https://doi.org/10.5664/jcsm.8946

JCSM | Journal of Clinical Sleep Medicine

EMERGING TECHNOLOGIES

Smart polymer implants as an emerging technology for treating airway collapse in obstructive sleep apnea: a pilot (proof of concept) study

Anders William Sideris, BMed, BMedSci (Hons)[1,2,3]; Gordon Wallace, PhD, AO, FAA, FTSE, FIOP, FRACI[4]; Matthew Eugene Lam, MBBS (Hons), BMedSci (Hons)[1,2,3]; Leon Kitipornchai, MBBS, FRACS (OHNS)[1,2,3]; Richard Lewis, MBBS, FRACS (OHNS)[5]; Andrew Jones, MBBS, FRACP[2,3]; Ali Jeiranikhameneh[4]; Stephen Beirne, PhD[4]; Lachlan Hingley, PhD[6]; Stuart Mackay, MBBS (Hons), BScMed, FRACS (OHNS)[1,2,3,6]

[1]Department of Otolaryngology Head and Neck Surgery, The Wollongong Hospital, Wollongong, New South Wales, Australia; [2]Illawarra ENT Head and Neck Clinic, Wollongong, New South Wales, Australia; [3]Illawarra Shoalhaven Local Health District Wollongong, New South Wales, Australia; [4]ARC Centre of Excellence for Electromaterials Science (ACES), Intelligent Polymer Research Institute, AIIM Facility, Innovation Campus, University of Wollongong, New South Wales, Australia; [5]Department of Otolaryngology Head and Neck Surgery, Royal Perth Hospital, Perth, Western Australia, Australia; [6]School of Medicine, University of Wollongong, Wollongong, New South Wales, Australia

Study Objectives: To assess the use of a novel magnetic polymer implant in reversing airway collapse and identify potential anatomical targets for airway implant surgery in an in vivo porcine model.

Methods: Target sites of airway collapse were genioglossus muscle, hyoid bone, and middle constrictor muscle. Magnetic polymer implants were sutured to these sites, and external magnetic forces, through magnets with pull forces rated at 102 kg and 294 kg, were applied at the skin. The resultant airway movement was assessed via nasendoscopy. Pharyngeal plexus branches to the middle constrictor muscle were stimulated at 0.5 mA, 1.0 mA, and 2.0 mA and airway movement assessed via nasendoscopy.

Results: At the genioglossus muscles, large magnetic forces were required to produce airway movement. At the hyoid bone, anterior movement of the airway was noted when using a 294 kg rated magnet. At the middle constrictor muscle, an anterolateral (or rotatory) pattern of airway movement was noted when using the same magnet. Stimulation of pharyngeal plexus branches to the middle constrictor revealed contraction and increasing rigidity of the lateral walls of the airway as stimulation amplitude increased. The resultant effect was prevention of collapse as opposed to typical airway dilation, a previously unidentified pattern of airway movement.

Conclusions: Surgically implanted smart polymers are an emerging technology showing promise in the treatment of airway collapse in obstructive sleep apnea. Future research should investigate their biomechanical role as an adjunct to treatment of airway collapse through nerve stimulation.

Keywords: smart polymers, sleep surgery, obstructive sleep apnea, airway collapse, nerve stimulation

Citation: Sideris AW, Wallace G, Lam ME, et al. Smart polymer implants as an emerging technology for treating airway collapse in obstructive sleep apnea: a pilot (proof of concept) study. *J Clin Sleep Med.* 2021;17(2):315–324.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Implantable 3-dimensional printed "smart" polymers are an emerging technology with potential applications in treating collapse in adult obstructive sleep apnea through mechanical airway manipulation. There is a paucity of devices that are commercially available or in research and development stage. Limited studies have investigated the use of implantable smart polymers in reversing the collapsibility of the pharyngeal airway by creating counter forces during sleep.

Study Impact: This paper describes an application of implantable magnetic polymer technology in an in vivo porcine model.

INTRODUCTION

Implantable 3-dimensional (3D) printed "smart" polymers are an emerging technology with potential applications in treating collapse in adult obstructive sleep apnea (OSA) through mechanical airway manipulation. There is a paucity of devices that are commercially available or in research and development stage. Continuous positive airway pressure,[1] positional and mandibular devices,[2,3] lifestyle modification[4] and weight loss,[5] surgical airway reconstruction,[6–8] and nerve stimulation[9,10] are not without limitations. In the last decade, commentaries have recognized few innovations in the field.

Previous work at our institution has allowed us to model the forces required to prevent airway collapse, which helps inform the development of new implant-based technologies to treat collapse in OSA.[11] Limited studies have investigated the use of magnetic implants in reversing the collapsibility of the pharyngeal airway by creating counter forces during sleep. One study has modeled such an invention in a human cadaveric model, indicating magnetic forces can produce anterior movement of the hyoid bone and increase airway caliber.12 To date this has not been tested in an in vivo model of drug-induced (or natural) sleep.

Various animal models have been utilized in modeling the upper airway[13–15] and in research assessing the forces required to collapse the airway.[16–19] We conducted an experimental in vivo porcine study aimed at assessing the use of a novel implantable magnetic polymer in reversing airway collapse, and identifying potential anatomical targets for airway implant surgery in a drug-induced sleep endoscopy model.

METHODS

Design of model: surgical approach and nasendoscopic assessment of airway manipulation

An experimental pilot study was performed using a live anesthetized porcine model. The surgical model included 4 key components (**Figure 1**):

1) Dissection and identification of target sites of airway collapse and airway landmarks (genioglossus muscle, hyoid bone, and middle constrictor muscle).
2) Stimulation of hypoglossal nerve and observation of airway manipulation pattern via nasendoscopy.
3) Implantation of magnetic polymer implant at target sites, application of magnetic force, and observation of airway manipulation via nasendoscopy.
4) Identification and stimulation of pharyngeal plexus and observation of airway manipulation via nasendoscopy.

Prior to data capture and implantation of devices, all raters were oriented with the normal upper airway porcine anatomy and sleep-induced collapse pattern during anesthesia via nasendoscopy. Surface markings were made at the mandibular symphysis and hyoid bone (approximately 8–11 cm caudal to the symphysis in a pig). A vertical incision was carried through subcutaneous fat and the platysma was identified. Both mylohyoid muscles were exposed, and the midline raphe was then opened to expose the vertical fibers of the geniohyoid muscles. These were separated in the midline to reveal the right and left genioglossi muscles (**Figure 1A**). The incision was continued inferiorly at the level of the geniohyoid to expose the hyoid bone, which was then skeletonized (**Figure 2A**).

