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ENT Masterclass®,
106 Nottingham Road,
Ravenshead,Nottingham
NG15 9HL England

www.entmasterclass.com

Welcome to Volume 12 Issue 1 of Journal of ENT Masterclass® 2019

I would like to welcome all our readers to the 12th edition of the Journal of ENT Masterclass®. The journal continues the mission of the Masterclass platform in providing global free education. This will be the second year that the journal is instantly available for online access and we are pleased with last year's success judged by the number of hits on the Masterclass website. We also plan to periodically add new material to the journal.

As for previous edition, our section editors have selected up to date comprehensive articles written by national and international authors. Previously uncovered areas have been incorporated to include image guided ablation of benign thyroid disease, infants with feeding and swallowing difficulties, paediatric reinnervation update and complications of dermal filler, amongst others. We are, as ever, immensely grateful for the hard work provided by the authors and section editors.

The Masterclass travelling club has extended even further than last year with trips to Manama, Lahore, Islamabad, Cape Town, Beijing, Chongqing, Bucharest, Tashkent and Lausanne. For 2020, there are equivalent travel plans with the highlight of the ENT Masterclass 'World Congress' that will be held in the grand avenue of the Palace of Parliament in Bucharest on the 21st and 22nd of August, 2020. The UK based ENT Masterclass courses in 2020 will continue as before with the flagship course at Doncaster in January, Emergencies', Thyroid and Radiology Masterclasses in June, Nurses Masterclass in October, GP Masterclass in November and Consultants Masterclass in December.

I hope you will enjoy reading this 12th edition, and please send us any comments and suggestions that you may have.

Mr Hesham Saleh, FRCS (ORL, H&N)
Consultant Rhinologist/Facial Plastic Surgeon
Charing Cross and Royal Brompton Hospitals, London.
Chairman, Editorial Board, Journal of ENT Masterclass®.
December 2019.

Image guided ablation of benign thyroid disease

John F. Curran, MBChB, B.S., Department of ENT, Greater Glasgow and Clyde Hospitals and
Omar Hilmi, FRCS-ENT/ORL-HNS, MBChB, Department of ENT, Greater Glasgow and Clyde Hospitals

Correspondence:
John F. Curran, Department of ENT, Monklands Hospital
Monkscourt Ave, Airdrie ML6 0JS

E-mail: john.curran1@nhs.net

Abstract
Non-surgical management techniques have been described to manage thyroid nodules, ranging from high energy and chemical ablation to fine needle aspiration. We performed a review of recent literature of available non-surgical techniques, describing and assessing acknowledged image guided ablation interventions by mechanism of action, indication, effectiveness, limitations, contraindications and the appropriate level of anesthesia or analgesia required. All interventions were deemed safe alternatives to surgrey, and while not as absolute as surgery, can provide an appealing alternative to some patients. Interventions can further be tailored to the patient and nodule morphology.

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Key words
Ablation, benign, thyroid.

Background
Thyroid nodules are common in the population with reported rates of up to 65% having significant nodules at autopsy[1]. Nevertheless, the majority of these are not clinically relevant and most patients do not require any interventional treatment. Clearly patients with malignancy or toxic nodules require treatment in the form of surgery and / or Radioactive iodine. Currently patients with compressive symptoms (breathing or swallowing problems) from large or multiple nodules are treated surgically as previously there has been no good "Medical" treatment for this problem. The vast majority of patients with significant thyroid nodules are not malignant, so a treatment that symptomatically improves them whilst avoiding the inherent risks of surgical intervention at little risk in itself is the holy grail for managing these cases.

In recent years, non-surgical techniques have been described to manage thyroid nodules. From high energy and chemical ablation to regular aspiration, these have the potential benefit of providing symptomatic relief for patients without the risks of surgical intervention. Further, they can be cost-effective in comparison as many only require normal cohort of clinic staff compared to inpatient or day case staffing for surgery. A recent review by Nixon et al looked into the efficacy and complications associated with many image-guided ablation techniques, as well as its role in the current management of thyroid disease[2]. The purpose of this article is to expand on this recent review and describe acknowledged image guided ablation interventions by mechanism of action, indication, effectiveness, limitations, contraindications and the appropriate level of anesthesia or analgesia required. We will be focusing on benign thyroid disease as most reported data of these image guided techniques on malignant disease do not have longer term outcomes available, though much of that data is encouraging in low risk primary and recurrent disease[2].

High-energy techniques
High intensity focused ultrasound
High intensity focused ultrasound (HIFU) is a minimally invasive procedure that uses a focused US beam to a target area, inducing coagulative necrosis[3,4]. The target area is very small thus allowing for precision but requires a significant amount of time to cover any large area, particularly given that a cooling interval should be performed between each short sonification[3]. Typical treatment times range from 45 to 60 minutes and in larger nodules may need to be repeated. It utilizes piezoelectricity via a high-frequency amplifier. Software uses areas marked by the user and develops a treatment unit with safety margins[5].

The procedure can be performed under conscious sedation with the patient supine and neck hyperextended. Local anaesthetic can be used to alleviate pain during the procedure, this is infiltrated around the thyroid gland[4]. Some users advocate general anesthesia to avoid patient movement and the need for recalibration between treatment pulses, but the procedure has also been described without analgesia or sedation[6].

Reported studies largely focus on solid thyroid disease such as toxic nodules, benign thyroid nodules or multinodular goitres. Outcomes are reported as better with smaller volume thyroid nodules[2,7,8] but Lang et al further demonstrates larger nodules can be treated with sequential sessions[9]. It has also been used to treat persistent or relapsed Graves' disease with variable levels of success[10].

Many studies recently have reported an overall effective volume reduction in benign nodular disease from reported between 48 – 84%[2,6,11-13]. With sequential sessions on larger nodules (>20mls), the overall volume reduction at 6 months improved from 48% to 57%[9]. Further, 95% patients reported reduced overall symptoms (measured by a visual analogue score) at 24 months in a recent study by Lang et al[11]. When patient outcomes were looked at in comparison to open thyroid lobectomy surgery, the symptom reduction rate was comparable[14].

There is reduced efficacy as the treated nodule volume increases. Using a single session technique, Lang et al reports a reduction of 78%, 68% and 48% in small (<10 mL), medium (10–30 mL), and large (>30 mL) nodules demonstrating decreasing effectiveness with size[9]. Further, nodules that are close to the skin, carotid sheath, trachea, oesophagus or recurrent laryngeal nerve are difficult to treat as they are near these critical structures which are outlined in the safety margins during treatment[2]. It is advised to maintain safe distance of 1.1cm from the tracheoesophageal groove to prevent recurrent laryngeal nerve injury[4,15].

HIFU is typically more expensive than other high energy-ablative techniques[6]. Additionally, only one machine is available on the market presently that is able to deliver this technique and as it utilizes real time ultrasound in the planning it requires some end user familiarity with ultrasound[4].

As HIFU requires no needle or injected substance, it can be said to be nearly non-invasive. Despite this, some patients suffer side-effects to the procedure such as temporary vocal cord palsy (1-4%[4,10]), pain during session, transient skin erythaema, minor swelling, cough, skin blisters (12%[16]), hypothyroidism[4], horner's syndrome[14] and haematoma[5].

Radiofrequency ablation
Radiofrequency ablation (RFA) involves the insertion of a needle into a thyroid nodule that conducts electrical energy from a generator to induce coagulative necrosis[2]. This incurs a locally controlled temperature of up to 100 degrees Centigrade[3]. This can be done as either a cranial-caudal or trans-ithmic approach. Hydrodissection with 5% dextrose beforehand has been shown to help protect critical structures[3,17,18]). Pescatori et al advise using an 18-19 gauge needle as this allows for better maneuverability and thus better results and less complications, however, they do recognize that a larger (14 gauge) needle may be more effective in larger nodules[19]. The cost of the generator is around $25,000 and an electrode is about $750 for each session[20].

RFA is recommended as a non-surgical, minimally invasive technique for large thyroid nodules that are cosmetically undesirable or causing compressive symptoms. It is effective in solid benign thyroid nodules, toxic thyroid nodules and cystic nodules. The latter has been evaluated as second line treatment in cystic nodules refractory to chemical ablation[2,21-23]). There has been a further report by Hong et al of it being an effective non-surgical treatment in paediatric patients with compressive or cosmetically unsatisfactory thyroid nodules[24].

Perithyroid lidocaine is the advised method of analgesia. This is preferred to GA or sedation as it allows for constant assessment of nerve function and will alert the clinician to any proximity to the trachea as the patient will begin to cough and ablation should be immediately ceased. Further, it allows the patient to swallow cold fluids to help prevent oesophageal injury during the procedure[23].

In benign nodules, a response rate of up to 91% has been reported with an effective (>50%) reduction of volume[2,25,26]). A recent UK study on 31 nodules demonstrating an average volume reduction of 67% at 6 months[27] and recent prospective Austrian study on 277 patients demonstrating similar results at 6 months (68%) and a reduction of 82% at 12 months[28]. Deandrea et al demonstrated that RFA is more effective on spongiform nodules when compared to mixed or solid patterns on Ultrasound findings (76% vs 67 and 66% respectively – P =<0.01)[29]. Patient satisfaction scores are high, with Jung et al, demonstrating 98% therapeutic success rate and an 95% volume reduction at 60-months in a prospective multicenter study on 345 patients[30].

In Toxic Nodules results are variable with 24-82% normalization of thyroid function according to recently published Korean guidelines on RFA[23].

In Cystic Nodules Sung et al demonstrated no significant difference in effectiveness to percutaneous ethanol in cystic nodules compared with RFA[31], and thus the consensus to utilize RFA as a secondary technique as it is less economical than percutaneous ethanol.

Toxic nodules that are larger than 20ml can have a reduced response to RFA[23,32] and nodule volume appears a significant predictor in efficacy in these nodules[33]. Similarly, in large volume benign lesions, a second session may be required to manage patient symptoms and achieve adequate reduction[2,34,35]. Further, in nodules with heavy calcification, adequate ablation can be technically difficult[23]. Caution is advised with pregnancy, serious heart conditions or patients with pre-existing contralateral vocal cord palsy[20].

In the hands of an expert service, RFA has a very low side effect profile (overall complication rate of 2-5%[20, 23,30]). Major complications including nerve injury (recurrent laryngeal (2%[36]), cervical sympathetic ganglion, brachial plexus (<1%[37]) and accessory nerve), nodule rupture (<1%[37]), permanent hypothyroidism (<1%[37]), abscess (<1%[37]) thyroid storm[18] and haematoma (1%[7]) have been reported[2,23]). Other minor complications include vomiting (<1%[37]), skin burn (<1%[37]), transient thyrotoxicosis[28], lidocaine toxicity, voice change (1%[37]), hypertension and pain (2.6%[37])[23]. Sim et al has demonstrated in a long-term follow-up nodule regrowth can occur in up to 24% of nodules at an average of 40 months post procedure[38].

From an economic standpoint, RFA is more expensive than radioactive iodine treatment (for toxic nodules) but comparable in expense to surgery[23,39,40].

Microwave ablation

Microwave ablation (MWA) is non-invasive technique that operates by transferring up to 100W (typically 30-50W) of energy at a via frequency (2,450MHz) along a cable into an inserted needle (antennae). This results in rotation of molecules and an increase in temperature as a result of increased kinetic energy[2,3]. Hydrodissection has been reported to be helpful in protecting critical structures – similar to other thermal techniques[41,42].

Local anaesthetic, usually in the form of lidocaine, is injected in the perithyroid space. Peri-procedural cardiac and observation monitoring is recommended.

For benign disease, being a relatively new technique, there are less reported studies on the use of MWA. These predominantly focus on solid benign thyroid nodules.

In reported studies on benign solid nodules, the volume reduction ranges from 45-90%[3,41-44]. A recent meta-analysis on MWA reports a 12-month reduction rate of 88.6%[45]. Vorlander et al demonstrated that results of MWA on benign solid nodules were comparable to RFA (54% and 51% respectively)[46]; this matched a further study that included a HIFU arm as well with all three being effective and without statistical significance in efficacy[47]. Further, a prospective trial by Zhi et al demonstrates MWA to be an effective alternative compared to surgery with a lower side effect profile and overall greater patient satisfaction[48].

At present, there is little research in the application of MWA to benign thyroid disease other than solid benign nodules. As it has been reported as effective as other thermal techniques in this regard, there is scope to assess its efficacy in other pathologies, such as toxic nodules.

Complication profile is similar to other thermal ablative techniques with reported overall complication rate of 6.6%[45]. Reported complications include subcapsular haemorrhage (40%[49]), pain (70%[49]), fever (30%[49]), voice change (3-9%[49]), recurrent laryngeal nerve injury (9% [49]), horner's syndrome[46], skin burn and thyroid dysfunction[3,5]. Nodule regrowth has been demonstrated by Wang et al with 16 of 110 patients experiencing this complication[50].

Laser ablation

Laser ablation (LA) is a thermal ablative minimally invasive technique that utilizes laser light to heat up local tissue to temperatures between 46C – 110C and, depending on the temperature used, induces a combination of carbonization, coagulative necrosis and subsequent fibrosis of thyroid tissue[3,51]. This is achieved by inserting an optical fibre into the target tissue through a needle under US guidance and energy is delivered by Nd:YAG laser or laser diode[3]. It can be operated with single needle or multiple but needles should be more than 1cm apart if the lesion is large enough to necessitate multiple needles[5,25]. The needle should be parallel to the long axis of the thyroid nodule[51].

Due pain associated with the procedure, it is typically performed with a combination of sedation and local anaesthetic. Local anaesthetic alone may be inadequate[2,52]). Despite this, some have suggested that no anaesthesia allows the operator to better monitor proximity to critical structures[51].

LA has been demonstrated as an effective treatment alternative of benign thyroid nodules and toxic nodules[53]. It has also been used in conjunction with aspiration in mixed or cystic nodules[54,55]. While a systematic review demonstrated that RFA may be superior to LA in nodule reduction[56], LA appears to be more effective in large volume nodules[2,57]. It has been reportedly used effectively in nodules that did not respond to other thermal ablative techniques[58].

Volume reduction is reported to range from 47-84% in solid benign disease[2,3,5,51,59]. The effectiveness can vary based on the ultrasound appearances of the nodule. Negro et al report a 5 year nodule reduction of 59.7% with solid nodules versus a 78.6% reduction in spongiform nodules[60]. In mixed and cystic nodules, aspiration combined with subsequent LA has been shown to result in an average nodule size reduction of 92% and a loss of the cystic component in 75% at an average of 45 months[61]. Oddo et al further reported that there is a significantly positive perception of the procedure in patients who have undergone it. 100% of patients stated their symptoms of discomfort had improved and there was a significant reduction of goiter symptoms using a validated thyroid patient reported outcome questionnaire (ThyPRO)[62].

Toxic nodules can be treated with LA but results show only 50% of cases achieving normalization of TSH[63]. There appears to be a correlation to reduction of volume and normalization of thyroid function[64], this improves to 87% with multiple cycles of LA, though this value is still less than what is observed with RAI[65]. Single session appears to be adequate in most cases for nodules under 5ml in volume, with Gambelunghe et al reporting about 90% of these patients were able to come off their methimazole[64].

Similar to RFA, there is suggestion that LA can be used in combination with RAI for the treatment of toxic nodules. In large nodules, a combined approach may have greater control of symptoms compared to only RAI (2, 66). Using this technique, Negro et al demonstrated in large volume nodules, both surgery and a combination of RAI and LA were comparable in efficacy of restoring normal thyroid function and both resulted an overall improvement in quality of life for patients (67).

From an economic standpoint, A solitary diode or Nd:YAG laser source costs between $15,000 – $20, 000 with the deposable components factoring in at $400 per session[20]. A further limitation is that nodules close to critical structures are more likely to be undertreated and subsequently experience regrowth[68].

A large volume retrospective study reports an overall complication rate of 0.9% (0.5% major and 0.5% minor complications[51,69]) but if considering only minor complications, a recent review collates this to be much higher at 38.3% those this is significantly higher than other reported complication rates[5,69]. The predominant reported complication is pain (10.6% - 13.4%[51,64]), both during (12%[69]) and after (5.4%[69]) the procedure[5]. Other reported complications include fascial effusion (3%[70]), skin burns (<1%[59,69]), hypothyroidism, cough (<1%[69]), dysphonia (<1%[69]), haematoma (<1%[69]), vocal cord palsy (1.6%[70]), pericapsular bleeding, pseudocysts (5%[70]), fever (6-7.7%[64,69]) and vasovagal reaction (<1%[69])[2,5]. Dossing et al has postulated that more serious complications may be more likely when more than one fibre is used during the ablation[61]. Nodule regrowth occurs in 5% of nodules[51].

Non-thermal Techniques: Aspiration

Therapeutic fine needle aspiration (TFNA) of a thyroid cyst is a well-established, minimally invasive procedure to treat benign thyroid disease for many decades[71]. It involves using large bore needle (18 – 27 gauge) into a cystic thyroid lesion. Initially diagnosed and treated on clinical suspicion of cyst[71], the development of US allowed the operator to ensure the cyst is evacuated, ideally, to dryness. Further, US allows good control of the needle by the operator to avoid vascular structures and is now considered mandatory[3].

Aspiration requires there to be a completely cystic or a thyroid nodule with a cystic component. Further, this method also allows for the operator to send off samples of fluids and tissue by means to FNA or core biopsy if there is still diagnostic uncertainty. Lastly, as previously described, it can be used as an adjunct with other non-invasive image guided therapies.

The range of reported effectiveness is highly variable. In 1966, Crile et al described clinical success in 94% (absent or significant reduction on clinical examination) of patients after up to two aspirations. With the advent of US assessment of therapy, this number is more likely between 14% - 89%[72-80.]

TFNA should avoided in hot nodules on scintigraphy or patients with coagulopathy[5]. Further, some reported studies required as many as 17 repeat aspirations, which can represent a significant burden both to patient and service[73].

The complication profile of TFNA is minimal, with infection, bleeding and haematoma (0.01%[81]) being the most commonly reported[44]. The largest issue is the rate of eventual recurrence after aspiration. This has been reported as high as 90%[2,82] and can result in a larger cyst[83].

Needle aspiration is well tolerated and cost effective[3]. It can often be done in a clinical setting and rarely requires any analgesia.

Percutaneous chemical ablation

Percutaneous chemical ablation (PCA) involves the use of a sclerosing agent injected into the cyst or nodule to invoke a thrombotic, coagulative necrotic and fibrosing response to the target tissue or cyst[2]. Most research describes the use of tetracycline or ethanol as the sclerosing agent. Other agents have been used, including hydrochloric acid[78], polidocanol[84] and arginine hydrochloride[85]. Current guidance advises the used of percutaneous ethanol in cases of relapsing and symptomatic benign cystic nodules[3]. While most studies used between 30 – 70% of the fluid extracted volume of ethanol[3,8], Halenka et al demonstrated injecting 20% worth of initial cyst volume in 95% ethanol achieves adequate response[86].

It is often performed in a similar manner to US guided FNA, with an added step of sclerotic agent injection. As such, it rarely requires formal anesthesia or more than simple analgesia. Lidocaine or saline can be injected before final withdrawal of the needle[54].

PCA has been predominantly described as a method to treat benign cystic thyroid disease. Percutaneous ethanol injection (PEI) been also used to treat solid and toxic nodules. PEI has also been described without US guidance in large cysts but the authors note this is more appropriate in the limited resource setting[87].

Resolution of the cystic nodules through the use of ethanol has been reported to range between 72% - 100%[75,78,80,82,86,88-91]. Comparatively, tetracycline, has a cure rate of 43% - 97%[75,79,89,92-95]. This demonstrates how the former has become the standard agent used in practice today for PCA. Further, it has reported that there was no statistical significance in resolution of cysts when comparing tetracycline and injected isotonic saline (43% and 47% respectively)[93], and in similar studies comparing ethanol and isotonic saline there was a significant difference of 77%-82% cure rate with ethanol and 36% - 48% with isotonic saline[96,97].

Other sclerotic agents have been mentioned including: hydrochloric acid a 37.5% cure rate[78], polidocanol with a 93% - 100% cure rate at 12 months[84,98], arginine hydrochloride had a 100% response rate after up to 3 treatments[85]. A further study by Gong et al demonstrated while no significant difference in cure rate was evident comparing polidocanol and ethanol, complication rates were higher with ethanol[99]. As previously stated, compared to RFA, several studies do not report any difference in volume reduction, symptoms or cosmesis[3,31,100].

Table 1. Suggested Optimal Minimally Invasive Treatment Based on Efficacy

Benign nodule type	Suggested Treatment
Solid nodule	RFA/MWA/HIFU
Large solid nodule	LA
Spongiform nodule	RFA
Mixed nodule	Aspiration + LA
Cystic nodule	Aspiration
Recurrent cystic nodule	1st line PEI. 2nd line RFA
Toxic nodule	RAI. RAI + RFA/LA in large nodules (>20mls)

Solid nodules treated with PEI have had smaller response rates to cystic thyroid nodules but still with response rates around 62.5%[101]. Zbranca et al describes the used of PEI on 6 patients with toxic nodules in which all patients were cured after 1 to 9 injections[75]. This matches initial previous work by Mazzeo et al[102] however long term follow-up has suggested that this value was closer to 80% in this series with a further 16% having a partial response[2,103]. While it may result in a lower rate of hypothyroidism, RAI remains more effective and as such, the preferred non-surgical treatment[2].

PEI may make any future surgery more wdifficult due to periglandular fibrosis[72] and this should be recognised should any future surgical planning take place. Economically it is very cost effective with the items required to perform PEI valued at between $50 - $100[20].

Reported complications with PEI include pain (21 - 73%[97,99,101,104]), ethanol toxicity 10% - 24%[90,99,104], bruising (2.5%[101]), temporary dysphonia (3 - 5%[97,101]), vocal cord palsy (<1%[2,103,105]) and horners syndrome (2.5%[101]). Severe complications related to extravasation from the nodule have been described, including peri-glandular fibrosis, laryngeal and skin necrosis[106], jugular vein thrombosis (3%[107]), graves' disease and graves' orbitopathy[10]. A Cochrane review has suggested that side effects were more likely in treatment of solid nodules as opposed to cystic[72].

Conclusion

Non-surgical management of benign thyroid nodules is feasible and there are a variety of options available that may be more appropriate given a certain nodule morphology or clinical picture (Table 1). It is important to recognise that these treatments aren't as absolute as surgical intervention, which is well tested and regarded as safe, but may be more appealing to or more appropriate for certain patients. Surgery has the benefit of not requiring potential further intervention for the same nodule as it is definitive and removes the fear of misdiagnosis by giving definitive histology on the nature of the lesion. Complications comparing each intervention and those of surgery are in Table 2. Cost for non-surgical intervention should be considered before being adopted by a health board. Surgical treatment cost can vary between countries with a hemithyroidectomy being charged as much as $30,000[20] in the USA but this includes pathology costs and staff time that is not included in the other treatment costs.

Table 2. Complication Rates of Non-surgical Intervention Treatments of Benign Thyroid Nodules

Complication	HFUS	RFA	MWA	LA	FNA	PEI	Surgical lobectomy
Pain	73	2.6	6.5-70	10.6 - 13.4	NR	21-73	?
Fever	NR	NR	30	6 - 7.7	NR	NR	NR
Infection/abscess formation	NR	<1	NR	NR	*	NR	0.5
Skin Blistering/burn/ erythaema	1-12	<1	2	<1	NR	NR	NR
Hypothyroidism	*	<1	1.3***	*	NR	NR	14
Hamatoma/bleeding complication	*	1	3-40	<1	0.01	NR	0.8
Voice change/dysphonia	NR	NR	3-10	<1	NR	3-5	5.3
Recurrent laryngeal nerve injury/vocal cord palsy	1-4	2	1-9	1.6	NR	<1	0.01
Nodule/cyst recurrence	NR	24	14	5	90	NR	NR
Estimated cost	NR	$750**	NR	$400**	Minimal	NR	$5617
Overall complication rate	10.8	2-5	6.6	0.9 - 38.3	Minimal	NR	2.6

*reported but no values given
**for disposable items only
***reported as 'thyroid dysfunction'

There remains further scope for research in terms of further robust direct comparison of these minimally invasive interventions. Further, some complication data can appear skewed when not wholly reported or in small number trials and as such, a meta-analysis of the available databases in reported studies would help sharpen these values.

By tailoring the intervention to the lesion and the patient, healthcare boards will be able to effectively offer non-surgical management options to patients with benign thyroid disease. While it may be unreasonable for each individual trust to be able to provide every intervention, utilizing a national framework would help address this issue.

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The use of percutaneous drains in head and neck surgery – An evidence-based approach

SY Hey[1], MBChB, MRCS, DO-HNS, PG Dip; **S Flach**[2], BSc, BM BCh, MSc, MRCS(ENT), PhD; **A Lim**[1], MBChB, BMSc; and **J Manickavasagam**[1,3], MBBS, MS(ENT), DOHNS, FRCS(ORL-HNS).
[1]Department of Otolaryngology, Ninewells Hospital, Dundee, UK;
[2]Department of Otolaryngology, Hospital of the Ludwig-Maximilians-University, Munich; Germany
[3]Tayside Medical Science Centre (TASC), University of Dundee, UK

Correspondence:
Miss Shiying Hey, Specialty Registrar
Department of Otolaryngology-Head and Neck Surgery
Ninewells Hospital, NHS Tayside, James Arnott Drive,
Dundee DD2 1SY

E-mail: shiying.hey@nhs.net

Abstract
Introduction: Percutaneous insertion of neck drains in head and neck (H&N) surgery is a common practice. However, their benefits remain controversial.

Aims: i) To provide an evidence-based approach on the use of percutaneous neck drain and, ii) to review the latest technical advancement in facilitating a drain-less approach

Outcome: The usage of neck drains and the timings of their removals can vary, dependent on the surgeons' preference and experience. With the advancement of energy-based devices and the ease-of-use of haemostatic agents, these are increasingly used in various H&N procedures with good outcomes and allow the routine omission of neck drains in selected cases. Furthermore, a drain-less approach has been shown to facilitate same-day or day case surgery, with the additional benefits in resource utilisation and patients' satisfaction.

Conclusion: Drain-less surgery is a safe and viable alternative for select H&N procedures. Instead of a routine practice, neck drains should be judiciously used in H&N surgery on an individual basis.

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Key words
Head and neck surgery, neck drain, day case, outpatient surgery, drainless surgery

Introduction
Drains are commonly used in post-operative care in head and neck (H&N) surgery. The aim is to obliterate potential dead space, drain any blood or fluid collection and to potentially reduce the risk of local infection.

In recent years, the use of surgical drains has decreased in many specialties including in H&N surgery, as the evidence of their benefits has been questioned[1-5]. This trend has been reinforced by increasing evidence of the viability and safety of the drain-less approach. As the use of neck drains is often the determinant of the length of hospital stay, the drain-less technique is further lauded for opening up the possibility for same-day discharge. With the additional benefits in resource utilisation and cost-effectiveness, it is not surprising that the drain-free approach is more widespread in privatised health care systems such as in the USA, Canada and Singapore, compared to the UK.

Nevertheless, the use of neck drains remains largely dependent on surgeons' preferences with practice variation seen dependent on the type of operation performed. In this article, we aim to provide an evidence-based approach on the use of percutaneous neck drains, as well as a review of the latest technical advancement in H&N surgery.

Use of drains in head and neck surgery
Traditionally, neck drains are inserted in major H&N procedures and necessitate at least an overnight hospital stay. Although in some units, patients are allowed to go home with neck drains in-situ and the aftercare to take place in an outpatient setting, this is yet to become a widespread practice[6].

The character and quantity of the drain output informs the healing process and facilitates early recognition of complications such as bleeding, anastomosis dehiscence, chyle leak etc. Although it aims to reduce post-operative morbidity, the neck drain itself can result in complications. These include trauma during insertion, patients' discomfort

or pain, a potential route of infection/sepsis at drain site, mechanical blockage/disconnection, damage or erosion of adjacent tissues, scar formation, fistula or rarely, metastasis at drain site[7].

When a drain is used, there is no general consensus on the optimal criterion to time its removal. Two main approaches include volume-based (25-30ml/24 hour, averaging to 1ml/hour) or duration-based removal, with no comparative study to demonstrate the superiority of one to the other[8,9]. The cut-offs used in both approaches can vary and were derived mainly from clinical observational studies. From a survey among the American Head and Neck Society members, further variability in practice, such as the site and number of drains used, is also a commonality, dependent on the preference and experience of the surgeons[10].

Energy-based devices in head and neck surgery

One of the main indications for neck drain insertion is to monitor for bleeding which risks airway embarrassment in the post-operative period. With advancements in energy-based devices (EBD) technology, the introduction of new instruments has increased the effectiveness of haemostasis. In H&N surgery, EBD have been shown to reduce intraoperative blood loss, dissection time and postoperative pain[11-14]. This has raised the question as to whether EBD could further promote a drain-less approach.

Among the commonly used EBD are Harmonic Scalpel, LigaSure™, THUNDERBEAT etc. These devices can omit the extensive clamp-and-tie process and reserve the standard ligation techniques for major vessel control. Multiple studies have compared various EBD but no significant differences have been demonstrated in patient outcomes[12,15-17]. Studies have shown that EBD can be safely used ≥2mm from an important structure[18-21]. However, operators should always be vigilant of the risk of collateral thermal injury from the heat emission.

Although no study has directly assessed the impact of EBD on neck drain usage, in a meta-analysis by Yao et al, no significant differences in drain output, postoperative complications and length of stay, between standard ligation techniques and Ligasure was demonstrated[22]. As the use of an EBD often provides additional confidence in the intraoperative haemostasis control, this could, in turn, facilitate the omission of the routine use of a neck drain. Although one should always bear in mind that a drain is not a substitute for good surgical technique!

