Arises: Mylohyoid line on the internal aspect of mandible.
- Insertion: Anterior ¾ (midline raphe)/ posterior ¼ (superior border of the hyoid bone).
- Action: Elevates hyoid, supports FOM, aids mastication/ swallowing.

Anterior Belly of Digastric (VC)

- Digastric fossa: Symphysis—fibrous loop around lesser cornu of hyoid.

Posterior Belly of Digastric (VII)

- Mastoid process.
- Fibrous loop around lesser cornu of hyoid.
- Sternocleidomastoid.
- Arises: Anterior and superior manubrium and superior medial third of clavicle.

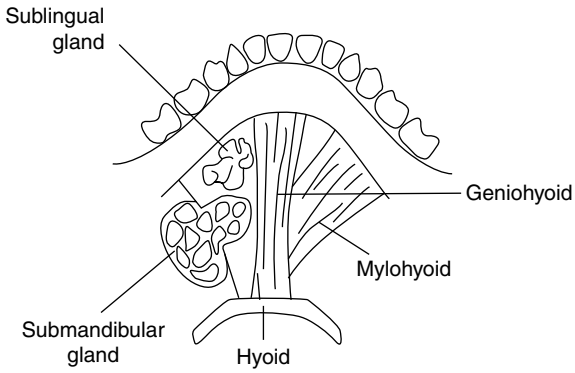


Fig. 18.6 Floor of mouth anatomy

- Insertion: Lateral aspect of mastoid process.
- Spinal accessory (C1–C5).

Omohyoid

- Arises: Suprascapular ligament and adjacent scapula.
- Insertion: Inferior border of the hyoid body.
- Ansa cervicalis (C1, C2, C3).

Facial Nerve (Fig. 18.7)

(a) Leaves pons at the cerebellopontine angle medial to vestibulocochlear nerve (VIII).
(b) Passes across the subarachnoid space inferior to internal auditory meatus.
(c) Enters the facial canal and two roots unite.
(d) Passes laterally into the medial wall of the middle ear.
(e) Turns 90 degrees at geniculate ganglion.
(f) Nerve then turns 90 degrees inferiorly, runs down medial wall of the mastoid antrum.
(g) Leaves middle ear via stylomastoid foramen.
(h) Passes between mastoid process and tympanic ring before passing between deep/ superficial portions of parotid gland.
(i) Within gland it is superficial to retromandibular vein/ external carotid.

Chorda Tympani

- Arises from facial nerve in facial canal, passes between tympanic membrane over handle of malleus.
- Leaves middle ear, emerges via petrotympanic fissure.
- Passes into infratemporal fossa, medial to spine of sphenoid which it grooves.
- Run antero-inferiorly deep to lateral pterygoid, and joins lingual branch of Vc 2 cm below skull.

Internal Carotid Artery

- Arises at bifurcation of common carotid artery (CCA) and continues upwards within carotid sheath
  - The carotid body lies behind bifurcation of the CCA (chemoreceptors).
- The ICA is lateral to ECA at origin, but soon slopes up posteriorly to lie medial.
- ICA has no branches & passes straight up beside pharynx to the carotid canal
  - Behind the ICA is the sympathetic trunk (outside the carotid sheath).
  - It is lateral with vagus nerve deeply placed b/w artery and vein.
  - Superficially, near its origin it is crossed by hypoglossal nerve.
  - Superior root of the ansa cervicalis runs downwards along it, within carotid sheath.

External Carotid artery (Fig. 18.8,18.9).

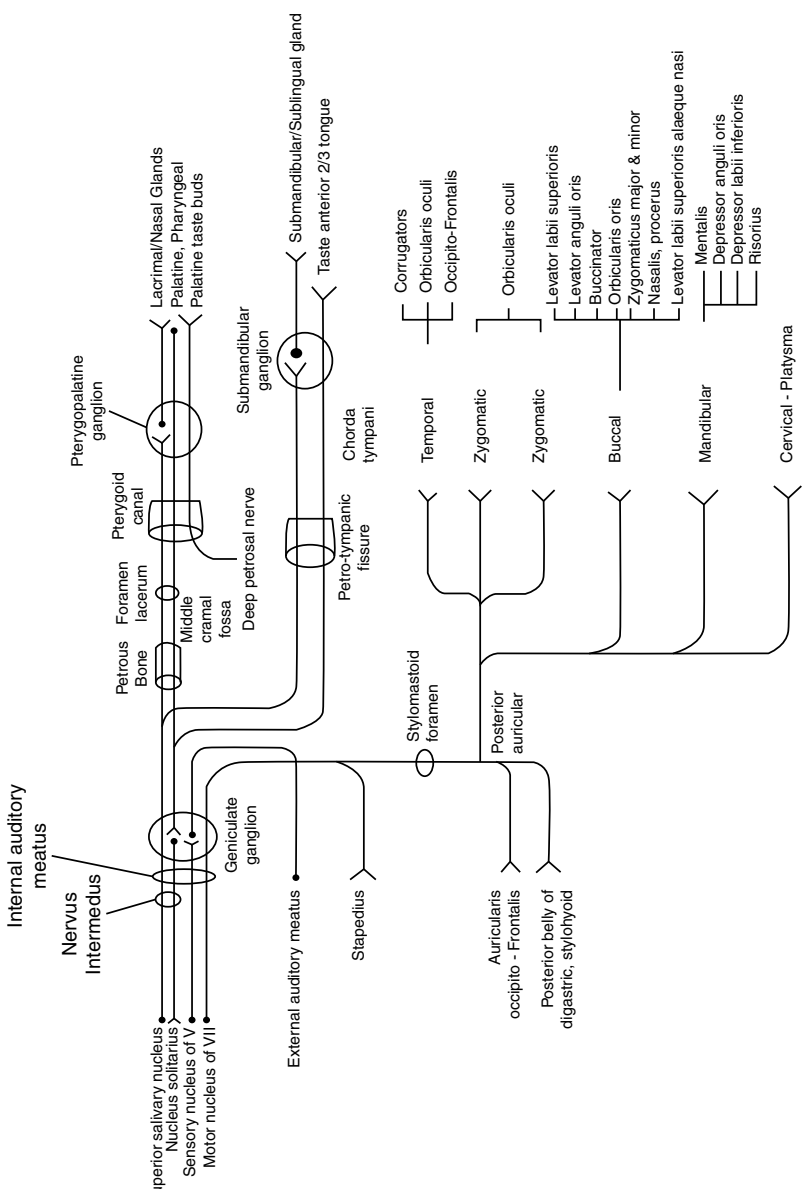


Fig. 18.7 Facial nerve

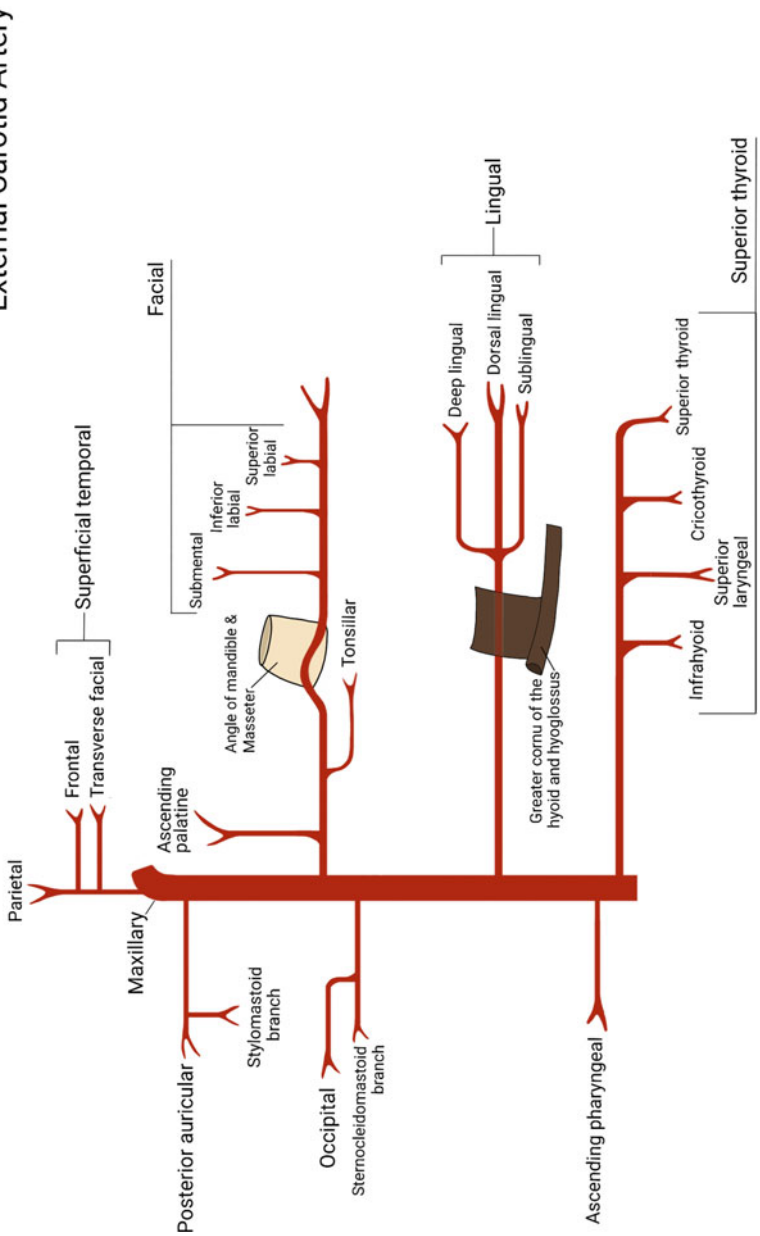


Fig. 18.8 External carotid artery

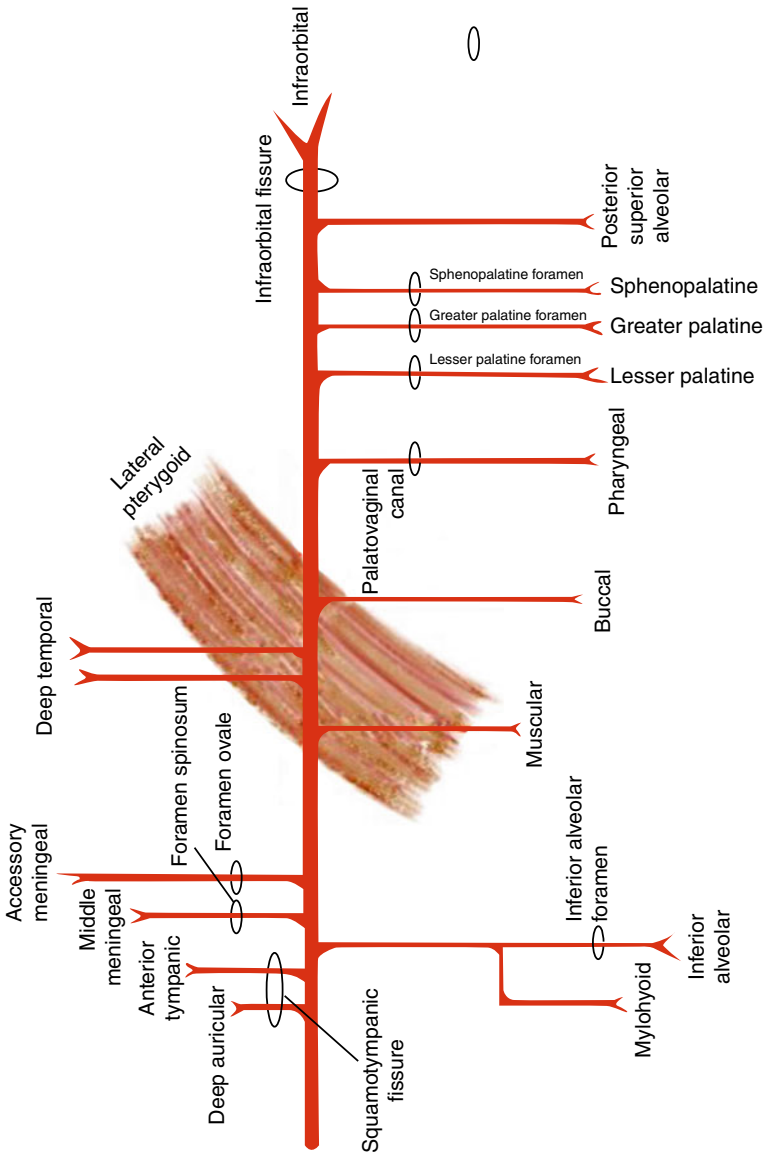


Fig. 18.9 Maxillary artery

18.2 Orbital Anatomy

Lacrimal Duct System

- 12 small ducts drain—drainage system
  - Superior and inferior punctum.
  - Lacrimal canaliculi.
  - Inferior and superior canaliculi (8 mm).
  - Lacrimal sac (12 mm long).
  - Medial orbital wall.
  - Medial palpebral ligament lies anterior and superior to the sac.
  - Nasolacrimal duct.
  - Inferior meatus.

