Health Policy Advisory Committee on Technology

Technology Brief

Hypoglossal nerve stimulation for sleep apnoea

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For further information, contact the HealthPACT Secretariat at:
HealthPACT Secretariat
c/o Access Improvement Service, Centre for Healthcare Improvement, Queensland Health
Floor 3, Forestry House
160 Mary Street, Brisbane QLD, AUSTRALIA 4000
Email: HealthPACT@health.qld.gov.au Telephone: (07) 3234 0624.

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This brief was prepared by Arlene Vogan from the Australian Safety and Efficacy Register of New Interventional Procedures – Surgical (ASERNIP-S).
TECHNOLOGY BRIEF

Register ID          WP097

Name of technology  Hypoglossal nerve stimulation

Purpose and target group  Patients with sleep apnoea

Stage of development in Australia
- Experimentally
- Established but changed indication or modification of technique
- Investigational
- Should be taken out of use
- Yet to emerge
- Nearly established

Australian Therapeutic Goods Administration approval
- Yes
- No
- Not applicable

International utilisation

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Impact summary

Hypoglossal nerve stimulation technology has been developed with the aim to treat obstructive sleep apnoea (OSA). The technology utilises an implantable device that electrically stimulates the hypoglossal nerve, leading to the contraction of the genioglossus muscle, the major muscle responsible for tongue protrusion. This prevents airway collapse and the development of upper airway obstruction during sleep. The technology is not currently registered on the Australian Register of Therapeutic Goods (ARTG), but has approval for use in clinical trials. The technology would be made available through hospitals via implantation by surgeons. The most common treatment for OSA, continuous positive airway pressure (CPAP), has been
associated with poor patient compliance. Hypoglossal nerve stimulation technology may provide patients with an alternative, more palatable, treatment option.

**Background**

OSA is characterised by repeated episodes of pharyngeal obstruction during sleep, including airway collapse (apnoea) or narrowing (hypopnoea), resulting in recurrent airflow cessation. Immediate symptoms of OSA include loud snoring, choking and gasping, and disrupted sleep. OSA is also associated with excessive daytime sleepiness, cognitive impairment, and cardiovascular and metabolic morbidities, resulting in a significant increase in mortality. Possible risk factors for OSA include obesity, gender (higher risk for males), craniofacial and upper airway abnormalities, alcohol consumption prior to sleep and night-time nasal congestion. Age is also a risk factor, with a higher prevalence between 40-70 years of age.

Diagnosis and severity of OSA is based on polysomnography (PSG; overnight monitoring of breathing abnormalities), which can detect apnoea or hypopnoea events. One common severity index of sleep disordered breathing is the apnoea-hypopnoea index (AHI), which is the number of events per hour of sleep. Mild OSA is defined as an AHI 5-19.9, moderate OSA 20-29.9, and severe OSA >30. An additional severity indicator used is the oxygen desaturation index (ODI), which is the number of events per hour of sleep where there is a greater than or equal to four per cent decrease in oxygen saturation. The subjective effects of OSA can be determined by patient questionnaires, including the Epworth Sleepiness Scale (ESS), a self-administered questionnaire that measures daytime sleepiness; and the Functional Outcomes of Sleep Questionnaire (FOSQ), a self-administered questionnaire that assesses the impact of excessive sleepiness on daily living activities.

Treatment options for OSA include CPAP, the delivery of air by a mask to maintain a constant pressure along the upper airway, preventing narrowing or collapse; use of oral appliances to stabilise the mandible and/or pull the tongue forward; and surgical options that target the upper airway.