Haemostatic agents in head and neck surgery

In addition to EBD in haemostasis control, the use of novel topical haemostatic agents has also gained momentum over the past few years. A number of studies have demonstrated that tissue sealants are not only safe to use in H&N surgery[23-25] but decrease the length of hospitalization and frequency of complications, consequently leading to sizeable cost savings[26,27].

A variety of the haemostatic agents have been developed, including fibrin sealants such as TISSEEL and ARTISS or, gelatine-thrombin matrix sealants such as FLOSEAL and SURGIFLO® [28-30]. These haemostatic agents can be directly applied, either therapeutically to an 'oozing' site or, prophylactically to a wound bed. They act through activation of the clotting cascade resulting in local haemostasis, an essential component in post-operative wound healing[23,31].

Encouraging evidence for the use of fibrin sealants in soft tissue H&N surgery was reported in a systematic review and meta-analysis by Bajwa et al[32]. This showed that fibrin sealants reduced the mean total volume of wound drainage. However, due to the poor methodology of the included studies and statistical heterogeneity, a statistically significant earlier drain removal or hospital discharge, as a consequence from the application of fibrin sealants, could not be demonstrated[27,32]. Although more evidence is needed to establish the direct effect of haemostatic agents with drain usage, it is possible to assume that these novel agents, in synergy with EBD, have the potential to replace the routine need for drain insertion in H&N surgery.

Thyroid surgery

Among all H&N procedures, evidence of drain usage is most concentrated in thyroid surgery, with a rationale to primarily minimise the risk of potentially life-threatening airway complication from bleeding, as well as to prevent seroma formation[33-35]. The incidence of haematoma in thyroid surgery and the need for re-operation is reported up to 1.5%[36]. While drains are often used for prevention of haematoma or airway compromise, it is worth noting that in a large bleed, the drain can be blocked off and does not necessarily alleviate the situation. Notably, in a meta-analysis by Tian et al, in comparing drain placement and no drain in patients undergoing total or partial thyroidectomy, no significant difference was found in haematoma formation between the two study groups[37]. Portinari et al also reported in their study that no decrease in re-operation rates was found when drains were used in thyroid surgery[38].

Furthermore, multiple studies including a meta-analysis by Woods et al indicate that drain use after routine thyroid surgery does not confer a benefit to patients[39]. Instead, drains may result in injury during insertion and increased scarring as well as cosmetic deficit[34]. Moreover, post-operative infection rate was found to be higher and hospital stay longer in those patients who had drain placement[37].

Based on the literary evidence, the routine placement of drains after thyroid surgery does not seem to be justified. Exceptions may be made however where the risk of haemorrhage is greater, particularly following resection of large goitres or when combined with central or lateral compartment dissections[36]. A decision on drain usage in thyroid surgery should be tailored to individual patients instead of being a routine practice.

Salivary gland surgery

Building on the promising results from drain-less thyroidectomy, parotid surgery too, has undergone changes over the recent years. Traditionally, postoperative management of parotidectomy included a drain due to the well vascularised wound bed and the risk of salivary leakage from an incised parotid. To address these concerns, the replacement of surgical drains with haemostatic agents in parotidectomy has shown favourable results. This has allowed parotidectomy patients to enjoy same-day discharge and eliminated the drain-related morbidity[40].

A randomized controlled study by Chua & Goh compared the safety and efficacy of partial superficial parotidectomies with fibrin sealants and pressure bandage to conventional surgery and drains[41]. The use of fibrin sealants for partial superficial parotidectomies resulted in lower hospitalisation costs, shorter duration of stay and no significant difference in morbidity compared to conventional surgical approaches with drains. In addition, Conboy et al and Coniglio et al both showed that parotidectomy without a drain can be undertaken safely and cost-efficiently in carefully selected patients[42,43]. Similarly, Hey et al reported drain-less parotidectomy techniques that allow same day discharge involving the prophylactic use of haemostats and postoperative application of a pressure bandage[44]. [Figure 1] [Figure 2]

Likewise, submandibular gland surgery has also been shown to have the potential for a drain-less approach. Bannister et al & Ujam et al both showed in their series the feasibility to replace drain with different types of haemostatic agents[24,45]. In agreement with these, Park et al showed that similar to thyroidectomy and parotidectomy, submandibular gland excision can be safely done with prophylactic use of fibrin glue without insertion of a drain[46].

In line with experiences from drain-less thyroid surgery, salivary gland surgery without drain insertion appears as a feasible and effective alternative to reduce patient morbidity and length of hospital stay.

Neck Dissection and Mucosal Head and Neck surgery

Neck dissection is a field in H&N surgery where practice can vary. Nevertheless, drains are ubiquitously used in neck dissection to apply a negative pressure to the potential space resulting from dissection, to allow fluid drainage and to inform and facilitate the healing process[47]. The average duration of drainage in uncomplicated neck dissection tends to be around two to four days[10]. However, there is a lack of consensus on the criteria used for drain removal, as mentioned previously. A randomized trial by Tamplen et al looked at patients undergoing unilateral or bilateral selective lateral neck dissection and concluded that a volume threshold for drain removal of 100ml over 24h after surgery appears to be safe[48]. Similarly, Flam and

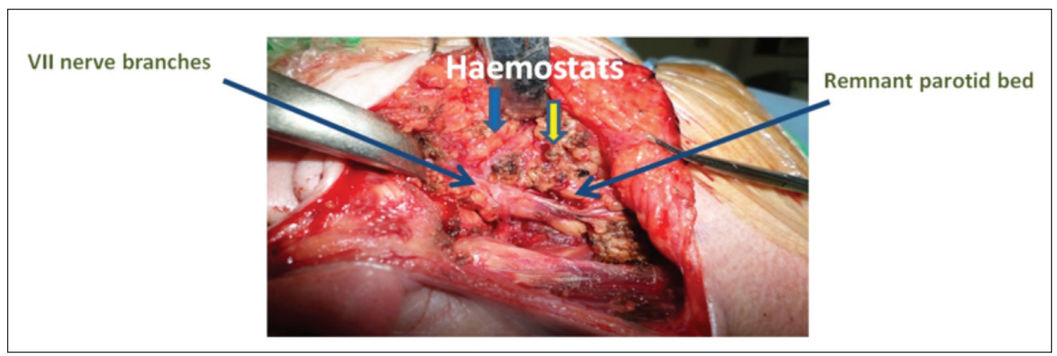

Figure 1: *(Original) The prophylactic intra-operative use of haemostats (applied as micro-dots) on parotid bed.*

Figure 2: *Post-operative application of a 'Balaclava' or pressure bandage for 48 hours. (Reproduced with owner's permission. This will be updated with further details in due course.)*

Smith suggested drain removal when the drainage rate is ≤50ml over 24h[49]. Kasbekar et al further showed that earlier drain removal can be achieved at the maximum drainage rate of 20ml in 6h (or 3.3ml per hour)[50]. Other studies have shown that the highest rate of drainage happens in the first 8 hours after surgery, thus advocating a drainage-rate criterion of ≤1ml/h over an 8-hourly period, to decide on the timing of drain removal[8]. Not only are there discrepancies in drain removal timing, but the use of drains in neck dissection tend to be reported in smaller numbers among heterogeneous studies, thus precluding any substantial conclusion to be drawn.

In contrast to the H&N procedures discussed above, there is no evidence to date reporting a drain-less approach in mucosal H&N resections involving the oral cavity, oropharynx, hypopharynx and larynx. Although haemostatic agents were shown to be safe for use in a small number of mucosal H&N cases[24], this has yet to replace the standard approach of drain insertion for postoperative care. In view of a paucity of literature in this field, this could represent the next phase of interest in drain-less surgery and further studies are required to ascertain its feasibility and safety profile.

Additional Potential Benefits

Same-day (also called day case or outpatient) surgery has been shown across specialties to be overwhelmingly endorsed by patients for smaller waiting time, less risk of cancellation, lower rates of infection and the comfort to convalesce in a familiar environment[51]. In comparison to other ENT sub-specialties, H&N surgery has a smaller range of procedures that are considered suitable for same-day discharge, with the neck drain often being a key determining factor. With an increasing appetite for day case surgery, the omission of neck drain can be deemed a safe and simple improvisation to facilitate day case surgery in select cases as outlined above.

The drain-less technique not only has the potential to enhance patients' experience but to facilitate cost saving by shortening the length of stay. This was shown by a phased feasibility study in the UK by Heyes et al, using the ARTISS agent to replace routine use of neck drains[30]. Likewise, in a case series by Conboy et al from Canada, drain-less technique used in parotidectomy resulted in a saving of $1775 per patient[42]. A similar comparative study from Singapore by Chua et al also reported that a drain-less technique could save $260 per patient, due to the significantly shorter hospital stay and the elimination of drain-related morbidity[41]. The cost-effectiveness could be further incentivised via the minimisation of re-admission and re-intervention rate, by facilitating the management of post-operative complications in an outpatient setting, as reported by Coniglio et al in their drain-less parotidectomy series[43].

Conclusions

Although percutaneous neck drains have been used with best intentions in H&N surgery for many years in the past, emerging evidence has demonstrated that drain-less surgery is a safe and viable alternative. With advancing technology in haemostatic agents and EBD, promising results have suggested that the routine omission of neck drain provides comparable outcomes. Furthermore, the drain-less approach confers additional advantages in patients' satisfaction and cost-effectiveness, with the latter being at the centre of focus in our current economic climate. As such, the judicious use of neck drains in H&N surgery is essential, both to rationalise resource utilisation and patients' experience.

Conflict of Interest

None declared

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A systematic multidisciplinary approach for assessing infants with feeding and swallowing difficulties: Understanding what the otolaryngologist brings to the team

Nikki Mills[1,2] BHB, MBChB, Dip Paeds, FRACS, IBCLC and **Melissa Keesing**[3] Bachelor of Speech and Language Therapy

[1]Paediatric Otolaryngology Department, Starship Children's Hospital, Auckland, New Zealand
[2]Department of Radiology with Anatomy, University of Auckland, Auckland, New Zealand
[3]Speech-language Therapy, Starship Children's Hospital, Auckland, New Zealand

Correspondence:
Nikki Mills
Paediatric Otolaryngology Department,
Starship Children's Hospital, Park Road, Auckland 1023, New Zealand

E-mail: nikki@adhb.govt.nz

Abstract

Assessment and management of feeding and swallowing difficulties in infants requires a multidisciplinary approach, tailored to the presenting problems and the specific needs of the infant and their family. Focusing on the potential contribution of the otolaryngologist to the multidisciplinary team, we present a four-stage assessment process that includes: (1) assessment of nutritional status; (2) assessment of anatomical structure and function; (3) assessment of general function (motor, sensory, cognitive); and (4) assessment of airway protection during swallowing. We expand on each stage to identify key questions, red flags, potential investigations and management options. Breastfeeding specific information is highlighted, recognising that increasing education and knowledge in this area is important in general otolaryngology training and that a traditional approach risks inadvertently undermining successful breastfeeding.

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Key words

Infant, breastfeeding, swallowing, aspiration

Introduction

Paediatric feeding disorders are commonly viewed from the perspective of a single professional discipline[1]. However, due to the complexity and heterogenicity of infants presenting with feeding and swallowing difficulties, their management requires diverse skills and experience. This paper presents a structured and practical approach to the assessment, diagnosis, investigation and management of paediatric feeding disorders within a modern multidisciplinary environment.

We use the term paediatric feeding disorder to include any impaired oral intake that is not age-appropriate and is associated with medical, nutritional, feeding skills and/or psychosocial dysfunction, with the broader definition of this term now including dysphagia[1].

Table 1: A 4-Stage Approach for ORL Assessment of Infant Feeding and/or Swallowing Difficulties: Overview and Key Questions

Stage 1: Assessing nutritional status
Are they achieving sufficient nutrition to grow?
What is the trend (in weight percentile) over time?
Stage 2: Assessing anatomical structure & function
Are there underlying congenital anatomical abnormalities contributing to difficulties with breathing, feeding, swallowing and/or airway protection?
Are further investigations and/or surgical intervention warranted?
Stage 3: Assessing general function: motor, sensory, cognitive
Are there concerns regarding how the infant achieves their nutrition: time taken, coordination, sensory anomalies, cognitive responses such as stress or aversion?
Stage 4: Assessing airway protection with swallowing
Is there sufficient concern to warrant investigation for possible aspiration?
Is aspiration +/- penetration visualised on VFSS +/- FEES?
Is there an appropriate cough response if penetration/ aspiration occurs?

Table 2: Stage 1: Assessing Nutritional Status
Key Questions
What was baby's birth weight?
What was the mode of delivery?
What is baby's current weight percentile and trend over time? (Plot on appropriate growth chart)
Do you think your baby eats/drinks enough?
Key Red Flags
Low percentile weight
Dropping percentiles
Parental concerns
Nutritionally inappropriate diet
Key Management Options/Suggestions
Involve Dietitian (to assess nutrition and intake and offer suggestions to optimise)
Involve Paediatrician (to exclude any potential medical causes for FTT)
Involve Lactation Consultant (if breastfeeding or feeding expressing breast milk)
Ensure strategies promote and protect the option of continued breastfeeding/EBM wherever possible

The make-up of a multidisciplinary team will vary based on local availability and expertise. The team needs to include the family as active participants; as their knowledge, perspectives, culture and goals will inform decisions on how best to treat the child. The disciplines included in the team will vary with the specific needs of the infant and family. For example, a social worker or mental health clinician may be able to assist with addressing the extraordinary psychological, social, and financial stress that can be associated with caring for an infant with feeding difficulties.

We draw on a review of the literature and 50 years of combined clinical experience in Otolaryngology (NM) and Speech-language Therapy (MK) to present a four-stage assessment process that includes: (1) assessment of nutritional status; (2) assessment of anatomical structure and function; (3) assessment of general function (motor, sensory, cognitive); and (4) assessment of airway protection during swallowing (Table 1). We then expand on each stage to identify key questions, red flags, potential investigations and management options. Adopting a structured approach reduces the risk of overlooking contributing factors (such as an undiagnosed co-morbidity) and helps to ensure that investigations and management plans are evidence-informed and effective. Although this article focuses on paediatric feeding issues in infants, many of the principles will be relevant at other ages.

Stage 1: Nutritional Status

The first stage involves assessment of the infant's nutritional status (Table 2). This involves taking a history of the child's nutritional intake and assessing their growth. Standard growth charts can be found in a child's health records book or electronic health record. These growth charts plot serial measurements of a child's growth (weight, length and head circumference). The World Health Organisation (WHO) Growth Charts are more appropriate for tracking the weight of breastfed infants than the USA CDC Growth Charts, which are based on populations with a high percentage of formula fed infants[2].

A weight below the 9th percentile or that drops across percentile-lines is considered a "red flag". However, having a healthy weight based on age does not exclude the presence of clinically significant feeding difficulties. Among new-born infants, concern should be raised if the infant loses more than 10% of their birth weight or does not regain their birth weight within fourteen days post-delivery[3]. A premature infant, born before 37 weeks' gestation, should have their weight plotted using their corrected age. Some specific populations, such as children with Trisomy[21], have modified growth charts that allow for altered expectations for growth associated with their condition[4].

With the transition from placental to enteral nutrition, it is expected that newborn infants will lose weight after delivery[3]. Healthy breastfed babies will commonly lose more weight than formula fed babies (average 6.6% versus 3.5%) and will take slightly longer to regain their birth weight (8.3 days versus 6.5 days)[5]. Research has shown that over 20% of infants delivered by caesarean section (versus 5% of non-caesarean delivered infants) will lose greater than 10% of body-weight[6] and it has been suggested that weight at 24 hours may be a more appropriate measure of "true" birth weight for these babies[7]. Babies born via caesarean delivery also have a higher incidence of breastfeeding difficulties, so early lactation consultant support is recommended for this group[8].

Faltering growth, previously known as failure to thrive, is a descriptive term and not a diagnosis. Causes for suboptimal weight gain need to be considered[9]. A referral to a dietician will ensure a thorough assessment of nutritional intake, with early involvement of a lactation consultant recommended for breastfed babies and referral for general paediatric review when appropriate.

Stage 2: Anatomical Structure

The second stage involves systematically assessing the infant for any anatomical anomalies or variants that may impact on feeding (Table 3), through history-taking, examination, and investigation as appropriate (Table 4). During this stage of assessment, an otolaryngologist's clinical skills and use of awake flexible trans-nasal endoscopy can provide valuable information on oropharyngeal anatomy and swallow dynamics for the multidisciplinary team.

Table 3: Anatomical "Checklist" for infant/paediatric feeding difficulties	
Airway	Nasal patency (fogging of mirror/ metal) Septum alignment Inferior turbinate appearance Rate, effort and noise of breathing (expose chest)
Craniofacial	Any dysmorphic features General head shape and size Mandible size and position Temporomandibular joint function
Oral cavity, Tongue & Frenulum	Hard palate height and contour Anterior "free length" of tongue (measured ventral surface: floor of mouth to tip) Lingual frenulum: – Height of attachment to ventral tongue – Length (between tongue and mandibular attachments) – Thickness/appearance General oral mucosal heath If post dental eruption: – Dental and gingival hygiene – Occlusion
Soft Palate & Uvula	Check for overt or submucous cleft
Tonsils	Size/Grade Presence of inflammation/infection

Airway:

Feeding and breathing are closely integrated in infants. Therefore all infants with feeding difficulties should be assessed for potential airway compromise and all infants with airway compromise assessed for potential feeding difficulties. Any increase in respiratory rate or effort will compromise the timing and coordination of the swallow, increase the risk of aspiration and potentially negatively impact nutritional intake. Compromised breathing can cause early fatigue during feeding, resulting in; short feeds of inadequate volume and calories and/or the infant falling asleep during feeds. Increased effort of breathing may also increase calorie expenditure.

Table 4: Stage 2: Assessing Anatomical Structure and Function
Key Questions
Does the baby have noisy breathing?
Is there any evidence of airway compromise?
If so, what level/s of the airway is/are affected?
Has a thorough clinical examination been performed?
Key Red Flags
Airway compromise: noisy breathing, nasal obstruction, increased work/rate of breathing
Parental concern
Chest wall shape deformity (eg pectus excavatum)
Velopharyngeal insufficiency: milk/liquid/food via nose during feeding
Difficulty latching at breast (weak, sliding off, maternal pain and/or nipple trauma)
Feeding interrupted by coughing +/- choking
Unlatching for "breathing breaks"
Any structural anomalies: including tongue, lingual frenulum, hard & soft palate
Investigations & Management Options/Suggestions
Ideally observe infant feeding – parent recorded video can be helpful
Consider flexible endoscopy (+/- FEES: recommend always performed with SLT)
If lingual frenulum appears restricted – ensure LC input, consider division if indications present
Investigations and/or interventions regarding any anatomical anomalies should be considered in the context of functional impact rather than appearance alone
Involvement of other specialties as appropriate

History taking should enquire about noisy breathing, with video recordings taken by parents helpful when the noise is not directly observable. Fogging of a cold metal tongue depressor (or mirror) held under the nares can assess nasal airflow. The infant's thorax should always be exposed to observe for an altered breathing pattern or resultant changes in the chest wall contour/shape.

Stertor is typically created by obstructed airflow within the nose or by dynamic collapse of the soft palate and/or tongue base. The infant may tend to mouth breathe and symptoms may be worsened when supine and/or sleeping. A breastfeeding infant with impaired nasal airflow will need to come "on-and-off" the latch frequently to breathe orally, which may cause nipple trauma and pain for the

Figure 1: *Adapted breastfeeding latching positions.*

mother. Bottle-fed babies with nasal airway compromise can usually be managed by reducing the flow rate of the teat and using “paced” feeding, which gives frequent pauses for “breathing breaks” by removing the bottle teat briefly from the infant’s mouth.

Minimal or mild symptoms are often caused by nasal mucosal congestion and can usually be managed with saline drops, non-invasive manual evacuation of nasal secretions and consideration of nasopharyngeal aspirate testing for viral-induced rhinitis. When more significant symptoms are present; congenital structural anomalies such as septal deviation, choanal atresia, piriform aperture stenosis and intranasal/nasopharyngeal masses need to be excluded[10]. If no surgical intervention is indicated, measures such as topical nasal steroids, humidification and/or a temporary nasopharyngeal airway could be considered.

Stridor is typically a higher pitched noise and is usually generated at the level of the larynx or supraglottis, and

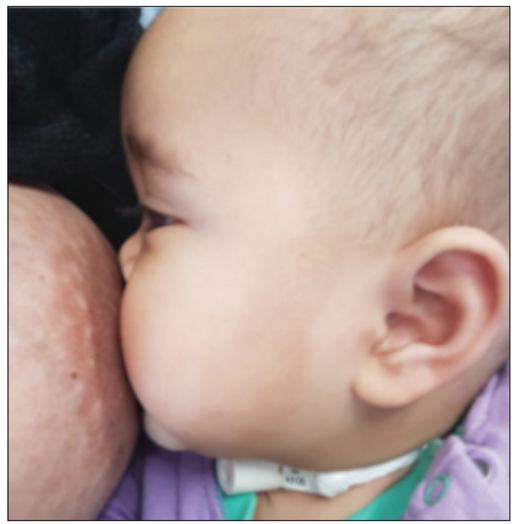

Figure 2: *Breastfeeding infant with tracheostomy.*

usually worsened by increased tidal volume and faster airflow, as occurs during feeding and crying. If there is a dynamic component to airway compromise, supine positioning will alter tongue base and epiglottic positioning, which will often worsen airway compromise. Positioning the infant semi-prone or “laid-back” position may improve their ability to breastfeed (as shown in Figure 1), with bottle-fed infants often improved in an upright or side-lying position. Remember that infants with complex airway problems, even those requiring tracheostomy, may be able to fully breastfeed once their airway is stabilised (see Figure 2).

Awake transnasal flexible endoscopy can be very helpful in determining the level/s of airway obstruction and can usually be performed in an outpatient clinic with appropriate personnel and monitoring. A vagal response can be stimulated, particularly in new-borns, so continuous oximetry is recommended, with immediate withdrawal of the endoscope if heart rate deceleration occurs to avoid causing an apnoeic event. Electronic recording with audio capture allows correlation of any noise generated during the endoscopy with the dynamic anatomy and the ability to analyse in slow motion. Laryngomalacia is the most common cause of neonatal stridor, but should only be diagnosed when endoscopically the supraglottic tissue collapse can be confirmed as generating the audible stridor. It is possible to proceed directly to Flexible Endoscopic Evaluation of Swallow (FEES) while the endoscope is in situ, but requires some pre-planning to ensure a speech-language therapist is present (see Stage 4).

Other investigations such as a chest x ray, overnight oximetry and an early morning capillary blood gas may be appropriate, together with a formal rigid airway endoscopy if there are concerns regarding tracheal anomalies or aspiration (see Figure 3).

Craniofacial

Midface hypoplasia is often associated with nasal airway compromise and micro/retrognathia with tongue base prolapse. Both can potentially create airway and feeding difficulties. These infants all warrant early multidisciplinary assessment of airway and feeding, with regular re-evaluation to assess for changes with growth and development.

Oral Cavity and Tongue

The biomechanics of breastfeeding require the infant’s tongue to elevate the nipple to the hard palate, contouring the dorsal surface around the nipple and creating a base-line intraoral vacuum[11]. Milk transfer then occurs by lowering of the mandible and tongue (en-bloc), increasing the strength of the intra-oral vacuum[12,13]. A biomechanical disadvantage is likely to exist when there is; retro/micrognathia, a high arched hard palate, a short anterior free length of the anterior tongue and/or restriction of tongue elevation caused by the lingual frenulum. An infant with any of these anatomical variants is more likely to

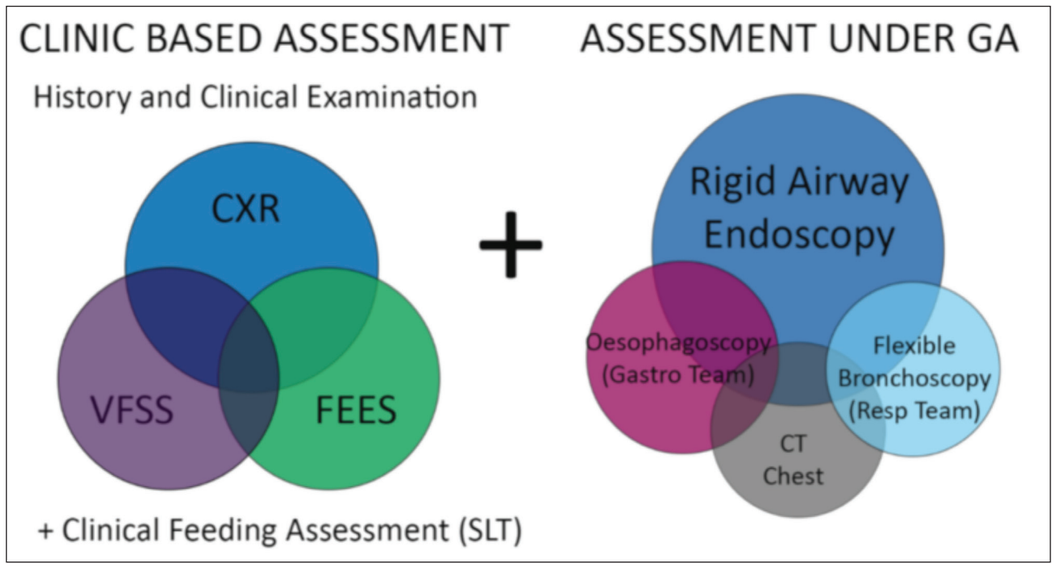

Figure 3: Assessment of an infant or child with complex feeding/airway difficulties +/- concerns.

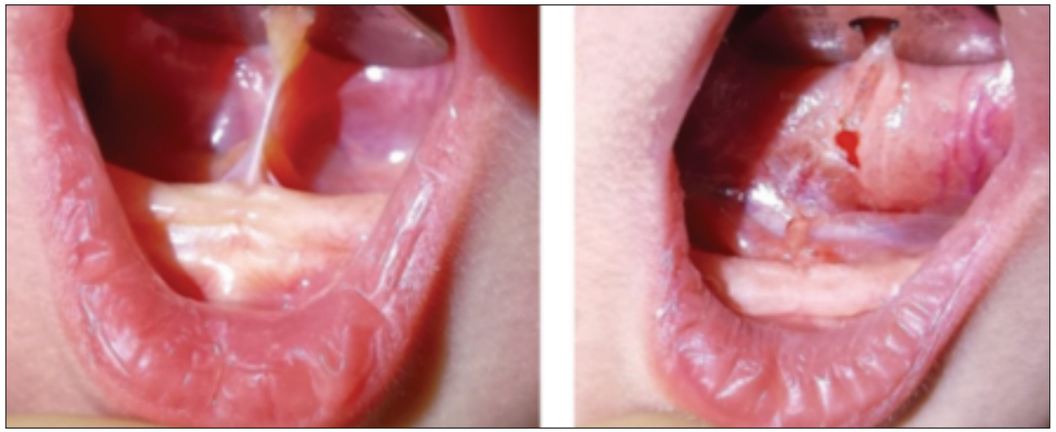

Figure 4: Tongue tie: before and after scissor division.

have challenges creating and sustaining an intraoral vacuum. These infants may have more difficulty transferring milk, presenting with; poor weight gain, long feeds, signs of frustration at the breast and/or difficulty establishing or maintaining maternal milk supply. The infant may develop compensatory strategies, including gripping the nipple with gums and/or using orbicularis oris muscle to help hold the nipple in the mouth, which may in turn cause maternal nipple pain and trauma.

When the lingual frenulum is restricting tongue elevation, some infants may benefit from surgical division (frenotomy, see Figure 4), with the best evidence for reduction in maternal pain with breastfeeding post-procedure[14-16]. However, it remains unclear which babies will benefit from frenotomy and there is concern regarding potential overtreatment in some countries[17-20]. Popular grading systems based on the height of attachment to the ventral surface of the tongue[21,22] do not correlate well with breastfeeding difficulties or outcomes following frenotomy. The lingual frenulum was previously thought to consist of a midline submucosal cord/band/string, but recently has been shown to be formed by a layer of fascia that spans the floor of mouth[23,24]. With tongue elevation, this fascia is brought under tension and dynamically elevates together with the overlying oral mucosa to create the fold of the lingual frenulum. With this new understanding of frenulum anatomy, further biomechanical research is needed to determine which morphological variants will benefit from frenotomy. Until clearer guidance is available, we recommend only proceeding to frenotomy when there is clinically apparent restriction of tongue elevation together with difficulties breastfeeding that have not improved with lactation consultant support.

Soft palate and uvula

The diagnosis of a cleft of the soft palate (particularly when partial or submucous) can be missed in newborns (Figure 5). Visualisation of the free edge of the soft palate and uvula is essential and usually requires a tongue depressor and headlight. A cleft of the soft palate is likely to cause an "air-leak" from the intra-oral space, preventing establishment of the intraoral vacuum required for breastfeeding. Although some infants with cleft palate can transfer milk at the breast, presumably aided by the maternal milk ejection-reflex creating flow, most need to be bottle fed using a specialised teat.

