Keon[8] described a patient with tumor enveloping the heart and infiltrating the pericardium who arrested during induction of anesthesia and was unable to be resuscitated. He recommends preoperative cardiac assessment in patients who are symptomatic. Our patient clearly does not fit this description.

Our fortuitious finding that laryngoscopy relieved the obstruction has not been reported previously, and should be added to the list of maneuvers to relieve these airway obstructions. The mechanism of action is not clear, but a possibility is proposed. Extrinsic compression of the airway may be accentuated by voluntary muscle relaxation and bronchial smooth muscle relaxation. Laryngoscopy might conceivably apply tension to the tracheal wall tending to straighten it out somewhat, much as pulling two ends of a rope will tend to straighten it and make it taut. This mechanism presupposes that the airway obstruction is primarily a passive one, in which bronchial smooth muscle relaxation allows the weight of the mass to compress the airway.

In conclusion, we want to reemphasize that patients with anterior mediastinal masses are at risk of developing airway obstruction. If this occurs under anesthesia, direct laryngoscopy may be carried out as an aid to relieve the obstruction.

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Treatment of Acute Respiratory Failure by Extracorporeal Carbon Dioxide Elimination Performed with a Surface Heparinized Artificial Lung

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Progressive states of acute respiratory failure (ARF) require mechanical ventilation, positive end-expiratory pressure (PEEP), and high inspiratory fractions of oxygen. Despite this treatment, however, hypoxemia continues to be a major problem. The carbon dioxide tension, on the other hand, can be maintained at near-normal levels, but it is at the expense of excessive hyperventilation, which, in its turn, entails high inspiratory pressures. Accordingly, high inspiratory pressures cause alveolar barotrauma,[1] and the ensuing regional pulmonary hypocapnia[2] and hypoxemia[3] most certainly determine the final outcome of ARF.[4]

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TABLE 1. Physiological Data

	1	2	3	4
SP s/d	75/42	80/40	100/80	120/80
PAP s/d	40/25	31/21		
PCWP	18	18		
CVP	5	15		
SVR	400	240		
PVR	104	37		
CO	10	15		
MV	15	17	8.4	10
Peak Airway Pressure	50	60	40	38
PEEP	5	8	5	0
Dyn. Com.	35	18	25	65

1 = day of admission; 2 = day 8 of conventional respiratory treatment; 3 = after 12 h of extracorporeal CO_2 elimination; 4 = day 9 of extracorporeal CO_2 elimination; SP, PAP, and PCWP = systemic, pulmonary artery, and pulmonary capillary wedge pressures in mmHg, s/d systolic, and diastolic; SVR and PVR = systemic and pulmonary vascular resistance in dyne · sec · cm^{-5}; CO = cardiac output in l/min; MV = minute ventilation in liters. Peak Airway Pressure and PEEP (positive end-expiratory pressure) are in cm H_2O, Dyn.Com. dynamic compliance is in ml/cm H_2O.

As an alternative to this conventional treatment, extracorporeal carbon dioxide elimination, combined with low pressure/low frequency ventilation, has been advocated. This is a technique which dissociates the natural respiratory function into two features: apneic oxygen diffusion accomplished through the resting diseased lung, and carbon dioxide removal accomplished through an artificial extracorporeal lung.[4] Such a treatment may, however, entail uncontrollable bleeding caused by the requisite iv administration of heparin. Moreover, such an administration limits the applicability of this technique, since it is incompatible with the treatment of head injuries. Therefore, before extracorporeal circulation can provide respiratory support on a prolonged basis, a thrombo-resistant surface needs to be introduced into the extracorporeal circuit. To produce such a surface, Larm *et al.*[5] developed a technique by which the heparin molecule is covalently attached to a primary amine. Accordingly, the material to be heparinized has to be furnished with primary amine groups (see Discussion).

