REVIEW ARTICLE

SUCTION PUMP ASSIST DEVICE IN CARDIOGENIC SHOCK

Lukman H. Makmun, Telly Kamelia, Ryan Ranitya, Zuswayudha Samsu

1 Department of Internal Medicine, Faculty of Medicine Universitas Indonesia, Cipto Mangunkusumo National General Hospital, Jakarta, Indonesia

2 Respirology and Critical Illness Divison, Department of Internal Medicine, Faculty of Medicine Universitas Indonesia, Cipto Mangunkusumo National General Hospital, Jakarta, Indonesia

ABSTRACT

Background: Cardiogenic shock is a critical condition where the heart fails to pump blood effectively, leading to high mortality rates. Mechanical circulatory support can temporarily reduce the heart's workload while maintaining systemic perfusion. This paper introduces the Suction Pump Assist Device, an early mechanical support system patented in 1979.

Aim: To present the design, working principle, and clinical relevance of the Suction Pump Assist Device and compare it to the modern standard, Extracorporeal Membrane Oxygenation (ECMO).

Method: The working principle of the suction pump assist device was analyzed and compared to ECMO, which has been the standard of care for cardiogenic shock since 2016. Results: The Suction Pump Assist Device operates on a principle similar to VA-ECMO, where venous blood is oxygenated externally and returned to the arterial system, thereby reducing cardiac workload and ensuring systemic oxygenation. While ECMO has evolved significantly with technological advancements, the fundamental concept remains consistent.

Conclusion: The Suction Pump Assist Device represents an early innovation in mechanical circulatory support for cardiogenic shock, sharing key principles with modern ECMO systems.

Keywords: Cardiogenic shock, Mechanical circulatory support, ECMO, Suction pump assist device

ABSTRAK

Alat Bantu Pompa Isap pada Syok Kardiogenik Latar Belakang: Syok kardiogenik adalah kondisi kritis yang ditandai dengan ketidakmampuan jantung untuk memompa darah secara efektif, sehingga menyebabkan hipoperfusi sistemik dan kegagalan organ multipel. Untuk mengurangi beban kerja jantung dan mempertahankan oksigenasi sistemik, dukungan sirkulasi mekanis diperlukan. Penelitian ini memperkenalkan Alat Bantu Pompa Isap, suatu inovasi awal yang dipatenkan pada tahun 1979 sebagai solusi sementara dalam menangani syok kardiogenik.

Tujuan: Penelitian ini bertujuan menampilkan desain, prinsip kerja, dan relevansi klinis Alat Bantu Pompa Isap serta membandingkannya dengan standar modern saat ini, ECMO (Extracorporeal Membrane Oxygenation).

Metode: Prinsip kerja alat ini dianalisis dan dibandingkan dengan ECMO, yang menjadi metode standar untuk penanganan syok kardiogenik sejak tahun 2016.

Hasil: Alat Bantu Pompa Isap bekerja dengan prinsip serupa VA-ECMO, yaitu menyedot darah dari atrium kanan, mengoksigenasinya secara eksternal, dan mengalirkan kembali darah ke aorta. Proses ini mengurangi beban kerja jantung sekaligus memastikan perfusi sistemik. Meskipun ECMO telah mengalami perkembangan teknologi signifikan, konsep dasar alat ini tetap relevan dalam manajemen syok kardiogenik.

Kesimpulan: Alat Bantu Pompa Isap adalah langkah awal inovatif dalam pengembangan dukungan sirkulasi mekanis untuk syok kardiogenik. Prinsip kerjanya menjadi landasan bagi teknologi ECMO modern yang digunakan saat ini.