Whitnalls Tubercle

Bony protuberance—lateral orbital wall 5 mm behind the rim

- Attachment of
  - Lateral horn levator aponeurosis.
  - Lateral palpebral ligament.
  - Lateral cheek ligament.

Muscles

- Inferior oblique
  - Anterior–posterior of orbital floor.
  - Movement: upwards and lateral.
  - Nerve innervation: CNIII.
- Superior oblique
  - By way of pulley and attached to sclera behind.
  - Movement: downwards and lateral.
  - Nerve innervation: CNIV.
- Superior rectus
  - Nerve innervation: CNIII.
  - Annulus of Zinn.
- Inferior rectus
  - Nerve innervation: CNIII.
  - Common tendons—Sclera 6 mm behind corneal margin.
  - Superior rectus.
  - Lateral rectus.
  - Nerve innervation: CNVI.

  - Medial rectus.
  - Nerve innervation: CNIII.
  - Levator palpebrae superioris.
- Lesser wing of sphenoid—anterior surface of superior tarsal plate.
- Voluntary: Oculomotor—raises eyelid.
- Involuntary: Sympathetic nerve.

Glands

- Sebaceous gland (eye)
  - Glands of Zeis.
- Sweat gland (skin).

Tenons Capsule

- Fascial structure subdivides the orbital cavity
  - Anterior (pre-capsular): fat/muscles/vessels/nerve.
  - Posterior (retro-capsular): ocular/globe.

Orbital Fat (Fig. 18.10)

- Anterior: Extra-canal—outside muscle (does not contribute to globe support).
- Posterior: Intra-canal fat 3/4—can be displaced (loss of globe support).
- Inferior rim to inferior tissues (12 mm).
- Inferior rim to optic canal (48 mm).
- Lacrimal bone—posterior and anterior crest where MCL (medial canthal ligament) attaches (anterior/ posterior).
- Lacrimal apparatus—drainage of secretion from lacrimal gland (superior/ inferior).
- Punctum: Connect to lacrimal sac.
- Horner's muscle attach to the post-lacrimal crest, contraction empties the sac.
- Overlying the sac is the fleshy mass tissue—caruncle.

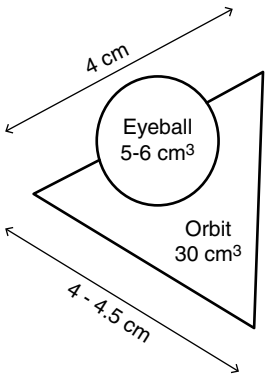


Fig. 18.10 Orbital distances

- Canthal tendons
  - Extensions of preseptal and pretarsal orbicularis.
  - Lateral slightly above medial.
  - Lateral tendon attaches to Whitnall's tubercle 1.5 cm posterior to orbital rim.
  - Medial tendon complex, important for lacrimal pump function.
  - Infra-temporal Region.
  - Anatomy.

Infratemporal Fossa (Fig. 18.11)

Space lying beneath base of skull between wall of pharynx and mandibular ramus

- Boundaries
  - Anterior: Infratemporal surface of the maxilla.
  - Posterior: Articular eminence of the temporal bone, carotid sheath/ styloid process.
  - Medial: Lateral pterygoid plate, Lateral pterygoid, superior constrictor,
  - Lateral: Mandible (ramus/ coronoid).
  - Superior: Greater wing of sphenoid below infratemporal crest adjacent to squamous temporal bone.
  - Inferior: ends when medial pterygoid attaches to the mandible, not floor.

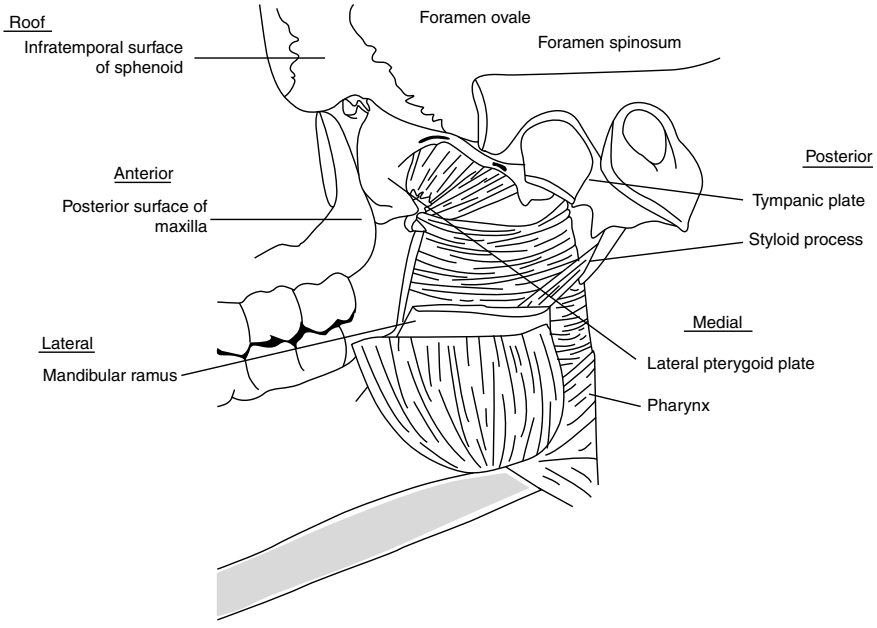


Fig. 18.11 Infratemporal fossa

- Foramen ovale– mandibular nerve.
- Foramen spinosum—medial meningeal artery.
- Alveolar canal—inferior alveolar nerve.
- Contents
  - Muscles: Temporalis, medial and lateral pterygoid muscles.
  - Arteries: Maxillary artery.
  - Veins: Pterygoid plexus of veins.

Nerves

- Vc—Mandible.
- Vb—Posterior superior alveolar nerve.
- Chorda tympani.
- Otic ganglion.
- Lesser petrosal nerve.

18.3 Pterygopalatine Fossa

Anatomy

- Small space between maxilla and the pterygoid process of sphenoid (Fig. 18.12)
- Pyramid shaped fossa—7 foramina/ fissures
  - Sphenopalatine foramen—nasopalatine nerve.
  - Palatovaginal fissure—maxillary nerve.

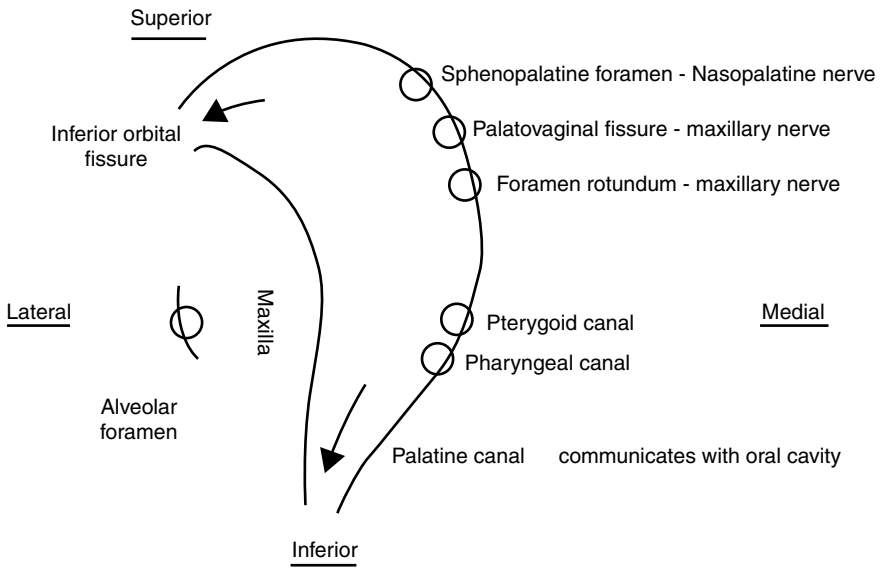


Fig. 18.12 Pterygopalatine fossa

- Foramen rotundum—maxillary nerve.
- Pterygoid canal.
- Pharyngeal canal.
- Palatine canal—communicates with oral cavity.
- Alveolar foramen.
- Inferior orbital fissure.

- Posterior—pterygoid process sphenoid bone Anterior—posterior wall of maxilla.
- Medial—palatine bone.
- Lateral—perpendicular plate of palatine bone.
- Roof: Sphenoid orbital process of palatine bone.
- Floor: Pyramidal process of the palate.

Parapharyngeal Space: (Fig. 18.13,18.14)

Anatomy

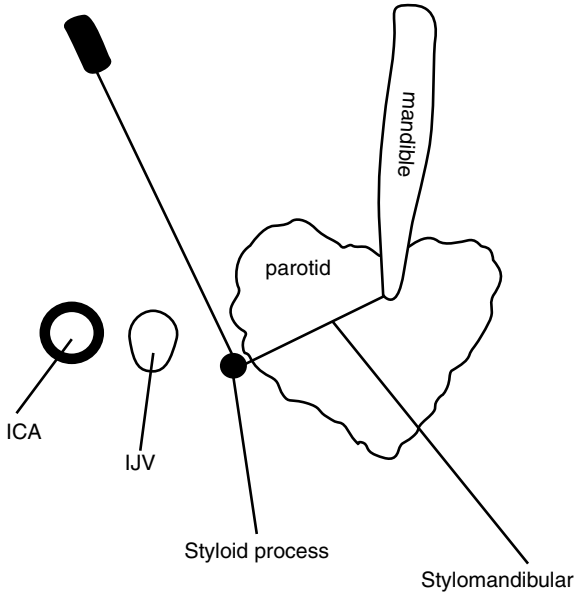


Fig. 18.13 Parapharyngeal space

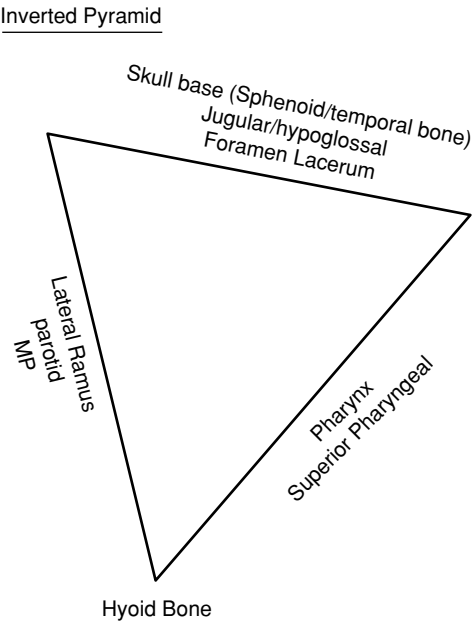


Fig. 18.14 Parapharyngeal space borders

Chapter 19
Salivary Glands

Abstract An overview of the management of pathology affecting the salivary glands.

Key Points

- Function of saliva.
- Saliva production.
- Salivary gland infections.
- Salivary gland stones.
- Sialendoscopy.
- Sialosis.
- Xerostomia and Sjögren's Syndrome.
- Mucocoele and ranula.
- Sublingual gland removal.
- Hypersalivation.
- Salivary neoplasms.
- Facial nerve.

19.1 Overview of Salivary Glands

Major salivary glands consist of three pairs:

- Parotid, submandibular and sublingual glands.
- Multitude of minor salivary glands distributed in the mucous membranes of the oral cavity and pharynx.

  They synthesise and secrete saliva, a fluid which provides mucosal lubrication, salivary electrolytes and antibacterial compounds that protect the oral mucosa and dentition.

A. Ahmed et al., *Oral and Maxillofacial Surgery*,
https://doi.org/10.1007/978-3-031-25473-4_19

Saliva is composed of

- 99% water.
- Minor amounts of electrolytes.
- Proteins including of amylase, lysosomes and albumin.

Parotid Glands

- Formed by serous acini, and produce 25% of saliva in the unstimulated state.
- When stimulated, they are responsible for approximately 50% of the total volume of saliva from the mouth.

The Submandibular Glands

- Composed of mixed acini with mucous and serous components.
- Unstimulated state, the submandibular glands produce the most saliva (approximately 70%).

The Sublingual Glands

- Predominantly mucous acinar cells and serous acini are rarely found.

The minor salivary glands are predominantly formed by mucous acini, and account for 5% of salivary production.

Infections

- Viral.
- Bacterial.

Acute/Chronic phases

Acute Suppurative Sialadenitis

- Common in parotid glands due to the reduced bacteriostatic activity of parotid saliva.
- Common causes of infection: Dehydration, duct stricture, stones and poor oral hygiene.
- Staphylococcus aureus, Haemophilus influenzae, and Escherichia coli (E.coli). Clinical features of acute suppurative salivary infection include:
- Symptoms: Pain, fever, and purulent discharge from the gland.
- 20% of cases bilateral.
- Management is dependent on the severity of infection.
- Mild cases: Rehydration, improved oral hygiene and repeated massaging of the gland. If purulent discharge is expressed from salivary duct, antibiotic management and a swab for MC + S.
- Severe presentations: incision and drainage may be considered.