TABLE 2. Physiological Data

	1	2	3	4
FI_{O_2}	0.4	0.8	0.6	0.4
$P(A\text{-}a)O_2$	164	438	327	140
Pa_{O_2}	72.3	57.8	60.1	102.5
Pv_{O_2}	30.4	51.2		
Pa_{CO_2}	45.6	74.3	44.3	42.6
Pv_{CO_2}	47.3	86.1		
$Q_s/Q_t \times 100$	23	65		
$V_d/V_t \times 100$	64	83		
Platelet	190	92	61	90
APT	22	44	42	59
Weight	67.7	71.8	71.4	67.8

1 = day of admission; 2 = day 8 of conventional respiratory treatment; 3 = after 12 h of extracorporeal CO_2 elimination; 4 = day 9 of extracorporeal CO_2 elimination; FI_{O_2} fractions of inspired oxygen; $P(A\text{-}a)O_2$, Pa_{O_2}, Pa_{CO_2}, Pv_{O_2}, and Pv_{CO_2} = alveolar-arterial oxygen difference, and arterial and mixed venous oxygen and carbon dioxide tensions in mmHg, Q_s/Q_t oxygen shunt is in % of cardiac output, Vd/Vt dead space tidal volume ratio is in %, Platelet is in millions/ml, APT activated partial thromboplastin time is in sec.

This technique was first tested in ten mongrel dogs.[6] All catheters, tubings, and hollow-fiber gas exchangers that make up the extracorporeal circuit were surface heparinized by Carmeda, Stockholm, Sweden (CBAS; Carmeda Biological Active Surface). No heparin was given to the animals, and, during a veno-venous bypass (500–700 ml/min) most of the carbon dioxide production was eliminated *via* the artificial lung, while oxygenation was performed through the natural lungs by oxygen diffusion. One liter of 50% oxygen in nitrous oxide was administered through a catheter placed at the level of the carina. Ventilation frequency was ½–1 breath/min, with a tidal volume of 500 ml. Extracorporeal circulation was performed during 24–30 h. Hemodynamics, systemic and pulmonary blood pressures, and cardiac output remained stable throughout the study. Blood gases remained normal, as did carbon dioxide elimination *via* the gas exchanger. Activation of coagulation was considered very moderate, as reflected by fibrinopeptide A concentrations. The equipment in two of the experiments was ethylene-oxide sterilized, and the toxicological tests were negative.

We describe here the use of such a system in a patient with ARF.

CASE REPORT

A 44-yr-old previously healthy woman was transferred to the intensive care unit with a 14-day history of symptoms of influenza, *i.e.*, cough, fever, and progressive dyspnea. Her ventilation was mechanically controlled. A flow-directed pulmonary catheter was inserted *via* the right brachial vein. Measurements revealed an extremely high cardiac output (10 l/min) and central and peripheral vascular resistances below normal (table 1). She was treated for a severe septic shock (temperature 40° C) with iv antibiotics and large doses of cortisone (methylprednisolone 2 g) in addition to iv administered adrenaline (0.1–0.3 $ug \cdot kg^{-1} \cdot min^{-1}$). A viral pneumonia was later confirmed by cold agglutinins, and a superimposed staphylococcus aureus infection was verified by culture. Pulmonary gas exchange deteriorated during the first 8 days due to the formation of abscess cavities in both lungs. A left-sided pneumothorax was treated with pulmonary drainage catheters connected to a subatmospheric pressure of 15 cm H_2O. This gave a pulmonary air leak of 3 l/min. On the eighth day of respiratory treatment, the patient was found to have a Pa_{O_2} of 57.8 mmHg and a Pa_{CO_2} of 74.3 mmHg with a FI_{O_2} of 0.8, a minute ventilation of 17 l, and a PEEP of 8 cm H_2O (peak airway pressure 60 cm H_2O) (tables 1, 2). Dynamic compliance was 18 ml/cm H_2O. Increases in minute ventilation were found to increase the broncho-pleural air leak, and, as the systemic blood pressure was, moreover, very sensitive to increases in inflation pressure, a further increase of tidal volume was considered

unwise. Cardiac output was 15 l/min with an oxygen shunt fraction of 65%[7] and a V_D/V_T of 83%.[8] Diuresis ceased on the eighth day. As the patient was found to fulfill the extracorporeal carbon dioxide elimination entry criteria (formulated by Kolobow *et al., i.e.*, FI_{O_2}: 0.6, PEEP > 5 cm H_2O, Pa_{CO_2}: 30–45 mmHg, Pa_{O_2}: <50 mmHg, O_s/O_t > 30%, and compliance: <30 ml/cm H_2O[5], a partial extracorporeal veno-venous bypass was established surgically after informed consent had been obtained from the family.