Bilateral hypoglossal nerves were exposed as they entered each genioglossus and confirmed functional with nerve stimulation at 0.5 mA, 1.0 mA, and 2.0 mA (Vari-Stim® III, Medtronic) by observation of tongue movement. The resultant increase in anteroposterior airway dimension at retrolingual level was observed on nasendoscopy. The original incision was extended laterally to enable dissection around the carotid sheath in the lateral neck. Next, exposure of the pharyngeal nerve plexus entering the constrictor muscles of the pharynx (including the middle constrictor muscle) was performed. The upper and lower subdivisions of the plexus to the middle constrictor muscles were confirmed with stimulation by observing contraction of these muscle fibers.

A biocompatible ferrous magnetic polymer was manufactured based off previous research.[11] Polycaprolactone/iron composite with 70 % wt iron content was prepared by dissolving polycaprolactone in chloroform at 40°C for 1 hour. Iron powder (mean particle size 1–6 μm, Sigma Aldrich, St. Louis, MO) was added to the solution under agitation at room temperature. The composite was then cast into a Petri dish to evaporate the solvent at room temperature overnight. The resultant polycaprolactone/iron film composite was cut into the small pieces and was melt casted into a cylindrical mold to make the implant disks. The resultant disks were circular, 20 mm in diameter and 3 mm in depth. The disks had drill holes inserted to allow suturing.

Magnetic polymers were implanted upon the genioglossus muscle (3.5 cm depth from skin) (**Figure 1B**) and at the hyoid bone (1.6 cm depth from skin) (**Figure 2B**) and on the middle constrictor muscle. At each site, implants were secured with absorbable sutures and tested against an external 102 kg or 294 kg magnet. Force was adjusted by varying the distance between the magnet and the magnetic polymer implant (**Figure 3**). The effect of the magnetic force was observed directly using nasendoscopy. The pattern and degree of movement of the internal airway was then graded by 4 subject matter experts (3 consultant otolaryngologists with subspecialty training in sleep surgery and 1 sleep physician). No widely accepted grading system for degree of airway movement exists, thus extent of airway movement was graded as small, moderate, or large. Direction of airway movement was described in the anteroposterior and mediolateral planes. During data capture, magnet manipulation at each force was repeated 3 times and each component of stimulation repeated 3 times.

Following observation of a previously unidentified pattern of airway movement on manipulation of the middle constrictor muscle, stimulation was applied independently to upper and lower branches of the pharyngeal plexus entering these middle constrictor muscle fibers. Stimulation was performed after noting the novel anterolateral rotatory movement of the airway with magnetic forces applied to the middle constrictor. Observing this effect produced the hypothesis that stimulation might produce a lateral airway wall effect that may be of clinical relevance in counteracting known lateral collapse patterns in OSA.

RESULTS

Target sites of airway collapse were successfully identified and confirmed with nerve stimulation. Pattern of airway manipulation was confirmed on magnetic manipulation or neural stimulation (**Table 1**).

Airway movement in response to magnetic forces applied to magnetic polymer implant

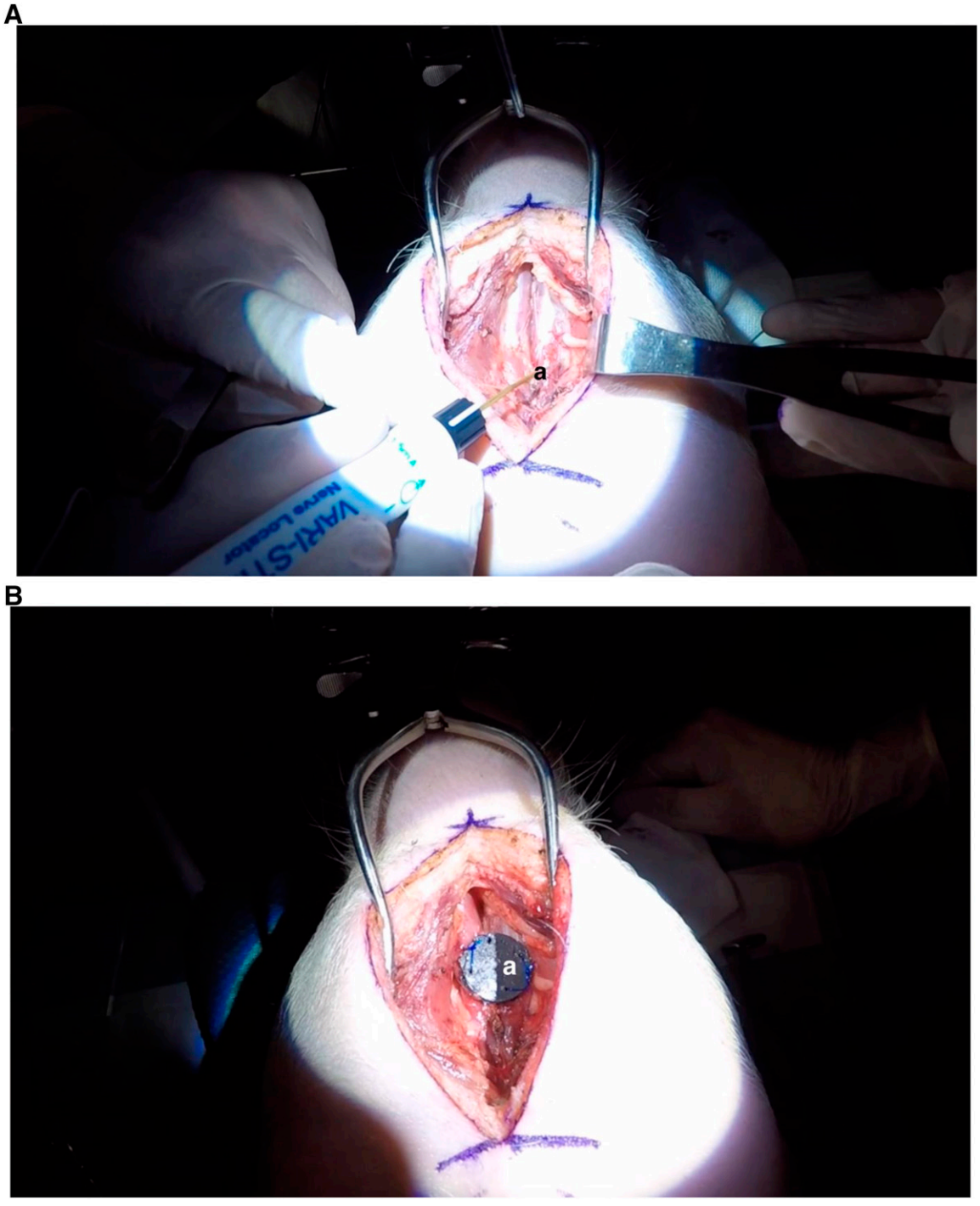
Magnetic polymer implanted to genioglossus muscle