Tonsils

Large tonsils are an uncommon cause of dysphagia in infants but can be an increasingly common cause of dysphagia as childhood progresses. Flexible endoscopy

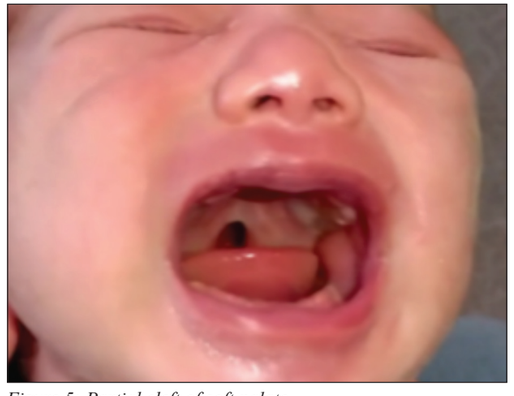

Figure 5: Partial cleft of soft palate.

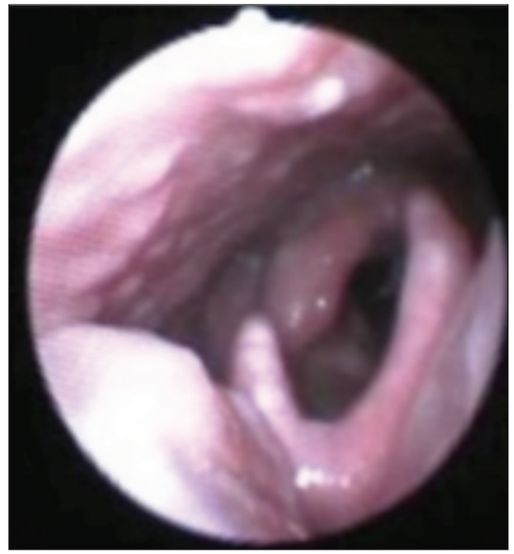

Figure 6: Large tonsils viewed via trans-nasal endoscopy.

can identify pendulous enlargement of the inferior poles of the tonsils which is not apparent trans-orally (see Figure 6).

Stage 3: General Function: Motor, Sensory & Cognitive

The third stage involves assessment of motor, sensory, and cognitive function (Table 5). Successful feeding requires a symphony of precisely coordinated neuromuscular activity from the lips to the lower oesophagus, mediated by cranial nerves, brainstem and the cerebral cortex[25,26]. Many factors can disrupt the reflexes which facilitate sucking, swallowing, and breathing in the early postnatal period[27]. Feeding and swallowing skills usually follow a pattern of developmental progress. However, problems are reported in 15 to 45% of typically-developing children, 70% with chronic medical conditions, and 80% of children with developmental disabilities[28]. An emphasis on feeding volumes and rigid timing schedules can create unrealistic expectations for parents and can override recognition of impaired feeding quality. Delay in identifying paediatric feeding disorders in children can result in impaired cognitive, physical, emotional and social development, can have an adverse effect on caregiver-child relationships and may lead to significant health and behavioural complications.

Table 5: Stage 3: Assessing General Function: Motor, Sensory, Cognitive
Key Questions
Do you have concerns about feeding your baby?
Do you think your baby eats enough?
Does your baby like to be fed?
Do you have to do anything special to feed your baby?
Key Red Flags
"Out of the ordinary" parental behaviours required to feed baby
Feeding taking extended time and/or being very frequent
Infant showing stress behaviours around feeding
Management Options/Suggestions
Early referral to Speech-language Therapist (SLT)

Dysphagia is defined as any disruption to the swallow sequence that compromises the safety, efficiency or adequacy of nutritional intake. It is a skill-based disorder and is distinct from behavioural feeding problems that may arise in children who have sufficient skills for normal eating and drinking[29]. To help distinguish symptoms or behaviours which are more indicative of a significant underlying problem, Barkmeier-Kramer et al identified a set of questions which correlate with the presence of paediatric feeding disorders (included in Table 5)[28].

A clinical feeding evaluation should be conducted in all infants with feeding and swallowing difficulties and will help guide when (and which) further investigations may be appropriate. It will include analysis of observable and audible oral phase skills and swallowing behaviours. Normal swallowing is divided into four main phases: (1) oral phase - sucking, chewing and propelling the bolus toward the pharynx; (2) oral-pharyngeal transit phase - triggering the swallowing reflex; (3) pharyngeal phase – bolus moving through the pharynx and (4) oesophageal phase – bolus moving through the oesophagus to the stomach. Ideally more than one full feed would be observed, assessing each phase of the swallow, any changes during the feed, feeding equipment being used, the infant's position, arousal levels and their physiological state before, during and after the feed. An important part of evaluation includes understanding the parent's perceptions and interpretation of their infant's behaviours during feeding.

Stage 4: Airway Protection with Swallowing

The fourth stage involves assessment of airway protection with swallowing (Table 6). Protection of the airway during swallowing is one of the primary functions of the larynx[30]. The infant larynx is positioned high in the pharynx, with the epiglottis often visible trans-orally (Figure 7)[31]. In this position, the epiglottis optimises airflow directly from the nasopharynx to the glottis and diverts milk directly into the piriform fossae. This adaptation optimises the airway

TTable 6: Stage 4: Assessment of Airway Protection during Swallowing
Key Questions
Does your infant cough/choke or gag easily or frequently during feeding?
Does your infant's voice change or breathing sound noisier or "wet" after feeding?
Does your infant cough frequently, with symptoms that persist over more than 2 weeks?
Has your infant required antibiotics for chest infections/ pneumonia?
Has your infant required admission to hospital for chest infections/pneumonia/breathing difficulties?
Has your infant had a CXR? Was it abnormal?
Has your infant had a feeding assessment by a speech-language therapist?
Key Red Flags
Chronic cough/recurrent chest infections/abnormal CXR
Clinical feeding assessment suspicious for aspiration
Management Options/Suggestions
If concerns that "harm" exists from possible aspiration proceed with investigations
Baseline CXR if not already done
Consider respiratory team referral
Speech-language therapy referral: Clinical Feeding Evaluation
Consider FEES +/or VFSS
Consider baseline overnight oximetry
Consider formal rigid airway endoscopy under a general anaesthetic (co-ordinated with other procedures as appropriate – see Fig. 3)
Consider therapeutic use of gravity (positioning during feeding)

during feeding and minimises the risk of aspiration whilst the infant is developing the skills and coordination required for a safe swallow.

Aspiration is defined as laryngeal penetration of any substance below the vocal cords[32,33]. Aspiration of saliva is thought to occur in around 50% of adults during sleep which is tolerated without apparent consequences if it is of small volume and there is normal immune function and tracheobronchial clearance[34]. It seems reasonable to expect that most infants will probably have (and will tolerate) occasional aspiration of small volumes as part of the normal "learning" process during swallowing. Improved airway protection with normal development probably reflects maturation of laryngeal chemoreceptors related to central neural processing rather than physical changes[35]. These protective reflexes occur as part of the transition

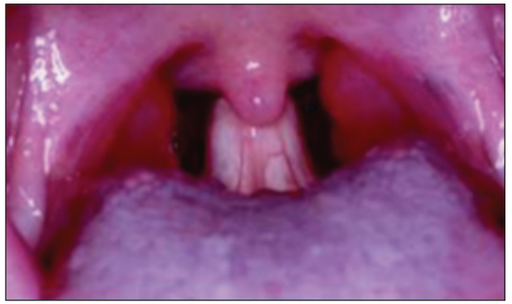

Figure 7: Normal epiglottis: visible on transoral view.

from the aquatic foetal environment, as immature apnoeic and rapid swallowing behaviours develop into a mature cough response for airway protection in the aerobic post-natal environment. Immaturity of these reflexes at birth may explain why infants with no other neurological or medical comorbidities will usually "grow out" of their aspiration with normal development. Unfortunately, there is no normative data regarding the volume or frequency of aspiration that would be considered "within normal limits" for children[36]. Potential for harm is probably related to a combination of: the volume aspirated; what substance aspirated; and individual patient factors that modulate their response to aspiration. As some populations have a much higher risk of developing chronic lung disease such as bronchiectasis[37], it is likely that the volume of aspirate required to cause harm will differ between individuals.

Although coughing or choking during feeds is suggestive of aspiration, there is a high prevalence of silent aspiration in infants, therefore the lack of overt signs does not exclude the diagnosis[38]. The decision to proceed with further investigations should be guided by the presence of clinical signs or symptoms suggesting the lower respiratory tract is compromised. The history may include description of a constant "rattly chest", "wet-sounding breathing", a chronic cough, frequent courses of antibiotics or hospital admissions for pneumonia. A baseline chest x-ray is recommended. When no lower airway harm is evident, the infant can be kept under review without a need for immediate investigation. When lower airway harm is evident, further investigation can confirm if aspiration is present and guide any recommendations to modify oral intake.

Aspiration can take several forms: (1) an anterograde event (during swallowing of a liquid or solid bolus); (2) a retrograde event (when material that has entered the stomach or stomach is refluxed back into the hypopharynx and then aspirated); or (3) aspiration of saliva (see

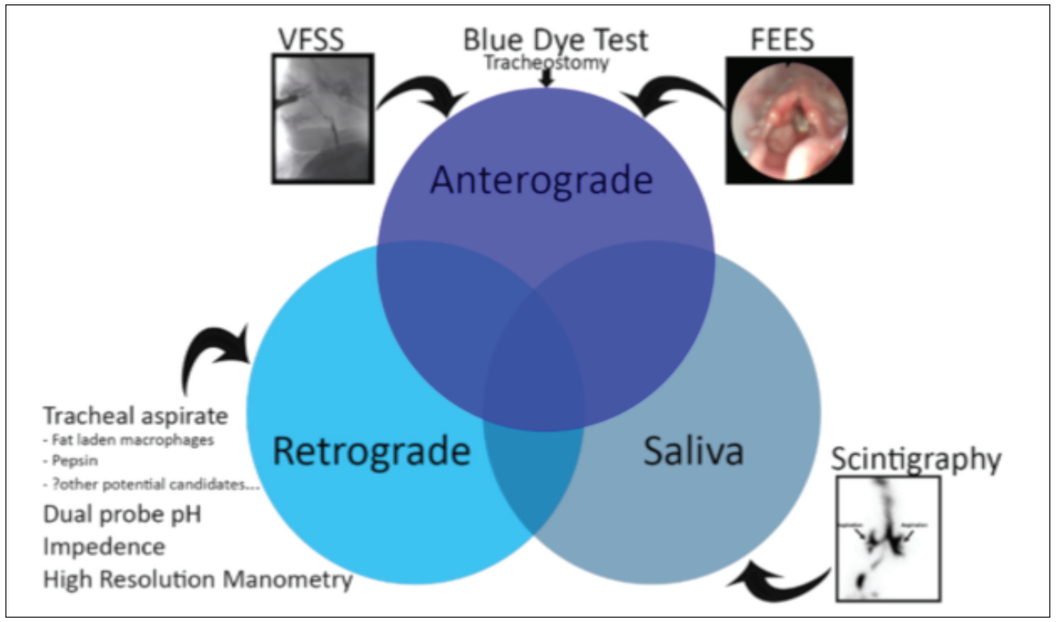

Figure 8: *Investigations for different types of aspiration.*

Figure 8). The key for evaluation for anterograde aspiration is an instrumental assessment of swallowing, which includes; Videofluroscopy Swallow Study (VFSS) and Flexible Endoscopic Evaluation of Swallowing (FEES). Both investigations provide complementary information, with different strengths and weaknesses (see Table 7).

For breastfeeding infants, FEES is the most appropriate investigation. Breastfeeding and bottle-feeding biomechanics differ, therefore VFSS (using a bottle) results cannot be extrapolated to be representative of the infant's swallow when breastfeeding. Remember that both VFSS and FEES essentially capture a snapshot in time and may not represent the range in swallowing that will occur with variables such as fatigue, distraction, or when an infant is unwell.

Other diagnostic procedures for assessment of aspiration may include rigid lower airway endosocopy, flexible bronchoscopy, CT chest and oesophagoscopy. A tracheal aspirate, collected at rigid airway endoscopy, can guide antibiotic therapy if warranted. There is currently no ideal biochemical marker whose presence in tracheal/bronchial aspirate distinguishes between anterograde and retrograde aspiration but there is ongoing research in this area. There is some evidence that in children with impaired swallowing (anterograde aspiration), the lung microbiome shifts toward oropharyngeal flora rather than gastric flora[39]. In the future, diagnosing the nature and severity of aspiration may be aided by the identification of specific bacterial biomarkers in samples from an individual's lungs, oropharynx and stomach.

The clinical feeding assessment, instrumental assessment of swallow together with input from the wider multidisciplinary team provides a general overview of an infant's feeding and swallowing presentation and help guide a plan for safe feeding. If any feeding restrictions are recommended there should be regular re-evaluation, as an infant's swallow and airway protection is expected to change with growth and development. It is recommended that FEES is used preferentially for follow up wherever possible, to limit the cumulative radiation exposure involved with repeated VFSS procedures.

Potential therapeutic interventions to reduce aspiration risk

When aspiration is present, both VFSS and FEES can be used to assess variability in airway protection by modifying bolus size, flow rates and/or consistency (by thickening). Traditionally, if these therapeutic options failed to adequately improve airway protection, an infant would be made nil by mouth and a nasogastric tube inserted. Our experience has shown (using FEES) that by altering

Table 7: Instrumental Assessment of Swallowing: Strengths & Weaknesses of VFSS & FEES

	VFSS	FEES	Both VFSS & FEES
Strengths	High sensitivity for aspiration: therefore considered "Gold Standard" Can assess oral, pharyngeal and oesophageal phases of swallowing	No radiation 3D view of anatomy Dynamic assessment of airway Can assess breastfeeding No alteration to liquids or food Can assess full duration of feed (if tolerated) Can assess saliva management Usually "portable" to location of patient	Compensatory strategies can be trialled and effect documented Capture & playback of video/ images - can give immediate visual feedback for parents
Weaknesses	Involves radiation: • Only a small "sample" of swallows recorded • not ideal for reassessments in short time period 2D view Foreign environment Contrast can alter taste and texture Not suitable to assess breastfed babies Not suitable for assessing saliva management Lack of validated scoring for paediatric patients	Patient tolerance of procedure variable Can stimulate vagal response (apnoea). Monitoring and access to resuscitation equipment recommended, particularly when performing on vulnerable infants Unable to assess oral or oesophageal phase "White out" during swallow – temporary loss of view Lack of objective measures for grading/reporting	Both require: • Time for procedure • Specialised equipment • Specific training/expertise • Patient tolerance/ co-operation Both potentially impact on the infant's behaviour and swallow Both assessments do not necessarily reflect the individual's full range of variability in swallowing that occur in that infant over a broader time Lack of normative data
Team	SLT & Radiology	SLT & ORL	
Location	Radiology Department	Clinic or bedside	

latching/feeding position, gravity can be used therapeutically to modify and improve airway protection during swallowing in some infants.

Laryngomalacia is the most common cause of airway compromise in infants and is often associated with dysphagia[40]. Both VFSS and FEES show high rates of penetration and aspiration in infants with laryngomalacia, with the majority improving or resolving after supraglottoplasty[41]. Infants with laryngomalacia often present with frequent coughing and choking episodes during feeding, necessitating pauses in feeding to recover which can lead to significant difficulties breastfeeding, infant distress or refusal to feed and/or suboptimal weight gain. These infants may improve both their dynamic airway (less stridor) and their ability to cope with milk flow (less choking with feeding) when in a more upright feeding position. With this adaptation, gravity tends to bring the tongue base forward, altering epiglottis position and reducing supraglottic dynamic airway collapse. FEES has shown that this more upright position also minimises the volume of milk flowing into the hypopharynx when the infant has normal pauses in sucking, which in turn reduces the risk of choking and/or aspiration. This is usually described as a "laid-back" breastfeeding position (see Figure 1), with many variations being effective. Lactation consultant input is recommended to assist with attaining comfort and competency using this adapted positioning. Similar positioning can be utilised in infants with low neurological tone, where the tongue base can cause airway compromise when feeding in a more supine (traditional cradle-hold) position.

Infants with unilateral vocal cord palsy have a high incidence of silent aspiration[42]. By positioning the infant side-lying for breastfeeding, with the affected vocal cord always positioned superiorly, we have shown using FEES that gravity ensures milk flow is diverted through the piriform fossa on the contralateral side (which has normal sensation and should elicit a normal cough response). By using this positioning at both breasts, most babies with unilateral vocal cord palsy will be able breastfeed with no compromise of their lower respiratory tract.

Table 8: Summary of recommendations: Infant feeding assessment

A multidisciplinary approach	Patient and family centred Culturally sensitive
Timely assessment	Infant feeding difficulties create extraordinary stress Adequate time allocation
Narrow window of time to establish/protect breastfeeding	Complex nature of feeding and swallowing problems Need for a thorough, methodological approach
Clinical Feeding Evaluation (CFE) by SLT	Cornerstone in evaluation of feeding and swallowing difficulties
Assess for airway compromise	Babies that don't breathe well = babies that don't feed well
Support breastfeeding	Support mothers who wish to breastfeed through early involvement of lactation consultant and tailored feeding positions
Complementary use of VFSS & FEES	Understanding the strengths and weaknesses of each
Understand the impact of gravity	Understand the potential therapeutic use of altering feeding position to modify dynamic airway compromise and improve airway protection with swallowing

Summary and recommendations

Infant feeding can be impacted by a broad range of factors. A systematic four-step approach to assessment ensures that important factors are not overlooked. Assessment should include: (1) assessment of nutritional status; (2) assessment of anatomical structure and function; (3) assessment of general function (motor, sensory, cognitive); and (4) assessment of airway protection during swallowing. Our summarised recommendations are outlined in Table 8. We emphasise the importance of actively supporting the possibility of breastfeeding when this is the mother's aim. New research continues to evolve our understanding in this complex area of clinical care. We embrace a team approach, continuing to learn from each other and from experience and we hope this outline encourages others to do the same.

Acknowledgements

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Implications of childhood obesity for the otorhinolaryngologist

Wai Sum Cho BMedSci, BM BS, MRCS (ENT), FRCS (ORL-HNS) and **Tawakir Kamani** MD, MSc, DOHNS, FRCS (ORL-HNS)
Department of Otorhinolaryngology – Head and Neck Surgery, Nottingham University Hospitals NHS Trust

Correspondence:
Wai Sum Cho
Department of Otorhinolaryngology, Head and neck Surgery
Nottingham University Hospitals NHS Trust, UK

E-mail: sam.cho08@googlemail.com

Abstract
The incidence of childhood obesity is increasing worldwide and has been described as an epidemic by the UK Royal Society of Public Health. This has implications for ear, nose and throat (ENT) surgeons both in terms of the increasing incidence of ENT pathologies secondary to childhood obesity and how obesity complicates their management. This article reviews the management of obese children from the perspective of an ENT surgeon.

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Key words
Obesity, childhood, management, otorhinolaryngologist

Introduction
Childhood obesity is rising in incidence worldwide and specifically in the United Kingdom (UK); which has one of the highest prevalence of obese children in Europe with almost 1 in 5 children leaving primary school obese[1,2]. This is believed to be secondary to a more sedentary lifestyle and increased consumption of a high calorie diet[3]. In children, obesity is classified based on having a body mass index (BMI) above the 95th centile in comparison with other children of the same age and sex[4]. This has significant public health implications as not only children who are obese are more likely to continue to be obese when they reach adulthood, but they are also at higher risk of developing psychosocial and medical issues[5]. For an otolaryngologist, managing obese children could be broadly divided into two main groups; a group where there could be implications when considering surgery in an obese child but also obesity itself leading to ear, nose and throat problems.

General anaesthesia for the obese child
Obese children have abnormal respiratory parameters leading to ventilation/perfusion mismatch and subsequently a higher likelihood of developing perioperative desaturation[6]. If volatile agents are used as part of a general anaesthetic, they may take longer to recover as these agents are known to accumulate in adipose tissue. Sevoflurane and desflurane have shorter recovery compared to isoflorane[6]. Interestingly there is no conclusive evidence that obese children are more difficult to intubate although they are more likely to need multiple laryngoscopies and are less likely to accept mask airways[6]. Considerations need to be taken with regards to dosing of medications for these children due to alteration in the drug volume distribution, metabolism and elimination from the body[7], 8. Initial dosing of medications especially opioids are best based on ideal body weight before being titrated as needed, as there is evidence to show increased respiratory depression and overdose when dose increases are given linearly based on patient's weight[8].

Operating on an obese child
An observational study in a large teaching hospital found that the majority of operations for obese children are being performed by ENT surgeons[9]. Whilst surgical time for performing surgeries such as adenotonsillectomy in obese children is similar to when performed on children who are not obese, there are other considerations that need to be taken into account when listing an obese child for surgery[9]. The potential for developing anaesthetic complications has been covered in the previous section and this could lengthen the time spent in theatre, subsequently affecting theatre turnover. Postoperatively, obese children are also found to be 2.3 times more likely to suffer from a post tonsillectomy bleed and a longer hospital stay[10,11]. For obese children with sleep disordered breathing (SDB), they are also more prone to have persistent SDB post adenotonsillectomy as described in the next section[12].

Obesity and SDB

Paediatric SDB is a condition caused by increased upper airway resistance and collapse leading to hypoxia and interrupted sleep from repetitive arousal[13]. The mainstay of treatment for children with SDB is adenotonsillectomy, which has been shown to be effective in reducing upper airway resistance and curing children of SDB[13]. Obese children have adenotonsillar hypertrophy, increased lymphoid tissue hyperplasia in the tongue base, and external compression of surrounding adipose tissue around the pharynx leading to a higher incidence of SDB in obese children[14,15]. Furthermore, when comparing tonsil size in terms of height, width and weight, Wang et al. (2010) found obese children to have larger tonsils when compared with their leaner counterparts[16]. The association between obese children and having more prominent lymphoid tissue is believed to be secondary to endocrine mediated somatic growth in these children[15]. This may explain why a significant number of obese children remain symptomatic from SDB despite initially showing improvement in their symptoms following adenotonsillectomy[17,18].

A prospective study by Mitchell and Kelly (2004) of 30 children assessed the outcome of obese children with SDB following adenotonsillectomy by performing polysomnography (PSG) pre and post surgery and found that 54% of children continued to have SDB[19]. Interestingly an imaging based study found lingual tissue hypertrophy is more marked in obese children following tonsillectomy which could also partly explain the persistence of SDB following tonsil surgery[14]. However, there are additional factors that could cause persistence of SDB symptoms in obese children following adenotonsillectomy. Obese children are likely to have higher mechanical load on the chest and reduced chest compliance leading to ventilation/perfusion mismatch and increased work of breathing[6,15]. These are hypotheses that this could lead to fatigue and further exacerbate SDB in obese children[15]. The cause of persistent SDB in obese children post adenotonsillectomy is therefore most likely multi-factorial and secondary to multi-level obstruction[18].

In spite of this, there is evidence to show that obese children with SDB will benefit from adenotonsillectomy. Mitchell and Boss (2009) found in their study of 40 children that there was an improvement in sleep parameters based on post-operative PSG and also improvement in the child's quality of life[20]. Adenotonsillectomy however did not improve any behavioural impairment[20]. The American Academy of Pediatrics guideline for management of SDB in children advocates that adenotonsillectomy remains the first choice of initial treatment for obese children in the presence of adenotonsillar hypertrophy[21].

In children who continue to be symptomatic post operatively, a repeat sleep study is indicated. Weight loss is encouraged and there may be a role for continuous positive airway pressure (CPAP) therapy for these children under the care of a respiratory physician[15].

Obesity and hearing loss

There have been observational studies both from a population based study and secondary care providers which showed a higher incidence of otitis media with effusion (OME) and acute otitis media (AOM) in obese children[22,23,24,25]. These children were more likely to have intervention in the form of grommets and repeated visits to the primary care practitioner for recurrent otitis media[22,23,24,25]. There are several theories exploring this association; Kaya et al. (2017) observed the presence of adenoidal hypertrophy in obese children with OME[23]. However, Kuhle et al. (2012) found a clear association between paediatric obesity and otitis media after adjusting for adenotonsillar hypertrophy in their population-based study of over 3000 children[25]. Another theory hypothesizes that adipose tissue causes an increase in pro-inflammatory cytokine interleukin-6 leading to a chronic inflammatory state and middle ear effusion[26]. A previous study into middle ear effusion fluid has shown a high (83%) incidence of increase in interleukin 6 supporting this theory[27]. Other theories include increased adipose tissue build up around the eustachian tube leading to eustachian tube dysfunction or gastroesophageal reflux, which is more common in obese children, leading to otitis media with effusion[28,29,30]. Dewan and Lieu (2018) performed a randomised double blind placebo controlled trial to assess the effectiveness of proton pump inhibitors (PPI) in treating OME but were unable to draw a conclusion due to small sample size[31].

A large cross sectional population based study in the United States found obese adolescents are more likely to suffer from high frequency sensorineural hearing loss and noise-induced hearing loss[32]. The cause for this is not fully understood although obesity is a known independent risk factor for age-related hearing loss in adults[33]. Adipose tissue secretes hormones and pro-inflammatory mediators and this is believed to cause end organ damage leading to hearing loss[33]. There is also a reported association with adipose tissue secreted adiponectin, which is lower in obese patients[33]. In an animal model, lower adiponectin levels were found to be associated with a lower blood flow to the cochlear[34].

Obesity and nasal obstruction

A large cross sectional study performed in France of over 6000 children aged 9-11 found a higher incidence of allergic rhinitis in children with a high BMI[35]. A similar finding was reported by Cibella et al. (2011) who performed a cross sectional study of 10 – 16 year old children in Italy[36]. The reasons for this are not well understood but it is widely reported that obese children are more likely to have asthma, suggesting a degree of atopy[35,36]. Kim et al. (2016) studied the role of diet and association with allergic rhinitis and found in their study of over 3000 children that allergic rhinitis was found to be significantly associated with a high fat and low carbohydrate diet[37]. The above cross-sectional studies performed have demonstrated possible associations, however there were numerous confounding factors and it was difficult to draw a definitive conclusion. For instance, a large multi-national cross-sectional study of more than 10,000 children combining affluent and non-affluent countries did not find an independent association between allergic rhinitis and paediatric obesity[38].

Conclusion

Childhood obesity is rising in incidence worldwide and this group of children are increasingly presenting to otorhinolaryngologists with multiple ear, nose and throat complaints. Greater understanding and awareness of this global issue will lead to a more effective, structured multi-disciplinary management of these children.

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Paediatric reinnervation update

Virginia Fancello[1] MD, **David Allen**[2] BSc MBBS MRCP, **Andrea Burgess**[3] FRCS ORL-HNS, **Hasnaa Ismail-Koch**[3] DLO, DM, FRCS ORL-HNS, **Kate J Heathcote**[4] FRCS ORL-HNS
[1] Laryngology Fellow, Poole Hospital NHS Foundation Trust.
[2] Consultant neurophysiologist, University Hospital Southampton NHS Foundation Trust.
[3] Consultant Paediatric Otorhinolaryngologist, University Hospital Southampton NHS Foundation Trust.
[4] Consultant ENT Surgeon, Poole Hospital NHS Foundation Trust.

Correspondence:
ENT Department, Poole General Hospital, Longfleet Road, Dorset BH15 2JB

E-mails: kateheathcote@gmail.com, virginia.fancello@gmail.com

Abstract
The management of unilateral or bilateral vocal fold palsy is challenging for otorhinolaryngologists. The ideal treatment should aim to restore the respiratory, phonatory, sphinteric and swallowing function without disruption of the laryngeal framework. After encouraging results for both unilateral and bilateral reinnervation in adults, these procedures have been performed in select paediatric patients, with the purpose of offering a long lasting treatment option in comparison with traditional procedures. With this review we hope to encourage further international collaborations and investigation in this field, since the benefits of the techniques appear to be particularly promising in paediatric patients.

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Key words
Vocal fold palsy, laryngeal electromyography, laryngeal reinnervation

Introduction
The availability of the paediatric flexible nasolaryngoscopes has made the diagnosis of vocal fold palsy (VFP) easier among children. Consequently an increased prevalence of paediatric unilateral VFP has been shown[1]. This data is also reinforced by the higher survival rates among pre-term, very low birth weight infants, who frequently require surgical treatments including patent ductus arteriosus (PDA) ligation with an associated high risk of recurrent laryngeal nerve injury resulting in a unilateral vocal fold paralysis.

In the past congenital or acquired infections represented a large share of the aetiologies, but the implementation of vaccination programs and antibiotic treatments has drastically decreased their incidence[2,3,4]. Nowadays iatrogenic left VFP seems to be mainly related to cardiovascular disease[5] with the estimated incidence of iatrogenic palsy following PDA ligation in babies under <1 kg ranging from 22.7 to 67%[6,7]. Right VFP is usually neoplastic or subsequent to a central lesion[8].

Bilateral vocal fold palsy (BVFP) is mainly neurological or idiopathic. Arnold Chiari Malformations are the most frequently associated condition in children diagnosed with BVFP[9,10].

Both unilateral VFP (UVFP) and BVFP are also complications following surgery for congenital tracheo-oesophageal malformations[11]. The early diagnosis of VFP is vital to improve morbidity and mortality in children with long-term respiratory complications following tracheo-oesophageal fistula (TOF) repairs.

However, despite all the heterogeneous information about the aetiology available in literature, the real incidence of VFP in children remains unknown and is therefore probably underestimated.

The management of VFP is challenging for otorhinolaryngologists, since the larynx is one of the main tools for human social interaction and therefore its functional impairment during growth can have dramatic consequences. The ideal treatment should aim to restore the respiratory, phonatory, sphinteric and swallowing function without disruption of the laryngeal framework.