Kata Kunci: Syok kardiogenik, Dukungan sirkulasi mekanis, ECMO, Alat bantu pompa isap, Oksigenasi

Correspondence : Telly Kamelia Respirology and Critical Illness Divison, Department of Internal Medicine, Faculty of Medicine Universitas Indonesia, Cipto Mangunkusumo National General Hospital, Jakarta, Indonesia Email :tellykamelia99@gmail.com HP : 0878 8159 7886

How to cite this article :

SUCTION PUMP ASSIST DEVICE IN CARDIOGENIC SHOCK

Introduction

Cardiogenic shock is a clinical condition in which the heart is unable to pump blood adequately. It usually occurs in of extensive acute myocardial cases infarction, also known as KillipIV. As a defense reaction, hormonal increases such as the ANS system (Autonomic Nervous System). The Adrenaline, Nor adrenaline levels increase, resulting in an increase in heart rate so that cardiac output can be maintained. However, if the pathological process continuous, then the situation worsens, the patient falls into a state of shock which is classified as cardiogenic shock. Clinical signs : blood pressure drops rapidly, pulse becomes faster, extremities are cold, consciousness drops.

The following action is administering drugs such as dobutamine, nor adrenaline or dopamine, and corticosteroids, but success remains low with a mortality rate of >90%.

The thought arose by removing some of the blood from the body, flowing it into the device to relieve the weakened heart function. At the same time, the blood that was removed was also given oxygen as it occurs in the physiological oxygenizing process in the lung. After that the blood is returned back into the body's blood stream.Thus,it is hoped that the heart can be helped temporarily by lightening its workload in order to pump blood into the body.This device is referred to as the Suction Pump Assist Device in Cardiogenic Shock.

Suction assist device-Pump in cardiogenic shock:

This device is intended to help patients with cardiogenic shock, because the mortality rate is very high which is reaching >90%.Even though it has been treated with hemodynamic drugs in the form of vasoconstrictors, positive inotropes or with the help of IABP (Intra Aortic Balloon Pressure) aids,the mortality rate remains high.



Sauerstoff oxygen .; Wasserbad Water bath. ; Aetzkalk watte caustic lime cotton to catch CO2. Priming volume : 135 cc blood or Dextran 6% or NaCl 0,9%.

Working principle: blood is suctioned from the vein, right atrium (RA) with pump A, flowed into the oxygenator, then after oxygenation flowed back by pump B to the Aorta. So, a certain volume of blood is removed from the blood stream in the cardiovascular system, thereby reducing the burden on the heart and at the same time providing oxygenisation. Pump A and B operate in synchrony based on the heart's frequency and ventricular contraction duration (LVET).Estimated HR 60/min and LVET 0.4 sec. Cardiac index is normal: 3.5 l/min/m^2 . or $SV = 40-70 \text{ ml/m}^2$ or approximately 60-100ml $(BSA 1,5m^2)$. It is estimated that pumpA will suck in $\frac{3}{4}$ of the SV (60 ml), which is = 45 ml. The rest of the blood will flow to the coronary and the whole body. Oxygen that is put into the oxidation boxis 0,8 I/ min. The air pump (M2) works to suck gas from the oxidation box at a speed of 0,6 1/min. Heparin was dripped at a dose of 1000 units/hour. The

blood was heated to a temperature of about 37 degrees. Celsius. Venous catheters were placed in the RA and for the aorta a pigtail catheter was used with the tip in the descending aorta approximately 2 fingers below the aortic knob and the aortic knob itself was located in the left para sternal V Clause.

Work procedure:

At the beginning, 3 x45 ml of blood, or 6% Dextran or 0.9% NaCl is inserted into the oxidation box. Oxygen is run, air is removed from the oxidation box. Thermal Bad and Heparin were also run.Pump A absorb 45 ml of blood from the RA and simultaneously pump B feeds blood back into the Aorta, starting in the systole phase (R wave of the ECG) with the rate 60 times /minute.

Calculation:

Based on normal physiology of human: Os/Qt=3%. Qt= Q total = blood flow in the Lungs vascular = approximately as much as HMV (Heart Min.Vol).=cardiac index./BSA Qt = 3.5 l/min/m^2 or = 5250 ml/min byBSA1.5m².

Qs=3 % Qt Qs = Q shunt. =3% of 5250 ml/min= 157.5 ml/min. Qp=Qt - Qs=5250 - 157.5 ml/min Qp=Qperfusion.