Chronic Suppurative Infection

- Secondary to unresolved acute infection, or persistently reduced salivary flow.
- Most common in the submandibular glands. Causes include:

  - Duct obstruction.
  - Salivary calculi.
  - Pain and swelling at mealtimes.
- Management of chronic infection includes the use of:
  - Sialendoscopy to remove the obstructing calculus, mucous plug, or dilate the duct.
  - In severe cases, removal of the gland may be necessary.

Salivary Stones

The submandibular gland is the most affected (80% of cases), followed by the parotid gland (10% of cases).

- Submandibular gland: More susceptible to stone formation due to longer duct length, saliva being more alkaline, high mucin and calcium phosphate concentration.
- Salivary stones are composed primarily of calcium carbonate and smaller quantities of magnesium and ammonia.
- 90% of stones of submandibular gland will appear radiopaque.
- 90% of stones of parotid gland appear radiolucent.

Management of Salivary Stones

Depends on the location and size of the obstructing stone.

- Conservative management techniques.
- Ductoplasty may be performed if stone is palpable.
- Sialendoscopy can be performed to remove the stone (stone is less than 6 mm in size and mobile).
- Lithotripsy can be used for suitable cases.
- Submandibular gland removal.

  90% of salivary gland stones occur in the submandibular gland, with 50% present asymptomatically.

Classic Signs and Symptoms

- "Mealtime syndrome"—with pain on the sight or thought of food.

Diagnostic Imaging

- Plain film imaging: OPG and occlusal radiographs.
- Depending on the size and location, plain film can show most stones, with 30% appearing radiolucent.
- CT will reveal relationship of duct to the gland, and presence of cervical lymphadenopathy.
- Ultrasound imaging can differentiate between solid and cystic form.

Management:

- Depends on the size and location of stone.
- Transoral delivery of stone with sialodochoplasty.
- Sialendoscopy facilitates removal of stone in suitable cases.
- Lithotripsy and retrieval of stone may also be explored. Extracorporeal lithotripsy is used mainly for fixed parotid stones, where the stone is greater than 6 mm in size and difficult to manage endoscopically.
- Excision of submandibular gland may be necessary.

All patients should be advised to maintain hydration, regular gland massage and analgesia. Antibiotics may be prescribed in cases of acute infection.

Sialendoscopy:

- Dynamic and minimally invasive, as well as acting as a diagnostic and therapeutic modality.
- The scope is usually 1.2 or 1.3 mm (the diameter of the Stensen's duct is 0.5–1.4 mm), with multiple ports for irrigation and instrumentation.
- Therapeutic indications include stone removal, duct dilation and for screening ductal system for residual stones after salivary lithotripsy.

Sialosis

- Chronic, bilateral non-inflammatory, non-neoplastic painless swelling of parotid gland.
- Secondary to autonomic neuropathy, and changes in salivary aquaporin water channels. Histologically, acinic cells are hypertrophic.
- Common causes of sialosis include diabetes mellitus, alcohol misuse and liver disease. Other causes such as drug misuse and bulimia are less common.

Xerostomia

Xerostomia Defined as

- Dry mouth resulting from reduced or absent salivary flow.
- Daily salivary production 0.5 – 1.5 L.
- 70% produced from the submandibular gland and 25% from parotid gland.

Causes of Xerostomia

- Congenital.
- Acquired.
- Sjögren's, sarcoidosis, diabetes mellitus, HIV, Hepatitis C and cystic fibrosis.
- Medications, antihypertensives, antidepressants and chemotherapy drugs.
- Chemotherapy, radiotherapy, graft versus host disease.
- Psychological causes: anxiety, exercise, dehydration and ageing.

Oral Complications

- Dry, cracked lips, angular chelitis and salivary gland enlargement.
- Difficulty eating and speaking, denture intolerance and dysgeusia.
- Increases infective processes: candida and dental caries.

Investigations

- Blood tests: Erythrocyte sedimentation rate (ESR), and C-reactive protein test (CRP) and autoimmune serology.
- Ultrasound, CT and MRI.
- Scintigraphy and sialography.
- Histopathology.

Management

Depends on cause

- Symptoms can be managed through hydration, sugar free gum, salivary substitutes and sialogogues.
- Systemic—Pilocarpine.

Sjögrens Syndrome

Autoimmune exocrinopathy—results in dry eyes and dry mouth.

Classification

- Primary—only exocrine involved gland.
- Secondary—60% associated with connective tissue disease (RA, SLE, scleroderma, polymyositis, primary biliary cirrhosis).

Pathogenesis

Lymphocytes infiltrate the gland—environmental antigen, HLA-B8, DR3 involved.

Clinical

- Peak onset 50 years old (90% females).
- Dry eyes, dry mouth, parotid enlargement.

Diagnosis

- 2016 American-European Consensus criteria for Sjögren's syndrome (Table 19.1)
- Based on the weighted sum of 5 items.
- A patient must have a total score of ≥ 4 to meet Classification Criteria for a Sjögren's diagnosis.

Investigations

- Salivary gland flow test.
- Schirmer test.

Table 19.1 Sjogrens scoring

ACR-EULAR 2016 Classfication criteris for Sjögren's		
Item #	Item to be scored	Weight
1	Labial salivary gland with focal lymphocytic sialadenitis and focus score ≥1/4 mm^2	3
2	Anti-SSA/Ro positive	3
3	Ocular staining score ≥5 (or van Bijsterveld score ≥4) in at least 1 eye	1
4	Schirmer's test ≤5 mm/5 min in at least 1 eye	1
5	Unstimulated whole saliva flow rate ≤0.1 ml/minute	1

- Sialography—punctate sialectasia, delayed emptying (snowstorm appearance).
- U/S—swiss cheese appearance.
- CT/MRI—internal hypodense areas—ductal ectasisa.

Management

MDT

- OMFS, ophthalmology, oral medicine, rheumatology.
- Artificial saliva.
- OH/diet/topical fluoride.
- Systemic—pilocarpine (muscarinic agonist).
- Ongoing review—6% risk of transformation to MALT lymphoma (5–15 years).

Bilateral Parotid Gland Enlargement

Sialosis

- Non-inflammatory, non-neoplastic, recurrent, bilateral swelling of salivary glands.
- Endocrine causes: Diabetes mellitus, acromegaly, hypothyroidism and pregnancy.
- Nutritional—general malnutrition, alcoholism, anorexia nervosa and bulimia.
- Antihypertensives and sympathomimetics are further neurogenic causes of sialadenosis.

Submandibular Gland Disease

Wharton's Duct

Wharton's duct is 5 cm long and arises deep within the submandibular gland. (Fig. 19.1).

- Exits on its medial surface between mylohyoid-hyoglossus muscle and between sublingual gland- geniohyoid muscle, to open into floor of mouth beside the frenulum.
- Duct is closely associated with hypoglossal and lingual nerves.

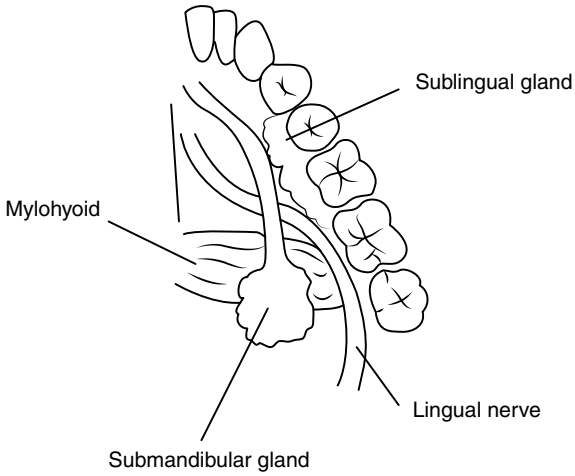


Fig. 19.1 Submandibular gland

- Hypoglossal nerve runs parallels to duct and runs deep to digastric muscle and submandibular gland.
- Lingual nerve courses laterally to the duct, and loops inferior and passes medial to duct.

Blood supply from facial artery, and venous drainage from facial vein.

Nerve Supply:

- Sympathetic supply is via superior cervical ganglion via the external carotid.
- Parasympathetic supply is from superior salivary nucleus via the chorda tympani, to lingual nerve via submandibular ganglion.

Histopathology of the gland will reveal mixed serous and mucous acini. The acini and intercalated duct is surrounded by elongated cells called myoepithelial cells.

Mucocoele/Ranula

Benign, mucous containing cystic lesion of the salivary glands.

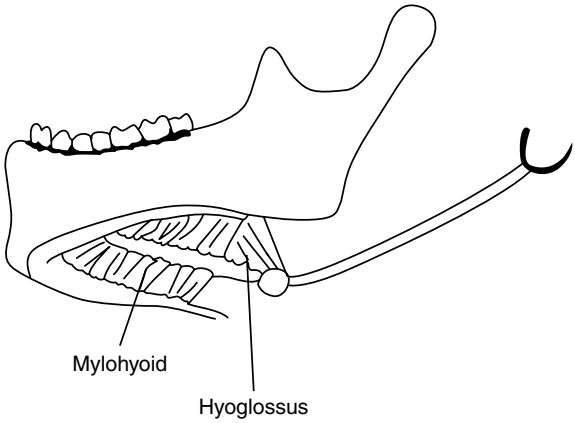
Mucocoele:

- Related to minor salivary gland.
- Two types of mucocoeles are recognised:
  - Retention type, in which the mucin pooling is confined within a dilated excretory duct or cyst.
  - Extravasation type, in which mucin is spilled into the connective tissues from a ruptured or traumatised salivary gland duct.

Clinically: They Present as

- Soft, painful swellings of variable size and colour (deep blue to pink).

Fig. 19.2 Mylohyoid



- Most common in lower lip, followed by the cheek, palate and ventral surface of the tongue.

  Dissection of the mucocoele with the associated mucous glands, as well as marsupialisation ± packing is recommended.

Ranula: (Fig. 19.2)

- Mucocoele in floor of mouth, typically from sublingual gland.
- Usually develop lateral to midline, unlike dermoid cysts.
- A "plunging ranula" extends through fascial planes, usually posterior to mylohyoid muscle into the neck, and can present as a cervical mass.

Management:

- Simple marsupialisation has a 61 to 89% recurrence rate.
- This is reduced to 10–45% if marsupialisation and packing is performed.
- For infants less than 3 months presenting with a ranula, aspiration is recommended.
- Sclerosant may also be injected, which is typically a mixture of low virulence strain of Streptococcus pyogenes incubated with benzylpenicillin.
- Removal of sublingual gland results in a 1% recurrence rate.

  The ranula should be aspirated 3–5 days prior to surgery (decompressing the ranula to a third/half of its size), and a longitudinal incision made into the ranula. The cyst should then be dissected around and freed. At the point of contact with the sublingual gland, a cutting diathermy is used to remove the portion of the gland to which the ranula is attached.

Sublingual Gland Removal

- Longitudinal incision is made through mucosa in a posterior-anterior direction midway between plica sublingualis and lingual aspect of the mandible. Incision

extends from second molar to the canine. Lateral and anterior sides of sublingual gland should be dissected, with careful protection of Wharton's duct. Lingual nerve should be identified as it crosses underneath the duct, and dissected from gland.

- Complications of procedure: injury to the duct (in 1–2% of cases), bleeding (1–2%), infection (1–2%) and lingual nerve damage (2–12%).
- For a plunging ranula, with sufficient aspiration and removal of sublingual gland should remove the source.

Hypersalivation

- Hypersalivation affecting outer third of oral cavity can impact seal of the oral commissure and alter anatomy.
- Management: medical and surgical techniques.
- Drugs: glycopyrrolate, and hyoscine patches are used to manage excess salivation.
- US guided injection of botulinum toxin to the submandibular glands have also been used.

Treatment of excessive salivary flow in patients with cerebral palsy can be managed by a combination of ductal repositioning and glandular excision.

Salivary Gland Neoplasms

- Neoplasms that arise in salivary glands are relatively rare, with a diverse range of histological and clinical behaviours.
- Most commonly arise in the parotid gland, followed by the submandibular and minor salivary glands.
- Parotid up to 85% of tumours 25–32% Malignant.
- Submandibular gland 8% of tumours 37–45% Malignant.
- Sublingual gland <1–5% of tumours 70–90% Malignant.
- Minor salivary gland 22% of tumours 45–82% Malignant.
- Malignant neoplasms are more common in submandibular and minor salivary glands.
- Salivary gland neoplasms are uncommon in children. Greater proportion (20–30%) of malignant neoplasms presenting in children are typically low grade mucoepidermoid carcinomas.
- Carcinomas are further classified as high grade, low grade or mixed depending on their histological picture.
- It is recognised that clinical behaviour rather than histology of the tumours provides a better treatment guide.
- Therefore, it is important to consider clinical factors in addition to histology and grade when planning treatment.
- Clinical examination commonly reveals a small palpable lump. Symptoms may present depending on the nature of the neoplasm.
- Signs and symptoms such as pain, rapid growth, fixity to surrounding structures, nerve involvement and cervical lymphadenopathy suggest malignancy.

