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Research Article

Design and optimisation of soft robotic actuators for augmented lung-ventilation

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ABSTRACT

Pulmonary rehabilitation through invasive ventilation involves the insertion of an endotracheal tube into the trachea of a sedated patient to control breathing via a ventilating machine. Invasive ventilation offers benefits such as greater control over oxygen supply, higher efficiency in supporting patient respiration, and the ability to manage airway secretions. However, this method also poses treatment challenges like ventilator-induced pneumonia, airway injury, long recovery times, and ventilator dependence. Here, we explore an alternative invasive ventilation technique using soft robotic actuators to mimic the biological function of the diaphragm for augmenting and assisting ventilation. We investigated two actuator geometries, each at two locations superior to the diaphragm. These actuators were tested on a bespoke ex vivo testbed that accurately simulated key diaphragmatic characteristics throughout the respiratory cycle. From this, we have been able to drive intrathoracic pressures greater than the 5 cmH_2O required for ventilation in a human male. Additionally, by optimising the placement and geometry of these soft robotic actuators we have been able to generate maximum intrathoracic pressures of (6.81 ± 0.39) cmH_2O.

1. Introduction

The thoracic diaphragm, a crucial muscle responsible for breathing, plays a pivotal role in the respiratory function of the human body. When functioning properly, it contracts and flattens, creating negative pressure within the thoracic cavity, allowing for inhalation. However, lung functionality can be compromised due to physiological conditions such as lung inflammation from infections like pneumonia, fluid buildup due to acute respiratory distress syndrome, aspiration of foreign objects, asthma attacks, traumatic lung injuries, airflow obstruction due to chronic obstructive pulmonary disease, neuromuscular disorders, and genetic conditions like cystic fibrosis. Resultantly, patients suffer from airway obstruction, weakened respiratory muscles, chest wall abnormalities, lung abnormalities, and respiratory centre dysfunction [1]. In instances such as diaphragm palsy [2], hyperinflation [3], and emphysema lung disease [4], the diaphragm may lose its ability to contract effectively [5]. Some non-physiological conditions can cause similar effects like environmental conditions (high altitude, air pollution, extreme temperature), external constraints (tight clothing or physical restraining of the chest region), and the use of mechanically driven positive pressure ventilation [6].

In severe stages of the above conditions, traditional treatment methods involve surgical interventions through procedures like diaphragm plication for diaphragm paralysis [7], lung volume reduction surgery for severe emphysema [8,9], tracheostomy for upper airway obstruction [10], lung transplantation [11], and bullectomy for removing large air sacs [12]. The general steps involved in all of the above procedures include surgical access of the trachea, post sedation use of mechanical ventilation, and close monitoring of the patients' vital signs. Though invasive ventilation is a lifesaver, it is a temporary solution until other treatment methods become effective.

Depending upon the severity of the patient, ventilators can be used for longer periods which can be uncomfortable for patients due to coughing, gagging, and injury induced by the ventilating tube. Additional limitations involve an inability to talk or eat, and the forceful pushing of air into the lungs due to positive pressure ventilation. Invasive ventilation also carries inherent risks and complications like ventilator-induced infections [13], lung collapse or pneumothorax [14], lung damage, and side effects of medications which may result in an inability to stop ventilator use [15]. Hence, there is a need to mitigate the negative effects of positive pressure ventilation while parallelly preserving the natural breathing and autonomy of the patient.

The presented work offers an alternative solution with the use of soft robotic actuation of the diaphragm. These actuators can augment diaphragmatic contractions, aiding in ventilation and

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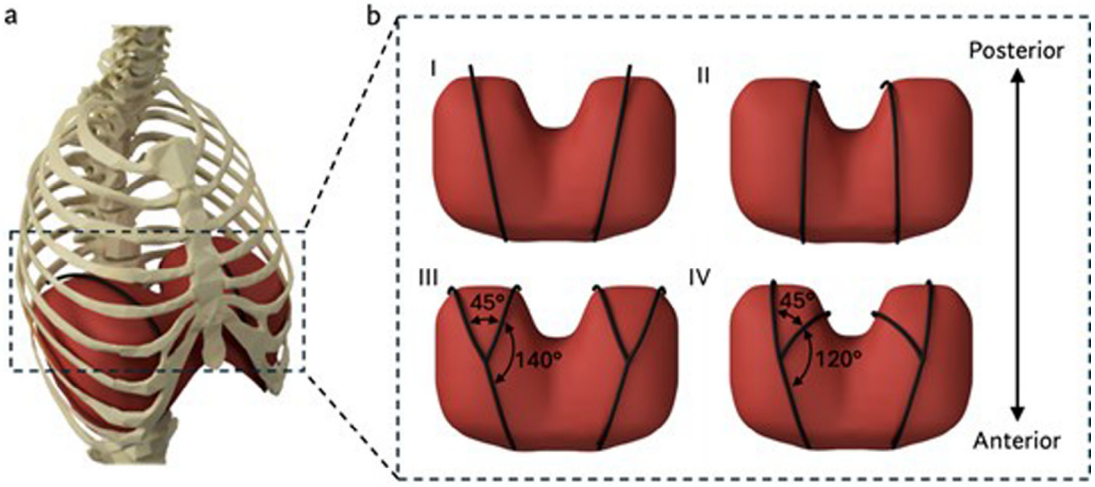