During enflurane anesthesia, wide-bore surface-heparinized chest-drainage catheters were inserted through a surgical cut-down into the right internal jugular vein (advanced 3 cm into the right atrium) and into the right saphenous vein (advanced 5 cm into the femoral vein). Venous blood from the right atrium was pumped (flow rate between 1–1.7 l/min) by means of a non-occlusive roller pump through a surface-heparinized 4.0 m^2 hollow fiber gas exchanger and returned to the patient *via* the femoral vein. To avoid stagnant blood flow, no drainage reservoir was used (see Discussion), and, to increase blood flow in the extracorporeal circuit, another non-occlusive roller pump re-circulated the blood in the gas exchanger at a flow rate of 3 l/min. Gas flow (non-humidified, 40% oxygen in nitrogen) through the gas exchanger was 13 l/min on average.

During the first 12 h, pH_a and Pa_{CO_2} returned to within normal limits (table 2). Pa_{O_2} increased, and it was, therefore, possible to decrease FI_{O_2} to 0.6 12 h later. However, it was still necessary to continue mechanical ventilation (8–10 l/min with a PEEP of 5 cm H_2O) due both to septicemia with an ensuing total carbon dioxide production of 370–390 ml/min (elimination *via* membrane lung + elimination *via* the Engström ventilator) and to a limited maximal elimination of carbon dioxide *via* the membrane lung of 210 ml/min (see Discussion). However, the mean airway pressure decreased from about 30 to 20 cm of H_2O, and the pulmonary air leak ceased.

An activated intravascular coagulation necessitated iv administration of heparin from the day of admission; and, even during the period of extracorporeal carbon dioxide elimination, heparin was administered iv in such quantities (15000–24000 *i.e.*/24 h) as were required to maintain the activated partial thromboplastin time (APT) at about 45 s and whole-blood clotting time (WCT) at 15–30 min. The platelet count remained unaltered as compared to the pre-extracorporeal circulation count (table 2). There was no bleeding from the cut-down or the arterial line. An epistaxis after the replacement of a gastric tube ceased spontaneously after 30 min. No bleeding was observed during tracheal suction. Unfortunately, it was necessary to remove the pulmonary artery catheter to make room for the wide-bore drainage catheters. Diuresis increased dramatically during the first 48 h, with an output of about 11 l.

After almost 9 days' treatment of extracorporeal circulation, pulmonary function had improved so that the gas exchange could be kept normal through the natural lungs with a mechanical ventilation of 10 l/min and a FI_{O_2} of 0.4. The extracorporeal bypass was, therefore, terminated, and the catheters were removed surgically. Weaning by the use of intermittent mandatory ventilation was started 1 day later, and, after another 15 days of mechanical ventilatory assistance, the patient was able to breathe spontaneously. Lung function tests (static and dynamic spirometry, carbon monoxide diffusion capacity, Tc-microsphere perfusion scintigraphy, Xe 133 radio-spirometry, inert gas elimination technique, and pulmonary artery catheterization) performed 1 month after decannulation revealed normal cardiac performance and a lung function within normal range.

Discussion

By using the CBAS technique for surface heparinization, we were able to perform prolonged extracorporeal circulation with a moderate amount of iv administered heparin, and so maintain a WCT of 15–30 min. In this way, the risk of serious bleeding was eliminated.

The covalent binding of active heparin implies that the heparin coating is inherently stable and, moreover, affords the material with platelet-compatible properties. The effect of the surface-bound heparin is believed to be of a catalytic character, *i.e.*, it takes up thrombin on the surface and accelerates the effect of plasma antithrombin III. Thus, the heparin surface initiates an immediate inhibition of the held thrombin. This means that the heparin surface in combination with plasma constituents takes active part in the clearance of thrombin from the plasma. With regard to uptake and inhibition of thrombin, the function of the heparin surface is found to be both qualitatively and quantitatively rather similar to that of the vascular endothelium.[9] This means that the heparin surface is dependent on the blood flow conditions, and that its capacity to clear the thrombin is more efficient, the higher the heparin-surface area/blood volume is. Blood, which most certainly contains minute amounts of active coagulation enzymes, will, therefore, clot in those regions of the extracorporeal system where the blood flow is stagnant and the prerequisites for action of the heparin surface are absent.