- Ultrasound (US) + FNAC primary diagnostic tool for salivary gland neoplasms. Benign disease can be identified in 90% of cases by using these modalities. If the primary investigations are not diagnostic, it is recommended to either repeat US guided FNAC or core biopsy.
- CT and MRI will delineate size, position and relationship of neoplasm in relation to local structures.

Management:

Submandibular

- Benign neoplasms of submandibular gland are typically managed with supra-capsular excision. Malignant neoplasms have poor survival as pre-treatment misdiagnosis is common.
- Some favour wider excision margins for adenoid cystic carcinomas, as they are difficult to excise with clear margins.
- For N0 tumours, a level 1 to 2a neck dissection is recommended, and for high grade tumours a level 1 to 3 neck dissection is recommended.
- High grade mucoepidermoid carcinomas, squamous cell carcinomas, and carcinomas ex pleomorphic adenomas have a high risk of metastasis.

Pleomorphic Adenoma

- Pleomorphic adenomas are mixed tumours of mixed of epithelial and myoepithelial cells. They are the most common tumours of the minor and major salivary glands.
- They present as a painless, slow growing mass, becoming less mobile as they increase in size.
- Diagnostic investigations include US + FNAC. MRI to determine extent of involvement of local structures.
- There is a wide spectrum of histopathological features. Tumour can extend into or through the capsule
  - Ductal epithelial component—ducts, tubules, solid sheets.
  - Myoepithelial component: myxoid, chondroid.

Management

Superficial parotidectomy, partial parotidectomy, extracapsular dissection.

Extracapsular Dissection

- Theory. Facial nerve is deep margin, so why not close margins in other areas.
- Reduced operating time, less transient facial nerve damage, less cosmetic deformity.

Prognosis: 95% cure rate. Incomplete removal can result in recurrence 4.6 yrs later, with a rate of 4%

- Why recurrence:
  - Spillage—wash with water—d/w MDT-observation vs. radiotherapy.
  - Lack capsule (pseudocyst),
  - Violation of capsule.

  Recurrent pleomorphic adenoma—usually multifocal. Consider radiotherapy. Localised removal.
  Why treat: Can undergo malignant transformation and growth if left (1% per year).

Warthin's Tumour

Warthin's Tumours are the second most common parotid tumours.

- Benign proliferation of entrapped salivary gland cells in lymphoid elements.
- Strong association with smoking.
- Bilateral presentation 4–6%.
- Histology usually shows ductal epithelium and lymphoid stroma. May have germinal centres.
- 5–10% recurrence after removal.
- Malignant transformation extremely rare.

Basal Cell Adenoma

- Present as small painless nodules, usually in the parotid 70% or upper lip 20%.
- Peak age group 60 – 70.
- 1–2% of salivary tumours.
- Can transform to basal cell adenocarcinoma.

Myoepithelioma

- Present similar to pleomorphic adenoma.
- Predilection for parotids and palate.
- 1% of salivary tumours.
- Closely related to pleomorphic adenoma, however:
  - Do not show ductal differentiation.

Do exhibit sharp demarcation of cellular cords from myxoid vascular stroma.

Malignant Salivary Tumours

Primary Tumour (PT)

- **pTX:** Primary tumour cannot be assessed.
- **pT0:** No evidence of primary tumour.
- **pTis:** Carcinoma in situ.

- **pT1:** Tumour ≤2 cm without extraparenchymal extension.
- **pT2:** Tumour >2 cm but ≤4 cm without extraparenchymal extension.
- **pT3:** Tumour >4 cm or tumour with extraparenchymal extension.
- **pT4a:** Tumour of any size invading skin, mandible, ear canal or facial nerve.
- **pT4b:** Tumour of any size invading skull base or pterygoid plates or encasing carotid artery.

Notes:

- Extraparenchymal extension is clinical or macroscopic evidence of invasion of soft tissues.
- Microscopic evidence alone does not constitute extraparenchymal extension for classification purposes.

Regional Lymph Nodes (PN)

- **pNX:** Regional lymph nodes cannot be assessed.
- **pN0:** No regional lymph node metastasis.
- **pN1:** Metastasis in a single ipsilateral lymph node ≤3 cm without extranodal extension.
- **pN2a:** Single ipsilateral lymph node metastasis ≤3 cm with extranodal extension or >3 cm but ≤6 cm without extranodal extension.
- **pN2b:** Metastasis in multiple ipsilateral lymph nodes ≤6 cm without extranodal extension.
- **pN2c:** Metastasis in bilateral or contralateral lymph node(s) ≤6 cm without extranodal extension.
- **pN3a:** Metastasis in a lymph node >6 cm without extranodal extension.
- **pN3b:** Extranodal extension in a single ipsilateral lymph node >3 cm or single contralateral node or multiple nodes with metastases, any with extranodal extension.

Acinic Cell Carcinoma

- Acinic cell tumours account for 3% of parotid tumours and are typically low grade.
- Present as a slow growing painful mass, with a variable history. They can be multifocal and can be bilateral in 3% of patients.
- 5 year survival rate of 90%-, and 20 year survival rate of 55%, with lymph node metastasis in 10% of patients.
- Management: Total conservative parotidectomy (preservation of the non-involved nerve). An elective neck dissection is not indicated.

Mucoepidermoid Carcinoma

- Mucoepidermoid tumours are epithelial, mucin producing tumours.
- Commonest salivary gland malignancy (4–9%), and 90% arise within the parotid gland.
- Commonest malignant salivary gland tumour in children, typically low-grade.
- Patients aged 30 to 50 years old, and almost always arise in superficial lobe.

- Histologically divided into:
  - Low Grade (5 year survival 86%).
  - Intermediate Grade.
  - High Grade (5 year survival 22%).
- 40% chance of lymph node metastasis in intermediate or high grade mucoepidermoid tumours. These are treated with adjuvant radiotherapy and/or neck dissection.

Adenoid Cystic Carcinoma

- 20–25% of all malignant salivary gland neoplasms.
- Slow growing. 2–6% occur arise in parotid gland, 15% arise in the submandibular gland.
- High-grade salivary malignancy, they have propensity for perineural invasion.
- They have variable histopathological appearances
  - Cribriform (pseudocystic spaces—with some reports of a better prognosis).
  - Tubular (small islands of cells).
  - Solid (islands of cells with small dense nuclei).
- Adenoid Cystic Carcinomas have a high rate of morbidity, due to local or distant recurrence (especially to the lungs).
- 20% result in pulmonary metastasis.

Management

- Excision must be made with the widest surgical margin (3 cm) and preservation of uninvolved nerves.
- Post-operative radiotherapy.
- For N0 tumours, neck dissection is not recommended as metastasis to the neck is rare.

Adenocarcinoma

- Adenocarcinomas are uncommon with 90% occurring in parotid gland.
- 5 year survival 75%.
- Histologically divided into the following:
  - Low Grade (papillary or mucinous).
  - High Grade.
- 40% chance of distant metastasis, with a 5 year survival of 19%.

Management

- Adenocarcinomas should be removed with wide local excision, and an elective neck dissection performed. Adjuvant post-operative radiotherapy for high grade adenocarcinomas should be considered.

Malignant Mixed Tumours 'Carcinoma ex Pleomorphic Adenoma'

- These are typically tumours with a history of multiple recurrences, managed with surgery and radiotherapy. They tend to be high grade with increase incidence.
- Management: Radical resection with adjuvant radiotherapy.

Squamous Cell Carcinoma

- Typically present in elderly populations (over the age of 70 years old) and have a very poor prognosis.
- Management is the same as for high grade mucoepidermoid carcinomas.

Parotid Gland

- The parotid gland is an irregular wedge-shaped gland located in the parotid compartment anterior and inferior to the auricle and overlies the masseter muscle. It wraps around the posterior edge of the ramus and extends posteriorly to the external auditory canal and mastoid tip. The parotid fascia incompletely covers the gland. Accessory parotid glands are found in 20% of patients.
- The parotid duct lies parallel to the zygomatic arch, approximately 1–1.5 cm below. Duct travels superficial to masseter muscle and turns at a right angle to pierce buccinator muscle. It empties into the oral cavity, opposite the second maxillary molar.
- Parotid gland receives both sensory and autonomic innervation. There are parasympathetic efferents from inferior salivary nucleus via the ganglion of the auriculotemporal nerve. There is sympathetic nerve supply from the superior ganglion.
- Blood supply of the parotid gland: branches of the external carotid artery, and the primary transverse facial artery. Venous drainage: via retromandibular vein.
- The paraparotid and infraparotid lymph nodes are located superficial to parotid capsule and drain the temporal region, scalp, auricle, ear and posterior nasopharynx.

Superficial Parotidectomy

Access is gained via the modified Blair incision, and a skin flap raised at the level of the parotid capsule. Greater auricular nerve is identified and posterior branch preserved. Parotid tail is freed from sternocleidomastoid muscle, and posterior parotid freed from external acoustic meatus.
Main trunk of the facial nerve is identified using landmarks:

1. Tragal pointer (TP): nerve lies approximately 1.0–1.5 cm deep and anterior/inferior to tip of TP.
2. Posterior belly of digastric: nerve lies approximately 1.0 cm deep to the medial attachment of the muscle to the digastric groove of the mastoid bone.
3. Tympanomastoid fissure: nerve lies 6–8 mm distal to the end point of fissure.
4. Retrograde approach, involves finding buccal/cervical branch and tracing backwards.

Complications

- Immediate complications include button hole penetration of skin, tumour spillage, damage to the facial and greater auricular nerve, haematoma and sialocele.
- Late complications include hypertrophic or keloid scar formation, neuroma, and recurrence and Frey's syndrome.

Frey's Syndrome

- Localised gustatory sweating -parasympathetic (to parotid) and sympathetic (sweat glands) -both cholinergic). Misguided during recovery. Usually 6 months later—not all need treatment.
- Starch iodine test—treatment—Botulinum toxin.
- Prevention: extracapsular dissection, thinner flap, temporoparietal fascia.

19.2 Facial Nerve

UMN/ LMN

1. UMN—forehead sparing.
2. Frontal region—bilateral.

House Brackmann Scale

3. I: Normal.
4. II: Slight weakness—complete eye closure.
5. III: Obvious weakness—complete eye closure, may not able to life eyebrow.
6. IV: Obvious weakness—incomplete eye closure, unable to life eyebrows,
7. V: Motion barely perceptible—slight movement.
8. VI: No movement, loss tone, no contraction.

Sunnybrook Facial Grading System.
Comprehensive scale for evaluation of facial palsy.
Consists 13 points—3 resting (total multiplied by 5), 5 voluntary movement (total multiplied by 5), and 5 synkinesis.
Composite score (range 0–100) = Voluntary movement score (−) Resting symmetry score (−) Synkinesis score.

Facial Nerve Palsy

- Capital Muscles used in facial expression—cannot fully restore symmetry. Facial nerve only cranial nerve spontaneously produces impulses during facial expression (Fig. 19.3).