Magnetic forces applied drawing the genioglossus muscle to skin revealed very minimal airway manipulation as noted on nasendoscopic examination of the airway lumen. When applying the 102 kg rated magnet, contraction of the airway was noted. With the 294 kg rated magnet, a small increase in anteroposterior airway caliber at the oropharyngeal level was produced by anterior movement of the oropharyngeal tongue (**Figure 4**).

Magnetic polymer implanted to hyoid bone

Magnetic forces applied drawing the hyoid bone to skin produced anterior movement of the lower oropharynx, vallecula, and lingual surface of the epiglottis, increasing anteroposterior diameter. With the 102 kg rated magnet, this movement was small. An increase in anterior movement of

Figure 1—Exposure, stimulation and suturing implant to genioglossi muscles.



(**A**) Exposure and stimulation of genioglossi muscles. a = left genioglossus muscle. (**B**) Suturing of implant to body of genioglossi muscles. a = site of magnetic polymer implant sutured to genioglossi muscles.

airway was observed when the 294 kg rated magnet was applied (Figure 5).

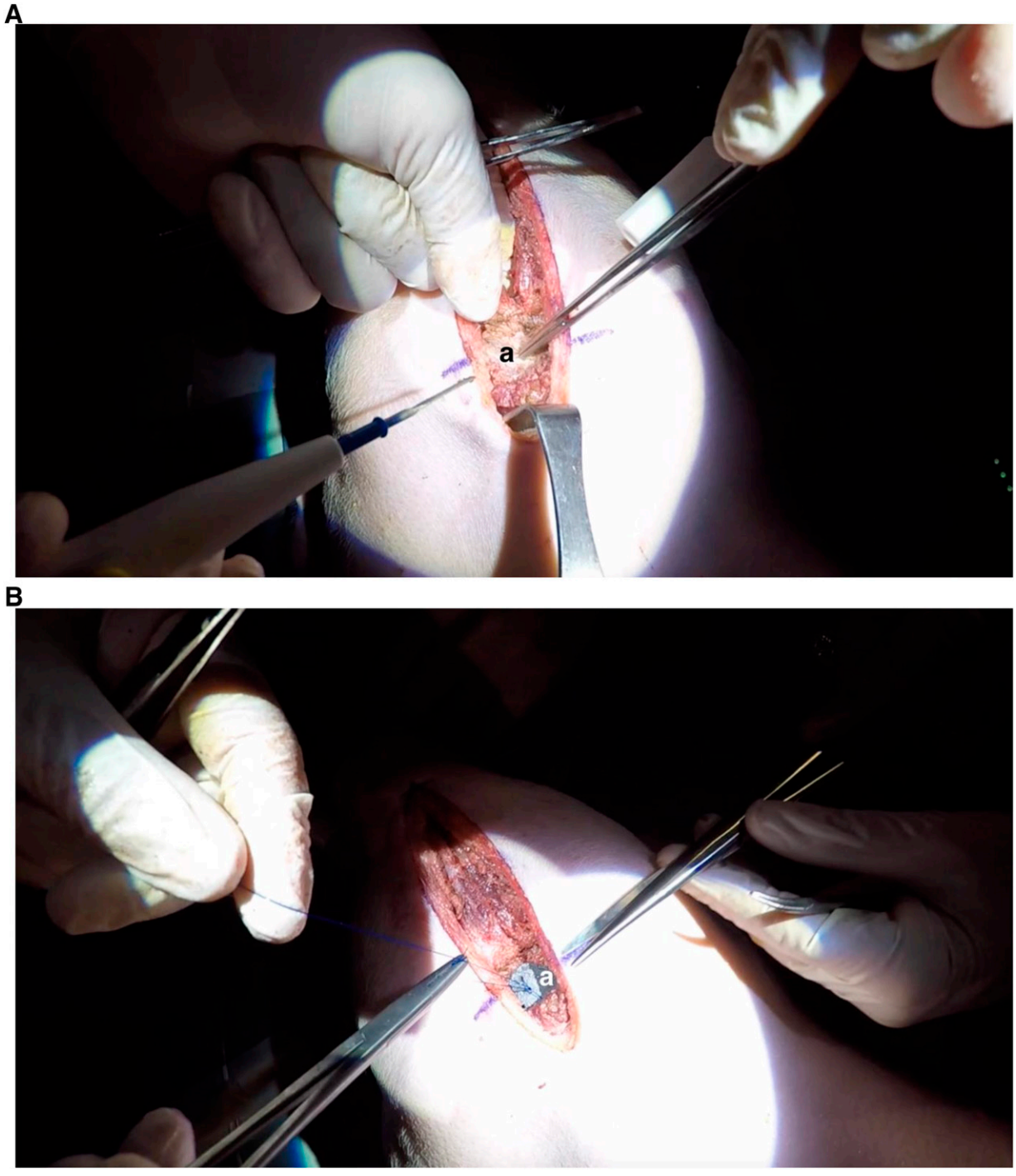
Magnetic polymer implanted to middle constrictor muscle

Magnetic forces applied to the middle constrictor produced small movements of the airway. Using the 102 kg rated magnet, minimal anterolateral (rotatory) movement of airway lumen was noted resulting in a small increase in anteroposterior diameter at the level of the middle constrictor muscles. This effect increased when exposed to the larger 294 kg rated magnet (Figure 6).

Airway movement in response to pharyngeal plexus stimulation

Stimulation of upper and lower pharyngeal plexus branches to the middle constrictor muscle produced contraction of the middle constrictor muscle at the respective levels (Figure 7).

Figure 2—Exposure, skeletonizing, and suturing implant to hyoid bone.



(**A**) Exposure and skeletonizing of hyoid bone (a). (**B**) Suturing of implant (a) to body of hyoid bone.