During deep sleep, the muscles of the throat relax, leading to a reduction in airflow. In OSA, complete relaxation of these muscles may cause the tongue to prolapse into the throat, causing the cessation of airflow. Tongue prolapse may be due to diminished neuromuscular activity in the genioglossus muscle. Therefore, the genioglossus muscle is a potential therapeutic target for OSA, as electrical stimulation can cause the protrusion of the tongue, stiffening of the anterior pharyngeal wall and prevention of upper airway blockage. Devices developed to stimulate this muscle directly were associated with improvements in the severity of the disease; however, these stimulation techniques would often arouse the patient, limiting the effectiveness of the treatment.
The genioglossus muscle can alternatively be targeted through the electrical stimulation of its motor nerve, the hypoglossal nerve. The branches of this nerve that innervate the genioglossus are predominantly motor fibres, and as such, stimulation of this nerve has the ability to activate the muscle with minimal sensory feedback.¹

Hypoglossal nerve stimulation systems: the HGNS® System (Apnex Medical, Inc., St. Paul, MN, USA); the aura6000™ system (ImThera Medical, Inc., San Diego, CA, USA) and the Inspire® Upper Airway Stimulation (Inspire Medical Systems, Inc., Maple Grove, MN, USA) consist of an implantable neurostimulator that delivers an electrical current to the hypoglossal nerve by a stimulation lead. Stimulation is synchronised with respiration sensing leads that measure changes in breathing. The neurostimulator is programmable through a computer interface and programmer head, with limited additional patient control (Figure 1). The device is surgically implanted under general anaesthesia, with the stimulating lead placed on the main trunk of the hypoglossal nerve. The neurostimulator is implanted in an infraclavicular subcutaneous pocket, and the respiratory sensing lead(s) are placed between the external and internal intercostal muscle (Figure 2).¹⁴

Clinical trials for the use of hypoglossal nerve stimulation therapy are being undertaken in the USA and Europe. The USA Food & Drug Administration (FDA) has granted investigational devices exception (for use in clinical trials only) for all three systems, with CE Mark approval in Europe granted to the Apnex HGNS® System and the ImThera aura6000™ System. Clinical trials for the Inspire® Upper Airway Stimulation system are underway in Europe.
Clinical need and burden of disease

The characteristic episodes of OSA lead to intermittent hypoxemia and the frequent interruption of sleep, which may result in long-term neurocognitive, metabolic and cardiovascular issues. Left untreated, patients with OSA are at increased risk of sudden death, hypertension, stroke, coronary artery disease, congestive heart failure, type II diabetes, depression, and decreased quality of life.

OSA affects millions of people worldwide, and prevalence is increasing with a greater incidence of obesity and an ageing population. In Australia, OSA is the most common chronic primary sleep disorder, affecting approximately 775,000 people in 2010 (4.7% of the population). Hospital separations for OSA (mainly overnight stays in sleep centres) have increased by 35 per cent between 2004-2005 and 2009-2010. In 2010-2011, private hospitals provided 86 per cent of hospital separations for sleep apnoea. Recent evidence from New Zealand indicates that Māori people have a higher prevalence of OSA than non-Māori, and that this population often presents with more severe forms of the disease. Obesity, one of the risk factors for OSA, is additionally higher in the Māori population than non-Māori.
In 2010, it was estimated that sleep disorders cost the Australian hospital system $96.2 million. Of this amount, 59.6 per cent has been attributed to OSA. The out-of-hospital cost of OSA was estimated to be $96.6 million. The cost to the health system for conditions attributed to OSA (cardiovascular disease, depression and anxiety, motor-vehicle and workplace injuries) was an estimated $408.5 million in 2010.10

**Diffusion of technology in Australia**

While hypoglossal nerve stimulation devices are not registered on the ARTG, clinical trials using the Apnex device have been undertaken at four clinical trial sites in Australia (Austin Health in VIC, St. Charles Gardner Hospital in WA, Repatriation General Hospital in SA and Westmead Hospital in NSW), with participation in a worldwide RCT at Austin Health and Westmead Hospital.1, 14 ImThera Medical, Inc. plans to submit an application to the TGA later in 2012.7

**Comparators**

CPAP is currently the universally-accepted gold standard treatment for OSA. The CPAP machine delivers a positive stream of air pressure that acts as a pneumatic splint to maintain the opening of the airway during sleep.5 The intervention requires patients to wear a nasal mask whilst sleeping. Compliance in the home setting is often poor, with only 40-60 per cent of patients using the treatment long-term or as prescribed.1