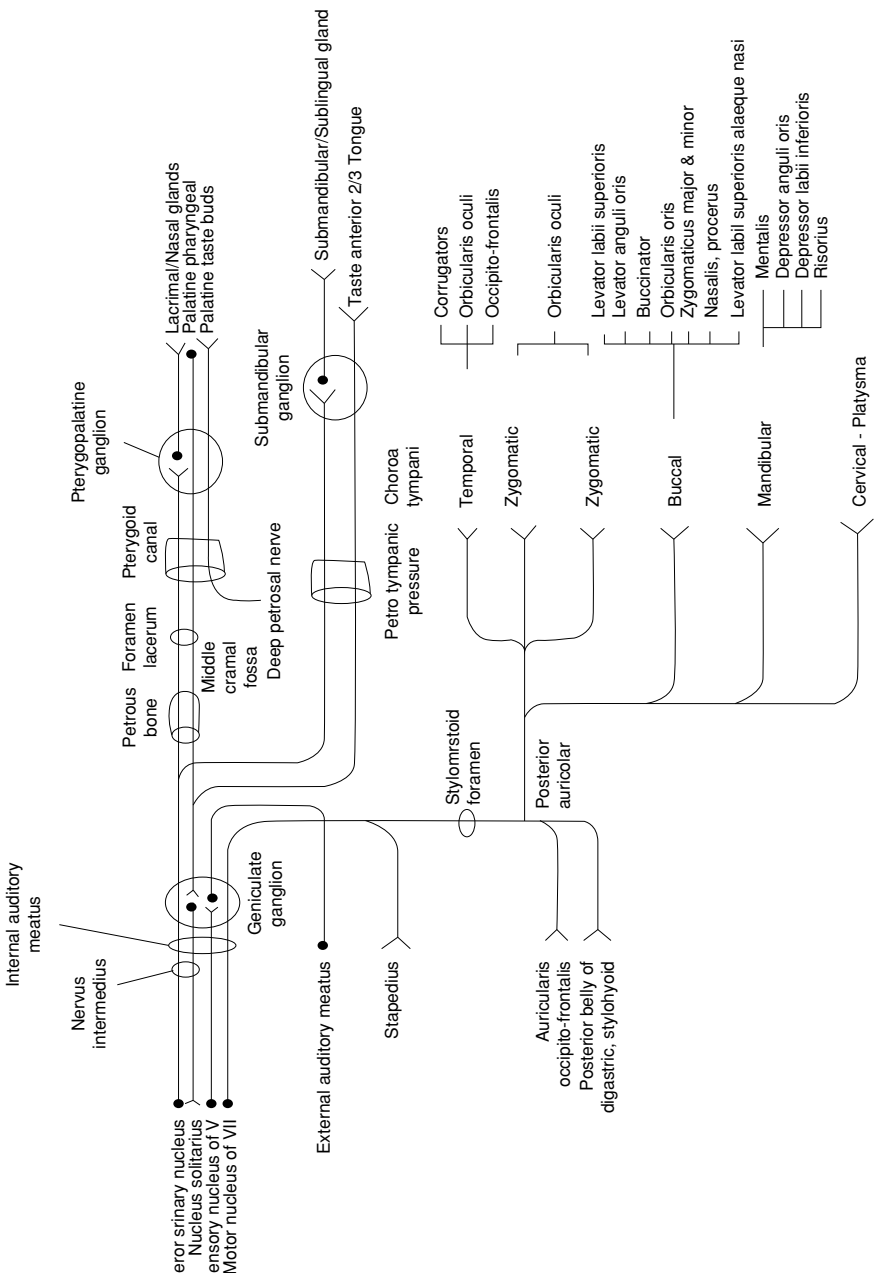


Fig. 19.3 Facial nerve

Table 19.2 Facial nerve differential diagnosis

History	Exam	Differential diagnosis
Recent Trauma	Head Injury	Fracture of temporal bone
Rapid onset of weakness	Forehead spared (neurological)	CVA
Rapid onset weakness (isolated)	Forehead affected	Bell's Palsy
Otalgia	Vesicles in ear	Ramsay's Hunt syndrome
Gradual onset	Abnormal neurology	MS/ MND
Gradual onset facial pain	Parotid lump	Carcinoma
Hearing loss	Sensory ataxia	CPA angle tumour
Ear disease	Conductive hearing loss	Cholesteatoma
Congenital		Mobius Syndrome
Bilateral LMN		Lyme disease (Borrelia Bergdorferi) Guillain-Barré Syndrome

Aetiology (Table 19.2).

(1) Central

- Vascular abnormality, tumours, trauma, congenital (Möbius).

(2) Temporal

- Bacterial/ viral, trauma, tumour, cholesteatoma, iatrogenic.

(3) Parotid

- Tumour, trauma, iatrogenic, intraneural spread.

Facial Nerve-Marginal Mandibular Branch

- Location to place skin incision to avoid marginal mandibular branch of facial nerve
  - Submandibular/Risdon approach.
  - Exact location differs.
    (1) Dingman and Grabb
      - 192 patients: branch below inferior border, posterior to where nerve crosses facial artery.
      - Cadaveric: anterior to that point—facial nerve above border 100% of patients.

    (2) Ziarah and Atkinson
      - 53%: Below mandible posterior to facial vessels.

  - 6%: Nerve continues to be below the inferior border anterior to facial vessels.

(3) 1.5–2.0 cm—Below inferior border -standard incision made.

Problems

- Ear: Hyperacusis.
- Brow: Ptosis/ failure to elevate/ lacrimal gland ptosis.
- Eyes: Lagophthalmos/ failure to close/ loss of corneal reflection/ loss tearing/ epiphora/ ectropion.
- Upper lip: Failure to elevate/ movement, food entrapment in buccal sulcus/ bite lips/ enlarged lips.
- Lower lip: Dribbles/ failure to purse/ incompetent lip.
- Tongue: No taste.

Clinical Examination

- History and examination.
- Observe for facial posture at rest.
- Hearing, taste, submandibular flow.

Investigations

- MRI/ CT/ plain films/neurophysiology.

Goals—Reconstruction

- Normal symmetry at rest.
- Normal symmetry with voluntary movement.
- Oral sphincter control.
- Expression of emotions.

Management

- Repair divided into stages.
- Acute facial nerve injury: need healthy nerve + healthy muscles
  - Direct repair.
  - Nerve grafting.
  - Cross facial nerve grafting.
  - Muscle rotation.
  - Nerve transfer.

Chronic:

- Cross facial nerve grafting to innervate free muscles.
- Procedures: Static and Dynamic.

Static

- Forehead
  - Brow lift.
- Eye
  - Artificial tears, taping, tarsorrhaphy, wedge excision.
  - Lateral canthopexy.
  - Eyelid closures with devices (weights/magnets).
- Upper face/lip
  - Facelift -SMAS.
- Lower lip
  - Wedge excision.
  - Facial slings.
  - Orbicularis oris advancement.
- Opposite face: Selective balancing neurectomy/ myectomy.

Dynamic

- Nerve transfer
  - Spinal accessory, hypoglossal.
- Muscles transfer
  - Temporalis: Fold over—arch sutures to lip, strips from coronoid to lip.
  - Masseter.
  - SCM: Detached from clavicle transposed to cover the mouth.
- Contralateral facial nerve = preferred choice.

Cross Facial Nerve Graft

Stage 1

- Sural nerve donor: Posterior lateral aspect leg
  - 2 cm posterior ankle—horizontal 1.2 cm incision.
  - 2nd: 4–5 cm proximal to first and slightly more posterior.
  - 3rd: 4–5 cm proximal.
- Adequate length = 10–12 cm.
- Facial nerve prep
  - Sural nerve grafted—2 branches of facial nerve from opposite side.
  - Buccal branch 1–2 branches cut and attached to sural—cross innervation.
- Long incision

  - Anterior parotid—nerve stimulation.
  - Find few branches—get present function.
  - Redundancy of innervation, to avoid any loss of function on normal side.
- Subcutaneous tunnel
  - Upper lip to opposite side.
  - Anastomosis between selected facial and sural—suture with microscope.
  - On weak side—nerve, fixed to parotid fossa/ can be identified when return 9–15 months later.

Stage 2

Muscle Transfer

- Pre-operative
  - Desired position of modiolus at rest, by pulling/checking intra-operatively.
  - Vector and extent of movement.
  - Required length muscle—modiolus to zygomatic bony prominence.
- Insertion of muscle graft
  - Gracilis/Extensor digitorum longus/Latissimus dorsi/Pectoralis minor.
  - Placed/divided into 2–3 portions—depending on tumour or fracture position.
  - Muscle length adapted with slight tension.
- Post-operative
  - 4 weeks, transcutaneous electrical stimulation of grafted muscle to avoid atrophy during new regeneration.

Labbé Procedure

- Lengthening temporalis myoplastysymmetry at rest and smiling.
- Coordinated/ spontaneous/ symmetrical smile.
- Insertion points of temporalis—nasolabial fold/ upper/ oral commissure.

Parapharyngeal Space

Clinical Presentation

- Usually asymptomatic.
- Unilateral Eustachian tube dysfunction-conductive deafness.
- Displacement of tonsils.
- Neurological
  - Vagus-vocal cord paralysis.
  - Hypoglossal.
  - Accessory.
  - Glossopharyngeal.
  - Horner's syndrome—miosis, anhidrosis, ptosis (Fig. 19.4).

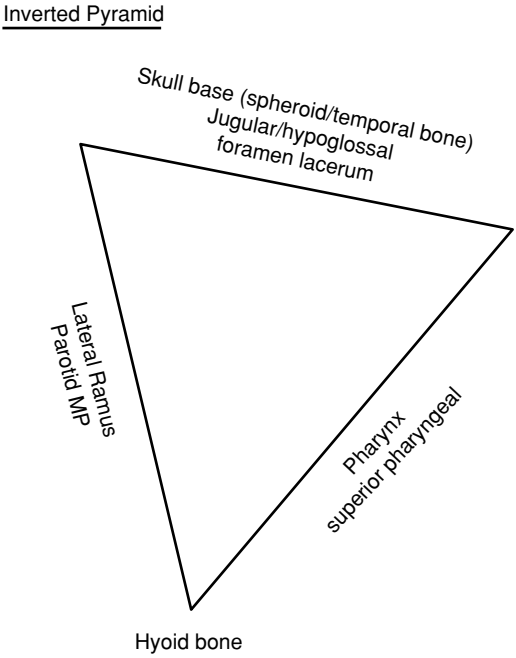


Fig. 19.4 Parapharyngeal space borders

Differential Diagnosis:

- Tumours (70–80% benign)
  - Salivary gland,
  - Neurogenic—Schwannoma, paraganglioma, neurofibroma.
  - Metastatic (SCC).
  - Lymphoreticular tumours.
- Lipomas.
- Haemangiomas.
- Aneurysm.
- Amyloid/Dermoid (Fig. 19.5).

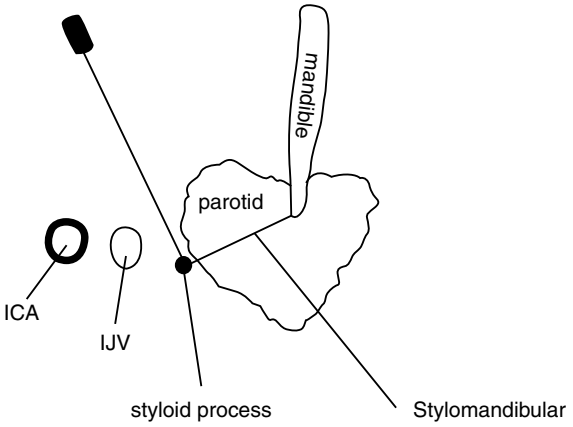


Fig. 19.5 Parapharyngeal space

Chapter 20
Oral Pathology

Abstract A summary of lesions of the jaws, their assessment and management.

Key Points

- Identification of lesions of the jaw.
- Odontogenic keratocyst.
- Dentigerous cyst.
- Eruption cyst.
- Radicular cyst.
- Gingival cyst.
- Nasopalatine cyst.
- Stafne bone cyst.
- Aneurysmal bone cyst.
- Solitary cyst.
- Ameloblastoma.
- Calcifying epithelial odontogenic tumour.
- Squamous odontogenic tumour.
- Odontoameloblastoma.
- Calcifying cystic odontogenic tumour.
- Odontogenic fibroma.
- Cementoblastoma.
- Clear cell odontogenic carcinoma.
- Ameloblastic fibrosarcoma.
- Fibrous dysplasia.
- Hyperparathyroidism.
- Paget's Disease.
- Ossifying fibroma.
- Cemento-osseous dysplasia.
- Osteogenesis imperfecta.
- Osteoma.
- Gardner Syndrome.
- Osteoblastoma.

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- Osteosarcoma.
- Chondroma.
- Chondrosarcoma.
- Ewing sarcoma.
- Multiple myeloma.
- Central giant cell granuloma.
- Langerhans cell histiocytosis.

20.1 Classic Appearance of Jaw Lesions

Multilocular Radiolucency—Posterior Body/Ramus

- Odontogenic keratocyst.
- Ameloblastoma.
- Odontogenic myxoma.
- Odontogenic fibroma.
- Giant cell lesions: central giant cell granuloma.
- Brown tumour of hyperparathyroidism.
- Cherubism.
- Aneurysmal bone cyst.

Radiopaque

- Chronic apical infection.
- Odontoma.
- Osteoma.
- Salivary stone.

Mixed

- Osteomyelitis.
- Calcifying epithelial odontogenic tumour.
- Cemento-osseus dysplasia.
- Osteosarcoma.
- Cementoblastoma: radiopaque with radiolucent margins.

Ground Glass Appearance

- Fibrous dysplasia.
- Hyperparathyroidism.
- Paget's disease of bone.

Associated With Crown of Tooth

- Dentigerous cyst.
- Odontogenic keratocyst.
- Ameloblastoma.
- Ameloblastic fibroma.

- Adenomatoid odontogenic tumour.
- Calcifying cystic odontogenic tumour.
- Odontogenic myxoma.

Differential Diagnosis of Jaw Swellings

- Cysts.
- Tumours: Benign (odontogenic/non-odontogenic).
- Giant cell lesions: Central giant cell/aneurysmal bone cyst/Brown tumour of hyperparathyroidism, cherubism.
- Fibro-osseous lesions.
- Vascular lesions.

Multilocular With Septi = Soap Bubble Appearance

- Ameloblastoma.
- Odontogenic keratocyst.
- Odontogenic myxoma.
- Aneurysmal bone cyst.

Cysts

(1) Epithelial

- Odontogenic
  - Developmental

    Gingival/odontogenic keratocyst/ Lateral periodontal/ dentigerous/ eruption.
- Inflammatory
  - Radicular/residual/paradental.
- Non-odontogenic

  Nasopalatine/nasolabial.

(2) Pseudocysts

- Stafne bone cavity, aneurysmal bone cyst, solitary bone cyst (traumatic).