Normally, stagnant blood flow is found in the drainage reservoir bag of the extracorporeal circuit. To avoid this stagnation, the reservoir bag was excluded from the circuit in our animal study, as well as in this human case. Hence, blood was drained from the right atrium by means of a roller pump. However, since the roller pump generates a subatmospheric pressure in the afferent line, which may cause the drainage catheter to suck itself into the vascular vessel wall, the tip of the drainage catheter was placed 3 cm into the right atrium (confirmed by x-ray) which has a larger diameter than the internal jugular vein. Furthermore, the catheter was equipped both with side holes and a hole at the tip. However, due to the risk of vascular vessel-wall damage, the maximal blood flow was restricted to 1.7 l/min. This figure was reached empirically by registering the pulsation in the drainage catheter. The restricted blood flow limited the elimination of carbon dioxide through the membrane lung to 210 ml/min. In healthy man, 210 ml of carbon dioxide equalize the metabolic rate. However, in this catabolic patient with septicemia, we found a carbon dioxide production of 370–390 ml/min, which forced us to continue mechanical ventilation with a minute volume of 8–10 l. Nevertheless, the mechanical stress on the pulmonary tissue decreased, and the air leak from the pneumothorax ceased. Stagnant blood flow was even found in the gas exchanger itself. In a previous study,[6] we found clot formation in the gas exchanger where blood flow had been stagnant, but, as compared to that found in gas exchangers used during

systemic heparinization, it was very moderate. In our human study, clot formation in the gas exchanger (after nearly 9 days) was equally moderate and found at exactly the same places as in the animal study.

To meet hemodynamic requirements, we believe that gas exchangers should be reconstructed; in the meantime, heparin should be administered iv in quantities that maintain an APT at about 45 s and a WCT at about 15 min. By introducing the new CBAS heparinization technique, it is our hope that extracorporeal carbon dioxide elimination can be applied in a safer manner and, thereby, earlier than is the case today where the method is merely a last-chance therapeutic tool. In this way, it should be possible on a broader basis to establish the effect and the right entry criteria of this method.

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Arnold-Chiari Malformation Type I Appearing after Tonsillectomy

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Acquired torticollis is often considered to be a benign transient muscle spasm. Persistent acquired torticollis, however, indicates an underlying disorder, such as a skeletal-muscular abnormality or a neurologic lesion. Hyperextension of the neck under anesthesia, especially during paralysis, in a patient with undiagnosed congenital cervical anomaly may be of potential harm. The following case illustrates the appearance of persistent torticollis after an elective tonsillectomy due to primary cerebellar tonsil herniation without cervical displacement of the medulla (Arnold-Chiari malformation type I).

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Key words: Anesthesia: pediatric. Complications: acquired torticollis; Arnold-Chiari malformation type I.

Report of a Case

An otherwise healthy, 8-year-old, 47-kg boy was admitted for persistent, painful right-sided torticollis. The torticollis developed during the first 24 h after a routine elective tonsillectomy at an outlying hospital 8 weeks before admission. The history was otherwise unremarkable except for a family medical history of multiple sclerosis. The physical examination was significant for obesity, painful right-sided torticollis, and no apparent gross neurologic defects. Otolaryngology consultation ruled out inflammation in the peritonsillar area. Initial reading of the radiograph and computed tomogram (CT) of the cervical spine showed no subluxation or osteitis. The patient was brought to the operating room for cervical traction by halo vest under general anesthesia to alleviate the torticollis. Anesthesia was uneventfully induced with thiopental, atracurium, and fentanyl iv, and maintained by inhalation of nitrous oxide and isoflurane; the trachea was intubated without cervical extension. The patient was discharged home the next day.

The neurosurgical consultant readmitted the patient a week later for further study. The neurologic examination revealed mild clonus, right-greater-than-left, bilateral hyperactive deep tendon reflexes in the lower extremities, and slight loss of fine movements in the right fingers. The halo vest was removed with resolution of the torticollis. Review of the CT scan revealed an Arnold-Chiari malformation (ACM) type I. Magnetic resonance studies delineated downward her-