Fig. 1. (a) A rendered representation of the actuators (in black) superior to the diaphragm for augmenting ventilation. (b) Rendered representations of the two actuator geometries (black) and their configurations. (I) Straight lateral. (II) Straight medial. (III) Branched wide. (IV) Branched narrow.

respiratory rehabilitation by aiding the creation of negative thoracic pressure. This approach is particularly beneficial for patients who are difficult to wean off ventilators due to critical myopathy or deconditioning [16]. The conceptual representation of this idea is shown in Fig. 1. Porcine studies [17] show that the utilisation of soft robotic diaphragmatic actuators offers improvements in respiratory function and pulmonary rehabilitation. Despite the benefits, there is limited data available on optimised designs for actuators which could provide ventilation capacity non-inferior to mechanical systems. Whilst this study involving porcine test subjects was physiologically accurate, it saw a large variation in responses between participants. In this study, we present the use of a bespoke ex vivo respiratory simulator to show the impact of the quantity and configuration of soft robotic actuators on the optimisation of intrathoracic pressures and to minimise the variability in ventilation response between different individuals.

2. Methodology

The foundational principle for obtaining accurate results was achieved by mimicking the physiological accuracy of the diaphragm and rib topology. As the abdominal and thoracic cavities are coupled by the diaphragm, it can therefore be understood that an increase in abdominal pressure would yield a corresponding decline in the intrathoracic pressure at low respiration frequencies [18,19]. The ex vivo simulator was specially designed to simulate the above configuration. This approach was used instead of mimicking the thoracic cavity as it enables rapid prototyping and testing. The volume of the abdominal cavity used for tests was (10890 ± 27) cm^3. It has been found using computed tomography (CT) scans that the volume of the human abdominal cavity is within the order of (7967.268 ± 2925.792) cm^3 [20], suggesting that the test bed used here is appropriate for replicating the higher bound of the human abdominal cavity. Since the abdominal volume varies with patient pathology, future studies are planned to investigate how the variation in abdominal volume affects soft robotic ventilation.

The test bed comprised of a silicone diaphragm constructed from Dragon Skin 10, mimicking the material properties of human muscle [21]. The diaphragm had a homogeneous thickness of 2 mm, replicating the upper end of the thickness of an adult diaphragm [22,23]. This was manufactured with a 3D-printed mould to ensure uniform thickness and a physiologically accurate topology. This synthetic diaphragm was clamped between 3D-printed PLA ribs which had mounting points allowing the actuators to be tested in various locations on the diaphragm (refer Fig. 1). Fig. 2 shows the experimental set up and the diaphragm and ribs mounted onto an air-tight box mimicking the abdominal cavity. The positive air pressure from the abdominal cavity was used to support the diaphragm and mimic an implicit thoracic cavity. Pressure inside the abdominal box was measured with a Bosch BMP180 barometer, which had an associated measurement error of 0.05 cmH_2O, and data was logged on an Arduino Uno.

To augment diaphragm contractions we employed hydraulic McKibben muscles [24–26] that replicated muscle contractions by generating a net force, causing the actuator to contract in length and hence resulting in a displacement of the diaphragm, see Fig. 3. In our proposed approach, we chose two configurations to pressurise the diaphragm wall. In the first configuration, two 280 mm McKibben actuators were individually positioned superior to the diaphragm over each dome, to minimise radial expansion into the thoracic cavity see, Figs. 3(a) and 3(b). The sleeve had a relaxed diameter of 9 mm and a pressurised diameter of 13 mm, this setup is able to achieve net contractions of 27%. Figs. 3(g) and 3(h) show the individual actuators in relaxed and pressurised states. The second configuration consisted of a branched (or Y-shaped) McKibben actuator which was again positioned superior to the diaphragm over each dome. The branched actuators were made up of three 160 mm long components joined together by a branched barbed connector. Figs. 3(e) and 3(f) show the individual images of branched actuators in stretched and contracted states. The Y-shaped actuators were designed to have greater contact with the diaphragm during pressurisation. This increased contact area aimed to achieve a greater reduction in intrathoracic pressure during inhalation. There were two positions proposed for each actuator geometry: straight lateral, straight medial, branched wide, and branched narrow, see Fig. 1 (I, II, III and IV respectively). Both configurations were selected to minimise conflict with other structures like the heart, oesophagus and aorta, which align with the mid-line of the diaphragm. Water was used as the medium to pressurise the actuators as it allows for greater pressure control and faster pressurisation times due to its incompressible nature. Also, for future in vivo testing water's biocompatibility offers added safety, as it poses no immediate danger to the patient in case of actuator rupture. Alternative solutions based on pneumatic pressure actuation [27,28] could cause damage within the thoracic cavity if used as the compression medium. The McKibben actuators were pressurised with a