The concept of laryngeal reanimation was developed in the early 1900s, but only in recent decades has reinnervation been applied in humans[12]. After encouraging results from both unilateral and bilateral reinnervation in adults, these procedures have been performed in selected paediatric patients, with the purpose of providing a more permanent treatment option in comparison with traditional procedures. It must be recognised however that the most common causes of unilateral and bilateral VFP in children differ to

those in adults; in adults usually the cause of laryngeal nerve injury is iatrogenic.

Pre-operative work up

In order to consider the option of laryngeal reinnervation, a comprehensive preoperative work up is crucial.

Direct dynamic and static evaluation of the larynx helps to define possible anatomical alterations or structural defects such as crico-arytenoid joint ankylosis, interarytenoid scars, interarytenoid webs or posterior glottic stenosis. A dynamic evaluation also helps to elucidate whether there is a breathing pattern coordinate with respiration and any paradoxical vocal fold movements. These conditions can be related to traumatic or prolonged endotracheal intubation, laryngeal trauma, inflammatory processes, radiation or caustic damage[13,14].

Magnetic Resonance Imaging, and in select cases Computed Tomography, may be necessary to investigate the aetiology and associated pathologies, particularly in cases of bilateral vocal fold palsy. Assessment of cardiorespiratory function is fundamental to plan the treatment and includes specialist paediatric respiratory and cardiac opinion. Chest radiography is important to assess the extent of any pre-existing lung injury and also to assess the diaphragm in cases suitable for bilateral selective reinnervation techniques.

Evaluation of swallowing together with the involvement of speech and language specialists and with videofluoroscopy is helpful to investigate feeding difficulties and aspiration. The assessment of voice is fundamental and should include a thorough assessment working together with paediatric speech and language therapists, to use the most appropriate, accurate tests for each child. Videos are obtained of voice, cough and maximum phonatory times. Paediatric voice related quality of life scores and paediatric voice handicap index scores should also be documented for all children with VFP.

Genetic investigations help to characterise clinical features and diagnose mutations related to neuropathy that can affect possible donor nerves which is especially important when reinnervation is to be contemplated[15].

Laryngeal Electromyography (LEMG), further developed for use in clinical practice by Hirano and Ohala in 1969[16], is a useful examination in children with vocal fold immobility. It can help to determine the degree/extent of denervation, spontaneous reinnervation, synkinesis and muscle atrophy, and in cases where the differential diagnosis between paralysis and fixation is challenging[17].

The examination of both thyroarytenoid muscles and posterior cricoarytenoids (PCA) is needed for a precise diagnosis, in particular in iatrogenic nerve damage, since it has been found that the recurrent laryngeal nerve (RLN) branch to the PCA is more frequently damaged during surgery[18].

Recent studies have shown that in iatrogenic VFP a waiting time of 6 to 9 months since the onset of the palsy may be more appropriate to establish the diagnosis, and confirm whether recovery is likely or unlikely[19].

The data obtained from these investigations is not only crucial in understanding the dramatic impact of vocal fold palsy in children but when considering treatment options. Therefore an early diagnosis of permanent nerve damage is key to plan an adequate management strategy or avoid unnecessary treatment if a possible recovery is expected.

LEMG can be carried out using total Intravenous Anaesthesia (TIVA) and also spontaneous respiration anaesthetic techniques. Simultaneous evaluation and palpation of the larynx can also be performed. Laryngotracheobronchoscopy during the same procedure allows other pathologies to be excluded, including not only the previously mentioned crico-arytenoid joint issues, but also laryngeal clefts which may be associated with TOF, subglottic cysts and stenosis in premature babies who may have had repeated intubations and tracheomalacia in babies with cardiac co-morbidities.

Self-assessment validated parental questionnaires in younger children such as the paediatric voice handicap index, paediatric voice related quality of life, Pediatric Eating Assessment Tool or STEP-CHILD are useful to evaluate the impact of voice and feeding difficulties on quality of life for children and their carers and provide an additional method for evaluating post-operative outcomes[20,21,22].

When the child's cooperation is possible voice analysis and a more accurate maximum phonation time should be recorded.

UVFP

UVFP is characterised by loss of glottic competence and secondary impairment of the actions for which it is necessary, such as voice and cough production, straining and Valsalva's manoeuvre.

Dysphonia is the leading symptom of UVFP, although neonates following PDA ligation may present with a weak cry and aspiration. Efforts to talk may be compromised by a loss of air volume through the glottic gap. In children and teenagers UVFP can represent a risk for social and emotional disturbance and isolation as well as physical and functional impairment[23].

Aspiration pneumonia may result in possible life threatening consequences particularly in pre-term infants with UVFP.

Surgery should be considered when conservative management has not been successful or when there is significant aspiration and dysphonia.

Injection mediatisation[24] should be carefully performed. In newborn infants and children, the vocal fold mucosa is thinner and the different layers are not developed. In addition the ligament is not clearly detectable in children under 4 years. A correct choice of the material (ideally short lasting) and meticulous injection are essential to avoid scarring, granulation or disruption of a growing vocal fold[25].

Type I thyroplasty[26] does not provide adequate permanent results. As the larynx grows, the implant may be displaced or the size may be inadequate. Moreover procedures under local anaesthesia and where patient cooperation is necessary to make fine adjustments, and especially on the larynx are likely to prove exceptionally difficult in a child.

Currently the most widely used and well accepted reinnervation technique for UVFP is based on ansa cervicalis to RLN neurorrhaphy.

This procedure reinnervates the muscles of the hemi-larynx and can restore vocal fold tone, bulk and tension. There is no restoration of movement coordinate with respiration and phonation; hence it is defined as "non-selective". The first satisfactory reports of this technique were published by Crumley in 1986[27,28]. Positive outcomes have also been described by Olson et al. (1998), Miyauchi et al. (1998), Lee et al. (2007), Lorenz et al. (2008), Marie et al. (2010) and Weng et al. (2011) in larger retrospective case series in adults[29,30,31,32,33,34]. All of these reports demonstrated that in adults over 17 years of age non-selective reinnervation could restore the physiological laryngeal phonatory function to near normal/ normal voice quality.

The application of this unilateral non selective laryngeal reinnervation (NSR) technique has been extended to the paediatric population. The first report was by Smith in 2008[35], who in 2015 published the largest report, on a cohort of 35 paediatric patients who underwent this surgery. Although the study revealed no significant association between age at time of surgery and outcome, a slight negative correlation was found with the length of denervation (a longer period of denervation resulting in a poorer outcome), though voice improvement was observed in all patients[36,37]. Zur and her group described their first results in 2012, with promising outcomes in 10 children under 14 years of age[38]. In 2015 Zur and Carroll published a comparison study between non selective reinnervation and injection laryngoplasty in 33 children with dysphonia. The study confirmed the hypothesis of long-term superior outcomes of NSR compared to injection laryngoplasty[39], and thus the group offer NSR as a standard treatment option in appropriately selected children. Furthermore in 2017 the same group reported on three paediatric patients affected by UVFP who underwent successful NSR for aspiration primarily with concomitant dysphonia[40]. Following comprehensive assessment, multidisciplinary discussion and encouraged by the results in the literature, the indications of dysphonia and aspiration causing significant medical and psychosocial issues resulted in the authors performing the first NSR in the UK in a 6 year old affected by UVFP, with successful outcome[41]. In all of the documented cases in the literature good outcomes have been reported, with resolution of aspiration and improvement of voice. Our team has successfully embraced this procedure, only recently introduced into the UK, with good preliminary results[42]. This technique has been offered in a fully evaluated structured way through the development of a management algorithm (Figure 1) with the aim to establish the prognosis and plan best treatment[43].

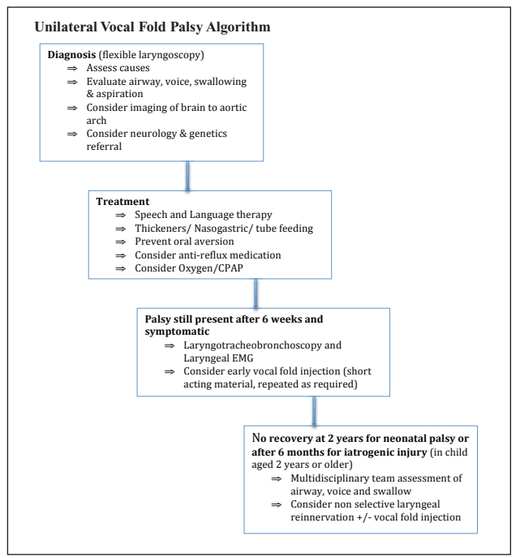

Figure 1: *A management algorithm for paediatric unilateral VFP.*

Table 1. A summary of the available evidence for NSR performed in children for unilateral VFP.

Author	Year	Number of patients	Findings
Ongkasuwan J et al.	2019	32	NSR performed at a younger age may have better voice outcomes compared to NSR performed 1 to 2 decades later.
Faoury M et al.	2019	1	The use of laryngeal EMG is crucial to predict outcomes and to choose the best treatment option. NSR may provide a permanent solution and should be considered in children as a management option.
Zur KB et al.	2017	3	NSR is a safe and effective option for the management of chronic aspiration pneumonia and dysphonia in patients with UVFP.
Farhood Z et al.	2015	3	There was statistically significant improvement in shimmer and Noise to Harmonic Ratio (NHR).
Smith ME et al.	2015	35	Denervation duration showed a slight negative correlation with postoperative outcomes. Voice improvement was seen in all patients.
Zur KB et al.	2015	33	The ANSA-RLN group showed better and longer-lasting perceptual and acoustic parameters in comparison with the injection and control groups.
Marcum KK et al.	2010	2	NSR previously described for older children and adults can be safely adapted for younger children (3 and 6 years old).
Crumley R et al.	1991	1	Improvement of pitch control in 8 years old patient treated with NSR.

Our group has now performed NSR in 5 paediatric patients, 2 male and 3 females, age range 2 to 16 years old, affected by iatrogenic UVFP. The postoperative outcomes in 3 have revealed a significant improvement in both voice and swallowing parameters and quality of life scores. In one case there has been no improvement following NSR and on reflection it is likely that this was due to a modification of the ansa nerve selection, therefore a revision procedure may be considered. We await the results of NSR following the most recent surgery. Table 1 summarises the findings of the recent literature following NSR in children. The procedure is currently offered to carefully selected children at our institution following a thorough multidisciplinary panel assessment and review.

BVFP

BVFP usually presents with life threatening stridor in neonates and 50 % of these children require a tracheostomy. Paediatric and laryngology specialists from our region have developed a prognostic and therapeutic algorithm for children with BVFP (Figure 2), to aid management in this often psychosocially complex group of children[43].

In addition to tracheostomy other surgical procedures may be considered for BVFP and include glottic widening procedures, such as lateralising sutures, cordotomy or arytenoidectomy. If BVFP is an option these techniques should be avoided as they result in permanent disruption of the laryngeal framework, with voice impairment and preclusion of a possible reinnervation.

Selective Laryngeal Reinnervation (SLR), as described by Professor Jean-Paul Marie[43], has the aim of restoring vocal fold movement coordinate with breathing and phonation. A root of the phrenic nerve acts as the donor for both posterior cricoarytenoid (PCA) muscles, with the goal of producing an inspiratory trigger on inspiration and consequent abduction of the vocal folds. The adductor muscles are reinnervated from a small branch of the hypoglossal nerve, active on phonation and swallowing. Both the recurrent laryngeal nerve and phrenic nerve are active on inspiration[44,45]. This specific function can be mutually beneficial in nerve reanimation surgery. This has been supported by a recent report in which the RLN has been successfully employed as a donor nerve in a patient with a diaphragmatic palsy[46]. This provides further validation to the theory on which the sophisticated steps of reinnervation are based. Marie has performed this technique in both paediatric and adult patients with promising results[47]. Other authors have published good outcomes in the adult population[48,49].

Concerns may be expressed regarding possible permanent damage to the phrenic nerve in a child. As such a comprehensive respiratory assessment is essential prior to proceeding with the surgery. In adults the entire phrenic nerve is commonly used to restore shoulder abduction in brachial plexus injury without major problems. In children there are series that describe the safe use of a phrenic nerve root for the same purpose without significant respiratory impact[51].

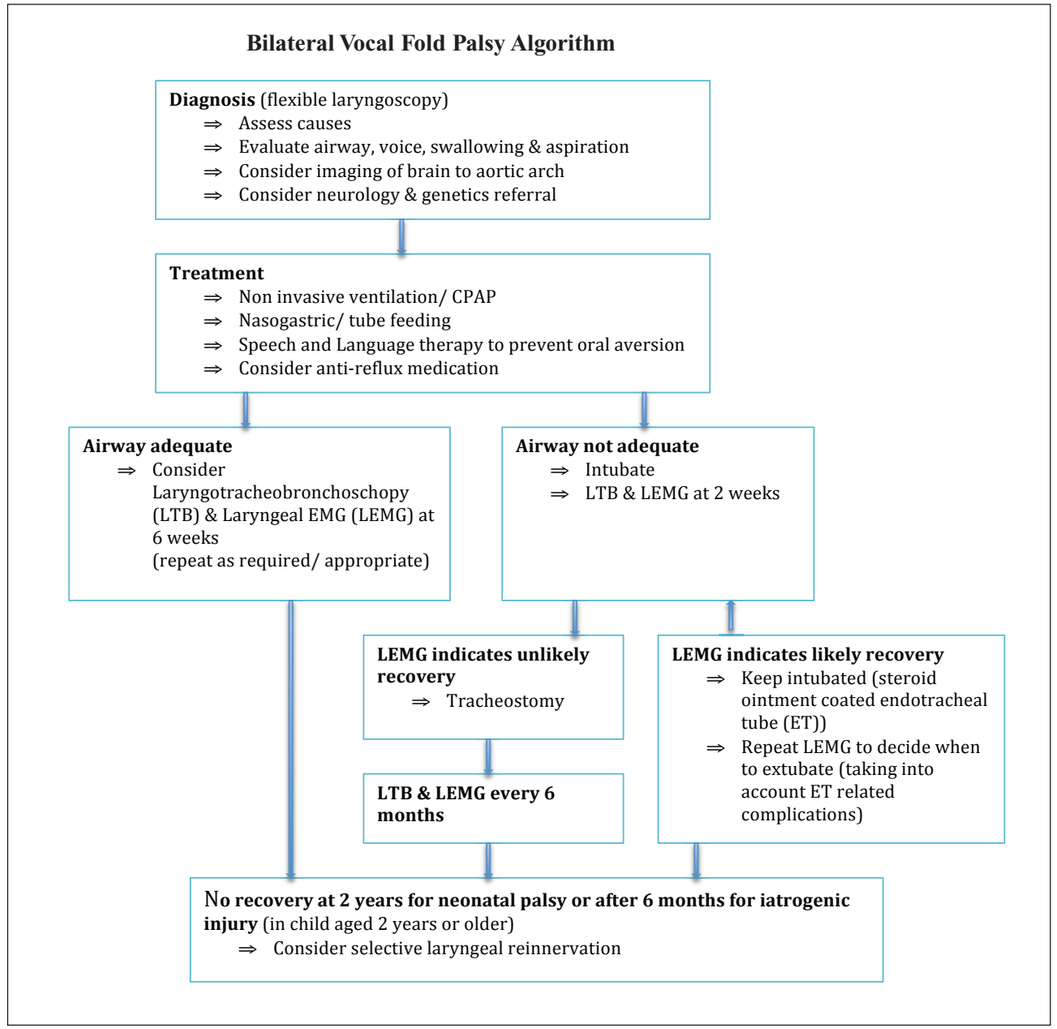

Figure 2: *A management algorithm for paediatric bilateral VFP.*

In his case series Marie (2018) described three selective laryngeal reinnervation procedures performed in children under 3 years of age[48]. Two of the children had idiopathic congenital BVFP and one iatrogenic BVFP; secondary to the cervicothoracic removal of a lymphangioma. Postoperatively the first two children have been decannulated and one has greatly improved with only slight stridor on demanding exercise. Selective reinnervation has also been performed in a 17 year old boy. He recovered bilateral inspiratory abduction and is now able to exercise without dyspnoea.

This procedure forms the basis of laryngeal reanimation and hence rehabilitation. In particular the benefits among paediatric patients can be impressive if restoration of physiological respiration and phonation is achieved. Further data and research is required in order to establish the most suitable children and best methods for

reinnervation techniques in order to obtain optimum reproducible outcomes.

Conclusion

VFP when symptomatic is associated with a high morbidity rate and often has a significant negative impact on the lives of children and their families. Therefore the best treatment options are mandatory. Laryngeal reinnervation, unilateral or bilateral, is sophisticated surgery with potential beneficial effects likely to be especially effective in the paediatric population, in particular when taking into account the rate of nerve regeneration in children compared with adults. This technique can be taught and performed by laryngologists and head and neck surgeons with microsurgical skills. However, these techniques have not yet been extensively embraced, and have only recently been introduced into the UK in both adults and children. Further studies in this field are crucial and an increased worldwide interest and collaborations in this approach are desirable, in particular when reporting techniques, data and outcomes.

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Crico-arytenoid joint fixation in juvenile idiopathic arthritis

Mohamed Shaker[1] MD, ENT Fellow, **Steven Frampton**[2] PhD, FRCS ORL-HNS, **Jonathan Buckland**[3] FRCS ORL-HNS, **Alice Leahy**[4] BA, MBBS, FRCPCH, MSc Cert Med Ed, **Hans de Graff**[4] MBBS, CCT in paediatrics
[1] University Hospital Southampton NHS Foundation Trust, UK.
[2] Locum Consultant ENT surgeon, Poole Hospital NHS Foundation Trust, UK
[3] Consultant ENT Surgeon, Queen Alexandra Hospital, Portsmouth, UK.
[4] Consultant Paediatric Rheumatologist, Southampton Children's Hospital, University Hospital Southampton NHS Foundation Trust, UK.
[5] University Medical Centre Utrecht, the Netherlands, Consultant Paediatric Rheumatologist, Southampton Children's Hospital, University Hospital Southampton NHS Foundation Trust, UK.

Correspondence:
Mohamed Shaker, ENT Department, University Hospital Southampton
Tremona Road, Southampton, UK. SO16 6YD

E-mail: shaker.orl@gmail.com

ABSTRACT
JIA represents the commonest rheumatic disease in the paediatric age group and may involve the cricoarytenoid (CA) joints resulting in stridor and potentially life threatening airway compromise due to bilateral CA joint fixation. Conversely in the paediatric population stridor as a presenting feature of JIA is rare with only a few cases reported in the literature. Otolaryngologists need to be aware of this potential diagnosis as multidisciplinary care is required for optimum long term follow up.

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Key words
Juvenile idiopathic arthritis, crico-arytenoid, fixation, stridor

Introduction
Juvenile idiopathic arthritis (JIA) (previously termed juvenile rheumatoid arthritis) is a term frequently given to chronic arthritis of unknown cause occurring in children younger than 16 years old and persisting for at least 6 weeks duration[1]. Diagnosis depends mainly on the exclusion of other causes of chronic arthritis[2]. JIA represents the commonest rheumatic disease in the paediatric age group with an incidence of 5 to 18/100,000 per year[3].

The recently (2019) proposed classification for JIA identifies 6 subtypes including: systemic JIA, rheumatoid factor positive JIA, enthesitis/spondylitis-related JIA, early-onset ANA-positive JIA, other JIA and unclassified JIA[3]. The first subtype (systemic JIA) affects approximately 5–15% of the general paediatric population in North America and Europe[1]. Systemic JIA is the commonest subtype and is characterised by daily fevers of unknown origin that may reach 39°C or higher once daily then decrease to 37°C or less between fever spikes. Fevers should persist for at least 2 weeks and be associated with 2 major criteria or 1 major criterion and 2 minor criteria in systemic JIA[3]. Arthritis and evanescent rash, are considered major criteria. Minor criteria include arthralgia for a duration of 2 weeks or longer (without associated arthritis), serositis, leucocytosis to 15,000/mm^3 or more with neutrophilia and generalised lymphadenopathy and /or splenomegaly and/or hepatomegaly[3].

JIA can involve any joint in the body, however there is a predilection for large joints[2]. Hand and feet small joints and ossicular joints in the middle ear may also be involved in the disease process[2].

Although involvement of the cricoarytenoid joint in the disease process is unusual, it can be a manifestation of early JIA[4,5]. In 50 cases of JIA, Abdel-Aziz et al. (2011) reported that cricoarytenoid arthritis was detected in 12% of the cases[4].

Aetiology
The cricoarytenoid joint is a synovial diarthroidal articulation between the cricoid and arytenoid cartilages[2,6]. The inflammatory process may ensue in the synovial membrane, then proceed to the articulating surface with resultant fibrosis and subsequent fixation[6].

Clinical Features
Upper airway symptoms in children with JIA include sore throat, hoarseness, dysphonia and phonatory disturbances[2]. Progressiively worsening symptoms including dyspnoea and inspiratory/expiratory stridor which may require intubation and mechanical ventilation may ensue. The disease process is usually more acute in children when compared with adults[2]. While adults can tolerate cricoarytenoid joint inflammation, children tend to present in a more urgent fashion, often necessitating intubation[2]. This is attributed to a narrower glottic opening with larger arytenoids and a higher amount of loose areolar tissue in children allowing inflammatory oedema to spread around the epiglottis and arytenoids[2,7]. Adults have less severe disease which does not usually result in airway compromise[2].

Cricoarytenoid arthritis should be considered in every JIA patient with chronic stridor and laryngeal obstruction[1,2,3]. Conversely cricoarytenoid arthritis may be the first sign of the disease, as reported in Case 1 preceding peripheral arthritis. JIA may also present with a maculopapular rash in addition to the joint pain, stridor and dyspnoea[2,3].

Diagnosis
Diagnosis of JIA is made mainly by eliminating other causes. History including familial history with emphasis on joint pain, morning joint stiffness, complete physical examination and documentation of any pathological features should be elucidated. The commonest manifestation of JIA is a unilateral swollen knee[9]. Inflammation of the synovial membrane, that is not always identical, may additionally involve other small and/or large joints[9]. Examination of the knee, wrist and ankle is essential in cases of rheumatoid factor positive JIA. Skin examination may show subcutaneous nodules. Hand examination may reveal boutonniere and swan neck deformities. Torticollis due to cervical spine involvement can also be seen. Asymmetric growth may ensue in advanced cases[9].

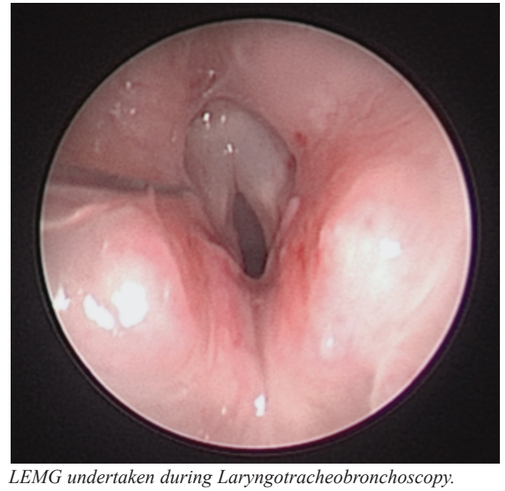
Examination of the oral cavity may show decreased oral opening, and micrognathia due to temporomandibular joint involvement. Paediatric hearing tests can detect conductive hearing losses due to ossicular joint involvement. Hoarseness due to cricoarytenoid joint involvement may also be heard[9]. Systemic examination may reveal pericarditis or myocarditis, splenomegaly, and lymphadenopathy especially in children with systemic JIA[9]. The diagnosis of JIA requires a thorough multidisciplinary assessment with attention to detail as the presentation may be subtle.

X-rays are the gold standard investigation for the detection of structural joint damage, growth and maturation disturbances of bones in JIA patients. However, sensitivity is low in detecting active synovitis and erosive changes in early stages of the disease. MRI is preferable to assess all features of synovial disease and is superior to conventional radiography in its ability directly visualise synovitis, cartilage, and early erosive lesions[8].

Laryngotracheobronchoscopy (LTB) with cricoarytenoid joint palpation and additionally laryngeal electromyography (LEMG) may be diagnostically helpful in cases of JIA. LTB with the expert use of a laryngeal probe allows the otolaryngologist to assess the extent of fixation. LEMG

Case 1:

A previously well 2½ years old girl developed progressive stridor over 5-6 weeks. Following critical airway compromise and unsuccessful medical management she was urgently intubated. On extubation she developed increased nocturnal work of breathing. Rigid laryngotracheobronchoscopy (LTB) revealed reduced movement of the vocal folds. Systemic steroids helped but she experienced multiple minor relapses and developed a painful knee joint. Magnetic resonance imaging (MRI) of the brain and laryngeal nerves and neurology assessment were unremarkable. Repeat LTB with laryngeal electromyography (LEMG) revealed reduced mobility of the cricoarytenoid joints but action potentials from the posterior cricoarytenoid (PCA) muscles. A diagnosis of juvenile idiopathic arthritis was confirmed. Treatment of both the knee and cricoarytenoid joints with steroid injections was performed intraoperatively. Postoperatively she was commenced on oral steroids which were converted to etanercept and methotrexate. She remains relapse-free after 21 months.



LEMG undertaken during Laryngotracheobronchoscopy.

allows assessment of the action potentials generated by the thyroartenoid (TA) muscles and posterior crico-arytenoid (PCA) muscles during respiration, hence demonstrating laryngeal nerve activity and aids diagmosis in terms of differentiating between CA joint fixation and a vocal fold palsy. Airway assessment under spontaneously breathing general anaesthesia also allows other pathologies both structural and dynamic to be excluded.

Management

Treatment of JIA includes a variety of drugs. The most commonly used are non-steroid anti-inflammatory drugs (NSAIDs), including ibuprofen, indomethacin, tolmetin and naproxen sodium. Other anti-inflammatory drug options include methotrexate and corticosteroids. Several case reports comment on effective treatment with non-steroidals[2,3].

Methotrexate 0.5-1mg/kg/week has improved the disease course significantly in JIA as well as in rheumatoid arthritis[8]. However, its effect may be delayed. The addition of 1mg/kg/day folinic acid or folic acid is advised to lower the effects on the bone marrow and control the side effects[8]. Oral or parenteral administration of steroids results in significant improvement of pain, swelling, sensitivity in the joints, carditis, hepatitis and lung disease. Biological drugs are used more in adults, however, recently there are reports of their recommended administration in children with limited usage. Etanercept, Adalimumab, Kanakinumab, Rilonacept, Tosilizumab and Rituximab are examples of these drugs which have been approved by the food and drug association (FDA) for JIA[8]. These drugs act mainly against Tumor necrosis factor-alpha (TNF-a). TNF-a is a cytokine which is directly involved in the pathogenesis of JIA and presents in high level in the serum and synovial fluid[8]. Etanercept is a dimeric fusion protein inhibiting human TNF receptor. It is the first treatment choice in patients with rheumatoid factor positive JIA. The main drawback is the local reaction at the injection site.Consequently it is better to administer the drug in multiple sites. Adalimumab is a human monoclonal antibody which inhibits TNF-alpha. Use of this drug in conjunction with methotrexate markedly accentuates its efficiency[8]. Kanakunimab is a monoclonal IgG1 antibody that acts as isoform of interleukin-1β. It is main advantage is that it does not cause much reaction at the injection site compared to other drugs. In addition it has a longer half-life when compared with other medications. Side effects include abdominal pain, vomiting and diarrhoea. Biological drugs have proven safe and effective in the treatment of JIA. Their usage has resulted in the reduction of the use of steroids and surgical intervention for JIA. Their limited usage is due to the lack of information regarding their long-term safety profile and cost, though they are effective drugs[8]. JIA is a complex disorder that needs multiple medications to achieve symptomatic control. The combination of anti-inflammatory drugs, and biological drugs should be made according to the severity of the condition[8].

Prognosis

Disease activity is rated based on a visual analogue scale (VAS), ranging from 0 (no activity) to 10 (maximum activity)[8].

The outcome for children with JIA is unpredictable. Pending no flare-ups during the first 5 years, the probability of permanently restricted joint mobility and cumulative joint damage is low. Aggressive medical management to achieve disease remission, is a key element to achieve a better outcome. A good preliminary response to medical treatment within the first 6 months, is an indicator for improved long term outcomes[5]. Early initial treatment is essential to prevent long lasting disability and complications of the disease[10].

Conclusion

JIA should be managed in a multidisciplinary team setting including paediatric rheumatologists, paediatricians, physiotherapists, ophthalmologists, and orthopaedic surgeons. Cricoarytenoid arthritis should be considered as a diagnosis in children presenting with chronic stridor, especially if JIA is present. Cricoarytenoid arthritis can be steroid-responsive, mimicking croup. LTB with cricoarytenoid joint palpation and LEMG can be diagnostically helpful. The mainstay of treatment in JIA is to achieve inactive disease or disease suppression without continued anti-inflammatory medication.

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Paediatric dysphagia: The role of the speech and language therapist

Jamie-Leigh Allen[1], Julia Robinson[1] and **Zoe Sherlock[2]**
[1] Paediatric Dietetic/ Speech and Language Therapy Department, Southampton Children's Hospital, Hampshire, UK
[2] Adult Dietetic/ Speech and Language Therapy Department, University Hospital Southampton, Hampshire, UK

Correspondence:
Jamie-Leigh Allen MRCSLT, RegHCPC
Paediatric Dietetics/ Speech and Language Therapy Department,
Southampton Children's Hospital,
University Hospital Southampton, Tremona Road, Southampton, Hampshire, SO16 6YD

E-mail: Jamie-Leigh.Allen@nhs.net

Abstract
We discuss the role of the Speech and Language therapist (SLT) in dealing with infants and children with swallowing disorders. This paper will outline the benefits of the holistic approach of the speech and language therapy swallow assessment and how it complements the ear, nose and throat (ENT) management in the multidisciplinary team (MDT) setting. This is highlighted by a case report.