(3) Cysts with soft tissues

- Dermoid, epidermoid, branchial, thyroglossal, salivary gland.
- Pathological cavities lined by epithelium and contain fluid/semi-fluid/gaseous content. Most cysts arise from odontogenic epithelium and grow by various mechanisms
  - Inflammation: Epithelial proliferation.
  - Bone resorption: Pressure/prostaglandins.
  - Alleviations in cystic osmotic pressure.
  - Genetic: PTCH gene—odontogenic keratocyst.

Odontogenic Keratocyst

- Aggressive behaviour with high recurrence rate.
- 11% of jaw cysts.

Aetiology

- 60% arise dental lamina rests of Serres.
- 40% arise reduced reduced enamel epithelium of dental follicle.
- Genetic association in odontogenic keratocyst: PTCH gene.

Clinical

- Any age: 5–90 years.
- Peak 20–40 years.
- M > F.
- 5% patients have multiple cysts (Gorlin-Goltz Syndrome).
- Mandible > Maxilla: 50% in 3rd molar region.
- Usually asymptomatic: can cause pain/swelling/displacement teeth/delayed tooth eruption/tooth mobilising, can cause bony expansion.
- Does not usually affect IAN sensation: can be displaced, slow growing in A-P direction.

Radiographic

- Well-defined radiolucency: unilocular/multilocular, can be associated with unerupted tooth (25–40%).
- May cause tooth displacement and root resorption.
- 40% in dentigerous position.

Investigation

- Aspiration: protein < 4 g/100 ml, thick yellow cheesy material (keratin)—cytology.
- Incisional biopsy: lining by ortho-keratinised (10%)/parakeratised (83%): epithelium some have both.
- High mitotic activity: growth neoplastic with invasion of medulla and not by bony expansion.
- Stratified squamous tall cuboidal/columnar 5–10 cell layers thick.
- Corrugated laminal surface ± daughter cysts (NB: presence keratin not enough for diagnosis).

Management

- Controversial: recurrence rate high (5–62%)—increase in Gorlin-Goltz Syndrome and mandibular lesions.
- Why?
  - Incomplete removal of original cyst lining.
  - Growth of new odontogenic keratocyst from residual odontogenic cell rests.
  - Development of unrelated odontogenic keratocyst in adjacent area.

- Epithelial islands/microcysts reported in overlying attached mucosa in all cases of recurrent odontogenic keratocyst.

Options

- Simple enucleation
  - Leads to high recurrence rate: not generally recommended.
- Enucleation with adjunctive treatment
  - Curettage: remove overlying mucosa.
  - Peripheral ostectectomy: ± methylene blue (guide).
  - Cryotherapy (using KY): can cause anaesthesia IAN (recovers in 3 months; recurrence 3%).
  - Carnoy's solution 5 min—mean bone penetration 1.5 mm in 5 min; < 10% recurrence

    Ethanol (absolute 95%) = 30 cc (6 parts).
    Chloroform = 4 g ferric chloride (3 parts).
    Glacial acetic acid = 10 cc (1 part).
    Ferric chloride 1 g.
- Marsupialisation
  - Formation of pouch and frequent irrigations or definitive procedure.
  - High degree co-operation, allow squamous metaplasia of cystic wall—subsequent removal easier.
- Decompression followed by enucleation ± adjunct
  - Nasopharyngeral tube sutured to keep open = 1.5 cm.
  - Halts expansion of cyst: appositional growth of bone occurs.
  - Issues

    Post-operative care = 1 year.
    Do not get true assessment of cystic lining—may miss ameloblastoma.
- Resection
  - Segmental/marginal (5 mm margin)—absolute indications

    Recurrent/extensive

    (a) Involvement of condyle.
    (b) Pathological fracture.
  - In areas of cortical perforation: reports of OKC found in soft tissues reported = remove.
- Follow-up
  - 6 monthly for 5 years, then annually to detect recurrence.

Basal Cell Naevus Syndrome (Gorlin-Goltz Syndrome)

- PTCH gene chromosome 9.
- Rare.
- Autosomal dominate.
- 40% new mutations.
- Multiple OKC (increased risk of recurrence) /BCC's (aggressive, poor response to radiotherapy).

Clinical

- Cutaneous: Palmar/plantar pitting, multiple milia/dermal carcinoma.
- Bone: Bifid ribs/vertebral and metacarpal abnormalities/kyphosis/sclerosis.
- Facial dysmophogenesis: Broad nasal bridge/hypertelorism/mandibular prognathism/frontal bossing.
- Neurological: Calcification falx cerebri/medulloblastoma/bridging of sella turcica/cataracts.

Investigations

- Thorough skin examination.
- Biopsy.
- OPG/skull/CXR/spine.

Management

- Dermatological referral.
- Genetic counselling.
- Conservative management:
  - Preservation of alveolus/teeth.
  - Often enucleation.
  - Remove 3rd molar.

Literature

- Cochrane 2010: no RCT: no conclusion reached.
- Blanas 2000: Systematic review
  - Enucleation 14–56% recurrence.
  - Enucleation + adjunct 1–8.7% recurrence (Carnoy's solution/decompression before enucleation).
  - Resection 0%.

20.2 Dentigerous Cyst/Eruption Cyst

- Developmental cyst that is attached to the cemento-enamel junction (CEJ) and encloses the crown of an unerupted tooth arising from reduced enamel epithelium (REE). (If partially erupted through bone = eruption cyst.).

Clinical

- 2nd most common jaw cyst.
- Age 10–30.
- M = F.
- 4% of unerupted teeth will develop dentigerous cyst.
- Mandible 8s > Maxilla 8s > Maxilla 3s.
- Generally asymptomatic, but can cause delayed eruption, bony expansion, tooth displacement/root resorption (rare), pain.
- Bilateral cysts with basal cell naevus syndrome/cleidocranial dysplasia.

Radiographs

- Well-defined, unilocular, radiolucent lesion.
- Surrounding crown—unerupted tooth.
- >5 mm = dentigerous cyst, tooth be may displaced.

Differential Diagnosis of Pericoronal Radiolucency

- Odontogenic keratocyst, ameloblastoma, ameloblastic fibroma, adenomatoid odontogenic tumour, calcifying cystic odontogenic tumour.

Investigations

- Aspiration—straw coloured fluid.
- If no fluids = solid lesion—incisional biopsy (could be ameloblastoma/tumour).
- ±CBCT/CT/MRI.

Histopathology

- Mucous cells.
- Stratified squamous epithelial lining (4–6 layers).
- Cilliated cells with sebaceous cells in epithelium.

Management

- Enucleation with removal of tooth.
- Marsupialisation (healing slow) = indication:
  - Allow tooth to spontaneously erupt or guided into functional position.
  - Risk of damage to IAN/developing tooth if enucleation.
  - To decompress a large cyst—reduces extent of surgery at a later date.

Prognosis

- Enucleation: curative.
- Transformation of epithelial lining of cyst into ameloblastoma is possible.

20.3 Radicular Cyst

- Periapical cyst: secondary to pulp necrosis.
- Develops from periapical granuloma at the apex of non-vital tooth.
- Stimulation of the epithelial cell rests of Malassez occurs.

Clinical

- Most common jaw cyst: peaks 4th–6th decade (often asymptomatic).
- Well-defined radioluncency: unilocular <1 cm—residual cyst if left after tooth removal.

Histopathlogy

- Straw coloured fluid containing cholesterol crystals protein >5 g/dl.
- Lined with non-keratinised stratified squamous epithelium.
- Rushton bodies ± dystrophic calcification and cholesterol clefts.

Management

- RCT small <1 cm/apical surgery.
- Extraction + curettage.
- Marsupialisation if large.
- Residual cyst: enucleation/marsupialisation (large).

Gingival Cyst Often in Adult/Lateral Periodontal Cyst

- Lateral periodontal cyst: Non-keratinised developmental cyst occurring lateral to the root of a tooth. Arises from dental lamina remnants (if multilociular, also known as botryoid odontogenic cysts).

Clinical

- Uncommon, any age, M = F.
- Ginigival cyst: Dome shaped fluctuant swelling within. Slightly inferior to the interdental papilla, may cause superficial erosion of cortical bone.
- Usually detected on radiographs as well-defined, round radiolucency along the lateral surface of a vital tooth (canine/premolar—90% buccal/labial).

Histopathology

- Lined with thin + non-keratinised epithelium with characteristics nodules of squamous cells on laminal aspect.

Management

- Enucleation and curettage.

Prognosis

- Only the multilocular variant has been documented to recur.

Paradental

- Original description: associated with impacted/partially erupted 3rd molar, attached to CEJ but not enclosing crown.
- Caused by inflammation in a periodontal pocket resulting in stimulation of the epithelial rest cells of Malassez or REE (reduced enamel epithelium).

20.4 Nasopalatine Duct Cyst

- Non-odontogenic cyst formed from proliferation of the epithelial remnants of nasopalatine duct.

Clinical

- Peak incidence 4th–5th decade.
- Female:Male 4:1.
- Present with nasal obstruction/deformity.
- Smooth fluctuant soft tissue masses between upper lip and nasal aperture with obliteration of nasolabial fold and elevation of nasal alar.
- Histopathology: Pseudostratified columnar epithelium/stratified squamous epithelium/goblet cells.
- Management: Excise through incision in buccal sulcus.
- Prognosis: Recurrence unlikely.

20.5 Stafne Bone Cyst

- Not true cyst, indentation of the posterior lingual mandible due to salivary tissues (developmental inclusion).

Radiograph

- Asymptomatic—incidental finding.
- Radioluncency beneath level of IAN.

Management

- No treatment—presence of salivary tissues confirmed via submandibular gland sialography.

20.6 Aneurysmal Bone Cyst

- Expansive, osteolytic pseudocyst of unknown aetiology.

Clinical

- Typically, < age 30; M = F.
- Mandible > Maxilla 2:1.
- Posterior > anterior.
- Varies from small localised, to aggressive destructive lesion invading soft tissues.
- Pain common >50% firm non-pulsatile swelling that may rapidly enlarge.

Radiograph

- Multilocular radiolucency with slightly irregular margins ± tooth displacement/resorption.
- Soap bubble appearance.
- Histopathology: Fibrous CT stroma—variable number of giant cells with with sinusoidal blood spaces.
- Management: Excision + curettage
  - Recurrence is common = enucleation and cryotherapy/resection.

20.7 Solitary (Traumatic) Cyst

- Empty cavity with no cyst lining.
- Incidental finding on radiograph.
- Found above IAN canal in body of mandible.
- Unknown osseous assertive necrosis after trauma (Harnet 2008).
- Scalloped border passing up between roots—commonest in young men.
- Management: Enucleation and establishment bleeding into lesion to encourage healing).

Fissural Cyst

- Arises from embryonic junctional epithelium at sites of embryological fusion.
- Rare in mouth—nasopalatine cyst (heart shaped).
- Same as nasopalatine/nasolabial (soft tissues).

20.8 Odontogenic Tumours

Classifications

A. Benign

(1) Odontogenic epithelium

- Ameloblastoma/calcifying epithelial odontogenic tumour (CEOT)/ squamous odontogenic tumour (SOT)/OKC/ adenomatoid odontogenic tumour (AOT)/ odontogenic keratocyst.

(2) Odontogenic epithelium + odontogenic mesenchyme

- Ameloblastic fibroma/ fibro-odontoma/ odontoblastoma/ odontomes/ calcifying cystic odontogenic tumour (CCOT)/dentinogenic ghost cell tumour (DGCT).

(3) Odontogenic mesenchyme + odontogenic epithelium

- Cementoblastoma/odontogenic fibroma/odontogenic myxoma.

B. Malignant

(1) Odontogenic carcinomas

- Intra-osseous SCC.
- Metastasising ameloblastoma/ameloblastic carcinoma.
- Ghost cell/clear cell odontogenic carcinoma.

(2) Odontogenic sarcomas

- Ameloblastic fibrosarcoma//fibroodontosarcoma.

- Only 4 benign tumours need possible resection as risk of recurrence high
  - Ameloblastoma/odontoameloblastoma, myxoma, calcifying epithelial odontogenic tumour.
  - Others treat with enucleation and curettage unless biologically aggressive tumour.

20.9 Ameloblastoma

- Benign, locally aggressive neoplasm consisting of odontogenic epithelium lying in fibrous stroma.
- Several subtypes: Affect recurrence.
- Probably arises from odontogenic epithelium (enamel organ, odontogenic cell rests of Malassez and Serres, reduced enamel epithelium).

Clinical

- Peak age 20–35 years.
- 75% in mandible.
- Common in men and Africans.
- Asymptomatic until swelling becomes obstructive.
- Tooth displacement/malocclusion.
- Nerve sensation not affected despite displacement.

Classification

- Peripheral/extra-osseous: Occurs in soft tissues.
- Solid/multicystic: Multilocular/root resorption/aggressive.
- Unicystic: Intraluminal/luminal/mural = less aggressive expands into surrounding bone.
- Desmoplastic.