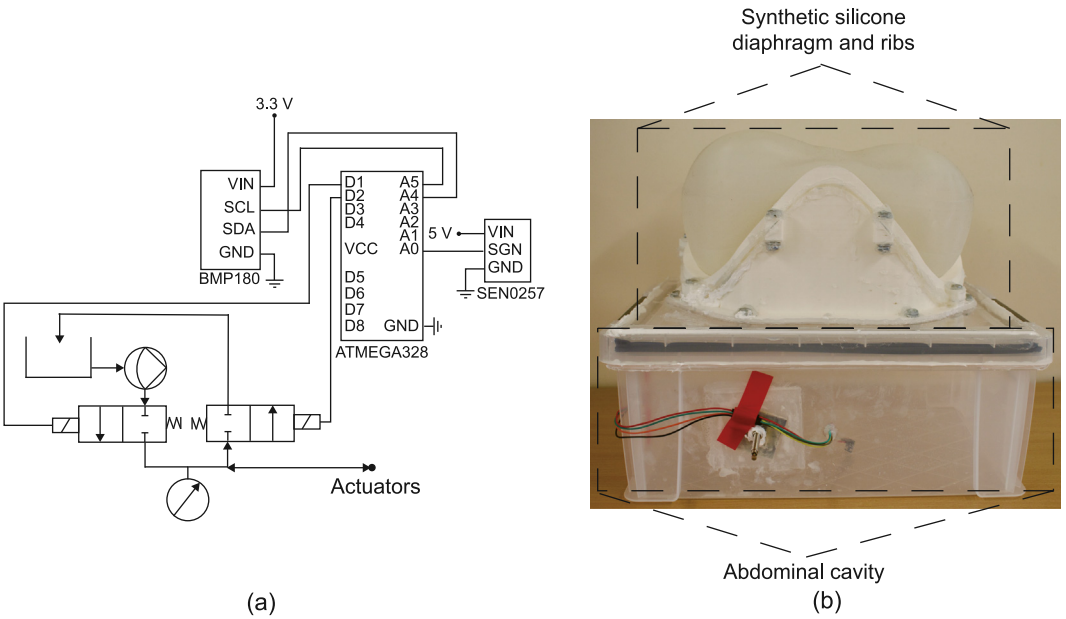

Fig. 2. The experimental setup showing the electrohydraulic schematics (a) used to control and measure the pressures of the actuators and abdominal cavity, and (b) the test bed showing the synthetic silicone diaphragm clamped to the ribs, which in turn are attached to the abdominal cavity.

Xylem Flojet diaphragm pump, and pressure was controlled using two inline solenoid valves. The actuator's hydraulic pressure was recorded with a hydraulic pressure sensor, which had an associated measurement error of 0.5% for all readings, and data was logged using an Arduino Uno.

Owing to the flexible nature of the actuators and their small cross-sectional profile, we propose to implant them with minimally invasive techniques such as thoracoscopic surgery [29]. Hence, the test bed was designed with a proposition to secure the McKibben actuators in place by stitching each end to the base of the diaphragm through mounting points, augmenting its biological function. To attach the actuators to their mounting points, a kevlar cord was threaded through the braid of the actuator's outer mesh. The hypothesis behind the proposed design was based on the pressurisation caused by the actuators contraction, which flattens the patient's diaphragm. This flattening increases the volume and decreases the pressure inside the thoracic cavity, from a baseline of -2 cmH_2O, to -7 cmH_2O for shallow breathing, and to -18 cmH_2O for deep breathing [30–34]. The reduced intrathoracic pressure draws air into the lungs, thereby stimulating inhalation. Upon release of pressure within the actuators, the diaphragm returns to its relaxed position, enabling exhalation. Additionally the elastic recoil of the actuators, with an attachment point to the central tendon, means that they can assist in exhalation, drawing the diaphragm up.