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Key words
Speech and language therapist, dysphagia, MDT, FEES, VFSS

Introduction
Paediatric dysphagia refers to babies and children with difficulties chewing, sucking and swallowing food, drink and saliva[1]. Dysphagia or feeding difficulties can present at birth or as a child progresses to larger volumes of liquids, during weaning and throughout their life following an event or change in medical status. Feeding concerns may be raised by parents or various healthcare professionals including midwives, health visitors, general practitioners (GPs), nurses and paediatricians. On going dysphagia can be associated with aspiration pneumonia, malnutrition, failure to thrive and potentially neurodevelopmental problems[2,3].

The SLT plays an integral role in the assessment, diagnosis and management of infants and children with dysphagia. Often children with dysphagia are referred to Speech and Language therapists (SLTS) who provide a holistic approach to the swallowing assessment. In the UK, SLT services are generally arranged in two tiers, hospital based and community based SLTS. There are NHS hospital based specialist paediatric SLTS who manage the swallow and communication needs of complex, acutely unwell or neurologically impaired inpatient and outpatient children. Close liaison between the services is essential to ensure continuity of care, particularly as feeding support may be required throughout childhood particularly as feeding skills and safety of swallow may change. The main focus of the initial assessment is to undertake a detailed case history and determine if there are any anatomical, physiological, neurodevelopmental or sensory and associated behavioural difficulties with eating and drinking at the pre-oral, oral and/or pharyngeal stage of swallowing.

The SLTS adopt a very holistic approach to feeding emphasising the need for an appropriate setting, neurodevelopmental skills as well as oro-pharyngeal motor skills. Observation of feeding is essential and the use of fibreoptic endoscopic evaluation of swallow (FEES) to look at the pharyngo-laryngeal phase has become increasingly useful as part of the initial assessment. Videofluoroscopic swallow study (VFSS) provides further valuable information but has the risk of radiation exposure so is not generally used in neonates and is restricted to specific cases.

SLT Assessment of swallow
General assessment
A child or an infant refusing or unable to feed is a very emotive situation and it is important to provide a supportive atmosphere and minimise stress for the child, carers and the child's wider support network e.g. school, nursery and playgroups. Introducing fun into feeding may help children who have had negative feeding experiences (e.g. related to medical conditions, force feeding, and lack of experience with oral feeding/ long term alternative methods of feeding) or are demonstrating oral aversive behaviours[4,5,6]. Optimising positioning through supportive seating and postures for children with poor core strength or reduced head control is essential when optimising feeding skills[7,8].

Observing a child's self-feeding skills (where appropriate) and encouraging them will assist with successful feeding and further development of their pre-oral and oral skills. Children with specific pharyngeal dysphagia may benefit from manoeuvres such as chin tuck, head turn or other compensatory strategies which may reduce the risk of aspiration however these should be assessed during the instrumental assessment.

Oral Phase
SLT assessment of oral stage involves offering the child a range of appropriate textures and consistencies while observing lip closure, lip, tongue and jaw movement including mastication, oral transit time, sucking which includes suck type, rhythm of the suck, suck-swallow-breath pattern and suck burst length/ duration subsequently relating these skills to the developmental stages of feeding.

Assessment of swallow
The pharyngeal phase of swallow is initiated at the point where the swallow reflex is triggered. The SLT may comment on the hyolaryngeal movement (elevation and excursion/ anterior tilt), timing of swallow trigger, number of swallows per bolus, wet sounding voice/ breathing post swallow and adverse signs of aspiration/ penetration which may include coughing, eye watering, gagging and colour change. Cervical auscultation may also be used when commenting on the phases of swallowing and swallow breath pattern. Cervical auscultation is part of the SLT's bedside assessment which can include palpation of the larynx, observation of saturation levels, heart rate and respiratory rate during feeding[9,10]. Where a bedside assessment may not provide full information of the child's dysphagia the SLT may recommend further instrumental assessment including FEES/ VFSS.

Fees
The Royal College of Speech and Language Therapists (RCSLT) position paper[11] describes the use of FEES in the SLT's assessment and management of dysphagia within paediatrics as well adults. The purpose of FEES can be to; diagnose dysphagia and assess the nature of the problem along with guiding dietary and behavioural management.

Although in adults the SLTS often perform the FEES independently of ENT surgeons, the complex developmental nature of paediatric swallow benefits from an MDT approach. The SLT's role during FEES is to assess the swallow function with a range of appropriate textures/ consistencies and provide recommendations regarding the swallow, whether oral feeding is advisable and whether any interventions are required to facilitate safe and efficient feeding. Observations can also be made on the child's neurodevelopment and the skills seen at the pre-oral (self-feeding skills versus being fed), oral stage (developmental stages of mastication, oral transit time, sucking which includes suck type, rhythm of the suck, suck-swallow- breath pattern and suck burst length and duration) as well as the pharyngeal stage of the swallow (see below). SLT's can also provide developmentally appropriate recommendations and goals for children, their family/ carers and for community SLTS including school teams.

During the pharyngeal phase, the following observations are made:

- The appearance of the tissues, base of tongue, velum, nasopharynx at rest and during swallow.
- Potential nasal obstruction causing mouth breathing.
- Any asymmetry of main structures.
- Presence of copious secretions (studies have shown pooled secretions have high correlation with aspiration[12]). We may describe them (foamy, thick, capacious). Amount of standing secretions: normal, excessive. Pooling within the laryngeal vestibule.
- Observing the movement and sensation of critical structures within the hypopharynx and larynx at rest and on swallow, any vocal fold immobility or laryngomalacia.
- Secretions during swallowing. Is there any evidence of overspill into the subglottis? How does the child react to the secretions? Are there spontaneous swallows to clear, ineffective attempts to clear or no attempt to clear these secretions?
- Is there evidence of airway protection during the swallow?
- Directly observed laryngeal penetration or aspiration.
- Can we improve the swallow by using a change of posture/position, flow rate of fluids, consistency of the diet and fluids, utensils and/ or bolus volume?

Following the FEES, the MDT often reviews recorded images and a feeding strategy developed with the family. This is integral as the family/ carers can understand the reasoning behind the recommendations given and feel involved in the decision making. Along with discussions with the family/ carers the SLT in the clinic will often hand over the recommendations to the child's community SLT who will be able to support the family/ child at home. Onward referrals may also be discussed and requested following the FEES clinic for example referrals to occupational therapy (OT), physiotherapy (PT), dietetics, gastroenterology and neurology. When determining if a

child is suitable for FEES a joint discussion with ENT/ SLT/parent/carers will occur and may include all or some of the following: some/ all of the wider range of medical teams involved with the child, the nursing team looking after the child, paediatricians, respiratory teams and cardiac teams. The parent is prepared as best as possible and is given the option to preferably stay or alternatively leave the room for the period of the assessment.

Strengths for using FEES within our clinical setting (inpatient and outpatient):

FEES allows the child to be assessed in a number of settings such as inpatient wards or outpatient clinics. FEES can be used safely and reliably when assessing laryngeal and pharyngeal aspiration in NICU infants (38 weeks+ gestational age)[13]. FEES enables assessment of very young children and neonates in a more natural position such as the parents arms, whilst breastfeeding, with which there is no other instrumental assessment of swallows that exists for this population[14]. Unlike with VFSS there is no "screening" time constraint with a FEES assessment that has proven helpful in determining issues that may be evident during the middle or end of a feed.

VFSS versus FEES

FEES is a very effective investigation that can be used in the inpatient and outpatient setting to provide useful advice regarding swallow. Its main limitations are that in our experience some children often age 2-7 find it difficult to tolerate and it does not examine the oesophageal phase of swallow[15]. VFSS examines all 4 phases of swallow and can be used to detect reflux and site of aspiration. VFSS however involves ionising radiation exposure therefore there are screening time constraints and repeated tests are restricted. In complex patients however, FEES and VFSS are complementary.

The following case is used to highlight the integral role the SLT has within the FEES assessment along with the repeatability and flexibility of the FEES assessment itself.

Case Study:

A 6 week-old baby girl was referred from the local ENT team to the tertiary children's hospital ENT clinic with a history of stridor post-bottle feeding, poor weight gain and nasogastric (NG) feeding. The differential diagnosis given was reflux, laryngomalacia, low tone or an underlying laryngeal cleft. There was no SLT involvement noted. Her past medical history included; term birth, 22q duplication, hypotonic, stridor, poor feeding and failure to thrive.

Her initial examination was unremarkable however laryngeal cleft could not be excluded on fibreoptic nasolaryngoscopy alone. She was commenced on anti-reflux medication, referred to a dietician with a plan for follow up by community SLT.

On review a month later, community SLT had advised pacing with bottles and to use the NG tube when she was not managing full amounts, coughing, spluttering, increased work of breathing, turning head away or getting fatigued. ENT referred to the joint ENT/SLT clinic for follow up.

During joint ENT/SLT initial clinic at age four months, parents reported that she has had four episodes of "bronchiolitis" but had been slowly continuing with bottles (with pacing), NG top ups and tasters of apple puree.

On FEES examination stertor was noted and the uvula was sucking in posteriorly to posterior pharyngeal wall. No vocal cord palsy or obvious stridor was detected. Increased secretions were noted at valleculae and pyriform fossa, frank aspiration seen with milk from the fast flow bottle (unable to trial slower flow teat due to reduced oral strength). No frank aspiration seen with apple puree from weaning spoon therefore recommended:

- NBM for milk (all via NG tube)
- IDDSI (international dysphagia diet standardisation initiative) level 4 (pureed)16 tasters to develop oral skills
- SLT suggested review of supportive seating by OT's
- Handover to community SLT service for ongoing therapy for hands to mouth play and oral skill development

At Joint ENT/SLT follow up clinic two months later (aged six months old) parents report that she has been managing her purees "very well" with improved head control, and now has supportive seating from OT. On going chest infections could be related to 22q immunodeficiency but chest x-ray showed right upper lobe shadowing suggesting possible aspiration (intra-swallow) or reflux. VFSS is now planned if the chest does not improve. Repeat FEES showed fatigue following IDDSI level 4 testing therefore not assessed with fluids. IDDSI level 4 revealed appropriate base of tongue movement and pharyngeal wall contraction, vocal cords normal, some nasal secretions noted however cleared on swallow, appropriate swallow trigger with IDDSI level 4 diet with no residue, no aspiration or penetration. Recommendations for her:

- Continue to promote oral intake of IDDSI level 4
- Referral back to her paediatrician for review of immunodeficiency
- Continued development of her gross and fine motor skills for sitting and feeding along with continued community SLT input.

Joint SLT/ ENT follow up clinic: seen at nine months of age, parents report increased puree intake, ongoing NG feeding but now able sit up with independent head control. FEES assessed her safe with IDDSI level 1 fluids which are now to be encouraged.

VFSS summary: penetration with IDDSI level 1 fluids but no frank aspiration seen. She therefore continued with IDDSI level 1 fluids using an open cup and regular pacing with single sips.

Conclusion

Joint ENT/ SLT FEES clinics enable ENT and SLT teams to develop robust and comprehensive services for children with dysphagia. Clear decisions regarding feeding strategies may reduce hospital stays, time with NG feeding and subsequent ongoing difficulties with adequate nutrition and hydration. The SLT's role enables the child to receive a holistic approach to feeding and increases communication with secondary care and community based teams regarding recommended input to develop fine and gross motor skills for feeding (for example seating/ hand to mouth play, weaning to solids advice and developmental appropriate feeding skills and thickener). The use of FEES and intermittent VFSS is also instrumental in the assessment and subsequent development and review of safe feeding strategies. This integrated care system across a network of providers offers new perspectives on feeding and the management of feeding throughout the child's life.

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Complications of dermal fillers

Dr Evelina Russell, MBChB BSc (Hons), FY2 Doctor, University Hospital Lewisham, London UK
Dr Aarthi Ravishankar, MBBS BSc (Hons), GPST2, University Hospital Lewisham, London, UK
Mr Alwyn D'Souza, FRCS (ORL-HNS), IBCFPRS, Consultant Facial Plastic Surgeon, University Hospital Lewisham, London, UK
Mr Anil Joshi, FRCS (ORL-HNS), IBCFPRS, Consultant Facial Plastic Surgeon, University Hospital Lewisham, London, UK

Correspondence:
Dr Evelina Russell
Education Centre, University Hospital Lewisham
Lewisham High Street,
Lewisham,
London SE13 6LH

E-mail: evelina.russell@nhs.net

Abstract
Facial rejuvenation is evolving rapidly and the use of injectable dermal fillers has been increasing. Although generally well tolerated, both short and long term complications can occur and can be serious. The main goal is to prevent them; however, this may not always be possible and good understanding of the management of complications is imperative for anyone performing the procedure.

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Key words
Injectable, dermal fillers, complications, management

Introduction
The use of injectable dermal fillers, which can temporarily eliminate facial lines, rhytides and defects, has become increasingly popular with patients seeking facial rejuvenation. In recent years, the number of providers offering dermal fillers has rapidly increased owing to the relative ease and lucrative nature of the procedure. Although the procedure is minimally invasive and generally has a good safety profile, complications can arise.

Adverse effects of dermal fillers tend to be minor and localised; however severe complications can occur. Complications can be divided into early and late and range from bruising to necrosis.[1,2] There are now over 60 dermal fillers available on the UK market and all have the potential to cause complications. They include hyaluronic acid (HA), poly-L-lactic acid, calcium hydroxylapatite and collagen fillers. Occasionally complications can be attributed to the selection of filler material, but in many circumstances can be related to incorrect technique and region selection. In order to provide safe care to patients and to achieve optimum outcomes, it is vital to have a full understanding of these issues.[1,3]

Complications: Early Onset
Early complications are those which occur within days to weeks of the initial injection.[4] Minor complications in the early stage include bruising, swelling and discomfort.[5] The use of local anaesthetic and recommendation of regular analgesia post procedure can reduce patient discomfort.[5]

Bruising
Bruising can occur within a few hours and may take several weeks to fully resolve. It is almost invariably minor; however, may be troublesome for the patient. Bruising is observed more frequently after injection into thin delicate skin, such as the lips and eyelids, and when into the dermal and subdermal planes.[6] The popular fanning technique of injection has been reported to increase the risk of bruising.[7] Steps can be taken to minimise the risk, including withholding medications known to thin the blood prior to the procedure such as aspirin, warfarin and non-steroidal anti-inflammatory drugs, as well as over the counter remedies such as St John's Wort, ginseng and fish oil.[6,8] Avoiding vigorous exercise for the first 24 hours to minimise hypertension, using fillers incorporating adrenaline (causing vasoconstriction), using the smallest gauge needle possible to deliver the filler and fewer injection sites, may all limit bruising.[6] Generally, bruising is self-limiting and will resolve. Advising the patient to immediately apply pressure and a cold compress for fifteen minutes to the area post-procedure and using vitamin K cream may treat bruising. Rarely, persistent hemosiderin staining may require treatment with pulsed dye light or potassium titanyl phosphate lasers.[6]

Swelling
Temporary swelling immediately post-procedure is normal and common. Treatment and prevention is as for bruising, and should settle within a week.[6] Oedema may be more significant when associated with hypersensitivity to the dermal filler, which may be an antibody-mediated or non-antibody mediated (delayed) reaction.[8] Some patients may develop hypersensitivity on initial or repeated exposure to the filler agent due to an immunoglobulin E (IgE) mediated response, which results in swelling, pain, erythema and itching within hours of injection.[8] Rapidly progressive angioedema is a medical emergency, but other patients may suffer angioedema that progresses more slowly but lasts several weeks and is not dangerous. For most, the swelling is short term and responds well to antihistamines. If antihistamines fail, oral prednisolone can be used. Chronic angioedema lasts more than 6 weeks and is challenging to treat and may require referral to immunology.[6,8]

Delayed hypersensitivity reactions mediated by T-lymphocytes can occur between 24 hours to several weeks post-procedure. Antihistamines are of no benefit and treatment involves removal of the allergen. Hyaluronidase can be used for HA fillers. Other fillers may require treatment with oral prednisolone whilst the filler resorbs, followed by laser or extrusion if ongoing.[8]

Importantly, malar oedema can occur following injection in to the infraorbital hollow and tear troughs. Injection of filler into the superficial compartment of the superficial suborbicularis oculi fat can impede lymphatic drainage of the compartment (which already has poor drainage due to the malar septum) leading to fluid accumulation in the infraorbital region.[8] Malar oedema is chronic and treatment resistant and therefore it has been recommended that injection into the infraorbital hollow is performed solely with HA, allowing for use of hyaluronidase if malar oedema is to occur.[8]

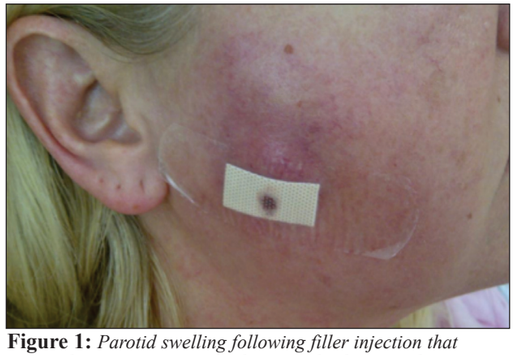

Figure 1: *Parotid swelling following filler injection that required excision to treat discharge and rule out malignancy, as fine needle aspiration was inconclusive.*

Swelling following dermal filler injection may present similarly to other facial swellings and need careful assessment. Further investigations such as fine needle aspiration may be required to determine the nature of the swelling and surgical intervention may be required in some cases (Figure 1).

Infection
Infection can be an early complication and cellulitis can occur following injection of dermal fillers due to inoculation of bacteria into the skin or entry of microorganisms through the disrupted skin barrier. Cellulitis presents with skin erythema, warmth and oedema around the injection site. It is important to distinguish this from hypersensitivity reaction which also causes erythema, but there is also usually an itch and the patient is apyrexial.[6] Abscess

formation is rare but requires treatment with antibiotics, incision and drainage. Unlike granulomas which shall be discussed later, abscesses are fluctuant with notable tenderness and warmth.[7] Antibiotics covering Staphylococcus and Streptococcus are the treatment of choice and may need to be given intravenously if the patient is systemically unwell or immunosuppressed.[6,8] Periorbital and midfacial infection require prompt treatment due to the risk of intracerebral spread.[6]

It is important to check for any history of cold sores as injections in the perioral region can lead to reactivation of the herpes virus. It has been suggested that a prophylactic course of aciclovir may be of benefit for those with a history of the virus. If infection does occur, aciclovir can be used if infection is recognised early and in combination with antibiotics if there is superimposed bacterial infection.[6]

Implant visibility
Other early complications include under and overcorrection and implant visibility.[3] Knowledge of the unique characteristics and mechanism of action of each dermal filler agent, in addition to correct technique, is crucial in placing the right amount of filler at the correct skin depth in order to avoid filler visibility or nodularity.[3] Injecting a filler agent too superficially can result in implant visibility[3]. Intervention is required in these events. Firm massage can be used to disperse excess HA or hyaluronidase can be injected. For other particulate dermal filler materials such as calcium hydroxylapatite or polymethylmethacrylate,

excess may need to be removed using dermabrasion or unroofing with a needle.[3]

Vascular compromise and necrosis

Skin necrosis following filler injection is a much feared complication. Necrosis is caused by vascular compromise resulting from obstruction of arterial or venous blood supply. The blood supply may be interrupted by inadvertent intravascular injection into an artery and embolisation, trauma to the blood vessel wall, or from external pressure of the filler onto the vessel wall causing compression.[9,10] Not only can vascular compromise result in skin loss and scarring, reports of acute blindness, stroke and death are made in the literature as a result of ocular and cerebral embolism.[6,11-15] The glabella is suggested to be the site at greatest risk of necrosis, but the nasolabial fold also carries a risk.[7] Recognition of vascular compromise and immediate treatment is vital in order to avoid serious adverse effects. Those performing dermal filler injections should also have a sound understanding of the anatomy of the vasculature surrounding the injection sites.

There are several factors which increase the risk of vascular compromise and those performing dermal filler injections can take measures to minimise them by doing the following:

1. Aspirating prior to injection to ensure the needle tip is not within a vessel.[16,17]
2. Avoid overcorrection and minimise the amount of filler volume used.[16]
3. Injecting at a low pressure.[17]
4. Avoiding deep injection of the filler product (larger blood vessels are located deep to the dermis).[6]
5. Use a blunt needle of the smallest size (blunt tip separates key structures including vessels rather than puncturing them, as with a sharp tip).[18]
6. Using a temporary product such as HA which has the option of hyaluronidase to quickly resorb some of the product.[18,19] Avoid the use of autologous fat injections which are highlighted in the literature as being associated with embolisation and visual loss.[6,12,13]
7. Avoiding scarred tissue areas (scars may fix vessels and make direction injection into the vessel easier).[20]
8. The glabella region should be reserved for those more experienced.[8]

The classical signs of impending vascular compromise are immediate-onset skin changes, with blanching, violaceous, or mottled appearance, and severe pain that is inconsistent with that typical of the injection.[6,9] There is also the possibility of delayed-type necrosis with symptoms occurring several hours after injection.[17] Swift recognition of vascular compromise and urgent intervention can potentially prevent progression to necrosis, and therefore if suspected, the injection should be stopped immediately. Aspiration of the filler can be attempted before taking steps to improve blood flow.[9] This includes massaging the area, application of warm compresses, as well as 2% nitroglycerin to promote vasodilation.[6-9] The topical nitroglycerin paste can be applied every one to two hours initially.[11] Hyaluronidase should be injected into the site HA fillers, and some suggest the use of hyaluronidase regardless of the filler used.[6] A course of aspirin to prevent further clot formation has been suggested, as well as low molecular weight heparin for more severe cases. The use of hyperbaric oxygen therapy may be helpful in patients with impending extensive skin necrosis.[11]

Once necrosis has occurred, good wound care with daily dressing changes are important to minimise scarring, as are antibiotics for any superadded skin infection. Antivirals should be considered if necrosis occurs around the mouth. Intralesional steroid injections, light dermabrasion and surgical revision may be considered for persistent scarring.[11]

The patient should be made aware of the risk of visual impairment and blindness. Direct injection of filler material into one of the distal branches of the ophthalmic artery (dorsal nasal, angular artery, zygomaticotemporal, zygomaticofacial, supratrochlear and supraorbital arteries) can lead to retinal artery occlusion.[21-23] Urgent referral to an ophthalmologist is needed if there are any concerns regarding vision following injection.

Complications: Late Onset

Late complications occur weeks to years after injection and comprise chronic inflammation and infection, nodules and granulomas, filler migration and scarring.[24]

Migration

Soft tissue fillers may migrate to a location away from their site of injection and may occur some years post-injection.[7,25] It can lead to mass lesions and swellings in other areas, including a 'popcorn lip' and patient dissatisfaction.[7] It can also result in inadvertent compression of other structures (Figure 2). Migration is more commonly associated with permanent and semipermanent fillers, such as calcium hydroxylapatite and silicone, rather than temporary fillers, which are reabsorbed before migration can occur.[7] However, cases of migration with temporary fillers such as HA are reported.[25] It may occur due to poor technique; large volumes injected under high pressure for example, and may be triggered by chronic inflammation or granuloma formation. Treatment options may include resorption with hyaluronidase or surgical removal.[26]

Nodules

Subcutaneous nodules are a known complication of dermal filler injections and usually trouble the patient. They may be non-inflammatory or inflammatory, and present as lumps weeks to months following treatment.[6]

Non-inflammatory nodules tend to be painless and palpable lumps that do not grow in size. They are usually localised to the injection site, but it is possible for the nodules to migrate.[9] Localised accumulation of filler is the most common cause of non-inflammatory nodules which may be due to overcorrection, injection of the filler too superficially or failure to discontinue the injection prior to removal of the needle.[8] Fillers such as HA, calcium hydroxylapatite and poly-L-lactic acid require injection mid dermis or deeper and nodules will form if injected superficial to this.[7] Whilst it may seem that a deeper injection is better, it is important to consider that the risk of vascular compromise will increase and the augmentation effect may not be as evident.[7]

Appropriate depth of HA injection is also important to avoid the complication referred to as the 'tyndall effect' which describes a bluish discolouration of the skin due to too superficial placement of the filler.[27] It occurs because of the light-scattering capacity of the filler material and is more likely to occur in areas with thin skin such as periorally and tear troughs.[28] It may be mistaken for a bruise, but does not resolve within a few days unlike bruising. Careful skin assessment pre-procedure and avoidance of areas of thin skin is important to prevent discolouration, and firm massage, aspiration and hyaluronidase injections can be used for treatment.[27]

Non-inflammatory nodules occurring after HA injection usually resolve with hyalyronidase. Those that form following injection with other filler types can be treated with massage in combination, with or without, either lidocaine or normal saline, before a trial of intralesional steroid injections. Further treatment options include injections of 5-fluorouracil (5-FU) and surgical excision as a last resort.[6]

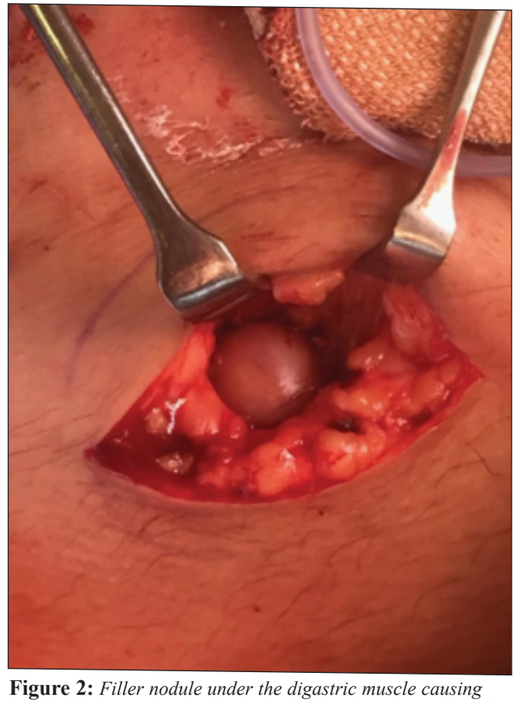

Figure 2: *Filler nodule under the digastric muscle causing restriction in mastication from cheek filler that migrated to the neck. The exact nature of the filler was not established.*

Non-inflammatory nodules should be distinguished from granulomas and biofilms, which occur as a result of inflammation around the foreign-body filler material and can be differentiated from a non-inflammatory nodule by tenderness, swelling, possible erythema and expression of pus.[9] Granulomas typically appear later than non-inflammatory nodules (several months to years as opposed to several weeks) and form in an attempt to contain any foreign material by enclosing it in a capsule of immune cells such as macrophages.[6] Intralesional corticosteroid injections remain the mainstay of granuloma treatment. Other therapies such as intralesional injections of 5-FU and hyaluronidase for HA fillers may be helpful, and surgical excision is required if other therapies fail.[6]

A biofilm is an accumulation of microorganisms that are either associated with a surface , such as a foreign implant, or attached to one another and form a living colony.[9] They typically present as chronic and recurrent infections at the injection site and although antibiotics may provide some temporary relief, usually definitive treatment is with removal of the filler and its biofilm.[9]

Although most complications including infection are not specific to a particular dermal filler, polyacrylamide gel (PAAG) is particularly biocompatible and provides bacteria an excellent material on which to multiply.[10] This can lead to late infections, abscesses and biofilms. If not

responsive to conventional antibiotic treatment, it is imperative to consider infection with atypical species.[10]

Conclusions

Dermal fillers are becoming increasingly popular. Clinicians should be fully aware of the signs and symptoms of complications and how to avoid them as much as possible. Adverse effects may occur early and appear to be minor but may still be concerning for the patient. Serious complications such as skin necrosis can be life-changing and even life-threatening. Good anatomical knowledge and proper technique can help to reduce the risk of complication, and when a complication does occur, the clinician should understand how to manage them from observation to surgical intervention.

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Mechanisms and treatment options for chronic non-allergic rhinitis

K. Van Gool, MD [1]; V. Hox MD, PhD[2]
[1] Department of Otorhinolaryngology, University Hospitals Antwerp, Belgium
[2] Department of Ear, Nose and Throat Disease, Cliniques Universitaires Saint-Luc, Brussels, Belgium.

Correspondence:
Valerie Hox
Department of Otorhinolaryngology
Cliniques Universitaires Saint-Luc
Avenue Hippocrate 10, 1200 Brussels, Belgium.

E-mail: valerie.hox@uclouvain.be

Abstract

Patients who present with chronic rhinitis are very frequently encountered in the physician's office. The best-studied form of chronic rhinitis is allergic rhinitis, however, the prevalence of non-allergic, rhinitis amongst the chronic rhinitis population is high and the disorder is an important cause of widespread morbidity. Despite this fact almost no diagnostic tests and very little treatment schedules are established for this patient group. Chronic non-allergic rhinitis covers an extensive range of differential diagnoses and comprises drug-induced, hormonal, occupational, gustatory, senile and idiopathic rhinitis. The causal factors and pathophysiological mechanisms are only defined for some of these forms. This review summarizes the causes of non-allergic rhinitis as well as the available options for diagnostic work-up and treatment strategies. It aims at providing a tool for a more precision-based approach of non-allergic rhinitis patients in order to obtain an improvement of their quality of life.