Practical

- Enucleation—peripheral and unicystic (intraluminal/luminal).
- Resection—locally aggressive solid types/multicystic/desmoplatic/mural unicystic/odontoameloblastoma.
- Resection + adjuvant—malignant = metastasised ameloblastoma/ameloblastic carcinoma + fibrosarcoma + odontosarcoma).

Radiograph

- Unilocular/multilocular radiolucency in tooth bearing areas.
- Tooth displacement/resorption.

Investigation

- Histopathology: Incisional biopsy.
- Islands of epithelium surround by palisaded columnar outer layer with reverse polarity of the nuclei—central parts maybe cystic (follicular or plexiform types).
- Akerman: Unicystic = 2 histological variants

  (a) Luminal: Cystic lesion lined by ameloblastic epithelium, may have intraluminal extension with no infiltration of fibrous wall.
  (b) Mural: Cystic wall infiltrated by tumour (follicular/plexiform).

- Absence of myxoid stoma—ameloblastic fibroma.
- OPG/CT/USS.

Management

- Tumour versus patient factors.
- Surgical resection primary treatment modality.

(1) Solid/multi cystic and mural unicystic

  - Resection with 1 cm bony margin + immediate reconstruction.

- Enucleation/curettage = 90% recurrence rate. Recurrence can take 5 years, may undergo malignant transformation.

(2) Unicystic: Enucleation + curettage.
(3) Peripheral

- Proliferation of odontogenic epithelium. Firm mass from gingiva not invading bone.
- Must exclude central ameloblastoma invading out = manage with excision with 2–3 mm margins.

(4) Ameloblastic carcinoma

- Bone resorption/mobile teeth—like SCC.
- 2–3 cm margins + neck dissection (prophylactic + therapeutic).

(5) Maxillary ameloblastoma

- Local resection with 1–1.5 cm margins as can be life threatening.
- Post-operative radiotherapy.

(6) Malignant ameloblastoma

- Metastatic focus of typical ameloblastoma.
- Usually lungs (inhalation theory) ± lymphadenopathy.

Recurrent

- Related to incomplete removals—Average time relapse 5–9 years.
- Mostly to bone: salvage resection based on original radiographs.
- Important to review previous histology in case of misdiagnosis.

Prognosis

- Maxilla more porous than mandible (thin/poor anatomical barriers)—also delay in diagnosis.
- 15% mortality (Nastri 1995).
- Soft tissue invasion—poor prognosis.
- Unicystic has better prognosis.

Children

- 76.5% unicystic versus adults 15%.
- Complicated by facial growth consideration/presence of unerupted teeth
  - Unicystic

    Enucleation for intra-luminal/luminal.
    Resection for mural.

  - Multicystic/solid/recurrence = resection.

20.10 Calcifying Epithelial Odontogenic Tumour (Pindborg)

- Rare/benign/locally aggressive epithelial odontogenic tumour arising from stratum intermedium—presence of amyloid and calcifications.

Clinical

- Age 30–50; M = F.
- Mandible > maxilla 2:1—especially posterior body.
- Mostly asymptomatic, may have jaw expansion.
- Variable behaviour: benign to locally aggressive (very rarely malignant).
- Prominent clear cell features in CEOT: more aggressive.

Radiograph

- Radiolucency with scattered opacities.
- Calcified amyloid.
- Often associated with impacted teeth.

Investigations

- Incisional biopsy.
- Strands/sheets of large polygonal epithelial cells.

Management

- No encapsulated = infiltrative.
- Surgical management = case specific—location/ size/ biological activity/ histological characteristics.
- Clear cell aggregates—often increase in aggressiveness with skip lesions.
- Resection with 1–1/5 cm margins and 1 uninvolved anatomical barrier.

Prognosis

- Recurrence <20% usually follows incomplete excision.
- Peripheral CEOT <2 cm—arising from gingiva/crystal ridge/painless/no bone resorption ± focal calcification = local excision with 5 mm margins including underlying periosteum—non-aggressive.

20.11 Squamous Odontogenic Tumour

- Rare/benign/low recurrence, treat with enucleation + curettage.
- Asymptomatic, multiple lesions in 20%.
- Radiograph:
  - Well-circumscribed semi-lunar radiolucency/produces severe bone loss (similar to periodontal disease).

Management

- Enucleation + curettage + extraction involved teeth.
- Circumscribed, round/irregular islands of squamous epithelium in fibrous stroma.

Prognosis

- Can recur, some developed into new SOT.
- Ameloblastic fibroma/fibrodontoma.

Clinical Features

- Children/young adults.
- M = F.
- Mandible > maxilla; posterior > anterior.

Radiograph

- Multilocular/unilocular.
- Opaque focus if fibro-odontoma.

Management

- Enucleation.
- Ribbons and strands of odontogenic epithelium.

Prognosis

- Recurrence 15%.
- Potential malignant change = ameloblastic sarcoma.

20.12 Odontoameloblastoma

- Ameloblastoma + odontoma.
- Management and prognosis as per ameloblastoma.
- Histology: Ameloblastoma + dentine/enamel formation.

Odontomas: Hamartomatous = Epithelium + Mesenchyme

- Compound: Denticles (small/separate/tooth like structures).
- Complex: Irregular mass of hard/soft tissues.
- Composite: Mixture of compound/complex.

Clinical

- Children/adolescents.
- Maxilla > mandible.
- Incidental/delayed eruption/cyst formation/displace teeth.

Radiograph

- Radiolucent/radiopaque lesion.
- Often associated with unerupted teeth.

Management: Enucleation With No Recurrence

Odontogenic Myxoma

- Benign locally aggressive may be infiltrative/locally aggressive.

Clinical

- Wide age range: age 20–60, M = F.
- Mandible > maxilla; posterior > anterior.
- Mostly asymptomatic.
- Jaw expansion ± tooth movement/malocclusion—similar to ameloblastoma, but more infiltrative growth.
- Variable behaviour: tend to be more aggressive in children and in the maxilla.

Radiograph

- Variable, well-circumscribed/diffuse.
- Often multilocular with 'soap bubble' pattern.
- May have cortical perforation, root displacement/resorption.

Histopathology—Incisional Biopsy

- Bland spindle cells in myxoid stroma infiltrating bony islands.

Management

- Resection 1–1.5 cm bony margins 1 uninvolved anatomic barrier.
- Enucleation/curettage—recurrences likely + tumour seeding.

20.13 Calcifying Cystic Odontogenic Tumour

- Benign, cell rests of Serres (dental lamina).

Clinical

- Any age, Female > Male.
- Maxilla > mandible.
- Asymptomatic/swelling.
- Early lesions radiolucent, then beomes mixed radiolucent/radiopaque.

Management

- Enucleation: Unicystic lesion, cuboidal/columnar cells with hyper-chromatic nuclei, polarised away from BM/ghost cells.
- Prognosis: No recurrence.

20.14 Odontogenic Fibroma

- Benign mesenchymal—central/peripheral.
- Very rare, any age Male = Female/mandible = maxilla.
- Asymptomatic and expansive
  - Well demarcated unlocular/multilocular.

Management

- Enucleation + curettage—mature mass of fibrous tissues: contain few epithelial cell rests ± calcified deposits.
- Prognosis: Excellent, recurrence very uncommon.

Cementoblastoma

- Benign neoplastic proliferation of cementum derived from PDL.

Clinical

- Usually age <25 Male = Female.
- Mandible > Maxilla; posterior > anterior.
- Initially associated with root of the tooth which remains vital.
- Can cause intermittent pain + cortical expansion.

Radiograph

- Well-defined, radiopaque, root affected with surrounding radiolucent halo.

Management

- Excision + extraction of tooth.
- Dense mass of mineralised cementum material with numerous reversal lines, thin fibrous capsule, continuous with PDL.
- Prognosis: Recurrence not seen.

20.15 Clear Cell Odontogenic Carcinoma

- Rare, aggressive, malignant; arises from embryonic ectoderm of dental lamina.

Clinical

- Age 40–80 years; Female:Male 4:1.
- Mandible > maxilla 3:1; Anterior > posterior.
- Jaw expansion/pain/tooth displacement/mobility ± neurosensory changes.
- Maxillary lesions may infiltrate/invade—nose/sinus/orbit/skull base.
- May metastasise.

Radiograph

- Poorly differentiated/unilocular/multilocular radiolucency ± tooth resorption/displacement.

Investigations

- Incisional biopsy.
- CT chest/neck.

Histopathology

- Poorly circumscribed.
- Consist of clear cells with central nucleus and well-defined cell membrane.
- Form large sheets separated by fibrous septa.

Management

- Resection 1.5–2 cm margins ± post-operative radiotherapy.

Prognosis

- 40% mortality over two years.

20.16 Ameloblastic Fibrosarcoma (Malignant)

Clinical

- Occurs age 20–30 years later than ameloblastic fibroma.
- Multilocular radiolucent lesion causing jaw expansion.
- Capable of neural invasion = paraesthesia.
- Slow growth, little tendency.

Investigations

- Incisional biopsy.
- Resemble ameloblastic fibroma, epithelial cell rests (benign)/mesenchyme (malignant).

Management

- 1–1.5 cm bony margins, radiotherapy has no value.
- Chemotherapy as adjunct if more aggressive behaviour evident.
- Prognosis: Good with resection
  - Systemic bone condition: bone Neoplasm.

20.17 Fibrous Dysplasia

- Non-inherited—genetic disorder.
- Bone: Replaced with abnormal fibrous connective tissues, in which immature tissue is formed.
- Onset 1st–2nd decade.

Classification

(a) Monostotic.
(b) Polyostotic.
(c) Craniofacial (2 or more bones of jaw/midface/skull complex in continuity).
(d) Syndromes associated
  - McCune Albright: Café-au-lait macules/precocious puberty ± increased thyroid.
  - Jaffe-Lichtenstein Syndrome: Skin pigmentation.
  - Kaban classified on clinical behaviour: quiescent/non-aggressive/aggressive.

Aetiology

- Gene defect involving Gsa subunit: Encodes protein required for bone maturation.

Clinical

- Adolescent/young adults: Slow painless swelling of bones, commonly found in maxilla.
- Disturbed occlusion/function, can cause neurological symptoms with compression.
- Periods of aching and quiescence.
- Classically burns itself out in late teens/early 20's.

Radiograph

- Initially radiolucent (unilocular/multilocular), eventually radiopaque or sclerotic ground glass (peau d'orange/fingerprint).

Investigation

- CT to visualise extension.
- Tc-99 bone scan (poly vs. mono).

- Biopsy: Bone replaced by fibrous tissue, woven bone and giant cells.
- Foci of irregular shaped trabeculae of immature bone.
- Genetic testing: Bloods (LFT/ALP/LH/FSH).

Management

- Depends on behaviour/size/aesthetic.
- Surgical treatment in growth phase on for functional—compression optic nerve/significant aesthetics.
- Optimal time of treatment: end 2nd–3rd decade, can stabilise in early adulthood.
- Pamidronate I.V. (twice yearly) = controlled progression and improved bone pain/QOL; 50% refill in osteoclastic lesions.

Surgical

- Contour excision (quiescent/nonaggressive).
- En bloc resection: ± grafting (aggressive/recurrent lesions).

Prognosis

- Regrowth following treatment 35–30%.
- <1% malignant transformation = osteosarcoma.

20.18 Cherubism

- Hereditary: Defect in bone remodeling, leads to 'cherub' appearance.
- Maxillary expansion with 'eyes turned to heaven', can be confirmed by genetic testing.

Classification—Motamedi

- Type 1: Mandible alone.
- Type 2: Mandible + maxilla (posterior).
- Type 3: Mandible + maxilla (entire).

Clinical

- Autosomal dominant (chromosome 4p 16.3).
- 40% Sporadic M > F.
- Begins age 2.5 years = fully expressed at age 5 years.
- Bilateral painless symmetrical facial swelling
  - Displacement of teeth.
  - Lymphadenopathy during rapid progression.
- Exposure of sclera = apparent upward gaze.
- Usually self-limiting = regression at puberty, at age 30 lesions often undetected.
- Can get serious complications—airway/orbital involvement.

Radiograph

- Symmetrical, well-defined multilocular radiolucency/multicystic.

Histology

- Vascular fibrous stroma, numerous fibroblasts, multinucleated giant cells (like hyperparathyroidism).

Management

- Conservative, aesthetic/function.
- Bone can be vascular.
- Use of calcitonin.

Prognosis

- Regresses with maturity—only partial bone regeneration on radiographs.

20.19 Hyperparathyroidism

(1) Primary

- Hyper-secretion of PTH by parathyroid adenoma.
- Increase in Ca/PTH/ALP.
- Decreased or normal phosphate.
- MEN 1 = pituitary adenoma, pancreatic islet cell tumour.
- MEN 2a = phaeochromocytoma, thyroid medullary carcinoma thyroid.

(2) Secondary

- Response to decrease in calcium ions: Renal failure/malabsorption.
- Decrease in Ca.
- Increase in PTH/ALP.

(3) Tertiary

- When parathyroid overreacting becomes autonomous, even when cause treated.
- Increase in Ca/PTH/ALP.