3. Results

The straight medial and branched narrow configurations were designed to drive greater intrathoracic pressures as they were placed closer to the crest of diaphragms two large domes [35]. This allowed the actuators to displace more of the diaphragm and therefore generate larger intrathoracic pressure differences. To support the silicone diaphragm, the abdominal pressure was first increased by 21.5 cmH_2O with respect to room pressure. From this baseline the actuators were pressurised for a third of a second, to replicate the natural breathing cycle [32], reaching a peak hydraulic pressure between 200 and 250 KPa. This increased the pressure in the rigid abdominal box. Once the actuators reached the target hydraulic pressure, they were depressurised and emptied, returning the system to its initial pressure. This was repeated three times to measure an average (or optimised) change in abdominal pressure, $\Delta P_{Abdominal}$. As the abdominal and thoracic cavities are coupled by the diaphragm we can therefore say that an increase in $\Delta P_{Abdominal}$ would yield a similar decrease in the intrathoracic pressure, $\Delta P_{Intrathoracic}$, at low respiration frequencies [18,19]. The pressure in the abdominal box, using the straight lateral actuator configuration, over 0.8 s can be seen in Fig. 4. Across all three trials, the abdominal cavity pressure profile remained consistent showing that the actuators are able to perform repeatable ventilation cycles. During the actuation period, $P_{Abdominal}$ rapidly increases from a baseline of 21.5 cmH_2O to a maximum pressure of 27.5 cmH_2O, hence $\Delta P_{Abdominal}$ is equal to 6 cmH_2O. From this point, the actuators were depressurised, allowing the pressure in the abdominal box to return to its initial pressure.

This same procedure was repeated once at a range of actuation pressures (50–250 KPa) for the four different actuator configurations. The results of these tests, showing how $\Delta P_{Abdominal}$ responds to the peak actuator pressure, can be seen in Fig. 5. Note the measurement error in $\Delta P_{Abdominal}$ is 0.05 cmH_2O for all readings, and the hydraulic actuator pressure sensor had a corresponding measurement error of 0.5%. The first notable result from this test shows that all actuator combinations exhibit a logarithmic pressure response. To the data a natural log curve, in the form,

$$\Delta P_{Abdominal} = A ln(\text{Peak Actuator Pressure}) - B$$

can be fitted. Where A and B are the fitting parameters which, respectively, are 2.9563 and 9.9952 for the straight lateral configuration ($r^2 = 0.89$, $P < 0.001$), 3.5522 and 13.5437 for the straight medial configuration ($r^2 = 0.94$, $P < 0.001$), 3.5050 and 13.1485 for the branched wide configuration ($r^2 = 0.93$, $P < 0.001$), and 4.0241 and 15.3824 for the branched wide configuration ($r^2 = 0.95$, $P < 0.001$). We can hence claim there are diminishing returns when ventilating at higher actuation pressures. In order to significantly increase $\Delta P_{Abdominal}$, and therefore drive deeper inspiration, we cannot rely solely on increased actuation pressures, we also need to explore other parameters such as the design and placement of the actuators. From here we can