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Key words

Non-allergic rhinitis, nasal hyperreactivity, capsaicin, nasal provocations

Introduction

The prevalence of chronic rhinitis is estimated to be around 30% of the Western population and is a significant cause of widespread morbidity, health care costs and reduced work productivity[1]. Chronic rhinitis is defined as a symptomatic inflammation of the nasal mucosa, leading to nasal obstruction, rhinorrhea, sneezing and/or nasal/ocular itch. Two of these nasal symptoms should be present for at least 1 h daily for a minimum of 12 weeks to define the chronicity[1]. Although sometimes mistakenly viewed as a trivial disease, symptoms of rhinitis may significantly impact a patient's quality of life[2].

While viral infection is the most common cause of acute rhinitis, allergy is the best-studied form of chronic rhinitis. Allergic rhinitis (AR) is relatively easy to diagnose by the combination of typical symptoms and positive skin prick tests (SPT) or the detection of allergen-specific IgE in the serum[3]. However, a large group of patients suffering from chronic rhinitis has no systemic signs of allergy and they are consequently classified as non-allergic rhinitics (NAR). This patient group forms a diagnostic and therapeutic challenge and probably accounts for about half of the total chronic rhinitis population[4]. In contrast to large-scale and well-conducted epidemiologic and immunologic studies on AR, data on the prevalence, pathophysiology and treatment of NAR is scarce. Currently, it is believed to be a heterogeneous patient population suffering from symptoms that are often indistinguishable from allergic rhinitis patients. This review summarizes the currently known etiologies of NAR and proposes a more accurate diagnostic work-up as well as therapeutic strategy.

Classification of NAR

Drug-induced rhinitis

The best known form of drug-induced rhinitis is 'rhinitis medicamentosa' which defines the nasal congestion that occurs with overuse of topical nasal vasoconstrictors[5]. The exact mechanism is poorly studied, but it is believed that recurrent nasal tissue hypoxia and negative neural feedback with chronic decreased α2-receptor responsiveness are involved[5,6].

In certain individuals suffering from a disorder of the in the eicosanoid synthesis, aspirin and NSAID's can induce rhinitis symptoms[6,7].

In addition, antihypertensive medication like methyldopa, hydralazine, guanethidine, ACE-inhibitors and α- and β-receptor antagonists down-regulate the activity of the sympathetic nervous system, possibly inducing nasal congestion. Also immunosuppresive medication, oral contraceptives and psychotropic agents can lead to nasal symptoms[6].

Smoking-related rhinitis

Unlike its' effect on lower airway physiology, the impact of tobacco smoke on the nasal mucosa is not well studied. Still, there is growing evidence that such exposure can have a significant impact on nasal function. Smokers show a higher prevalence of chronic rhinitis compared to non-smokers[8] and several authors have reported that tobacco smoke exposure overall is associated with acute and chronic nasal symptoms[9,10]. Chronic cigarette exposure leads to mucosal recruitment of CD^8+ T lymphocytes[11], as well as decreased cilia beat frequency[12]. Several components of cigarette smoke such as formaldehyde and acrolein act as a local irritant on the nasal mucosa[13].

Senile rhinitis

Senile rhinitis is the characteristic clinical picture of elderly patients, suffering from a persistent clear rhinorrhea, often in the absence of other nasal symptoms[14]. Senile rhinitis is believed to be caused by an age-related dysregulation between the sympathetic and parasympathic nervous systems that innervate the nasal mucosa, causing a cholinergic hyperreactivity, since anticholinergic drugs are effective in these patients[15].

Rhinitis linked to systemic auto-immune disease

The systemic diseases presenting most frequently with sinonasal involvement are the vasculitic diseases Eosinophilic Granulomatosis with Polyangitiis (EGPA, previously Churg-Strauss syndrome) and Granulomatosis with Polyangiitis (GPA; previously Wegener's granulomatosis)[16,17]. Over 75% of patients with EGPA and GPA present with rhin(osinus)itis symptoms, usually nasal obstruction and chronic recurrent infections[18]. In GPA patients this is commonly associated with crusting and bloody discharge; EGPA patients' lesions are less erosive[19]. Also, a small percentage of sarcoidosis patients can develop sarcoid of the nose causing symptoms of nasal obstruction, rhinorrhea or crusting[20]. Less frequently, systemic lupus erythematosus, relapsing polychondritis and Sjögren syndrome may present with difficult-to-threat rhinitis and rhinosinusisitis in addition to other organ involvement.

Hormonal rhinitis

The most prevalent form of hormonal rhinitis is pregnancy rhinitis, which has been estimated to have a cumulative incidence of 22% by a large multicenter study[21]. Pregnancy rhinitis typically starts during the second month of pregnancy, usually disappearing rapidly after delivery. But nasal congestion can even occur in conjunction with the rise in serum estrogens that occur at ovulation in the normal menstrual cycle[22,23]. The pathophysiology remains largely unexplained, but estradiol has been shown to increase vasodilation and vascular leakage by stimulating nitric oxide production[24] in addition to its' general pro-inflammatory effects such as induction of eosinophilic migration and degranulation[25].

Increased nasal secretion in hypothyroidism has been reported on an anecdotal basis[22]. It has been proposed that nasal symptoms occur in acromegaly, however, a Swedish study could not demonstrate the induction of nasal congestion in response to a growth hormone treatment[26].

Occupational rhinitis

Occupational rhinitis is defined a rhinitis attributable to a particular work environment[27]. Occupational agents are either biological proteins that induce a classic IgE-mediated allergic inflammation (high molecular weight [HMW] agents) or low molecular weight (LMW) agents. LMW sensitizers are capable of activating the adaptive immune system leading to a sensitization to the agent[28]. They are mostly chemicals; but also, several drugs, metallic agents and wood types own this sensitizing capacity and only a minority of these agents induce detectable antigen-specific IgE, thus complicating diagnosis.

The non-sensitizing LMW agents are addressed as airway irritants. A single exposure to high concentrations of irritant induces an acute toxic effect on the respiratory mucosa[29], but recently there is increasing evidence that also long-term exposure to lower concentrations of irritants can induce a more chronic dysfunction of the nasal mucosa[10]. Mechanisms responsible for irritant-induced rhinitis are not well known and are thought to involve epithelial damage[30] and neurogenic inflammation[31]. The transient receptor potential (TRP) A^1 channel that is expressed on the sensory nerve endings of the non-adrenergic, non-cholinergic (NANC) neural system in the nasal mucosa has emerged as being a major irritant detector[31,32].

Idiopathic rhinitis

In about 50% of NAR patients, the causal factor of their rhinitis remains undetermined. These patients are adressed as idiopathic rhinitis (IR) patients, formerly known as intrinsic or vasomotor rhinitis[33].

IR patients often report nasal hyperreactivity (NHR) as a key feature[34] which is defined as the induction of nasal symptoms upon encounter of environmental stimuli, such as temperature/humidity changes, strong odours, cigarette smoke and other respiratory irritants[35,36]. Although it is present in all types of rhinitis (infectious rhinitis, AR and NAR), it is the specific hallmark of IR.

The two most plausible pathophysiological hypotheses are non-IgE mediated inflammation[37] and/or neural dysfunction[38]. In the latter group, it is believed that nasal symptoms are induced upon local release of neuropeptides such as substance P (SP) upon stimulation of the sensory nerves of the NANC system (Figure 1). A mechanistic study of steroid-unresponsive IR patients showed an upregulation of the $TRPV^1$-SP signaling pathway in their nasal mucosa in comparison to healthy controls[39].

Local allergic rhinitis

Multiple studies suggest that a subgroup of NAR suffer from a local allergic rhinitis (LAR). Despite a negative test for systemic specific IgEs, some patients react positively to a nasal allergen provocation test with the induction of a Th^2-type mucosal cell infiltration similar to AR. In some of these patients, allergen-specific IgEs can be detected in the nasal mucosa[40,41]. However, a lot of uncertainty exists about the prevalence of LAR and the exact pathophysiology and diagnostics.

Diagnosis of NAR:

By definition, a patient suffering from chronic rhinitis with a negative SPT and serum specific IgE test, suffers from NAR. Several explorations can help with further differentiation of diagnosis:

History:

For most subtypes of NAR, medical history is the key for diagnosis. Full evaluation should always include a determination of the pattern, triggers, duration of the

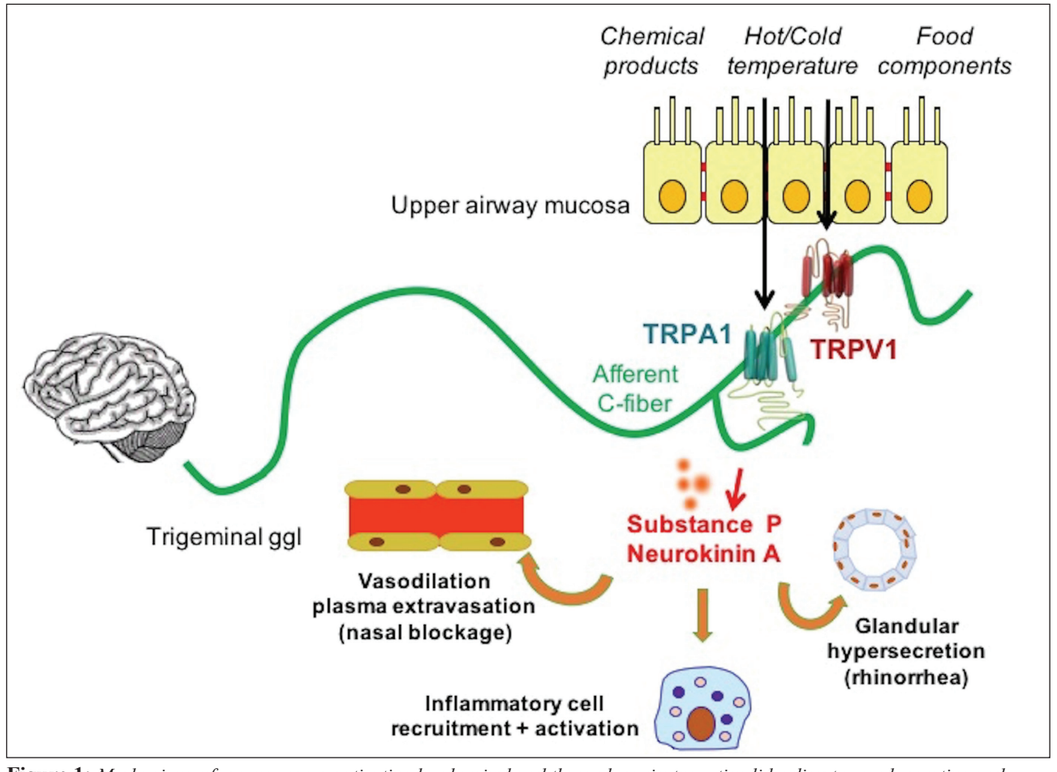

Figure 1: *Mechanisms of sensory nerve activation by chemical and thermal respiratory stimuli leading to nasal secretion and blockage via antidromal release of neuropeptides upon activation of TRP channels. TRP: Transient Receptor Potential (original, adapted from ref 28).*

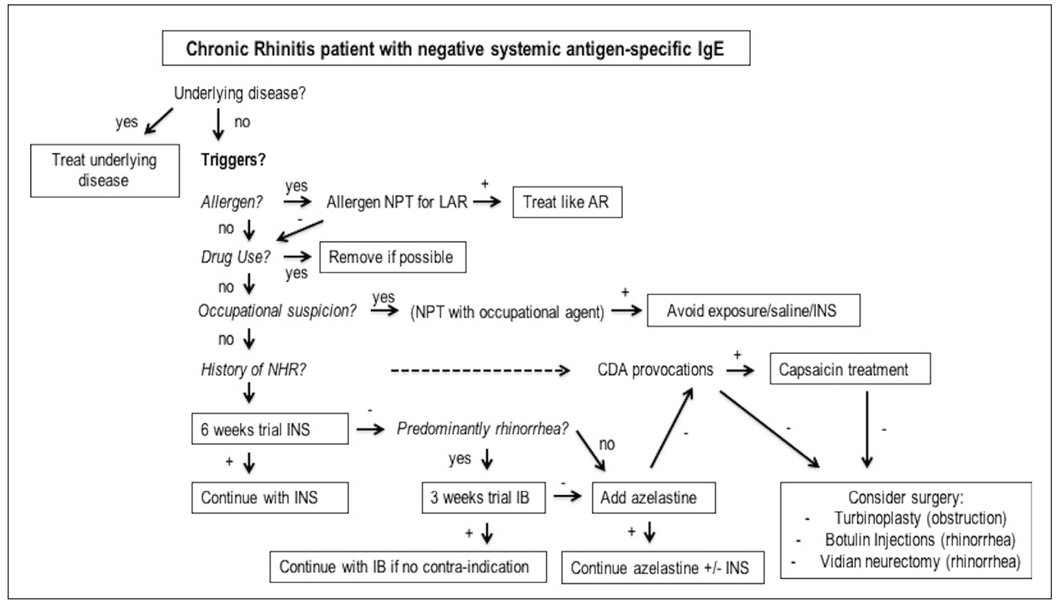

Figure 2: *Proposed algorithm for treating patients with NAR. NPT: Nasal Provocation Test; AR: Allergic Rhinitis; LAR: Local Allergic Rhinitis; INS: intranasal steroids; CDA: cold dry air; NHR: nasal hyperreactivity; IB: ipratropium bromide.*

symptoms as well as response to medications, comorbidities, environmental and occupational exposures, including tobacco.

Nasal endoscopy:

Every chronic rhinitis patient should undergo a nasal endoscopy, mainly to exclude rhinosinusitis, tumours or foreign bodies. Signs of infection, crusting, and/or significant structural abnormalities can help in the differential diagnosis.

Nasal patency measurements

Rhinomanometry, acoustic rhinometry and Peak Nasal Inspiratory Flow meters objectively measure nasal airway resistance. All techniques correlate relatively well with subjective sensation of nasal blockage and with each other[42], however, these methods are mainly considered useful tools in provocation testing in NAR patients.

Nasal sampling:

Currently, nasal sampling (cytology and nasal biopsies) is not implemented in the routine diagnostics for chronic rhinitis but may help to distinguish between an inflammatory or non-inflammatory pathology, which could be directive in choosing a therapy.

Nasal provocation tests

During nasal provocation tests, the nasal mucosa of the patient is exposed to the suspected provocative agent and consequent changes in nasal patency and symptom scoring can prove the link between the administered agent and the symptoms[45].

In case of a typical history of AR in the absence of systemic IgEs, provocation with the suspected allergen is the method of choice to diagnose LAR[46].

When occupational rhinitis is suspected, nasal provocation testing with the suspected agent is the golden standard for making a definite diagnosis[28].

To diagnose NHR, several provocation tests have been explored in the past, including metacholine, histamine, hypertonic saline and capsaicine[34]. However, most of these substances are ineffective in discriminating IR patients from healthy controls or are too patient- or examiner-unfriendly. In 1998, the group from Amsterdam showed that intranasal exposure to cold dry air (CDA) was the most reliable method for measuring NHR symptoms[47]. Recently these results were confirmed by Van Gerven as well as Segboer and colleagues who demonstrated that even a short protocol with CDA exposure has a high sensitivity and specificity for the diagnosis of NHR[36,48].

Treatment options for NAR patients

A therapeutic algorithm including trigger avoidance, medical and surgical treatment options for NAR patients is proposed in Figure 2.

Trigger avoidance:

If there is evidence for drug-induced rhinitis, discontinuation or change of medication should be considered 50 and is mandatory in the case of rhinitis medicamentosa.

In case of occupational rhinitis, environmental control is the mainstay of therapy, also to avoid progression of disease towards the lower airways. This is achieved by removing the etiologic agent, improving ventilation, wearing protective masks, or changing the work site[28].

If an underlying hormonal or vasculitic disorder is present, management of the underlying condition is the key step in dealing with the upper airway symptoms[51].

In IR patients suffering from NHR, it is recommended to avoid exposure to ‘unspecific’ triggers such as airconditioning, cigarette smoke, strong odours, pollution, cleaning agents and even frequent nose blowing[34].

Nasal saline lavage:

Because of its beneficial effects as an ‘active placebo’[52], isotonic nasal saline douches should be considered in most NAR patients, especially in case of vasculitic diseases and occupational rhinitis[28,51].

Intranasal corticosteroids:

Intranasal steroids (INS) appear to be useful in treating NAR in cases where inflammation is evident, like vasculitis-linked rhinitis.

Although there are no studies available to prove their beneficial effect in occupational rhinitis, therapeutic options include the daily use of INS in case of rhinitis due to HMW agents and LMW sensitizers[53].

Inconsistent results have been reported on the efficacy of INS in the treatment of IR patients. A double-blind, placebo-controlled study from Lundblad et al. showed a symptom improvement of 56 % with local momethasone furoate in over 300 IR patients[52]. Dockhorn also demonstrated that topical steroids significantly reduced symptoms in NAR compared to placebo[54]. A similar study by Blom and colleagues tested fluticonasone proprionate in 65 NAR patients, but found only a small decrease in nasal symptoms, which only reached significance for sneezing[44]. The contradictory findings of this latter study, might be explained that all patients were IR patients referred to a tertiary center because of previous unresponsiveness to local steroids[44].

Currently, in IR patients with no information on nasal inflammatory markers, a trial with INS for at least 6 weeks is still the first therapy of choice[55,56].

Cholinergic antagonists:

In cases where disease is caused by an overactive parasympathetic system leading to nasal glandular hypersecretion, cholinergic antagonists have proven to be effective. Ipratropium bromide (IB) has shown to be effective in reducing both the severity and the duration of rhinorrhea in NAR[57]. It is the first therapy of choice in senile rhinitis and it can be beneficial for rhinorhea symptoms in patients suffering from other types of NAR[54].

Botulinum toxin (BT) also has an acetylcholine inhibiting effect and several studies have shown a beneficial effect of exposing the nasal mucosa (injections or direct application) on rhinorrhea in IR patients[58].

Antihistamines:

Two double-blind, placebo-controlled studies showed a beneficial effect of the antihistamine molecule azelastine in IR patients[59,60] reducing nasal obstruction, rhinorrhea, sneezing, postnasal drip, nasal congestion and anosmia with a response rate between 82 - 85% in over 200 IR patients[59,60]. The mechanism of action has not been unravelled, but might involve anti-inflammatory characteristics[60] and reduction of substance P release[61] in addition to it’s histamine 1 receptor antagonism[49]. The combination formulation of intranasal azelastine with fluticasone proprionate has been shown to be effective in reducing symptoms in a population of both allergic and non-allergic rhinitics[62], however, studies investigating its efficacy in a specific NAR population are currently lacking.

Capsaicin

Since 1991, several clinical trials have proven that repeated intranasal application of capsaicin reduces symptoms and NHR in IR patients[63]. The therapeutic effects are most likely caused by a neurogenic desensitization leading to a reduction of mucosal nerve fibers and a downregulation of the TRPV1-SP nociceptive signaling pathway[39]. Since capsaicin clearly targets the nervous system, it is an interesting therapeutic option in steroid-unresponsive IR patients. Van Gerven and colleagues showed that rhinitis

symptoms improved in 80 % of these patients after a one-day high-dose intranasal capsaicin treatment[39].

Surgical intervention

It is generally accepted that surgery for NAR should only be considered in those patients who respond insufficiently to medical therapy.

Laser turbinectomy significantly decreased both subjective and objective symptoms in NAR patients[64,65] and therefore turbinate surgery can be an effective symptomatological treatment option in NAR patients with nasal obstruction who present with hypertrophy of the inferior turbinates.

Vidian neurectomy has been proven effective in dealing with rhinorrhea as well as nasal obstruction associated with IR[66] but comes with surgical risks. Therefore it should be reserved for patients with important quality of life reduction and performed by experienced surgeons.

Conclusions

In this review, we emphasize the diagnostic and therapeutic challenge of NAR. A consensus on classification, diagnostic work-up and treatment for the affected patients is needed, especially in the light of more treatment options becoming available nowadays. In this way, we can account for the growing need for precision medicine in upper airway disease.

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Endoscopic repair cerebrospinal fluid (CSF) rhinorrhea

Karan Jolly – MBChB DOHNS MRCS(ENT), Otolaryngology Specialist registrar (ST6), **Georgios Tsermoulas** - MUDr FRCS(NS), Consultant Neurosurgeon and **Shahzada K Ahmed,** BSc (hons) DLO FRCS (ORL-HNS) PhD, Consultant Otolaryngologist
University Hospital Birmingham.

Correspondence:
Georgios Tsermoulas - MUDr FRCS(NS), Consultant Neurosurgeon. University Hospital Birmingham.

E-mail: tsermoulas@uhb.nhs.uk

Abstract
Cerebrospinal fluid (CSF) rhinorrhea is the result of an abnormal communication between the subarachnoid space and the sinonasal cavity, through a skull base defect. The vast majority of traumatic leaks will heal with conservative measures with surgical intervention reserved for patients who fail to respond to conservative management due to risk of meningitis. A careful diagnostic work up includes testing nasal fluid for Beta trace protein and identifying the site of leak with high resolution CT and or MRI imaging. Intracthecal injection of fluorescein can be extremely helpful in the diagnosis and also used intraoperatively to aid repair. The results of transnasal endoscopic repair have high success rates and are usually the first line treatment strategy.

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Key words
Csf, rhinorrhea, repair.

Introduction
Cerebrospinal fluid (CSF) rhinorrhea is the result of an abnormal communication between the subarachnoid space and the sinonasal cavity, through a skull base defect. Persistent CSF leaks are divided into traumatic and non-traumatic. Between 80-90% of CSF rhinorrhea cases are related to trauma, this being either a head injury or iatrogenic following endoscopic endonasal surgery[1]. Non-traumatic causes include skull base tumours, raised intracranial pressure and congenital skull base defects. The risk of meningitis is a concern amongst these patients and can range from between 10% to 37% if managed conservatively, underscoring the importance of early detection and timely repair[3,4,5]. Historically, open intracranial approaches have been used to manage such cases, however advances in endoscopic endonasal surgery have revolutionised treatment of CSF rhinorrhea due to significantly less morbidity and higher success rates ranging from 87% to 100%.[6]

Accidental trauma
Traumatic CSF leaks can be a result of head injuries with anterior skull base fractures. This is seen in 15 to 30% of cases of skull base fractures and more frequently in comminuted fractures. These leaks often occur through the cribiform plate of ethmoid sinus roof due to tightly adherent dura in these areas[6]. Most patients (80%) will present with CSF rhinorrhea in the first 48hrs and 95% of these patients will manifest within 3 months[7]. In such cases, leaks rarely require treatment as up to 85% heal spontaneously with conservative management[7]. Surgical intervention is usually indicated in patients who fail to respond to conservative management due to risk of meningitis.

Iatrogenic trauma
Iatrogenic CSF rhinorrhea accounts for 16% of traumatic cases[6]. It can occur following routine endoscopic sinus surgery as well as more advanced skull base surgery. The most common site of injury is the lateral cribiform lamella with other sites being sphenoid sinus and posterior fovea ethmoidalis. Risk of CSF leak following functional endoscopic sinus surgery is quoted as 0.5%, increasing with more complex skull base procedures such as clival tumours and revision surgery[8]. The majority of these will be repaired immediately at the time of injury or will be transferred to a skull base centre for surgical repair.

Non-traumatic
Spontaneous leaks account for the majority of non-traumatic leaks. The exact pathogenesis of spontaneous CSF rhinorrhea is unknown, however it is thought to be related to elevated intracranial pressures (ICP), commonly due to idiopathic intracranial hypertension (IIH)[9]. Patients with IIH are classically middle aged overweight women and present with headaches, visual disturbance and papillodema. There is an increased prevalence of this disease in the western world over the last few decades, most likely as a result of the obesity epidemic[10]. Spontaneous leaks secondary to sustained raised ICP are thought to result from increased dural pulsation with remodelling and thinning of the skull base creating an osteodural defect in pneumatised parts of the skull base[6]. However, although elevated ICP has been implicated in spontaneous leaks, it is not the case in all patients with spontaneous CSF rhinorrhoea[11,12]. Historically nontraumatic spontaneous leaks accounted for 4% of CSF leaks, however more recent data suggests spontaneous leaks may be more common, ranging from 20.8% to 40% of all CSF leaks[10].

Other causes of non-traumatic leaks include tumours, mucoceles or infective processes eroding the skull base. Congenital causes can occur with or without raised ICP and these include encephaloceles, persistent craniopharyngeal canal (with or without tumour) and congenital widening of diaphragma sella[13].

Diagnosis
It is very important to have a high index of suspicion based on the history of presenting patients. This includes a recent history of trauma or surgery, which holds true for the majority of patients with CSF rhinorrhea. The most common clinical manifestation is persistent clear rhinorrhea, often unilateral, made worse by bending over or Valsalva manoeuvre[11]. Some patients may report a history of headaches in the presence of raised ICP or intracranial lesions.

A quick bedside test for CSF fluid is the presence of a double ring sign when drops of the fluid is placed on absorbent filter paper or the “halo sign” on a pillowcase. Bedside glucose detection using test strips is not recommended due to its lack of sensitivity and specificity[6]. The gold standard test for CSF fluid is to test for Beta 2 transferrin by immunofixation electrophoresis, with a sensitivity of 94% to 100% and specificity of 98% to 100% [12]. If there is a high index of suspicion or once the nasal discharge is confirmed as CSF, imaging is required to help locate the precise site of leak.

Imaging

Computed tomography: (Fig 1)
High resolution (0.5mm slice thickness) CT (HRCT) of the paranasal sinuses and skull base is the first line imaging modality, offering detailed osseous anatomy with greatest spatial resolution to pinpoint a site of dehiscence. HRCT has a sensitivity of 88% to 95% in identifying skull base defects with confirmed CSF leak[14]. HRCT is also useful in delineating sinonasal anatomy for surgical planning and for use of intraoperative image guidance navigation. At the time of imaging an active leak does not have to be present to identify an osseous defect, however in the presence of multiple fractures or defects it can become difficult to identify which defects are responsible for the CSF leak[10]. Another limitation of HRCT is that is offers poor soft tissue detail.

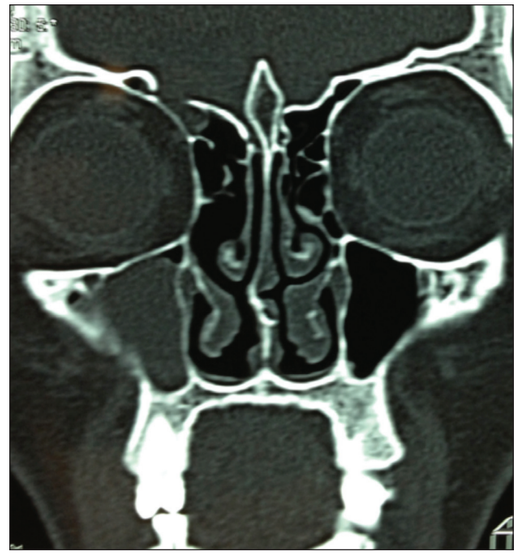


Figure 1: *Coronal Computed Tomography scan showing bone defect in right anterior skull base with opacified right maxillary sinus.*

Having said that, if only one clinically correlating osseous defect is identified, no additional imaging is required before proceeding to surgical repair[15] unless there is concern that there may be a meningocoele or meningoencephalocoele.

Magnetic resonance Cisternogram: (Fig 2)
Coronal Magnetic resonance Cisternography (MRC) is performed with heavily T2 weighted (T2w) fat saturated images and serves as a complementary imaging modality alongside HRCT in cases of suspected intracranial herniation, due to poor osseous detail[16]. A positive finding will highlight a CSF column communicating from the subarachnoid space with or without any meningeal or brain herniation. Sensitivity of MRC imaging in identifying the source of leaks is up to 94% [9,17].

Computed tomography cisternography (CTC):
This involves the use of intrathecal non-ionic iodinated contrast with scans taken in the prone and supine position,

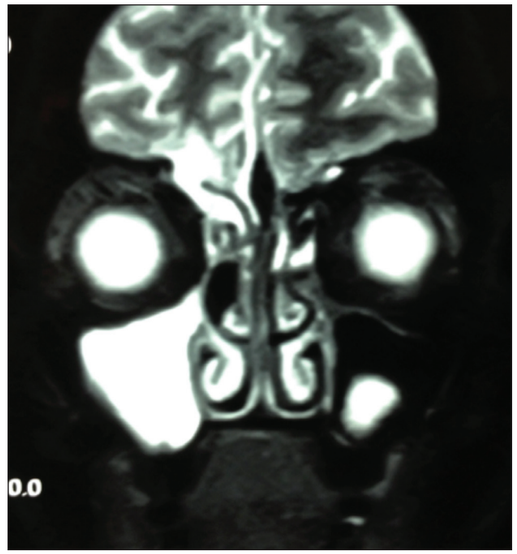


Figure 2: *T2 CIS Magnetic Resonance Image showing fluid column in continuity with CSF space and right anterior ethmoid sinus and also filling the right maxillary sinus.*

with supine images also taken before contrast injection for comparison. A positive study will identify extracranial fluid or soft tissue adjacent to the osseous defect with pre and post contrast scans showing interval contrast pooling. CTC historically was the study of choice in identifying CSF leaks however now is rarely used in cases of multiple osseous defects or when other imaging modalities fail to show any defect. Disadvantages of this CTC include high radiation dose due to multiple scans, potential adversity from contrast and that patients have to be actively leaking for a positive scan resulting in low sensitivity rates[16].