Concurrent nasendoscopy revealed contraction and increased rigidity of the lateral walls of the airway. The resultant effect was reduction of the anteroposterior collapse noted without stimulation. Sequential increases in stimulation amplitude (0.5 mA, 1 mA, 2 mA) revealed increasing lateral wall rigidity in a similar pattern. There was no apparent difference in airway contraction pattern between upper and lower branches of the pharyngeal plexus to the middle constrictor musculature.

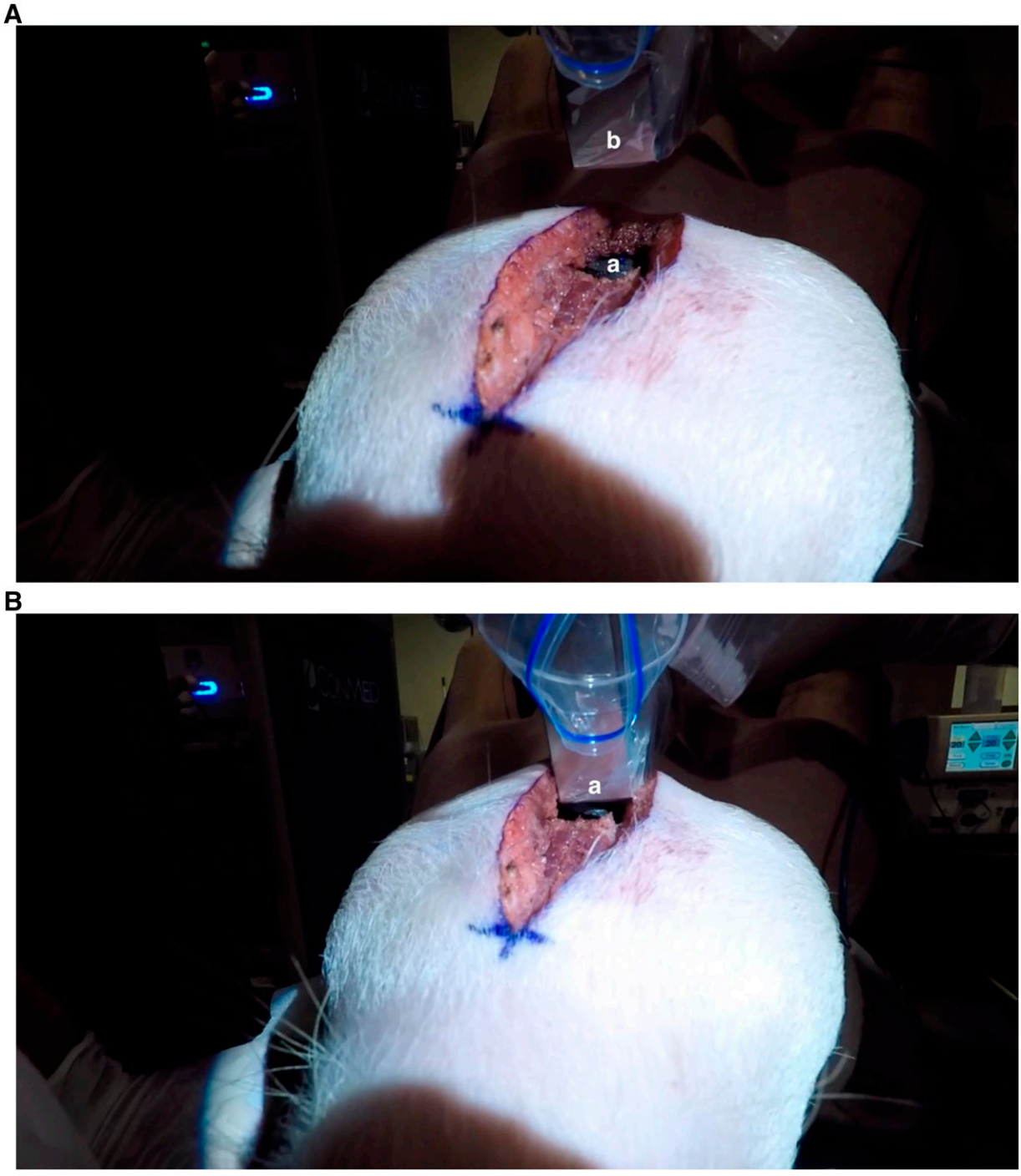
Reproducibility of airway movement assessment

There was 100% agreement between all raters with regard to resultant airway movements as recorded in each section above.

DISCUSSION

The implantable magnetic polymer produced endoscopically apparent airway movement when stimulated by an external

Figure 3—Application and movement of magnet.



(**A**) Magnet (b) applied to magnetic implant (a) on hyoid bone. (**B**) Magnet (a) moving anteriorly, bringing the airway forward toward the skin.

magnetic force. However, the force required to produce these movements was large and may be technically complex in human OSA applications.

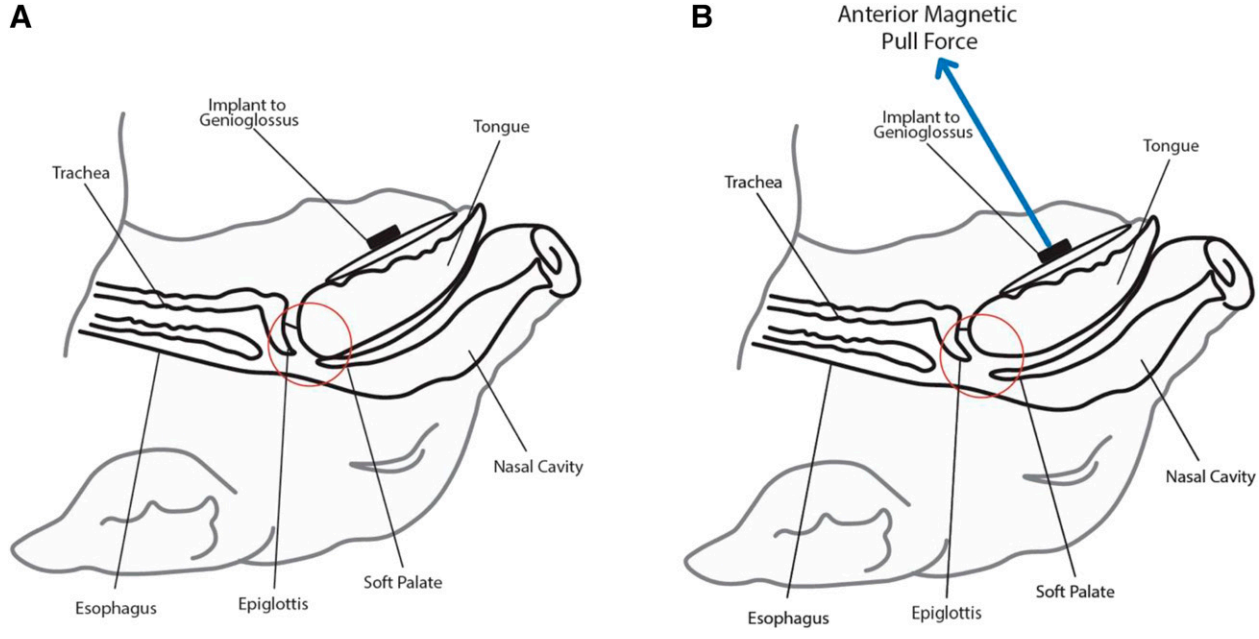
Magnetic manipulation of the middle constrictor muscles revealed a previously unidentified pattern of anterolateral airway movement. Stimulation of the pharyngeal plexus branches caused tonic contraction of the lateral pharyngeal wall and an apparent static rigidity of the lateral airway walls, preventing collapse.