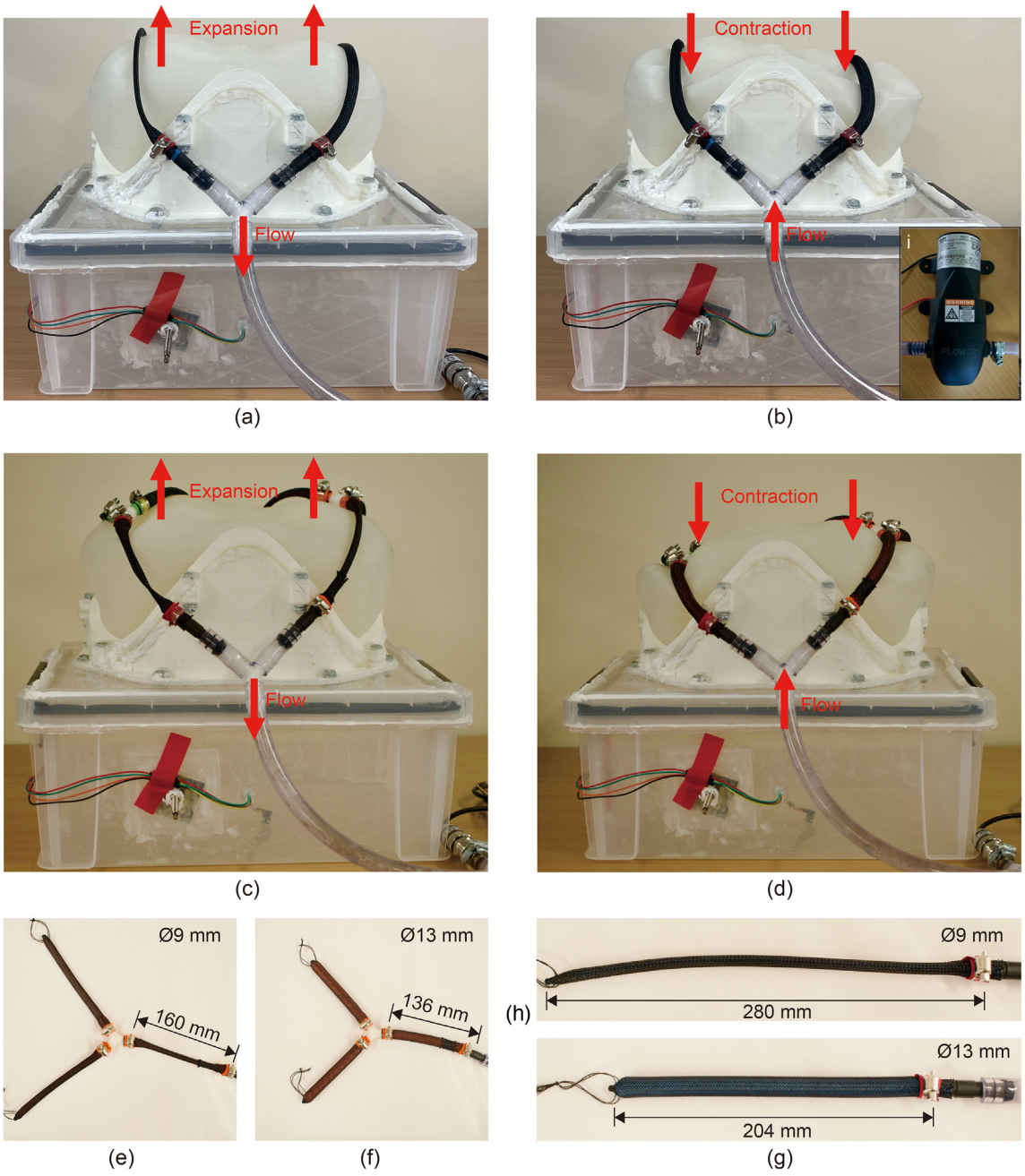

Fig. 3. The experimental setup. (a) The respiratory simulator with non-pressurised actuators in the straight lateral configuration. (b) The respiratory simulator with pressurised actuators in the straight lateral configuration. (c) The respiratory simulator with non-pressurised actuators in the branched wide configuration. (d) The respiratory simulator with pressurised actuators in the branched wide configuration. (e) Non-pressurised branched actuators. (f) Branched actuators pressurised to 250 KPa. (g) Non-pressurised straight actuators. (h) Straight actuators pressurised to 250 KPa.

quantify how each actuator position increases $\Delta P_{Abdominal}$ and hence decreases $\Delta P_{Intrathoracic}$. By repeatedly running the same test at the same actuation pressure, we can find the maximum change in $\Delta P_{Abdominal}$. Fig. 6 shows $\Delta P_{Abdominal}$ and $\Delta P_{Intrathoracic}$ for all different actuator configurations at an actuation pressure of 250 KPa. Note that the error in $\Delta P_{Abdominal}$ is found from the standard deviation over multiple readings. From repeated trials, we can use the measured change in $\Delta P_{Abdominal}$ to infer the corresponding reduction in $\Delta P_{Intrathoracic}$. The change $\Delta P_{Intrathoracic}$ for all actuator configurations is as follows, -5.83 ± 0.27 cmH_2O for the straight lateral configuration, -6.15 ± 0.46 cmH_2O for the straight medial configuration, -5.95 ± 0.47 cmH_2O for the branched wide configuration, and -6.81 ± 0.39 cmH_2O for the branched narrow configuration.

Hence it can be understood that both the geometry and location of the actuators over the diaphragm play pivotal roles in creating intrathoracic pressure. By targeting the medial parts of the diaphragm, with the straight medial configuration, we are able to increase $\Delta P_{Intrathoracic}$ by 5.5% compared to the straight lateral configuration. In comparison, the branched geometries, when targeting the posterior parts of the diaphragm in the narrow configuration, can increase $\Delta P_{Intrathoracic}$ by 14.5%, compared to the wide configuration. Given an intrathoracic pressure difference of 5 cmH_2O is needed for respiration we can say that all actuators