Contrast enhanced Magnetic resonance cisternogram (MRC):

This technique employs intrathecal gadolinium with subsequent T1 weighted sequences. Like with CTC, a positive study will show contrast leakage through dural and osseous disruption. Similar to CTC, MRC also requires HRCT for interpretation. MRC is particularly useful in cases of slow flow or intermittent leaks and offers increased sensitivity in comparison to HRCT with non-contrast MRC[14,18]. In high flow leaks sensitivity has been reported up to 100% with slow flow leak sensitivity being between 60-70%[19]. Strengths of this technique include less radiation and easier interpretation in comparison to CTC due to better soft tissue/ bone differentiation. Although gadolinium has been used safely worldwide at low doses, there is potential risk of neurotoxicity and so patient selection is very important[18,20].

Intrathecal fluorescein: (Fig 3)

The use of intrathecal fluorescein is off-licence yet can be very useful in identifying the location of CSF leaks intra-operatively. Commonly 0.1ml of 10% fluorescein (10mg) is diluted in 10ml of CSF and injected intrathecally via a catheter over 10 minutes. This can be directly visualised intra-operatively and enhanced with the use of a blue light filter. Side effects reported from the use of fluorescein such as seizures are dose dependent and associated with much higher doses. Generally speaking, no side significant side effects have been reported with doses less than 10mg[2].

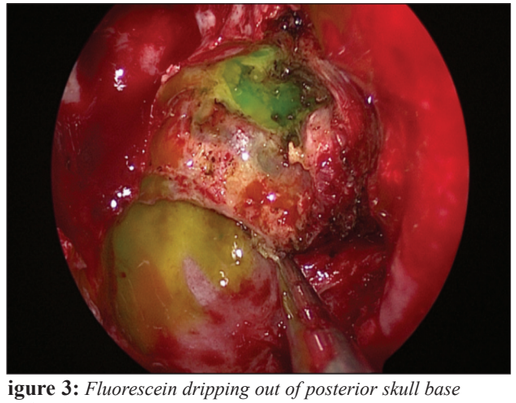
Management

Conservative management:

The vast majority of traumatic CSF rhinorrhea can be managed conservatively for up to two weeks, with up to 85% of CSF leaks healing spontaneously[22]. Failing this, the risk of meningitis necessitates for definitive surgical repair. Conservative measures include bed rest, laxatives, and the avoidance of anything that will increase ICP such as lifting heavy weights.

The use of a lumbar drain may also be considered but this carries additional morbidity and the evidence for its use is limited. The use of prophylactic antibiotics is a controversial topic and practice can vary due to conflicting evidence[21].

A recent evidence based review conferred no added benefit from the use of prophylactic antibiotics in traumatic leaks[21]. All patients should also be given pneumovax vaccination to prevent meningococcal meningitis.



igure 3: *Fluorescein dripping out of posterior skull base defect (arrow).*

Surgery

Open:

Open approaches to anterior skull base repair is far less common than historically. This can be either with an intracranial or extracranial approach. Open approaches are only rarely indicated. These may include: large encephalocoeles, in patients with extensive multiple defects and for leaks associated with intracranial lesions or haematomas. Leaks difficult to manage endoscopically, such as those in the posterior frontal sinus wall, may sometimes require an open cranial approach. However, these techniques are associated with a significantly higher rate of morbidity compared to purely endoscopic approaches[23].

Transnasal Endoscopic:

Advances in endoscopic sinus surgery have led to it being the preferred method of repairing CSF leaks, due to significantly reduced morbidity and excellent outcomes. Success rate from endoscopic repair ranges from 70% to 100% on first attempt and 86% to 100% in revision/ redo surgery[21]. A variety of graft material can be used for repair of the skull base, including fat, bone, allografts, free mucosal or fascial grafts, vascularized flaps as well as synthetic grafts and sealants to hold the repair in place. There is currently no evidence that supports one material to be superior to another and their use very much relies on site and size of defect as well as surgeon's preference[21]. However, in the presence of large defects (>3cm) or high flow leaks, vascularized grafts (eg nasoseptal or pericranial) confer improved outcomes with lower post-operative leak rates[21,24], especially in tumour surgery in which patients may receive post-operative radiotherapy. In reality, surgeons often use a multilayer technique with variety of different materials as underlays and overlays around the defect, with nasal packing to hold the repair in place. The use of lumbar drains has previously been commonly used in the perioperative period, however the current evidence does not support the routine use of lumbar drains[21]. Complications of repair include headache, meningitis, pneumocephalus, haematomas, abscess formation and recurrence[25]. In cases where localising the defect is difficult, intrathecal fluorescein can be used intra-operatively to confirm the exact location of the dural defect and observe CSF leaking using a blue light filter. Complications such as seizures have rarely been reported[26] after its use but this is eliminated when lower concentrations of fluorescein are used.

Conclusion

Trauma still continues to be the most common cause of CSF rhinorrhea. Early diagnosis and swift intervention is the key to managing these patients. In most cases the site of leak can be identified with appropriate imaging and repaired with a transnasal endoscopic approach.

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Orbital and optic nerve decompression

O.C Okonkwo[1] MBBS MRCS FRCS ORL-HNS and **S K Ahmed**[1] MBBS PhD FRCS ORL-HNS
[1] Department of ENT and Skullbase Surgery, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, B12 2GW

Correspondence:
O.C Okonkwo, Department of ENT and Skullbase Surgery
Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, B12 2GW
E-mail: o.okonkwo@nhs.net

Abstract
Background: The concept of orbital decompression was first described in 1890 by Julius Dollinger.

However, in recent decades the endoscopic approach to the orbit and optic nerve has evolved.

The underlying pathologies that lead to increased intraorbital pressure secondary to extrinsic compression of the globe or that lead to optic nerve compression are varied, but they are linked as they all impact on optic nerve perfusion pressure and can cause optic neuropathy.

The indications for orbital decompression can be acute or chronic. However, optic nerve decompression should only be considered if there is evidence of compressive optic neuropathy with deteriorating visual acuity, unresponsive to medical treatment, in the presence of an intact optic nerve.

Prior to surgery thorough work up in conjunction with an ophthalmologist is essential.

We discuss developments in surgical techniques for orbital and optic nerve decompression.

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Key words
Orbit, optic nerve, decompression, endoscopic

Anatomy
The orbit is a rigid anatomical structure made up of 7 bones, bound by 4 bony walls and bound anteriorly by the eyelids and orbital septa.

The posterior aspect of the orbit narrows to a confluence forming the orbital apex where the optic canal, superior orbital fissure and inferior orbital fissure transmit nerves and vessels into the orbit.

The rigid boundaries of the orbit create a fixed capacity of approximately 30ml and there is little room to accommodate any significant increase in volume of the globe or extra ocular soft tissue. As a consequence, changes in volume are associated with extrinsic compression of the globe and sequalae such as proptosis, diplopia and visual loss.

Likewise, the optic canal which runs through the lesser wing of sphenoid and transmits the optic nerve and ophthalmic artery, is a fixed capacity bony canal and any change in volume of its contents can result in compressive optic neuropathy.

This often manifests as slowly progressive decrease in visual acuity, with dyschromatopsia; a relative afferent pupillary defect; visual field defect; and optic atrophy or oedema.

Background
The concept of orbital decompression was first described in 1890 by Julius Dollinger. The traditional external approaches are well established i.e. trans conjunctival, transcranial and lateral orbitotomy. However, in recent decades the endoscopic approach to the orbit and optic nerve has evolved, initially endonasally, as pioneered by Kennedy et al in 1990[1] and more recently via endoscopic transorbital neuroendoscopic surgery (TONES).[2] These approaches are minimally invasive and allow good access and visualization of ocular structures.

Aetiology
The underlying pathologies that lead to increased intraorbital pressure secondary to extrinsic compression of the globe or that lead to optic nerve compression are varied, but they are linked as they all impact on optic nerve perfusion pressure and can cause optic neuropathy (see table 1). The most commonly encountered of all these pathologies is thyroid eye disease

Indications
The indications for orbital decompression can be acute or chronic. (See table 2) The indications for optic nerve decompression are, however, more tenuous and the literature is inconclusive. There have been studies to show

Table 1: Underlying Aetiology of Globe and Optic Nerve Compression				
Inflammatory disorders	**Trauma**	**Neoplasia**	**Benign masses**	**Infection**
Thyroid eye disease	Bony displacement	Sella/parasellar masses	Mucocele	Abscess
Ocular myositis	Hematoma	Orbital/orbital apex masses	Meningiomas	Post septal cellulitis
Systemic inflammatory disease	Oedema	Nasal/paranasal masses	Fibrodysplasia	

that it is of no benefit in traumatic optic neuropathy due to the high rate of spontaneous resolution in visual acuity[3]. However, the literature suggests that optic nerve decompression should be considered if there is evidence of compressive optic neuropathy with deteriorating visual acuity, unresponsive to medical treatment, in the presence of an intact optic nerve[4,5].

Work up

Opthalmological assessment is essential prior to the procedure. This includes measurement of visual acuity, assessment of colour vision, evaluation of proptosis and assessment of eye movements and diplopia.

A CT scan evaluating the orbits and sinuses is required to review relevant anatomy and the surgeon should identify the middle turbinate attachment, location and course of the anterior ethmoid artery, and the presence of an Onodi cell as the optic nerve may course through the lateral aspect of the cell.

Furthermore, MRI imaging of the orbit and brain is the imaging modality of choice when assessing the optic nerve and soft tissue within the orbit.

Procedure

The goals of surgery are in optic nerve decompression are to reduce or reverse vision loss. Orbital decompression has the additional goals to prevent ocular surface damage; relieve orbital pain and congestion; reduce proptosis, diplopia, lid retraction, chemosis, lid oedema, and fat prolapse.[6]

There are 4 areas in which orbital decompression can be achieved (See table 3). Each has unique considerations and can impact on subsequent rehabilitative ocular surgery e.g. eyelid and strabismus procedures, therefore we feel these patients should be managed in conjunction with ophthalmology colleagues. In this article we will focus on transnasal orbital decompression.

Technique

Endoscopic visualization via the trans nasal route allows access to the medial aspect of the orbit, orbital floor and orbital apex making it an ideal approach for accessing both intra and extra conal structures located in the medial and posterior aspect of the orbit. It has the advantage that it is low morbidity when compared to other techniques[7] and does not leave a scar. However, it can be associated with new onset or worsening of pre-existing strabismus, double vision and globe dystopia, therefore patients must be counselled on the possible need for subsequent strabismus surgery.

Orbital Decompression

After adequate decongestion of the nose the surgeon first performs a large middle meatal antrostomy with removal of the uncinate process anteriorly and bony removal extending posteriorly to the posterior maxillary wall. The superior limit is the orbital floor and inferior limit is the superior margin of the inferior turbinate.

Then a complete spheno – ethmoidectomy is performed to expose the medial wall of the orbit from the skull base superiorly to the roof of the maxillary sinus inferiorly; and anteriorly from the maxillary line to the face of the

Table 2: Indications for orbital decompression	
Acute	**Chronic**
Acute optic neuropathy	Disfiguring proptosis
orbital compartment syndrome	chronic pain/discomfort
corneal decompensation	congestion
acute globe subluxation	corneal exposure/ulceration
Severe orbital inflammation	Progressive orbitopathy not responding to other measures

Table 3: Areas of Orbital Decompression and Surgical Access	
Fat Compartment	Transcutaneous Transconjunctival
Orbital floor	Transorbital Transcutaneous Transantral Transnasal
Medial Wall of Orbit	Transorbital Transantral Transnasal
Lateral Wall of Orbit	Tranorbital Transconjunctival

sphenoid sinus posteriorly (Figure 1). During this exposure one should be careful not to penetrate the skull base; traumatise the anterior or posterior ethmoid arteries; or inadvertently enter the orbit or sphenoid sinus.

The lamina papyracea is carefully removed, commonly using a Cottles elevator, with care taken to avoid trauma to the periorbita (Figure 2). Fragments are removed using the Blakesley forceps.

An inferiormedial orbital strut is preserved (Figure 3) to keep the eyeball in the same axial position reducing the risk of double vision.

If an inferior decompression is required, the medial aspect of the orbital floor is often thicker than the lamina papyracea and is thinned carefully with a high speed diamond burr with the inferior orbital nerve used as the

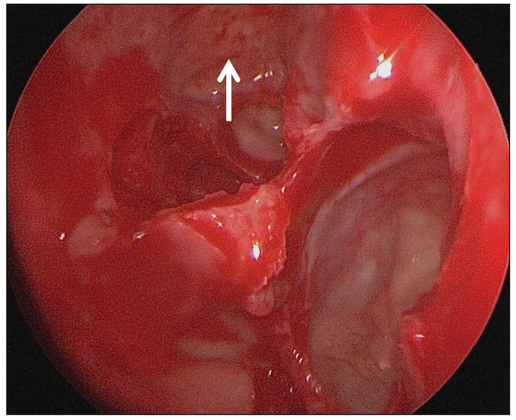

Figure 1: *Image of left skull base after complete ethmoidectomy (white arrow)*

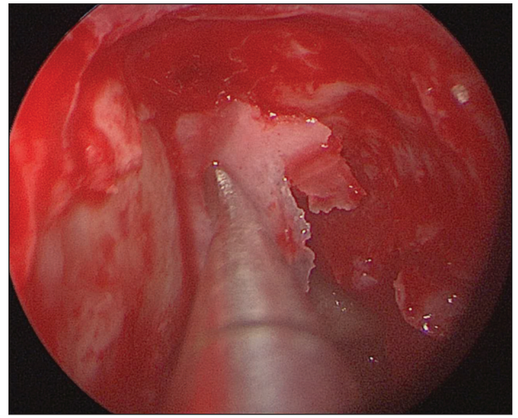

Figure 2: *Left lamina papyracea being removed with cottles elevator.*

posterior lateral limit. The periorbita is carefully lifted off the floor of the orbit and the floor is then carefully removed with a j shaped curette or sickle knife.

The periorbita is then incised and septations broken down with gentle blunt dissection so that fat prolapses into the nasal cavity. (Figure 4).

Be aware that posteriorly there is less extraconal fat, so the medial rectus is very quickly encountered and can sometime be seen through the periorbita.

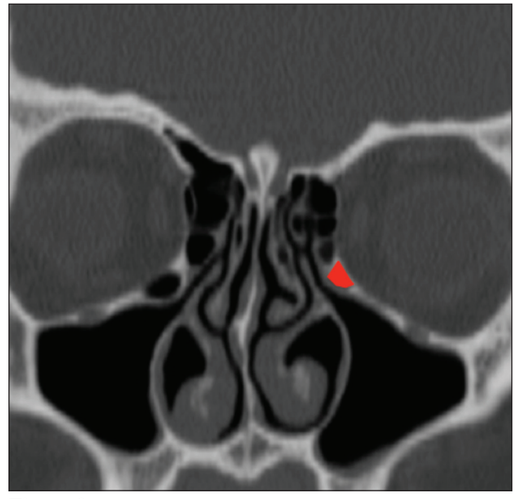

Figure 3: *Coronal CT scan showing inferomedial strut of bone that is preserved between the medial orbital wall and inferior orbital walls.*

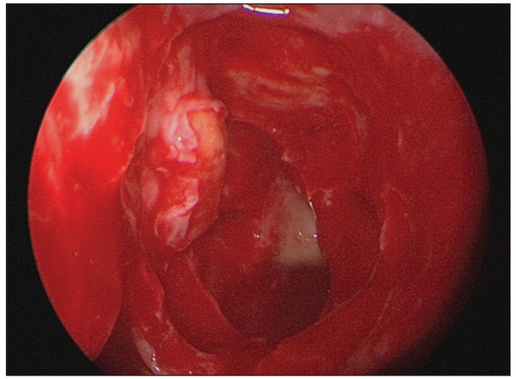

Figure 4: *Left sided periorbita incised allowing orbital fat to herniate into ethmoid cavity.*

When decompression surgery is performed, expected benefits include reduction in exophthalmos, periorbital puffiness (swelling and fat prolapse) and lid retraction. Results are generally proportional to the extent of bone removal and fat herniation into the sinuses.[7]

Other positive effects of decompression include a decrease in intraocular tension and relief of pain, improvement in pre-existing strabismus and cure of postural visual obscuration in patients with orbital and optic nerve micro vasculopathy.[8]

Optic Nerve Decompression

Decompression of the optic nerve employs similar techniques to orbital decompression.

A complete spheno-ethmoidectomy is performed, and lamina papyracea is elevated posteriorly to the orbital apex and the optic tubercle (the thicker bridge of bone between the ethmoid and sphenoid).

The optic nerve is identified in the sphenoid sinus and the optic tubercle is drilled to eggshell thickness and removed to expose the optic nerve sheath (Figure 5).

Using this technique, it is possible to decompress 180-270 degrees around nerve.

Some authors advocate incision of the optic nerve sheath along the optic nerve and through the Annulus of Zinn in order to relieve idiopathic intracranial hypertension. Place the incision(s) at the superomedial quadrant, as the ophthalmic artery is located in the inferomedial quadrant of the optic canal.

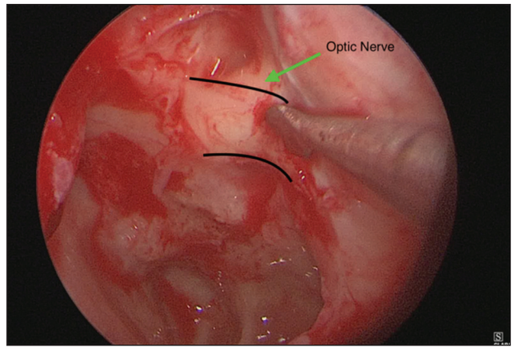

Figure 5: *left optic canal decompression.*

However, opening the optic nerve sheath is controversial and exposes the patient to CSF leakage as well as the ophthalmic artery to injury and should therefore be reserved for very specific cases.[9]

Summary

Transnasal surgery gives good access to the medial orbital compartment and optic nerve

The indications for orbital and optic nerve decompression must be carefully considered

Decision making must be done in conjunction with an ophthalmologist

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Auricular prominence and otoplasty – An overview

Abdul Nassimizadeh[1] (MBChB, BMedSci, MRCS (ENT), **Mohammad Nassimizadeh**[1] (MBChB, BSc, MRCS) and **Grace Lee Peng**[2] (MD FACS)
[1]University Hospital Birmingham, Mindelsohn Way, Edgbaston, B15 2TH
[2]Facial and Reconstructive Surgery, 120 S Spalding Drive, Beverly Hills, CA USA 90212

Correspondence:
Abdul Nassimizadeh

E-mail: anassimizadeh@gmail.com

Abstract

Auricular prominence is a common cosmetic variation which has significant psychological effects on school-aged children. Though the definition prominence itself is highly subjective, it is well documented that alterations in ear size, shape, position and projection having significant influences on overall individual appearance. It can ultimately lead to poorer educational performance and longer lasting psycho-emotional issues. While moulding techniques are commonplace in the first six months of life, thereafter there needs to be the consideration of potential surgical intervention if required. These can be divided in cartilage-preserving and cartilage-cutting techniques. In this article we give an overview of auricular prominence, ear anatomy, clinical evaluation, historical management, current treatment options, and potential complications.

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Key words

Otoplasty, Pinnaplasty, Auricular Prominence, Bat Ears, Ear Protrusion

Introduction

Affecting 5-10% of the population, auricular prominence is a common cosmetic deformity, with ear size, shape, position and projection having significant influences on overall individual appearance.[1,2,3] Though there have no significant associated functional deficits, it has a profound psychological impact, especially in a younger demographic and school-age children.[3,4]

These individuals have been found to have difficulties in social integration, manifesting as behavioural problems and worsening school performance.[3] Being bullied, teased and ostracized eventually leads to feelings of inadequacy, as well as eventual social and economic disadvantages from lack of attendance or engagement with education. Ultimately the backlash from this cosmetic deformity can lead to psycho-emotional issues lasting a lifetime.[5,6,7]

Ear correction surgery is the fourteenth most frequent cosmetic surgery in females, and sixth in males.[8] With evidence highlighting enhanced self-confidence, as well as positive subjective impacts on general health and well-being, in addition to improved school performance, parents often refer their children for surgical opinions. It has been shown that early treatment has greater positive impacts.[1,3,9]

It was for the aforementioned reasons that the National Health Service (NHS) released guidelines for consideration of surgery in those under the age of nineteen years. However, in this economic climate there has been an increased scrutiny regarding most forms of aesthetic surgery including otoplasty.

Anatomy

The architecture of the auricle primarily involves the helix, antihelix, concha, tragus and lobule. There are also ancillary structures such as the antitragus, intertragal incisures and Darwin's tubercle.[3] While the otic placode presents during the third week of gestation, the external ear continues to grow even after skeletal maturity with ear length increasing for a longer duration than width.[2,10] Even with increasing age, gradual microscopic changes with regards to cartilage cell density and numbers of elastic fibres play important roles when considering intervention, such as decreased skin elasticity and resilience.[11]

With regards to innervation, the auricle receives its supply from the auriculotemporal branch of the trigeminal nerve, facial nerve, glossopharyngeal nerve, Arnold's nerve from the vagus and nerves from the second and third cervical plexus. Vascular supply is typically from the superficial temporal artery, the posterior auricular artery and the lesser occipital arteries.[10]

There have been numerous anthropomorphic studies with regards to defining the ideal ear, finding common distances and angles. The distance between the lateral helix to the scalp should ideally be 20mm, producing an auriculocephalic angle ranging from 20 to 30 degrees. The conchal bowl extends to a depth of 15mm, and produces a conchoscaphal angle which is normally less than 90 degrees.[3,10,12,13]

When considering vertical height and width, the latter should be approximately 55% of the former. If the typical height is approximately 60mm, this would make the width around 35mm. Irrespective of ethnicity, males have been found to have longer and wider ears when compared to females across all age groups.[2,3,14,15]

Auricular Prominence

In this review we will mainly consider prominauris, the most common indication worldwide when performing otoplasty.[4,10] Though, it must be noted that there are other aetiology resulting in malformation which require surgical intervention.

Prominauris is typically seen when the auriculocephalic angle is greater than 30 degrees or the conchoscaphal angle is equal to or greater than 90 degrees.[4,16,17]

It can be inherited in an autosomal dominant pattern, with questions regarding family history important during consultation. The two main factors resulting in this condition include a poorly developed antihelical fold and a hypertrophied conchal cartilage but it can also include other alterations including a flat scapha or prominent lobule.[4,10,18,19] These irregularities may occur in isolation, but most frequently produce protrusion together in varying degrees.

If considering the mechanism, when the antihelical fold is inadequately curved, there is a prevention of the normal posterior folding of the helical-scaphal unit, which in turn lengthens the distance between the mastoid skin and the helical rim, leading to greater ear prominence. Specifically, this variation leads to prominence of the upper and middle thirds of the ear. In contrast, conchal hypertrophy deepens the bowl and displaces the helical rim, forcing the auricle away from the scalp and brings forward the middle third of the auricle.[20,21]

Optimal Age of Intervention

It is universally accepted that the ideal time of therapeutic or operative intervention is between the ages of four and six.

Between these ages, the external ear reaches approximately 85% of its maximal width and 50 to 60% of its maximal height, in addition to having a patient demographic not yet fully subjected to bullying.[3] By the age of six, the ear is technically classified as a mature ear. Cartilage pliability is still malleable enough to contribute to higher rates of success and delaying management to this age has not shown to contribute significantly to psychological morbidity long term, though there are studies showing poorer quality of life leading up to this point from peer ridicule.[6,7,20,21] If the patient is far older, the auricular cartilage becomes more calcified and less malleable, resulting in higher rates of failure.[7]

It is important to note we are considering patients who have grown beyond the age of nonsurgical moulding or splinting techniques. While these are an effective avenue in the first few weeks of life and up to six months, beyond this the rigidity of the cartilage makes it resistant to conventional moulding techniques.[22,23]

Patient Evaluation and Analysis

There are numerous anatomical variables which are used to evaluate the auricle. When analysing the ear in isolation it is important to consider whether in general it is oval, round, triangular, rectangular or variations and combinations of these shapes. The position and size of the intertragal notch must be noted, with significant variations noted between individuals. With regards to the lobule, it can be attached or free, as well as variable in size and shape.[2]

However, analysing the ear without consideration of the face globally will eventually lead to suboptimal surgical results. The Frankfort horizontal plane is an axis used to gauge spatial relationships between the ear, eyebrow, eyes and nose.[23,24] This passes through the inferior orbital rim to the top of the tragus. Above this, the superior edge of the ear should theoretically be level with the eyebrow and lie parallel to the Frankfort line. Below it, the lobule should lie at the level of the nasal tip, with the overall length of the ear equal to that of the nose, from the nasion to the subnasale.[3,23,24,25]

There is no requirement for pre-operative imaging in patients with normal acoustic function, but pre-operative photographs must be taken anteriorly, posteriorly and laterally.

As mentioned previously, psychological impact of auricular prominence is well noted and must be addressed during the consultation. It is important to manage expectations of surgical outcomes, as well as garnering an insight into the patient's viewpoints and understanding. Self-confidence and social interactions are two topics which should be spoken about individually. There are also calls for psychological evaluation, especially in a paediatric population pre-operatively, with hopes of engagement in the decision-making process.[3,7,26]

Aesthetic Goals for Intervention

The primary goal of therapeutic intervention is to restore acceptable auriculocephalic, conchoscaphal and conchomastoidal angles.[18] The results should be reliable, stable and satisfactory for the patient, as well as creating a closely symmetrical result.

More specifically, McDowell has previously listed a set of goals with regards to otoplasty.[27] These included to correct the protrusion of the upper, middle and lower third of the auricle, with protrusion of the upper third the most important. If examining the patient anteriorly, the helix should extend beyond the antihelix, at least up to the mid-ear. The helix should have a smooth regular line. The postauricular sulcus should not be markedly distorted. The ear should not be placed too closely to the head and the positions of the two ears should match closely, ideally within 3mm at any given point.

Historical Intervention

Dieffenbach reported the first documented technique of otoplasty in 1845, where he described the resection of retro-auricular skin with subsequent conchomastoid fixation.[28] He did this when managing auricular prominence post-traumatically, but it only corrected the cephaloauricular angle and there was significant recurrence.[17] The technique was enhanced in 1881, by Ely who included conchal and triangular fossa cartilage excision as a second-stage procedure.[17] Luckett addressed the antihelical fold and its restoration in 1910, where he added a posterior excision of skin and cartilage along the antihelical fold, but it resulted in an unsightly sharp antihelical border.[29] In 1952, Becker modified the reconstruction with a tubing technique, where he made a cartilaginous tube to accentuate the antihelix, with similar reports elsewhere at the time.[30]

But it was in 1963, when the Mustarde technique was developed that the modern era of otoplasty fully took flight. He advocated the use of mattress sutures without the excision of any cartilage.[31] Furnas, in 1968, elaborated further by introducing a suture technique for the conchal bowel which was widely popularised.[32] Since then, there have been a variety of different techniques using these baseline principles alone, in conjunction or with further modifications.

Current Surgical Intervention

With over 100 reported procedures in the literature there is likely no gold standard, but we will attempt to give a brief overview of popular management options.[17,33] Techniques can be subdivided in cartilage-preserving and cartilage-cutting, though most surgeons use a combination of these.

Both techniques require access, using a postauricular incision eventually hidden in the postauricular sulcus. Though skin is excised in some techniques, with adipofascial tissue left behind, others advocate that this is not necessary and may result in hypertrophic scarring. The scaphal region and mastoid fascia are both exposed when raising anterior and posterior skin flaps.

Though some surgeons prefer operating under local anaesthesia in adults, general anaesthesia is commonly required in children.

Cartilage Preserving

Mustarde's and Furnas's techniques are both cartilage-preserving. Mustarde's technique involves full thickness horizontal mattress suturing through the anterior perichondrium along the posterior cartilage using non-absorbable sutures to re-create the antihelical rim, but not conchal bowl. A cartilage tattoo with methylene blue can be used to mark the locations if required. There are typically three to four sutures required when recreating an antihelical fold placed about 10mm apart.[17,34] The Furnas technique on the other hand focused on the management of excessive conchal cartilage.[32] Four permanent conchomastoid sutures are placed, avoiding the anterior conchal skin and help with relocation.

Cartilage Splitting

Cartilage-splitting techniques were started by Gibson and Davis, who noticed that when the cartilage was incised it tended to warp to the opposite side.[35] There have been a large number of subsequent modifications involving scoring, scratching, scraping, filing, rasping or abrading.[36,37,38] Criticism of these techniques include the creation of sharp contours, but with modifications, this is gaining greater advocacy when used appropriately.[18]

There has also been the rise of incisionless otoplasty such as transdermal abrasion of the cartilage at the site of the desired fold and percutaneous Mustarde-type sutures, as well as laser-assisted cartilage remodelling, however these are not yet commonly encountered.[39,40,41] It is also important to note that there are non-surgical options becoming available using plastic or metallic splints which remain in place for 2-12 weeks.[3]

Surgical Complications and Sequelae

These can be divided into early and late complications. The most concerning early complication is haematoma formation leading to cartilage and skin necrosis, ultimately

forming a cauliflower deformity. This can also occur secondary to overtightening of sutures and occasionally excessive pressure from the dressing. Other early complications include bleeding, infections including perichondritis and dehiscence.

Late complications can include excessive scarring and keloid formation, a hidden helix anteriorly, suture extrusion, hypersensitivity and deformities such as a telephone ear deformity, reverse telephone ear or a vertical post deformity. The most common aspect to consider in patients, however, remains dissatisfaction and poor aesthetic outcomes. Residual asymmetry is apparent six months post operatively and loss of correction occurs in 6.5 to 12% of cases, requiring re-operation.[42]

Conclusion

It is clear that otoplasty is a useful surgical management option for patients with auricular prominence, and can provide a vast improvement in patient quality of life. There are variable management options which must be considered during consultation for optimal outcomes.