Clinical

- Asymptomatic to severe symptoms - stones/moans/groans/bones.
- Increase in calcium ions: Thirst/N + V/constipation/depression/psychosis/weight loss.
- Bone: Pain/renal stones/radiolucencies.
- Urinary: Polyuria/renal calculi.
- Neurological:Memory loss/coma.

Radiograph

- Variable: Single/multiple radiolucencies (bone demineralisation with fibrous replacement).
- Osteoporotic appearance, partial loss of lamina dura.

Investigations

- Bloods/Histology: Osteoclastic resorption bony trabeulae/multicellular giant cells.
- Brown tumour: Haemosiderin and extravasated blood cells.

Management

- Primary: Surgery.
- Secondary: Manage underlying disease, phosphate binders (calcium carbonate/ aluminium hydroxide).

20.20 Paget's Disease/Osteitis Deformans

- Polyostotic disease, rarely leading to clinical symptoms.
- Aetiology unknown—primary osteoclastic dysfunction.

3 Phases

- Resorptive.
- Vascular.
- Sclerotic.

Clinical

- Age 50; Male:Female 3:2.
- All bones, mainly axial skeleton (vertebrae).
- 20% facial bones—mandible: maxilla 2:1.
- Asymptomatic/symptomatic.
- Neurological complaints—headache/auditory, visual, paralysis weakness.
- Intraoral: Bony enlargement. poor prosthetic adaptation, tooth displacement, malocclusion.
- Teeth often ankylosed.
- Vascular phase: Post-extraction haemorrhage.
- Sclerotic phase: Bone dense/avascular—prone to infection post-extraction.

Investigations

- Increase in ALP + Ca.
- Normal phosphate + PTH.

Radiograph

- Stage disease: Early osteolytic later 'cotton wool' pattern radiopaque pattern with hypercementosis, loss of lamina dura and obliteration of PDL spaces.

Histopathology

- Disorganised bone with reversed lines—mosaic (biopsy not always necessary as risk bleeding).

Management

- Milder—none, only analgesics.
- Pain/deformity controlled by inhibiting osteoclasts
  - Calcitonin (I.V., nasal spray): decreases calcium opposes PTH.
  - Bisphosphonates (oral ethidronate /I.V. pamidronate).

Prognosis

- Rarely fatal—heart failure/pathological fracture.
- 5% risk of sarcoma—6 months review with radiograph
  - Rapid expansion—serum ALP.
  - New radiolucent lesions.

20.21 Ossifying Fibroma

- Benign neoplastic bone: Excessive growth/bone destruction/recovery.
- Juvenile (aggressive): < age 15—commonly invades orbits/sinus.

Clinical

- Uncommon 3rd–4th decade, arises tooth bearing regions.
- Mandible > maxilla (premolar area).
- Growth variable, can be expansive.

Radiograph

- Well-circumscribed, sharply defined borders—roots displaced/resorbed.
- Radiolucent—mixed—radiopaque.

Investigation

- Histopathology not diagnostic.
- Fibrous connective tissue with well differentiated spindle fibroblasts.

Management

- Variable behaviour, recurrence 1–63%.
- Enucleation: Early/small/well demarcated.
- Resection: Large (5 mm margins, excision into sinus/nasal cavity).

Juvenile

- Non-aggressive: Enucleation.
- Aggressive: Rapid cortical thinning/perforation/teeth displacement = resection.
- Prognosis: 20–50% recurrence rate.

20.22 Cemento-Osseous Dysplasia Cementoma

- Reactive/dysplastic response of bone and cementum to unknown stimulus.
- Periapical/focal/florid/familial gigantiform cementoma.

Clinical

- Common Female > Male; middle age.

Radiograph

- Periapical area: Vital tooth—opaque mass with thin radiolucent ring.
- Florid: Expansion of lesion, multiple quadrants.

Investigation

- Bloods: Exclude Paget's (ALP).

Management

- Periapical/focal: No treatment.
- Florid: Avoid extractions tooth ankylosed to bone cementum.
- Fracture retained tooth—secondary infection.

20.23 Osteogenesis Imperfecta

- Brittle bones and blue sclera.
- Inherited defect—defect in collagen type I.

Classification

- IA: Multiple fracture/blue sclera/deafness.
- IB: IA + dentinogenesis imperfecta—tooth wear (AD).
- II: Severe—early death.
- III: Normal sclera, early death—cardiac.
- IV: Normal sclera, mild/moderate fragility—dentiogenesis imperfecta.
- Investigations: Genetics.

Management

- Brittle bones—defect mineralisation, blue sclera (decrease in collagen fibre thickness).

- Aortic dilation/hyper mobility/bowing long bones/easy bruising.

Treatment

- Bisphosphonates if facial fracture variable rate of bone regeneration and healing.
- Dentinogenesis imperfecta: Onlay/full crown coverage.

20.24 Bone Tumours

Classifications

A. ***Benign***

(1) Bone forming

- Osteoma/osteoblastoma.

(2) Cartilage forming

- Chondroma/chondroblastoma.
- Central giant cell granuloma.
- Ossifying fibroma.

B. ***Malignant***

(a) Primary

- Osteosarcoma.
- Chondrosarcoma.
- Fibrosarcoma.
- Myeloma.

(b) Secondary = metastatic.

20.25 Osteoma

- Periosteal versus endosteal
  - Endosteal—Asymptomatic, incidental finding on radiographs.
  - Periosteal—Asymptomatic, slow growing, hard mass.
- Rare, 2nd–5th decade—except Gardner syndrome.
- Radiographs: Well-circumscribed, radiopaque.
- Management: Surgical excision, asymptomatic monitor.
- Prognosis: No recurrence.

20.26 Gardner Syndrome

- Rare autosomal dominant charcaterised by
  - GI tumours.
  - Skin and soft tissues tumours.
  - Multiple osteomas and dental abnormalities.

Clinical

- Osteomas: Eespecially facial/skull (frontal bone), multiloculated masses, tear drop shape.
- Intestinal polyposis (adenomas): 100% malignant transformation.
- Skin/soft tissues: Sebaceous cysts, epidermal/desmond cysts, fibromas, tumours.
- Teeth: Odontomas, supernumeraries.
- Investigations: Gastroenterology/dermatology/radiographs.
- Management: Prophylactic colectomy, fibrous tumour excised with 1 cm margins.
- Prognosis: Good with early diagnosis.

20.27 Osteoblastoma/Osteoid Osteoma

- Benign neoplastic proliferation—rapid onset/cause pain.
- Osteoblastoma >2 cm, osteoid osteoma <2 cm.

Clinical

- Rare, cortical pain (severe), expansion is usually in age >30.

Radiograph

- Thin radiolucency surrounds a variable calcified central tumour mass.

Management

- Excision with 5 mm margin, may recur.
- Rare cases malignant transformation.

20.28 Osteosarcoma

- Malignant mesenchymal.
- Secondary radiation, loss p53.

Clinical

- Age: peaks in young/elderly.
- M = F.

- Common in long bones, rare in jaws.
- Expansion of bone, radiopaque (wide PDL space = mobile teeth, paraesthesia, pain.

Radiograph

- Sclerosing type: Sunray apperance.
- Osteoclastic: Radiolucency.
- Combination: Radiolucent/radiopaque mass.

Investigations

- Biopsy, CT/CXR—early lung mass.

Histopathology

- Variable: bone/cartilage/fibrous tissue.

Prognosis

- Neoadjuvent chemotherapy 3 cycles: interim CT—surgery = 5 year survival 50–80%.

20.29 Chondroma

- Benign tumour of cartilage—or low grade chondrosarcoma.
- Extremely rare, mostly found in mandibular condyle.
- Irregular radiolucent lesions ± foci calcification.
- Management: Conservative surgical excision/wide local excision (low grade chondrosarcoma).

20.30 Chondrosarcoma

- Malignant: Produce cartilage.
- Central arises within bone, peripheral/surface bone, extra-osseous-mesenchymal (high grade).

Clinical

- Peak at age 20–40 years.
- Swelling/mass/tooth mobility.
- Anterior maxilla > posterior mandible.
- Dental: Loosening/exfoliation.
- Sinonasal: Epistaxis/nasal obstruction.

Radiograph

- Radiolucent/mixed/radiopaque.
- Can cause widening of PDL and sunburst appearance (like osteosarcoma).

Management

- Radical excision.
- 5 year survival 15–68%.

20.31 Ewing Sarcoma

- Commonly found in pelvis/lower limbs, thought to be of neuroectodermal origin.

Clinical

- Average 11 years.
- Swelling/loosening of teeth/paraesthesia—like osteosarcoma.
- Systemic symptoms: Fever.

Radiograph

- Radiolucent with poorly defined margins.
- Classic "onion skin" periosteal reaction uncommon in jaws.

Investigations

- Biopsy > 5% glycogen granules.
- CD 99—stain.

Management

- Extremely aggressive: Metastasise (lung/liver/lymph nodes and other bones).
- Multi-agent chemotherapy/surgery.
- Radiation not typically used, unless margin unclear (radiation induced sarcoma).

Prognosis

- Head and neck better than other locations.

20.32 Multiple Myeloma

- Malignant proliferation of plasma cells, non-functioning immunoglobulin.
- Most common primary malignancy in bone: mean age 63.

Classification

- Multiple myeloma.
- Plasmacytoma: solitary lesion of bone.
- Extra-medullary plasmacytoma: 80% in head and neck (sinonasal/oral mucosa).

Clinical

- Bone pain, especially vertebral fracture.
- Increase in calcium ions.
- Decrease in platelets (epistaxis/anaemia/renal dysfunction).
- Jaw (12–30%): Pain/swelling/paraesthesia/loosening tooth/pathological fracture.
- 10% have systemic amyloidosis.

Radiograph

- Multiple, sharply demarcated lesions—'punched out' in skull.

Investigations

- SPEP (M spike of immunoglobulins protein).
- Urine electotropheresis (Bence-Jones protein).
- Increased serum Ca, increased ESR, BMAT.

Histopathology

- Proliferation of plasma cells.

Management

- Chemotherapy/steroids/localised radiotherapy for painful lesions/bone marrow transplant if unresponsive.
- Median survival = 3 years.

20.33 Central Giant Cell Granuloma

- Benign proliferation of fibroblasts and multinucleated giant cells that occurs almost exclusively in jaws with unknown aetiology.
- Uncommon: Mostly found in age 30; F > M 2:1.
- History: Identical lesions to hyperparathyrodism/cherubism/Noonan syndrome/NF1.

Clinical

- Painless expansion or swelling/variable growth/perforations with extension into soft tissues (uncommon).
- Aggressive CGCG: May cause pain/and rapid growth, root resorption, cortical perforation.

Radiograph

- Multi-/unilocular radiolucency with well-demarcated margins.
- May displace teeth/resorb roots.

Investigations

- Exclude high flow vascular lesion.
- Incisional biopsy/curettage.
- Blood to exclude PTH.

Histopathology

- Uniform fibroblasts in stroma containing varying amount of collagen with giant cells.

Management

- Differential giant cell lesions
  - CGCG.
  - Brown tumour.
  - Cherubism.
  - Aneurysmal bone cyst.
  - Osteoblastoma.

Aggressive If

- Size >5 cm.
- Recurrence.
- Show: Rapid growth/cortical thinning or perforation/tooth displacement/resorption
  - Small/non-aggressive: Curettage (recurrence 15–20%).
  - Aggressive: Need surgical margins (increased recurrence 75%, especially in males/younger patients).

Medical

- Steroids: Intralesional triamcinolone for 6 weeks—may get response then surgical excision if required.
- Calcitonin: Daily for 12 months, nasal spray/SC—inhibition of osteoclastic activity, should reduce tumour size in 6 months then surgery required as do not get complete remission.
- Interferon alpha: Suppresses FGF, only if all else failed as side effects—fever/rash/hair loss/neutropenia).
- Cochrane 2009: No evidence RCT to support.

20.34 Langerhans Cell Histiocytosis

- Antigen presenting cells.

- Classification (Lichtenstein)
  - Localised (bone): Eosinophilic granuloma.
  - Disseminated

    Chronic: bone/skin/viscera (Hand Schuller Christian disease).
    Acute: bone/visceral/often lethal.

Clinical

- Mandible more than maxilla.
- Mild bone pain/swelling/tooth mobility.

Radiograph

- Early: Localised, punched out radiolucencies with no calcification.
- Late: 'Floating teeth' if alveolar bone affected.
- Hand Schuller Christian disease: Affects sphenoid bone, triad
  - Skull lesions.
  - Diabetes insipidus.
  - Exophthalmos.

Investigations

- Incisional biopsy.
- Histology: Langerhans cells (Birbeck granules).
- Skeletal survey.
- Bloods: FBC/LFT/U + E/INR/APTT.

Management

- Chemotherapy—disseminated.
- Resection with 5 mm margins and radiotherapy non-accessible lesions 60 Gy.
- Intralesional and systemic steroids have been used.