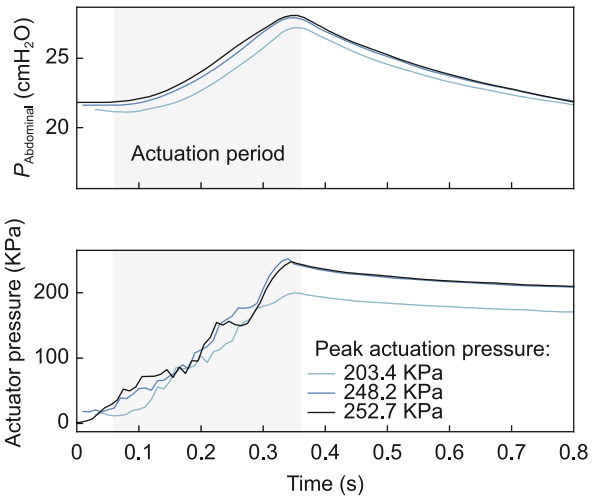

Fig. 4. The abdominal pressure (above) and the actuator pressure (below) throughout the ventilation cycle. Between 0.05 and 0.34 s the actuators are pressurised and push on the diaphragm, causing the abdominal pressure to increase. After reaching a set pressure the actuators depressurise returning the system to its normal pressure. This was done at peak actuation pressures of 203.4 KPa, 248.2 KPa, and 252.7 KPa.

fulfil this requirement [30–34]. What is noteworthy though is that the narrow branch geometry can generate similar pressure differences at lower peak actuation pressures, meaning that the narrow branched configuration can be used at an actuation pressure $\tilde{2}0$ KPa lower than other geometries. This results in lower forces on the diaphragm suture points. The key clinical result to be drawn here is that soft robotic muscles can generate sufficient intrathoracic pressures to ventilate a patient.

4. Discussion

The work done here has built upon previous studies showing that artificial muscles can be used for ventilation [17]. Specifically, we have demonstrated that all actuator configurations tested here (straight lateral, straight medial, branched wide, and branched narrow) can drive intrathoracic pressures greater than the 5 cmH_2O needed to ventilate humans [30–34]. Despite the benefits, implantation of soft actuators are distant from clinical translation due to variable responsiveness across subjects caused by biological, anatomical, and implantation differences for specific diseases. Additionally, there is a relational dependence on the rate of individual breathing and soft robotic ventilation effectiveness. Different individuals have specific respiratory biomechanics which results in variability in adaptation to soft robotic ventilation implants.

The above results provide the following three insights, firstly by targeting the posterior portion of the diaphragm with compression, larger intrathoracic pressures can be driven. Secondly, using actuators with larger contact areas, deeper intrathoracic pressures can also be driven. Finally, we have shown that additional ventilation pressures cannot be generated by simply increasing actuation pressure, the amount and location of the actuators have a significant impact. Whilst the diameter of the actuators can affect contraction length [36], other intrinsic variables can have greater impacts on contraction length, such as actuator braid angle, diameter, and stiffness. Therefore the properties of this can be optimised in further studies. Additionally, as the inflation speed (due to pump actuation) controls the ventilation profile, we propose keeping a constant predetermined flow rate, regardless of the patient's respiratory demand. By decreasing the diameter and increasing the inflation speed the ventilation profile will diverge from nominal physiological conditions, as tested here. Regarding the inhalation pressures, as the diaphragm has a large central tendinous area an increased diameter will not draw more on the diaphragm as the tendon pulls the rest of the diaphragm with it.

An important contribution of this study shows that by conducting an ex vivo study, (as opposed to in vivo) we have been able to generate repeatable results by removing subject variation enabling comparisons to be made between the quantity and location of actuators. An optimisation of configuration for understanding the variability in the diaphragm ventilation is tested. Different configurations of actuators have shown the potential to help reduce variable responses in soft robotic ventilator systems. It is noteworthy that the appropriateness of each configuration would vary between different patient populations, pathologies, and associated disease stages. For example, a high BMI individual suffering from respiratory failure, a chest infection, or a fat embolism may benefit from a branched actuator, with a distribution of forces resulting in lower peak forces at the diaphragm mounting points. Whereas a patient with a similar condition but a different pathology being low BMI may be satisfied with the straight configuration.

The presented work is unique in comparison to existing state of the art technology in lung ventilation. A traditional non-invasive ventilation method includes the utilisation of a mask for delivering ventilation support without an endotracheal tube. The current trends of invasive lung ventilation comprise of traditional positive pressure ventilators which are capable of supporting patients through protective lung ventilation for reducing injury and changing ventilation frequency [37]. These ventilators are capable of monitoring patient conditions through advanced feedback systems like Electrical impedance tomography, esophageal pressure monitoring, lung mechanics calculation etc. Under extreme respiratory failure, extracorporeal membrane oxygenation is used to bypass the lungs for gas exchange. Some other advanced methods of lung ventilation involve phrenic nerve stimulation through electrical [38] and magnetic stimulation [39,40] which may not be useful for all lung failure conditions, such as diaphragm palsy.