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Facial palsy in childhood

Matthew Rollin, FRCS (ORL-HNS)
Consultant ENT Surgeon
Imperial College Healthcare NHS Trust
London, UK

Correspondence:
Mr Matthew Rollin FRCS (ORL-HNS)
Department of ENT / Head & Neck Surgery
Imperial College Healthcare NHS Trust
London W2 1NY

E-mail: m.rollin@imperial.ac.uk

Abstract

The facial nerve is the most commonly morbid cranial nerve. It contains motor, sensory and parasympathetic fibres, and has a complex course which is a source of vulnerability. Although facial palsy is less common in children than in adults, idiopathic palsy is the most common diagnosis in both groups and is regarded as having a better prognosis in children than in adults, irrespective of treatment. There is a paucity of high-quality evidence to inform the treatment of children with facial palsy; most recommendations are based on evidence from adult studies. The literature generally supports medical over surgical therapies; oral steroid and anti-infective agents are the cornerstones of management.

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Key words

Facial palsy, paediatric, Bell's palsy, paralysis, neuropraxia

Pathophysiology and Epidemiology

There are many potential causes of facial palsy, the more common being infectious, traumatic, congenital, neoplastic and idiopathic. Approximately half of acute cases are idiopathic and are given the label "Bell's Palsy".

Bell's Palsy

In one study of 170 patients aged 18 years or under, Bell's palsy was the final diagnosis in 42%, trauma in 21%, infection in 13%, congenital palsy in 8% and neoplasia in 2%[1]. The estimated prevalence of Bell's Palsy is 20 per 100 000 people per year[2].

One widely accepted theory is that Bell's palsy occurs secondary to axonal spread and multiplication of a latent neurotropic virus, causing neuropraxia through inflammation and demyelination. Since there is no effective method to test for the presence of such a virus within the nerve clinically, however, most patients with idiopathic palsy are given "Bell's Palsy" as a diagnosis[3].

There is no geographical, racial or gender predilection in Bell's palsy, but there is a threefold increased risk in the third trimester of pregnancy and first post-partum week[4], and a fourfold increased risk in persons with diabetes[2].

Infection

Herpes Simplex Virus is widely thought to be the neurotropic virus behind Bell's palsy in both children[3,5] and adults[6,7]. There is some direct evidence for this. One case-control study examined samples of endoneurial fluid and posterior auricular muscle in patients undergoing decompression surgery for idiopathic palsy. HSV-1 genomes were identified in 11 of 14 cases, and in none of the controls[8]. In a prospective case-control study of children with acute unilateral palsy, positive PCR and ELISA for HSV-1 genomes were significantly more likely in cases than controls[3].

Herpes zoster infection has also been linked to facial palsy. In societies with low immunisation rates, acute zoster viraemia has been identified with PCR or serology in up to 37% of cases, including both zoster sine herpete (without skin signs) and herpes zoster oticus (Ramsay-Hunt syndrome).

Acute otitis media (AOM) was once considered a major cause of facial palsy in childhood, although the incidence has fallen from approximately 2% in the pre-antibiotic era to 0.16% more recently[9].

Studies in endemic areas have found Lyme disease to be a common cause. In one paediatric study in an endemic area of the United States, Lyme disease was the causative factor in 50% of cases, followed by idiopathic palsy in 26%, AOM in 12%, varicella (6%), zoster (4%) and coxsackie virus (2%)[10]. Similar findings were reported from a paediatric population in a Lyme-endemic area of Scandinavia[11].

Lyme disease is infection with bacteria of the genus Borrelia – typically Borrelia burgdorferi. The vector is a tick (genus Ixodes, usually in nymphal form). In Europe, Ixodes ricinus (the castor bean tick) is the usual culprit, and transmits the infection more rapidly than Ixodes scapularis, the typical North American vector. The disease is not known to be transmissible between people, via other animal vectors, or through faeco-oral routes.

Facial palsy may be the only symptom of Lyme disease[12], can be unilateral or bilateral, and usually resolves within eight weeks if the underlying infection is treated[13]. It is the most common cranial neuropathy in Lyme meningitis[14,15], but can occur secondary to Lyme disease without meningeal involvement. One theory is that this relates to direct invasion of the nerve trunk by Borrelia; however, evidence is weak, comprising retrospective studies showing that a high percentage of children with confirmed neuroborreliosis and unilateral palsy had an ipsilateral source such as a tick bite in the head & neck region[11,16].

HIV infection rarely causes facial palsy in children directly, although where it does, it typically occurs during the seroconversion illness[17]. Where HIV infection leads to reduced cellular immunity, other opportunistic infections may result in palsy[18].

In areas without access to vaccination programmes, compression from mumps parotitis may be a common cause[19].

Other infections which have been linked to onset of facial palsy in children include CMV, EBV, adenovirus, rubella, mumps, influenza B, coxsackie virus and Rickettsia (Mediterranean spotted fever)[19,20,21]. Palsy in the presence of purulent otorrhoea unresponsive to typical antibiotic therapy raises the possibility of tuberculosis[22,23].

Congenital

Congenital facial palsy may in reality be traumatic and perinatally acquired. The facial nerve position relative to the infant mastoid, the mandibular angle and the maternal sacrum increase its vulnerability during birth; instrumental delivery compounds the problem. The incidence is 1.8 per 1000 live births, and risk factors include large head size (birth weight greater than 3500g), forceps-assisted delivery and prematurity[24,25].

True congenital palsy can occur in isolation, but is commonly associated with multiple cranial nerve palsies, and multisystem dysmorphia. It can occur secondary to branchial arch development sequence disorders[26]. Two genetic loci for developmentally-derived congenital palsy have been identified, designated hereditary congenital facial paresis 1 (chromosome 3q21–22) and 2 (chromosome 10q21.3–22.1)[27,28,29].

Möbius (Moebius) syndrome typically comprises unilateral or bilateral congenital facial and abducens palsy, although cranial nerves III, IV, V and VIII can also be affected[30,31]. Anatomical analysis has shown hypoplasia of associated brainstem nuclei and nerves[32]. Up to one third of affected individuals have associated intellectual problems and/or autism[33]. Genetic linkage analysis points to a locus at 13q12.2–13, although distinct causative genes have yet to be identified[34,35]. It is possible that Möbius and the other facial paresis syndromes overlap and share common aetiology.

Goldenhar syndrome (oculo-auriculo-vertebral dysplasia) is a group of congenital anomalies affecting structures arising from the first and second branchial arches. Congenital facial paralysis is notable in more severe forms[36].

Congenital Asymmetric Crying Facies is caused by unilateral agenesis or hypoplasia of the depressor anguli oris. 45% of cases are associated with developmental anomalies in other systems[37].

Facial palsy has been noted in 38% of patients with CHARGE syndrome[38], and some reports detail patients with an aberrant facial nerve course, complicating cochlear implantation[39].

Trauma

The greater superficial petrosal nerve tethers the geniculate ganglion, leaving the tympanic and mastoid segments of the facial nerve susceptible to shearing forces during sudden head movements, particularly deceleration injures. Despite this, the rate of palsy in paediatric temporal bone fractures is low. One study of children up to 14 years of age found facial weakness in 3% of 72 fractures[40].

Neoplasia

Neoplasia in childhood is thankfully rare. The two most common causes of malignancy-associated facial palsy are infiltration of the temporal bone by leukaemia[41,42], and rhabdomyosarcoma[43,44,45]. Neoplasia of the facial nerve is rare in childhood.

Other aetiologies

Cholesteatoma should be considered if the onset of palsy is gradual. Tympanic membrane examination will often reveal the diagnosis, but congenital cholesteatoma can present with neurological or hearing impairment before the tympanic membrane is involved[2].

Melkersson-Rosenthal syndrome classically comprises facial paralysis, episodic facial swelling and a fissured tongue; although the majority of cases are “atypical” forms, without one component of the triad. Onset is typically in adolescence, with recurrent episodes of facial paralysis thereafter[46]. Although one report noted presence of perivascular granulomas in oedematous tissues[47], the cause is unknown and treatment therefore controversial.

Neurosarcoidosis and Guillain-Barre syndrome have been linked with subacute bilateral facial palsy in children[48]. Palsy has been reported as part of an otological presentation of granulomatosis with polyangiitis (GPA)[49]. It has also been linked with severe systemic hypertension of childhood and adolescence, particularly in combination with headache, altered conscious level, vomiting, convulsions or other focal neurology[50,51,52].

Diagnosis

The history should cover onset, rate of deterioration, associated symptoms such as taste sensation, hyperacusis and headache, ear discharge, recent infections or illnesses, tick bites and recreation in woodlands, and HIV risk factors.

Relevant past medical history includes diabetes mellitus or other immune-modifying diseases, history of chronic ear disease, and immunisation history.

Physical examination should include the facial nerve, parotid, ear, neck and other cranial nerves as appropriate. In a young child who will not comply with examination, the palsy may not be apparent until the child cries. Forehead sparing can be detected by the presence of skin creases and paralysis of the lower eyelid, which may cause ectropion and tear spillage; loss of midfacial muscle tone may cause droop of the cheek and loss of the nasolabial fold. Oral continence may be lost, and speech may become distorted as plosive sounds are undermined by air escape.

The typical history of idiopathic palsy includes involvement of all peripheral branches, with acute onset over 24 – 48 hours. Classically there is rapid progression, reaching maximal clinical weakness within three weeks from symptom onset. There may be a prodrome, including ear pain and altered hearing level, but this does not occur commonly enough to be considered typical.

Painless, nontender swelling and erythema of the face progressing to facial palsy suggests Lyme disease[53], as do presence in a Lyme-endemic area, recent recreation in woodlands, and onset in spring or early summer. The typical course of Lyme disease includes onset of a non-pruritic non-tender rash approximately one week after infection. It can take the form of erythema migrans, and a bullseye rash pattern is considered highly suggestive of Borrelia infection.

Congenital palsy will usually come to attention shortly after birth. Prolonged traumatic labour, instrument delivery, head & facial injury, periauricular ecchymoses and haemotympanum may point towards a traumatic cause, but do not in themselves exclude the presence of developmental anomalies.

Topographical testing to determine the site of a facial nerve lesion has little clinical relevance and carries no prognostic value. It is therefore principally of interest in the study of the history of medicine.

Serology

Lyme serology is negative for four to six weeks after initial infection and is highly sensitive and specific thereafter. It is rare for palsy to develop before the patient is seropositive. Lyme serology is recommended for all children with acute-onset facial palsy in the spring, summer and early autumn.

Lumbar puncture

Clinical suspicion of meningitis should provoke lumbar puncture. The CSF concentration of anti-Borrelia antibody is diagnostic of neuroborreliosis. CSF analysis can also aid the diagnosis of Lyme disease without meningeal involvement, as the majority of children with isolated Lyme disease-associated palsy have abnormal findings, including elevated WBC and/or protein levels. These findings are clearly not pathognomonic, however, therefore the decision to obtain CSF should be made in consultation with paediatric infectious disease specialists, microbiologists and the child’s carers[11,54,55].

Imaging

Exact imaging techniques should be discussed with an experienced radiologist; a rule of thumb is that MRI is useful in the evaluation of the intraparotid and intracranial facial nerve, and CT is useful in the evaluation of the temporal bone. Contrast enhancement of the geniculate ganglion on MRI may be seen in idiopathic palsy; however caution is advised, as contrast enhancement of the first genu and proximal tympanic segment may be normal[2,56]. Contrast enhancement of the labyrinth in the context of facial palsy suggests infection with herpes zoster, even in the absence of vesicular eruptions[57].

Coexisting facial and abducens palsy mandates imaging of the brainstem, as the nuclei are co-located in the pons. Imaging is recommended in the slowly-evolving palsy,

and also with those individuals showing a typical onset of idiopathic palsy but failing to show any improvement at six months[58].

Presence of forehead sparing (facial palsy with preserved temporal branch function) mandates imaging. Facial movement may be voluntary or emotional in origin, and the lower motor neurone pathway is common to both. Dissociation of voluntary facial movement from emotional movement therefore suggests a supratentorial problem and mandates imaging.

Imaging is also indicated if clinical findings suggest cholesteatoma, chronic otitis media, mastoiditis, temporal bone trauma or neoplasia.

Electrophysiology

Electrophysiological tests can determine the extent of facial nerve dysfunction, and therefore do have prognostic value. They may also assist in determining when surgical decompression may be recommended and when facial reanimation should be considered. If performed in series, they may allow monitoring of recovery, and so may guide management. In the case of palsy noticeable shortly after birth, electrophysiology can help distinguish between traumatic and congenital lesions.

In the first few days after onset of palsy, assessment of the blink reflex (via stimulation of the supraorbital nerve and the trigeminofacial reflex pathway) can confirm the peripheral location of the lesion, and assess the degree of axonal conduction block, thereby holding prognostic value[59].

Electroneuronography (ENoG, also known as a motor nerve conduction study) compares nerve conduction between the two sides, and therefore requires a functioning contralateral nerve. The main trunks are stimulated at the stylomastoid foramen, and compound muscle action potentials (CMAPs) are detected at the nasolabial fold. Their amplitude in comparison to the unaffected side estimates the degree of axonal loss[59]. Wallerian degeneration normally begins within 3 days, CMAPs generally reach their minimum level between 7 and 14 days post-onset – hence ENoG is most useful between 3 and 21 days post-onset. An amplitude reduction of 95% or more within this window is associated with a poor prognosis[59,60,61].

Electromyography (EMG) is more useful than ENoG in cases of delayed paralysis and can be used in bilateral palsy. The patient is asked to attempt facial movement, and motor unit potentials are measured in the orbicularis oris and orbicularis oculi muscles. Fibrillation potentials are seen in the context of Wallerian degeneration, and polyphasic potentials indicate early reinnervation although, in this situation, clinically obvious muscle movements may not occur for another three months. Electrical silence is a poor prognostic indicator.

Classification

Several classifications of nerve injury exist; the most commonly used are the Seddon and Sunderland classifications[62].

Treatment

Treatment should be guided by the pathophysiology, severity and timescale of the palsy. In all cases, the patient and their carers should understand the importance of eye care. If the patient is unable fully to close their eye, they are at risk of corneal abrasion, and should be treated with artificial lubricants during the day, and ointment and patches at night. Taping the eyelid is no longer advised, owing to the possibility of the tape slipping and abrading the cornea. Further measures are not usually required in the acute setting; tarsorrhaphy and gold weight implantation may be required if the palsy is prolonged.

Idiopathic (Bell's) palsy

Direct evidence is poor in the paediatric population, as there are no high-quality trials, however the prognosis is generally favourable[63,64]. The mainstay of treatment in adults is early administration of oral steroid – a recommendation supported by several high-quality trials[65,66,67,68,69,70,71] and meta-analyses[72,73,74,75,76]. Trials have reached differing conclusions on the merits of antiviral agents administered with steroid therapy; antiviral monotherapy was found to be no better than placebo.

In the absence of high-quality evidence in children, national bodies and learned societies recommend early intervention with oral steroid. A typical regime might be Prednisolone 2mg/kg od (maximum 60mg) starting within 3 days of symptom onset, continuing for 5 days, followed by a short taper[77].

Although large well-regarded clinical trials have not found additional benefit with added antiviral therapy, some trials have found benefit, particularly for (adult) patients with severe palsy[68]. Some bodies therefore also suggest combining steroid therapy with an oral antiviral such as valaciclovir 20mg/kg (maximum 1g) tds for 7 days. Expert opinion has not reached consensus on this point[77].

Ramsay-Hunt syndrome

Patients with herpes zoster oticus should be treated with antiviral therapy and oral steroid.

Other infections

AOM and neuroborreliosis should be managed according to local protocols, in consultation with expert microbiologists. Borrelia is often treated with doxycycline, or with amoxicillin plus cefuroxime.

Surgical intervention

There are no high-quality studies concerning early surgical management of facial palsy in children. One non-controlled study in adults evaluated surgical decompression of the labyrinthine segment via a middle cranial fossa approach, showing benefit in patients with severe palsy (defined as ≥90% reduction in CMAPs on electrophysiology)[78]. Another study reported that early surgical decompression was not associated with improved recovery but was associated with reduction in future episodes of palsy[79]. Guidance from the American Academy of Neurology in 2001[80] and AAO-HNS in 2013[81] do not recommend surgical decompression, owing to the paucity of high-quality evidence and the potential for surgical complications.

Children with congenital or permanent palsy may be offered surgical intervention, either for static appearance or reanimation[82,83]. Static procedures aim to achieve oral and labial symmetry at rest; reanimation techniques aim to allow some facial expression. These techniques cannot restore normal function, although even a modest improvement may be of great psychological benefit[83,84,85,86,87]. The timing of such procedures is best decided in a multidisciplinary team.

Prognosis

True congenital facial palsy has a relatively poor prognosis, owing to underdevelopment of crucial structures. Conversely, perinatally-acquired traumatic palsy has a good prognosis, with nearly all studied patients showing some improvement over time[88].

Most studies have investigated the prognosis of idiopathic palsy, and observational studies of children have been conducted. Overall, the current literature finds that the majority of children recover with minimal or no dysfunction, and that likelihood of complete recovery correlates with severity, with partial/incomplete palsies more likely to recover to normal function than dense/complete palsies. Typically, improvement is seen within six months[88,89,90,91]. The largest cohort of children studied to date was 463, within an overall cohort of 2570 in the 25-year Copenhagen study; 90% of children recovered full facial function[88].

Prognosis is generally more favourable if some recovery is seen within 21 days of onset[92]. If no improvement has been seen within 4 months, a diagnosis of idiopathic palsy should be questioned, and confirmatory investigations arranged[93]. Severe lesions, requiring extensive axonal regrowth, are more likely to lead to axonal misdirection, and thereby synkinesis or "crocodile tears".

The House-Brackmann grading system is intended to be a shorthand for severity, and thereby provide prognostic information. Although the literature bears this out, one study (in adults) has suggested that muscle electrophysiology provides superior prognostication, particularly in more severe cases[94].

Work is underway to determine reliable prognostic thresholds based on electrophysiology. One study reports that, of those (adult) patients with CMAPs >30% of normal, 84% recovered normal facial function[95]. CMAPs <10% of normal are generally agreed to represent poor prognosis, although even in this group up to one-third of patients may show near-complete recovery[91]. There are no recent studies evaluating surgical decompression; those conducted 30–40 years ago agreed that surgical intervention was unlikely to alter prognosis, and carried significant risks[91,95,96].

Recurrence of idiopathic palsy is unusual in all age groups, and should provoke further investigation, including imaging and serial blood pressure measurement[51]. One series reported a 6% recurrence rate (11 of 182 children), of which two cases were associated with Melkersson-Rosenthal syndrome[97].

Key points

- Idiopathic (Bell's) palsy is the most common cause of acquired facial palsy in childhood.
- Other important causes of acquired palsy include hypertension and Lyme disease.
- The anatomy of the childhood facial nerve accounts for its vulnerability during birth trauma.
- Imaging and electrophysiology are the most valuable investigations.
- The prognosis of idiopathic palsy in childhood is generally good.
- Treatment involves supportive measures, particularly eye care, oral steroid therapy and possibly antiviral therapy. Most evidence for this comes from studies conducted in adults.

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Abstracts: ENT Masterclass Trainees' Gold Medal 2019

Accuracy of clinical coding and financial renumeration for endoscopic sinonasal procedures: Multidisciplinary changes through a two cycle quality improvement project

Mr Bassem Mettias, Mr Joshua Whittaker, Miss Katie Midwinter, Mr Yujay Ramakrishnan
Institution: ENT, University Hospitals Nottingham NHS trust, UK.

Correspondondence:
Mr Bassem Mettias (ENT ST5 East Midland Trainee)
Email: bassemadel@hotmail.com Contact number: 07426813089
Address: ENT department, University Hospitals of Leicester, Royal Infirmary Hospital

ABSTRACT

Background
Clinical coding is the process of translating medical terminology into an international syntax familial to the non-medical staff. Health resource grouping (HRG) is the method to summarize disease diagnosis and procedure into informal units of Hospital Episode Statistics (HES).

Aims
Assess the accuracy of clinical coding in endoscopic sinonasal procedures. Investigate the effect of intervention to improving the accuracy.

Methods
A two-cycle service evaluation including all rhinology patients operated on at a single tertiary centre in the UK in 2017. Each cycle lasted 4 months. The operation notes were reviewed by two clinicians briefed in clinical coding to generate the 'standard' codes template. The 'original' clinical codes assigned and their related HRG retrospectively obtained. A Second cycle piloted the generated multidisciplinary standard template (MST) to improve accuracy between a rhinology firm who used the template and another who didn't.

Results
45 and 49 procedures were recorded in the 1st and 2nd cycle respectively. From the first cycle, accuracy of original coding was 62%. Of the miscoded procedures 18%, 9% and 11% were over, under and wrongly coded respectively. The inaccuracy in coding resulted in a tariff over-payment of £109.92 per procedure. In the second cycle, streamlining coding procedures significantly improved in the firm who used MST.

Conclusion
There were significant inaccuracies in translation of endoscopic sinonasal procedure into clinical coding. The inaccurate financial remuneration cause constrained on healthcare systems. MST is easy to interpret by clinicians and non-medical staff. It can reduce errors in coding and enable more accurate funding allocation to hospitals.

Streamlining tonsillitis and peritonsillar abscess treatment. The new 4 hour target!

Timothy Williams, Emily Heywood, Mona Saad, Mark Waller, Glen Watson
E-mail: twilliams23@icloud.com
Institution: ENT Department, Royal Hallamshire Hospital, Sheffield Teaching Hospitals

Introduction
There has been a significant rise in tonsillitis and peritonsillar abscess related admissions and complications. This can be attributable to a substantial reduction in tonsillectomies since the introduction of procedures of limited clinical effectiveness (POLCE). As a result there has been an increasing amount of bed days associated with these common ENT presentations with significant financial burden. Data suggests there have been no net savings as a result of these additional bed days.

Objective
To create, implement and evaluate an evidence based 4 hour treatment bundle for the management of tonsillitis and peritonsillar abscess.

Method
All patients who presented to a large teaching hospital with tonsillitis or peritonsillar abscess over two, one month periods were included, between which the 4 hour treatment bundle was implemented. This treatment included the use of intravenous fluids, analgesia, antibiotics, corticosteroids, plus needle aspiration in peritonsillar abscess patients. Patients were reassessed on completion of treatment bundle. Outcomes measured were overnight admission and re-admission rates.

Results
Admission rates prior to the introduction of the treatment bundle was 75% with 3 readmissions. Admissions reduced to 25% after the introduction of the treatment bundle and reassessment with no readmissions.

Conclusion
The introduction of a 4 hour treatment bundle safely reduces admission rates of patients with tonsillitis and peritonsillar abscess. This data is being used to introduce an ambulatory day unit on the ward, to further provide a streamlined, safe treatment service to reduce pressure on beds as well as reduce financial burdens associated with patient admissions.

Canalostomy as a surgical approach to local drug delivery into the inner ears of adult and neonatal mice

Guopeng Wang, Jingying Guo, Lu He, Shusheng Gong
Department of Otolaryngology-Head and Neck Surgery, Beijing Friendship Hospital, Capital Medical University, Beijing 100050, China
Email address: guopengent@163.com
All authors have agreed the contents of the paper for submission.

Objective:
Local delivery of drugs into the inner ear is a promising therapy for inner ear diseases. In this study, we aimed to evaluate the effectiveness and safety of the drug delivery through semicircular canals (canalostomy) in both adult and neonatal mice.

Methods
A fast-green dye or adeno-associated virus serotype 8 with the green fluorescent protein gene (AAV^8-GFP) was inoculated into the inner ear of mice through canalostomy. Following surgery, animals underwent swim tests and auditory brainstem response (ABR) measurements. Then inner ears were harvested for morphological studies and immunohistochemistry.

Results
The canalostomy facilitated broad distribution of fast-green dye in both cochlea and vestibular end-organs. In mice after AAV8-GFP injection, no signs of vestibular dysfunction were found, and there were no changes of ABR thresholds after surgery. Extensive GFP expression and no morphological lesions were detected in the cochlear and vestibular end organs. Robust GFP expression was found in inner hair cells, marginal cells, spiral ganglion neurons, vestibular hair cells, vestibular supporting cells and vestibular ganglion neurons.

Conclusion
Canalostomy is an effective and safe approach to drug delivery into the inner ears of adult and neonatal mice and may be used to treat human inner ear diseases in the future.

Neuroprotective effects of NMDA-Rs blocker on the Auditory Cortex in salicylate-induced tinnitus

Yu Song[a], Shan Xiong[a], Furong Ma[a] , Lanqun Mao[b]
[a] Department of Otolaryngology Head and Neck Surgery, Peking University Third Hospital, Beijing, 100191, PR China
[b] Beijing National Laboratory for Molecular Sciences, Key Laboratory of Analytical Chemistry for Living Biosystems, Institute of Chemistry, the Chinese Academy of Sciences, Beijing, 100190, PR China

Abstract
Object
To examine the effect of administering NMDA-Rs blocker at different times on the changes of neurochemicals and electrophysiology in the auditory cortex (AC), the center of auditory perception, in a rat tinnitus model induced by salicylate (SS).

Method
This study simultaneously monitored the dynamic change of ascorbate and glutamate in the AC during SS-induced tinnitus and its response to intraperitoneal administration of MK-801 by in vivo microdialysis with an online electrochemical system (OECS) and high-performance liquid chromatography (HPLC).

Result
We found that the levels of both ascorbate and glutamate were more significantly suppressed in the groups of MK-801 given at 30 min pre or post SS injection than the levels in the group of MK-801 given at 60min post SS injection compared with the SS only injection group. Electrophysiological recording performed on the SS-injection animals revealed that the spontaneous firing rate (SFR) of neurons in the AC was dramatically increased. The animals treated with MK-801 showed a significantly attenuation of hyperactivity in AC both in the groups of MK-801 given at 30 min-pre or -post SS injection and 60min-post SS injection.

Conclusion
These findings suggest that NMDA-Rs are involved in the pathological mechanism underlying salicylate-induced tinnitus, and also indicate that therapeutic effects on tinnitus are depend on the administration time of the blocker of NMDA-Rs , which might advance studies on understanding the therapeutic potential of NMDA-Rs antagonist in tinnitus therapy.

Outcomes of Parotidectomy from a single surgeon in a District General Hospital setting

Prathibha Nanoo, Afeef Aslam, Muhamad Quraishi
Presenting Author: Prathibha Nanoo
ENT Department, Doncaster Royal Infirmary, Thorne Road, Doncaster DN2 5LT
Email: prathibha.sprasad@gmail.com

Abstract

Introduction

80% of all salivary gland tumours occur in parotid. 80% of these are benign pleomorphic or mixed adenomas. Purpose of parotidectomy is to remove abnormal growths in parotid gland. During the procedure the facial nerve is at risk and great care is taken to preserve this.

Aim

To evaluate the outcomes of patients who had surgery under a single surgeon for parotid lumps from April 2004 to Nov. 2018

Methods

A retrospective case notes review of parotid surgeries, by a single surgeon, was performed. Data collected included patient demographics, presentation, radiological and histological investigations, operative findings, outcomes, complications and histopathology.

Results

Between 2004 – 2018, 339 patients underwent parotidectomies. 95.6% of the parotid tumours were benign. 59% of these were pleomorphic adenoma. FNA could accurately confirm diagnosis in 62.5% cases. Temporary facial weakness was noticed in 33% of operated cases. Permanent palsy was seen only in one which was preoperative. Other postoperative complications (seroma, wound infection, Frey's) found in 13% of the studied group.

Conclusion

Outcome rates were comparable with published data. The incidence of postoperative complications is influenced by the pathology, with inflammatory lesions significantly increasing the risk of facial nerve dysfunction and other complications. Overall, the incidence of permanent facial paralysis was less than 1%, but temporary nerve palsy was common at 33%, with most patients regaining normal function within 1 year of the operation. In most cases facial weakness improves to normal or near-normal levels within 6 weeks.

A Study Evaluating The Effects of Throat Packs During Nasal Surgery – A Randomised Controlled Trial

Ali Al-lami, ENT ST8 Surrey & Sussex Healthcare NHS Trust, East Surrey Hospital, Canada Avenue, Redhill, RH1 5RH. aklami@doctors.org.uk. The randomized controlled trial was carried out at Peterbrough City Hospital.

Objectives

The aims of this study were to identify differences in post-operative nausea and vomiting (PONV) and throat pain between throat packed and non-packed patient groups in nasal surgery.

Methods

This was a prospective, double blind, randomised controlled trial. Patients were randomised into throat and non-throat pack groups. A validated PONV questionnaire was completed 6 hours post-operatively. Visual analogue scores (VAS) for throat pain were completed in recovery, 2, and 6 hours post-operatively.

Results

80 patients were enrolled (40 into each group based on power calculation). Mean PONV score for the throat pack group was 2.75 and the mean PONV score for the non-packed group was 0.36. The difference in PONV was not statistically significant (P value = 0.375, 95% confidence interval (CI) = -1.19 - 3.32).

With regards to throat pain VAS scores, in recovery, the mean scores for the throat packed and non-throat pack groups were 2.5 and 1.3 respectively. Statistical analysis showed significant difference with the throat pack group experiencing more throat pain in recovery (P value = 0.018 (95% CI = 1.13 - 2.52). At 2 hours and 6 hours post-operatively, statistical analysis showed no difference in the mean throat pain VAS scores for the throat packed group (2.1 and 2.3 respectively) and non-throat packed group (2.3 and 1.4 respectively).

Conclusion

The use of throat packs in nasal surgery does not confer PONV reduction benefit. The use of throat pack however is associated with a small but statistically significant more throat pain in the recovery period.