The constrictor muscles are conventionally understood to contract and produce propulsion of food boluses during swallowing. The mechanism of this is not completely understood, although a previous study in cats has demonstrated an increase in velopharyngeal and oropharyngeal cross-sectional area in the lateral direction with glossopharyngeal nerve stimulation.[20] The same study demonstrated a decrease in cross-sectional area with stimulation of the pharyngeal branch of the vagus nerve.[20]

Table 1—Resultant airway movements on nasendosopic examination from magnetic forces (1,2,3) or stimulation (4).

Magnetic Force or Stimulation	Resultant Airway Movement on Nasendoscopic Examination
1. Genioglossus to skin	102 kg: Contraction of airway.
	294 kg: Small anterior movement of anterior wall increasing anteroposterior diameter at retrolingual level.
2. Hyoid to skin	102 kg: Small anterior movement of lower oropharynx, vallecular, and lingual surface of epiglottis, increasing anteroposterior diameter at this level.
	294 kg: Moderate anterior movement of lower oropharynx, vallecular, and lingual surface of epiglottis increasing anteroposterior diameter at this level.
3. Middle constrictor anterior movement	102 kg: Small anterior movement of anterolateral wall of airway increasing anteroposterior diameter at level of middle constrictor muscle.
	294 kg: Moderate anterolateral rotatory movement increasing anteroposterior diameter at level of middle constrictor muscle.
4. Stimulation pharyngeal plexus branches to middle constrictor	Sequential increase in lateral wall tone and rigidity at 0.5 mA, 1.0 mA, and 2.0 mA with anteroposterior collapse of airway noted when stimulation removed.

Figure 4—Illustration of anterior magnetic pull force, genioglossus muscle.



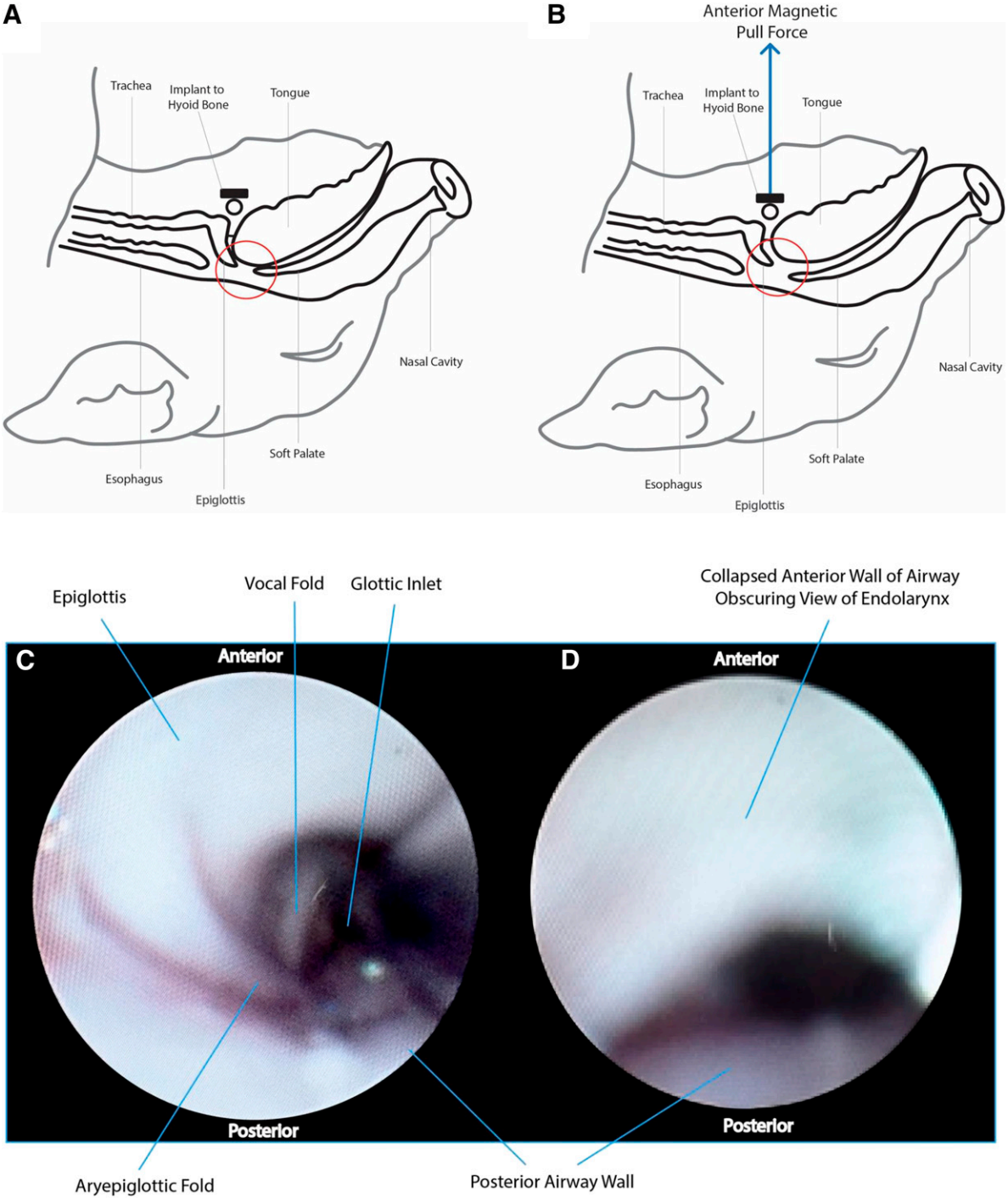
(**A**) Implant sutured to genioglossus muscle with no anterior magnetic pull force, red circle illustrating airway obstruction at the oropharynx. (**B**) Magnetic anterior pull force produced anterior movement of anterior wall, increasing anteroposterior diameter at retrolingual level.