5. Limitations

Although this work shows that the diaphragm can be augmented with soft robotics actuators to ventilate, there still exist several significant hurdles towards clinical trials. The main hurdle is that of biocompatibility. Currently, there exists no proven way to prevent tissue irritation between soft robotic actuators and tissue. Over time, this irritation could cause scarring or release of debris due to consistent rubbing of the actuator and diaphragm wall. For ex vivo studies, we used static Kevlar strands to secure the actuators in place. However, Kevlar is not biocompatible and will cause significant irritation to tissue if implanted. Future research in exploring biocompatible actuators [41,42] and exploring surgically safer methods of implantation could result in minimising tissue damage.

The following considerations underscore the need for continued research and development to overcome these hurdles before advancing to clinical trials.

(1) Development of an effective and safe method of implantation and attachment to the diaphragm.
(2) Exploration of the potential impact of the actuators when stitched in place with a form of nonabsorbent suture.
(3) Rigorous testing is required to ensure that the actuators do not interfere with the function of internal organs, such as the heart and lungs.

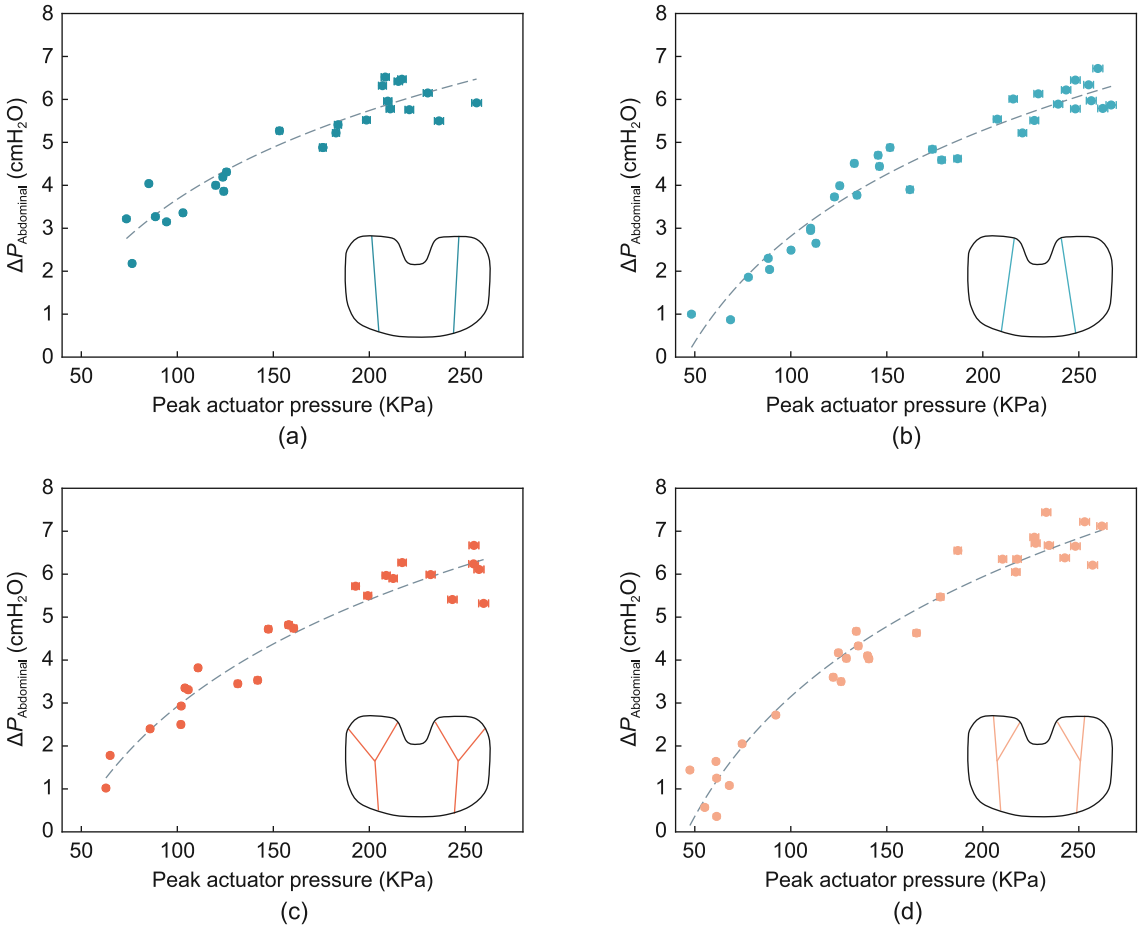

Fig. 5. How $\Delta P_{Abdominal}$ responds to the peak actuator pressure for all four actuator configurations. (a) Straight lateral. (b) Straight medial. (c) Branched wide. (d) Branched narrow.