A variety of upper airway surgical procedures are options for patients with severe OSA who do not respond to CPAP. One of the more commonly performed procedures in Australia is uvulopalatopharyngoplasty (UPPP), a highly invasive procedure wherein the uvula, a portion of the soft palate, and tonsils (if present) are removed in order to enlarge the airway.5 Medicare Benefits Schedule (MBS) claims for UPPP (item number 41486) have remained constant over the past five years, with approximately 1200 claims per year.15 Long-term follow-up studies have suggested that the initial effect of surgery may lessen over time.16

Due to the limited effectiveness of treatment alternatives, Eastwood et al (2011) suggested that patients may not be seeking the required medical advice.1 Consequently, the number of patients who report for sleep investigations may be an underestimation of the true burden of disease.

**Safety and effectiveness**

Four studies assessing hypoglossal nerve stimulation were included in this brief: two case-series studies that examined safety and preliminary effectiveness;1, 4 and two additional studies that used the same patient pool as one of the case-series,
Eastwood et al (2011)\textsuperscript{1}, with an aim to characterise physiological changes in response to therapy.\textsuperscript{9,17} All studies were manufacturer-sponsored.

Van de Heyning et al (2012)\textsuperscript{3}

**Study description**

This prospective multi-centre case-series study (level IV intervention evidence) aimed to examine the safety and preliminary effectiveness of the Inspire\textsuperscript{®} Upper Airway Stimulation system. The study was conducted in two stages: in the first stage, patients were enrolled based on broad selection criteria (n=22); while additional patients were recruited for the second stage based on the outcomes of stage one (n=9). Patients who met the inclusion criteria (stage one: moderate to severe OSA, failure of or intolerance to CPAP treatment, body mass index (BMI) <35 kg/m\textsuperscript{2} and AHI ≥25 events/h; stage two: as in stage one but BMI ≤32 kg/m\textsuperscript{2} and AHI ≤50/h) underwent surgical implantation of the hypoglossal nerve stimulation system, under general anaesthesia. Nine patients were implanted with the Inspire\textsuperscript{®} Upper Airway Stimulation system in phase two of the study; however, one patient was excluded from the analysis within the period of six months post-implantation due to the inability to activate the tongue with amplitude within the allowable range.

Therapy was activated four weeks post-implant, at which point the device was adjusted to produce optimal results. Therapeutic titration was made at 2- and 4-month post-implant sleep studies if necessary. Study (effectiveness) data were recorded at baseline and six months post-implant. Three participants were excluded from the effectiveness analysis, two had explantation of the device (see Safety section below), and one was lost to follow-up. Responders were predefined as those with an AHI reduction of at least 50 per cent from baseline, and an AHI less than 20 at six months post-implant.

**Safety**

Two (6.4%) serious device-related adverse events were reported during the 6-month post-implant period. One participant experienced pain and swelling immediately post-implant which resolved with antibiotic treatment. The other experienced a delayed device-related post-implant infection and required device explantation. One (3.2%) additional participant had the device removed due to inadequate tongue response. The majority of non-serious adverse events were due to postoperative pain and stiffness (7/31) and sore throat (4/31). All non-serious events resolved without intervention.

**Effectiveness**

The effectiveness of the therapy was assessed during in-laboratory polysomnography (PSG) and the completion of the ESS and FOSQ questionnaires. In
the first stage of the study, there was no change in baseline AHI when all patients were considered. Of the 20 patients who were assessed at six months, six (30%) met the responder definition, with significant reduction in AHI and ODI compared to baseline \( (p<0.001) \); note that 14 patients did not meet the responder definition. It was identified that the responders had significantly lower AHI \( (26.1 \pm 5.0 \text{ for responders compared to } 51.1 \pm 16.8 \text{ for non-responders, } p<0.01) \) and BMI \( (27.8 \pm 1.8 \text{ responders, } 30.7 \pm 2.6 \text{ non-responders, } p<0.05) \) levels at baseline. Statistical analyses determined the predictors of therapy to be a combined criteria of AHI less than 50/h and BMI of less than 32 kg/m² \( (p=0.01) \). Of the 11 participants who met this criteria, six (55%) were responders. Additional participants were recruited for the second stage of the study on the basis of this new criteria \( (n=9) \). Placement of the stimulating electrode changed between the stages: placement in the second stage was only around the medial division of XII hypoglossal nerve branch, which innervates the genioglossus muscle. In the first stage, the electrode was placed prior to the branching of the XII branch into its medial and lateral parts; the latter innervates other muscles of the tongue. One participant was excluded from 6-month assessment due to explantation of the device (see Safety section above). In the eight participants assessed at six months, AHI was reduced significantly from 38.9 at baseline to 10 (-74%; \( p<0.01 \)). A significant improvement in ODI was also observed \( (p<0.01) \). In total, seven of eight (87.5%) participants met the responder definition.