This finding could be applied in management of certain airway phenotypes predisposed to collapse planes around the constrictor musculature by preventing occlusion of the caudal oropharynx and hypopharynx via stiffened pharyngeal musculature and improved underlying tone. It highlights an area of potential further research.

Implantable magnetic polymer airway augmentation is an experimental treatment approach that showed some effect in this study. Further research may reveal it or other smart biopolymer implants as an effective adjunct to hypoglossal nerve stimulation or other therapies. Clarity is needed to define appropriateness and degree of magnetic stimulation input to optimize its effect in the respiratory cycle. Inputs from stimulation devices or pressure (and even airflow) feedback from the walls of the collapsing airway, possibly measured by smart biopolymer implants, may be necessary.

The utility of implantable 3D-printed biopolymers in preventing airway collapse in OSA lies in their capacity to expand or stiffen airway components in response to various stimuli. In addition to augmentation by magnetic forces, the material properties of polymers can be modified by temperature,[21] pH stimulation,[22] and electroconductive means,[23,24] to achieve the desired mechanical effect on the airway. A recently published study described an in vitro simulation model of airway collapse for experimental testing of smart polymers,

Figure 5—Illustration of anterior magnetic pull force, hyoid bone, with endoscopic views.

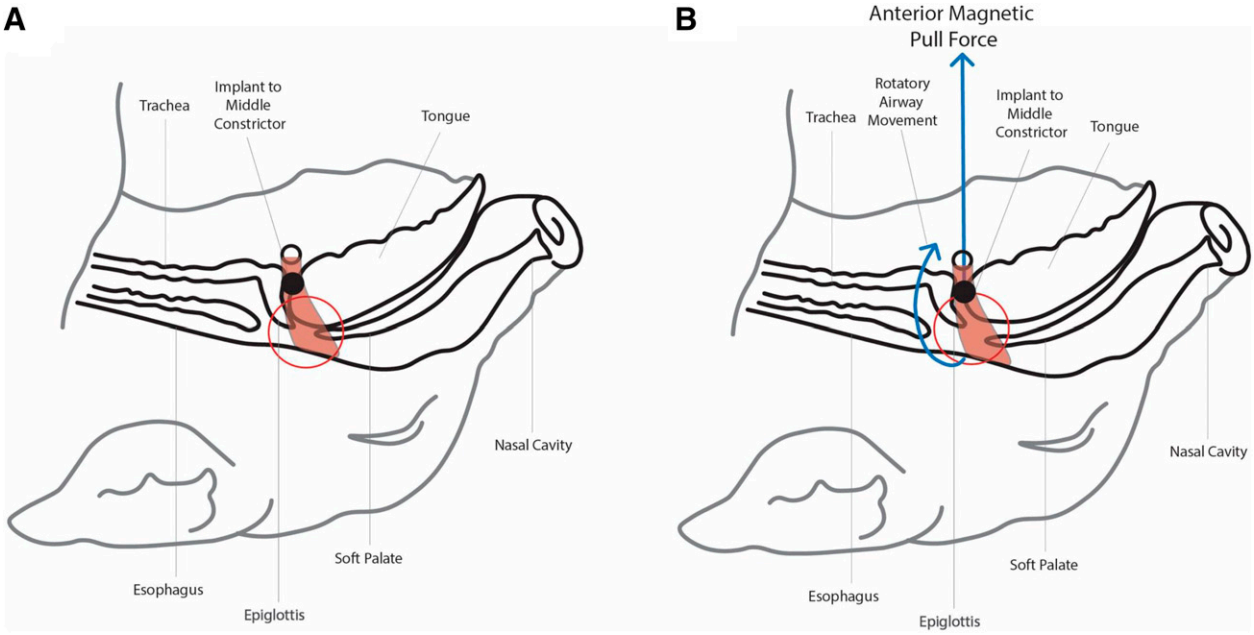


(**A**) Implant sutured to hyoid bone with no anterior magnetic pull force, red circle illustrating airway obstruction at the oropharynx. (**B**) Magnetic anterior pull force producing anterior movement of lower oropharynx, vallecular and lingual surface of epiglottis, increasing anteroposterior diameter at this level. (**C**) Endoscopic view of porcine larynx during application of anterior magnetic force at level of hyoid bone (increased patency of airway). (**D**) Obstructed endoscopic view of porcine larynx without application of anterior magnetic force at level of hyoid bone (collapse of airway).

providing proof of concept of 3D-printed scaffolds preventing airway collapse[11]