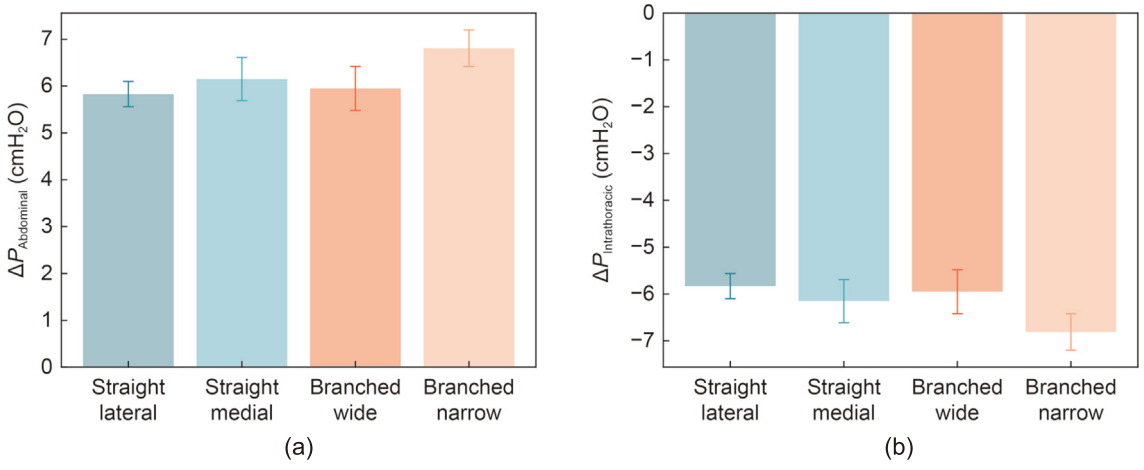

Fig. 6. (a) The maximum increase in $\Delta P_{Abdominal}$ that each actuator configuration can achieve at 250 KPa. (b) The maximum decrease in $\Delta P_{Intrathoracic}$ that each actuator configuration can achieve at 250 KPa.

(4) Exploring surgical stitching of the actuators along the length of the diaphragm to minimise rubbing and to embed a smooth, biocompatible, coating on the actuator's exterior to prevent irritation where rubbing may occur.
(5) Repeating the above studies for variation in the diaphragm topology (including volume variations for the abdominal cavity), corresponding to physiological changes between patients. This may result in finding the relationship between the change of thoracic pressure response with each actuator position and configuration.
(6) While the shape of the actuators favours thoracoscopic surgery, efforts to miniaturise the control system for portability are essential. For instance, using a miniaturised hydraulic pump [43,44] with a fixed volume of water could eliminate the need for a large reservoir, enhancing the practicality of the system for implantation.
(7) Refining the implantation process and miniaturising the control system for portability are critical steps towards translating this technology into clinical practice.

6. Conclusions

To conclude, the use of soft robotic actuators to augment the diaphragm presents a promising alternative for managing severe respiratory conditions without the need for intubation. Through ex vivo testing, we have demonstrated the capability of these actuators to generate intrathoracic pressures sufficient for ventilation, exceeding the threshold of 5 cmH_2O [30–34] required for respiration in humans. Furthermore, our investigation into different actuator geometries and positions has revealed insights into optimising intrathoracic pressure differentials. Specifically, by targeting the posterior portion of the diaphragm and by using branched actuators that have larger contact areas, we can increase the intrathoracic pressure by 17% compared to other geometries and locations. In some cases, patients do not wish to be supported by a machine but their poor health conditions do not allow them to be weaned off. While surgical options remain viable, the utilisation of soft robotic actuators offers a less invasive and potentially more effective solution for managing diaphragm-related challenges. Here we improve upon the limitations in previous studies by using an ex vivo respiratory simulator to controllably and reliably explore the geometry and position of these soft robotic actuators for optimising the maximum intrathoracic pressures.

Despite the challenges for clinical translation, the potential benefits of using soft robotics actuators for ventilation warrants continued research and development efforts. By tackling these issues, ultimately in vivo tests can be carried out to validate and realise the full potential of soft robotic diaphragmatic augmentation as a transformative therapy for severe respiratory conditions.

CRediT authorship contribution statement

Christopher Michael Hofmair: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Data curation, Conceptualization. **Kunal Bhakhri:** Writing – review & editing, Writing – original draft. **Manish Chauhan:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Project administration.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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