Of the participants who were followed up at six months \( (n=28) \), ESS improved from 11.0 at baseline to 7.6 after six months \( (p<0.01) \), and FOSQ score improved from 89.1 to 100.8 \( (p=0.02) \).

**Eastwood et al (2011)**

**Study description**

The aim of this single-arm, open-label case-series study (level IV intervention evidence) at four clinical sites in Australia was to examine the safety and effectiveness of the Apnex HGNS® System in the treatment of OSA, with long-term follow-up to three years; the publication reports data to six months. Patients \( (n=21) \) who met the inclusion criteria (moderate to severe OSA, failure of or intolerance to CPAP treatment, age 21 to 70 years, BMI ≤40 kg/m² and AHI between 20 and 100/h with ≥15 events/h occurring in non-rapid eye movement sleep with a predominance of hypopnoeas) underwent surgical implantation of a hypoglossal nerve stimulation system. Only 21 of 33 patients assessed for eligibility were enrolled and all patients were overweight or obese (mean BMI 32.7 kg/m², range 26.7-38.7). The surgery took a mean of three hours \( (189 \pm 58 \text{ minutes}) \).

Therapy was initiated 30 days post-implant, with daytime and overnight studies used to determine the effectiveness in reducing OSA severity. Baseline data were
recorded one month post-implant, with sleep studies repeated again at three and six months. The severity of OSA was defined by AHI at baseline (one month) and three and six months post-implant. Participants additionally completed five questionnaires at these time points, including the FOSQ, ESS and others relating to changes in quality of life, measures of sleep quality and levels of depression. It appears that the study used a shortened version of the FOSQ questionnaire (range 0-20 compared to 0-120 for the full version); however, this was not explicitly reported.

Data to 12 months have recently been presented that show efficacy is maintained.

**Safety**

All adverse events were reported regardless of severity or whether the event was device or therapy-related. The primary safety endpoint was the rate of freedom from serious adverse events at implant and at three and six months. Adverse events were considered serious if they resulted in patient death, life-threatening illness or injury, permanent impairment of body structure or function (including medical or surgical intervention to prevent), or in-patient hospitalisation (> 24 h).

There were no deaths in the study, nor were there any unanticipated adverse effects related to the device. Two serious adverse events were observed (9.5%): the device was explanted in one participant due to a procedure-related haematoma and infection while the second required an additional procedure for lead replacement due to a cuff dislodgment (considered both a procedural- and device-related event). A third patient elected for explantation of the device in order to proceed with an alternative surgical treatment.

Numbness and pain at the incision site were the most common procedure-related adverse events, with five (23%) and three (14%) respective reports. Of the therapy-related adverse events, eight patients (38%) had abrasions on the ventral surface of the tongue due to movement over the lower incisors. These were short in duration and treated with plastic guards placed over the mandibular teeth. Resolution occurred in all cases. The rate of freedom from serious adverse events at three months was 90.2 per cent (19/21) and at six months, 85.2 per cent (18/21).