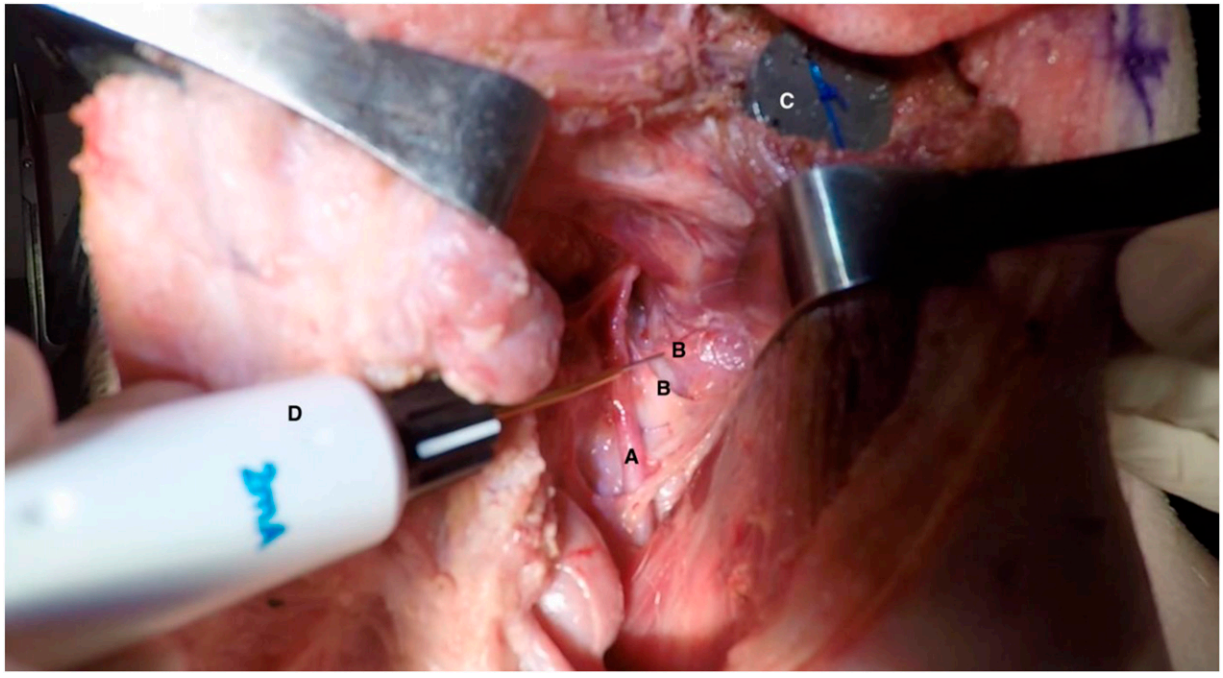
Innovative designs might incorporate multiple polymer sheets along the length of the pharyngeal wall to prevent airway collapse, with such sheets acting in a synergistic manner on target muscle groups and/or augmenting the tensile properties of specific tissue layers. Furthermore, an enhanced understanding of the role of the pharyngeal constrictors in OSA and research in ex vivo and in vivo animal models addressing this unknown may shed light on the

Figure 6—Illustration of anterior magnetic pull force, middle constrictor muscle.



(A) Implant sutured to middle constrictor muscle with no anterior magnetic pull force, red circle illustrating airway obstruction at the oropharynx. **(B)** Magnetic anterior pull force producing anterolateral rotatory movement of oropharyngeal wall, increasing anteroposterior diameter at this level.

Figure 7—Exposure and stimulation of branch of pharyngeal plexus to middle constrictor muscle.



(**A**) Internal carotid artery. (**B**) Branches of pharyngeal plexus to middle constrictor muscle. (**C**) Magnetic polymer implant. (**D**) 2 mA nerve stimulator.

role of implantable devices targeted at multiple site of collapse (such as the pharyngeal constrictors and genioglossi) to produce synergism.

The study has important limitations. Firstly, this pilot study was performed in one animal subject with the intention of informing further animal (and ultimately, human) studies. Secondly, the generalizability of these results to natural sleep is limited by drug-induced sleep conditions. Use of magnetic augmentation techniques should be assessed during natural sleep to further establish effect, prior to human translation. However, this poses its own challenges with regard to method of data capture and feasibility in an animal model.

Thirdly, the airway movement grading system used is subjective. Currently there is no available validated objective scoring system for assessing change in airway collapse nor has an accurate, reliable, objective method for intraoperative measurement of airway dimension and diameter been established. Accepted standard assessment of airway anatomy and configuration of collapse remains interpretation by field experts, as in our study.[25] Level one evidence supports the robustness of this method, with up to 89% interobserver agreement noted.[26]

Fourthly, the clinical significance of anatomical patterns of airway collapse and strategies to reverse them is affected by other physiological elements that could not be tested in this model. These include the effect of such movements on respiratory outcomes (eg, ventilation) and whether the noted movements result in altered airway lumen resistance pressures. It stands to reason from first principles that increase in anteroposterior diameter should decrease ventilation pressure requirements, and stiffening of lateral airway walls should optimize ventilation by preventing airway wall collapse. However, further data are required to confirm this concept.

Finally, a method for measuring pressure changes (in the airway wall or lumen) has not yet been established in an anesthetized animal model. Previous work at our institution has established a simulated model of upper airway dynamic collapse but has not yet been translated to an in vivo setting.[11] Changes in ventilation pressure and volume requirements with airway manipulation are a possible avenue to attain these data and should be studied in future experiments. Our evaluation was intentionally designed with consistent ventilation conditions to allow valid comparisons between forces and anatomical positions of manipulation.

CONCLUSIONS

Surgically implanted smart polymers are an emerging technology for treatment of airway collapse in OSA. Future research should investigate their biomechanical role as an adjunct to treatment of airway collapse through nerve stimulation.

ABBREVIATIONS

3D, 3-dimensional
OSA, obstructive sleep apnea

REFERENCES

1. Epstein LJ, Kristo D, Strollo PJ, Jr., et al. Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009;5(3):263–276.
2. Ngiam J, Balasubramaniam R, Darendeliler MA, Cheng AT, Waters K, Sullivan CE. Clinical guidelines for oral appliance therapy in the treatment of snoring and obstructive sleep apnoea. *Aust Dent J.* 2013;58(4):408–419.
3. Permut I, Diaz-Abad M, Chatila W, Crocetti J, Gaughan JP, D'Alonzo GE, Krachman SL. Comparison of positional therapy to CPAP in patients with positional obstructive sleep apnea. *J Clin Sleep Med.* 2010;6(3):238–243.
4. Thomasouli MA, Brady EM, Davies MJ, Hall AP, Khunti K, Morris DH, Gray LJ. The impact of diet and lifestyle management strategies for obstructive sleep apnoea in adults: a systematic review and meta-analysis of randomised controlled trials. *Sleep Breath.* 2013;17(3):925–935.
5. Greenburg DL, Lettieri CJ, Eliasson AH. Effects of surgical weight loss on measures of obstructive sleep apnea: a meta-analysis. *Am J Med.* 2009;122(6):535–542.
6. MacKay SG, Carney AS, Woods C, et al. Modified uvulopalatopharyngoplasty and coblation channeling of the tongue for obstructive sleep apnea: a multi-centre Australian trial. *J Clin Sleep Med.* 2013;9(2):117–124.
7. MacKay S, Carney AS, Catcheside PG, et al. Effect of multilevel upper airway surgery vs medical management on the apnea-hypopnea index and patient-reported daytime sleepiness among patients with moderate or severe obstructive sleep apnea: the SAMS Randomized Clinical Trial. *JAMA*. 2020;324(12):1168–1179.
8. MacKay SG, Chan L. Surgical approaches to obstructive sleep apnea. *Sleep Med Clin.* 2016;11(3):331–341.
9. Eastwood PR, Barnes M, MacKay SG, et al. Bilateral hypoglossal nerve stimulation for treatment of adult obstructive sleep apnoea. *Eur Respir J.* 2020;55(1):1901320.
10. Eastwood PR, Barnes M, Walsh JH, et al. Treating obstructive sleep apnea with hypoglossal nerve stimulation. *Sleep*. 2011;34(11):1479–1486.
11. Hingley L, Jeiranikhameneh A, Beirne S, et al. Modeling the upper airway: A precursor to personalized surgical interventions for the treatment of sleep apnea. *J Biomed Mater Res A*. 2020;108(6):1419–1425.
12. Rosenbluth KH, Kwiat DA, Harrison MR, Kezirian EJ. Hyoid bone advancement for improving airway patency: cadaver study of a magnet-based system. *Otolaryngol Head Neck Surg.* 2012;146(3):491–496.
13. Brennick MJ, Pack AI, Ko K, Kim E, Pickup S, Maislin G, Schwab RJ. Altered upper airway and soft tissue structures in the New Zealand Obese mouse. *Am J Respir Crit Care Med.* 2009;179(2):158–169.
14. Forbes RB, Murray DJ, Albanese MA. Evaluation of an animal model for teaching fibreoptic tracheal intubation. *Can J Anaesth.* 1989;36(2):141–144.
15. Ram B, Oluwole M, Blair RL, Mountain R, Dunkley P, White PS. Surgical simulation: an animal tissue model for training in therapeutic and diagnostic bronchoscopy. *J Laryngol Otol.* 1999;113(2):149–151.
16. Kairaitis K, Verma M, Amatoury J, Wheatley JR, White DP, Amis TC. A threshold lung volume for optimal mechanical effects on upper airway airflow dynamics: studies in an anesthetized rabbit model. *J Appl Physiol (1985)*. 2012;112(7):1197–1205.
17. Kairaitis K, Stavrinou R, Parikh R, Wheatley JR, Amis TC. Mandibular advancement decreases pressures in the tissues surrounding the upper airway in rabbits. *J Appl Physiol (1985)*. 2006;100(1):349–356.
18. Kairaitis K, Howitt L, Wheatley JR, Amis TC. Mass loading of the upper airway extraluminal tissue space in rabbits: effects on tissue pressure and pharyngeal airway lumen geometry. *J Appl Physiol (1985)*. 2009;106(3):887–892.
19. Goding GS, Jr., Eisele DW, Testerman R, Smith PL, Roertgen K, Schwartz AR. Relief of upper airway obstruction with hypoglossal nerve stimulation in the canine. *Laryngoscope*. 1998;108(2):162–169.
20. Kuna ST. Effects of pharyngeal muscle activation on airway size and configuration. *Am J Respir Crit Care Med.* 2001;164(7):1236–1241.
21. Pérez-Madrigal MM, O'Reilly RK. Thermally switching on/off the hardening of soaked nanocomposite materials. *ACS Cent Sci.* 2017;3(8):817–819.
22. Bazban-Shotorbani S, Hasani-Sadrabadi MM, Karkhaneh A, Serpooshan V, Jacob KI, Moshaverinia A, Mahmoudi M. Revisiting structure-property relationship of pH-responsive polymers for drug delivery applications. *J Control Release*. 2017;253:46–63.
23. Fishman JM, Wiles K, Lowdell MW, De Coppi P, Elliott MJ, Atala A, Birchall MA. Airway tissue engineering: an update. *Expert Opin Biol Ther.* 2014;14(10):1477–1491.
24. Chen H, Müller MB, Gilmore KJ, Wallace GG, Li D. Mechanically strong, electrically conductive, and biocompatible graphene paper. *Adv Mater.* 2008;20(18):3557–3561.

25. Eggerstedt M, Urban MJ, Chi E, Ritz EM, Losavio P. The anesthesia airway evaluation: Correlation with sleep endoscopy findings. *Am J Otolaryngol.* 2020; 41(2):102362.
26. Carrasco-Llatas M, Zerpa-Zerpa V, Dalmau-Galofre J. Reliability of drug-induced sedation endoscopy: interobserver agreement. *Sleep Breath.* 2017; 21(1):173–179.

ACKNOWLEDGMENTS

The authors thank the Garnett-Passe and Rodney Williams Memorial Foundation, Melbourne, Victoria, Australia; the Large Animal Research and Imaging Facility, Preclinical Imaging and Research Facilities, South Australian Health and Medical Research Institute, Gillies Plains, South Australia, Australia; Australian National Fabrication Facilities (ANFF) Materials Node; Dr. Sepidar Sayyar for access to facilities and staff in preparation of the magnetic polymer samples; Hatrik House (hello@hatrikhouse.com), Sydney, Australia; and Mrs. Sophia Athas for graphic design service in creating illustrations and figures.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication August 5, 2020
Submitted in final revised form October 15, 2020
Accepted for publication October 16, 2020
Address correspondence to: Anders Sideris, Department of Otolaryngology Head and Neck Surgery, The Wollongong Hospital, Loftus St., Wollongong, NSW, Australia; Email: anders.sideris@gmail.com

DISCLOSURE STATEMENT

All authors have seen and approved this manuscript. Work for this study was performed at Large Animal Research and Imaging Facility, South Australian Health and Medical Research Institute, Gillies Plains, South Australia. This study was funded by Garnett-Passe Conjoint Grant 2016-2018, Garnett-Passe and Rodney Williams Memorial Foundation. The authors report no conflicts of interest.

EDITOR'S NOTE

The Emerging Technologies section focuses on new tools and techniques of potential utility in the diagnosis and management of any and all sleep disorders. The technologies may not yet be marketed, and indeed may only exist in prototype form. Some preliminary evidence of efficacy must be available, which can consist of small pilot studies or even data from animal studies, but definitive evidence of efficacy will not be required, and the submissions will be reviewed according to this standard. The intent is to alert readers of *Journal of Clinical Sleep Medicine* of promising technology that is in early stages of development. With this information, the reader may wish to (1) contact the author(s) in order to offer assistance in more definitive studies of the technology; (2) use the ideas underlying the technology to develop novel approaches of their own (with due respect for any patent issues); and (3) focus on subsequent publications involving the technology in order to determine when and if it is suitable for application to their own clinical practice. The *Journal of Clinical Sleep Medicine* and the American Academy of Sleep Medicine expressly do not endorse or represent that any of the technology described in the Emerging Technologies section has proven efficacy or effectiveness in the treatment of human disease, nor that any required regulatory approval has been obtained.