**Effectiveness**

The primary effectiveness endpoints were the mean change from baseline in the AHI and FOSQ score at three and six months post-implant. The most common definition of surgical success includes the postoperative reduction of AHI to less than 20 events/h and a greater than 50% postoperative reduction of AHI. Twelve of 21 participants (57%) met these criteria after six months of therapy. Overall, AHI decreased from 43.1 at baseline to 19.1 (-56%) at three months (p<0.001), and 19.5 (-55%) at six months (p<0.001). Additionally, participants with BMI less than 35 kg/m² were observed to have a lower AHI at six months post-implant, compared to
those with a BMI greater than 35 kg/m². The total FOSQ score changed from 14.4 at baseline to 17.0 at three months \((p<0.001)\), and 16.7 at six months \((p<0.001)\). FOSQ scores that exceed a 2-point change were considered to be clinically meaningful improvements in daily life activities.

The secondary effectiveness endpoints included therapy utilisation, changes from baseline measurement of other PSG-based measures that relate to sleep disordered breathing and sleep architecture, and changes in the questionnaire scores. Compliance with the therapy was high, with use on 89 ± 15 per cent of nights, at an average of 5.8 ± 1.6 hours per night. The ESS score changed from an abnormal sleepiness baseline score of 12.0 to 7.9 and 8.1 at three and six months respectively \((p<0.001)\). These scores are within the normal range on the ESS. Changes in the other sleep-related questionnaire scores reflected some improvement in sleep, but restoration to normal range was not observed for all cases.

Schwartz et al (2012)

**Study description**

The aim of this study (level IV intervention evidence) was to characterise airflow responses to hypoglossal nerve stimulation therapy. Participants who met the eligibility criteria \((AHI \geq 20)\) underwent surgical implantation of the device \((n=30)\), a number of these participants were reported in the Eastwood et al (2011) study but it was unclear how many). The differences between alternating stimulated and unstimulated breaths during sleep were analysed. Therapy was also applied with increasing amplitudes.

**Effectiveness**

Therapy was observed to increase maximal inspiratory airflow relative to unstimulated adjacent breaths at mid- and high-level amplitudes. At the mid-level, inspiration was observed to be flow limited, with flow limitation abolished at the high-level amplitude. The airflow response increased from \(215 \pm 21 \text{ mL/s}\) without stimulation to \(509 \pm 37 \text{ mL/s}\) upon stimulation. The authors concluded that the therapy “produced marked dose-related increases in airflow without arousing patients from sleep. Increases in airflow were of sufficient magnitude to eliminate inspiratory airflow limitation in most patients...suggesting potential efficacy across a broad range of OSA severity.”

Goding et al (2012)

**Study description**

The aim of this study (level IV intervention evidence) was to characterise the changes in the airway spaces of the pharynx during hypoglossal nerve stimulation. Participants who met the inclusion criteria (moderate to severe OSA, failure or
intolerance of CPAP treatment, BMI ≤40 kg/m² and AHI 20-100 events/h) underwent surgical implantation of the device (n=26, 17 participants were in the Eastwood et al (2011) study). Cinefluoroscopy image acquisition started and stopped approximately one second before and one second after stimulation in participants under general anaesthesia. Measurements of the pharyngeal lucency at the inferior and superior borders of the mandibular body were recorded (PLIMB and PLSMB).

**Effectiveness**

Prior to stimulation, PLIMB and PLSMB were not visible in any of the participants. The average width of the PLIMB with stimulation was 9 ± 3 mm, with a width of 13 ± 5 mm for PLSMB. The presence of PLIMB and PLSMB indicate an increase in the amount of total airway available. The authors concluded, “In a majority of subjects examined, hypoglossal nerve stimulation resulted in opening of the retropalatal space. Hypoglossal nerve stimulation produced pharyngeal opening in the retrolingual area and anterior to the palate independent of BMI.”

**Cost impact**

The manufacturers of the hypoglossal nerve stimulation systems have been contacted regarding the costs of the device; with the cost of the aura6000™ system indicated to be US$30,000, with a 15 year estimated lifetime. Other costs related to the therapy include the costs of the surgery, anaesthesia and postoperative and follow-up sleep studies. During the Australian clinical trial, the average surgery time was three hours; however, two manufacturers have indicated an estimated surgery time of approximately 90 minutes; with an overnight hospital stay at the discretion of the physician. Clinical advice suggests that with experience and expertise in implantation of the device, surgical time would be expected to decrease, and most patients would be discharged on the same day, given the completion of surgery by mid-afternoon.

In comparison, the average cost of a CPAP machine, accessories and spare parts in Australia in 2010 was $1591.60. These can be purchased or rented at the patient’s expense; some states may offer subsidies for CPAP treatment, and some private health insurance companies assist with the cost of the machine.

A cost-utility analysis performed by one of the manufacturers has concluded that the therapy is “a highly cost effective intervention for OSA from a health system perspective and ‘dominant’ from a societal perspective”. The incremental cost effectiveness ratios (ICERs) were calculated for both the hypoglossal nerve stimulation therapy and CPAP in comparison to no treatment, and were based on the assumption that the therapy is approximately as effective as CPAP. As the therapy consists of an implantable device, with no tubes, noise or need for a mask, the added convenience of the therapy has been estimated to lead to higher
compliance, resulting in improved cost effectiveness. The cost-utility analysis may be highly optimistic, and premature given the limitations of the evidence base.

Implantation of the device requires no special instrumentation, and can be performed by surgeons familiar with the surgical techniques and neuroanatomy of the upper neck, which are typically ear, nose and throat, maxillofacial and neurosurgeons.

**Ethical, cultural or religious considerations**

Access to surgeons and surgical lists in the public sector may be a limiting factor.

**Other issues**

All studies included in this brief were manufacturer-sponsored.

Training techniques for optimal placement of the electrode around the hypoglossal nerve must be developed for better effectiveness and to avoid nerve injury.

Upcoming manufacturer-sponsored clinical trials:

- ImThera Medical, Inc. has conducted a safety and effectiveness study for the aura6000™ system, which was scheduled for completion at the end of 2011. Safety and effectiveness results at 12 months have been published during the completion of this brief. The study included 14 participants and observed significant reductions in the AHI and ODI indices. Device-related faults that resulted in re-operation were reported, in addition to two cases of transient tongue paresis.

- Inspire Medical Systems, Inc. has a large (n=900) randomised Phase III trial underway with a 12-month follow-up period. The estimated study completion date is March 2014. Details specifying the type of intervention and the nature of the control group were not provided.

- Longer term follow-up of the HGNS® System from the Eastwood et al (2011) study is estimated for completion in May 2013. The safety and effectiveness trial for the same device conducted in the USA is due for completion in 2013. Apnex Medical, Inc. is also recruiting for a Phase III randomised clinical trial undertaken predominantly in Australia and the US (estimated n=132, completion in 2017).

**Summary of findings**

The use of hypoglossal nerve stimulation devices in the treatment of OSA appears to be relatively safe; however, the effectiveness data are based on small, low-quality studies. Study participants who exhibited positive treatment effects were observed to have lower AHI and BMI levels at baseline, and as such, may not be representative
of the population with moderate to severe OSA. Therefore, large randomised controlled trials are required in order to adequately determine the effectiveness, in terms of clinical response rates; and safety, including the rate of explantation and infection. As the included studies were small with relatively short follow-up, clinician advice indicated that the rate of these complications is likely to increase. Consequently, should the therapy be introduced, a record of the implanted devices should be maintained on a central registry. Evidence to date only supports the use of the technology for the treatment of mild to moderate (not severe) OSA, and that at the quoted price, is unlikely to be offered to patients in the public sector. Compared to the non-invasive comparator CPAP, use of the technology may pose great harm at considerable cost for benefit which is not currently proven.

HealthPACT assessment:

Based on the low level, small feasibility studies included in this brief and the use of the device at an experimental level only, HealthPACT recommended that the technology be monitored for 24 months.

Number of studies included

All evidence included for assessment in this Technology Brief has been assessed according to the revised NHMRC levels of evidence. A document summarising these levels may be accessed via the HealthPACT web site.

Total number of studies 4
Total number of level IV studies 4

References


**Search criteria to be used**

Hypoglossal nerve stimulation, obstructive sleep apnoea