=5092.5 ml/min.

HR=60/men or 1/sec.

So Qp = 5092,5/60 ml/sec

= 84,9 ml/sec

= 85 ml/sec.

For every 1 cardiac action, the volume of blood ventilated is 85 ml and the duration is 1 sec.

RR (Respiration Rate) = 20/min. or = 1/3 sec.

Every time you breathe, the breath volume is 500 ml and the alveoli air volume is 350 ml. Breathing frequency is 20/min,or 1/3 sec. This means that every 3 seconds, new air enters the lungs and at the same time the old air is discharged out. So 350 ml of alveoli air volume ventilates $3/1 \ge 350$ ml of blood = 255 ml of blood in 3 seconds.

350ml of air containing 20% 02 =70 ml O2. in duration 3 sec.

In **3 sec**, **255 ml** blood will take **70 ml O2**(= O2 uptake in 3 sec).

Or in 1 sec, **85 ml blood** will take 1/3 x 70 ml O2

= **23 ml O2/sec** (=O2 uptake per second).

In Oxygenator box:

Priming volume is 3 x 45 ml fluids (eg.blood or Dextran 6%, or NaCl 0,9%)

Blood is suctioned from the vein system (RA), amount 45 ml/sec then be inserted into oxygenator box.

Frequency of pump action = 60/min. (same as Heart rate) Duration of ventilation = 3 sec. Blood amount 3x 45ml=135 ml The amount of O2 required: 135/255 x 70 = 37 ml in 3 sec.

So O2 is put into the oxygenator box: $(60/3 \times 37) : 1000 = 0,74 \text{ l/min.}$

RQ(RespiratoryQuotient = 0.8)

Means that each CO2 release//02 intake, amounting to 0.8x 37 ml = 29,6 ml in 3 sec..

or = (60/3 x 29,6) : 1000 = 0,6 l/min.

So pumping air out with a frequency of **0,6** l/min.

The RQ value is determined with AGD based on the 02-CO2 diagram. 02 administration and air pump can be adjusted individually according to AGD and DO2



"excessive hypo- and hyperoxemia should be avoided" and that "gas blender should be adjusted to target slight hyperoxemia after the oxygenator (150 mmHg)"(Winiszewski et al., 2022). CO₂ is more soluble and more diffusible than oxygen so the amount removed is controlled by the gradient between the PCO₂ in the flowing blood (typically 45–50 mmHg) and the CO₂ in the ventilating gas (typically zero).

Additionally factors that influence to oxygenation.

Carbon Dioxide and Oxygen

Cardiogenic shock can be defined as a failure of global oxygen delivery DO2 to meet oxygen consumption (VO2), resulting in tissue hypoperfusion. DO2 represents the oxygen delivery and can be calculated by the product of the total oxygen content in arterial blood (CaO2) and cardiac output (CO). CaO2 is estimated from hemoglobin (Hb) concentration (in g/dl), the amount of oxygen bound to it (oxygen saturation in percentage), and the partial pressure of oxygen (PO2 in mmHg) dissolved in the plasma. (Dias Claúdio et al., 2023).

Extracorporeal Life Support Organization (ELSO) Interim Guidelines for Venoarterial Extracorporeal Membrane Oxygenation in Adult Cardiac Patients, the experts stated that



Nitric Oxide

The intrapulmonary distribution of ventilation and blood flow is a major determinant of the efficiency of transpulmonary oxygenation and determines the partial pressure of oxygen in systemic arterial blood (Pao₂). In the normal lung, a low oxygen tension constricts the vascular bed in hypoxic regions and redistributes blood flow toward lung regions with better ventilation and a higher intra-alveolar partial pressure of 2004)Endogenous oxygen.(Ichinose et al., nitric oxide (NO) has vasodilator effects on both systemic and pulmonary circulation; conversely, inhaled NO only dilates pulmonary smooth muscle(Jacobson, 2002).

Conclusion:

Working principle: protect part of the heart function by sucking blood from RA enriched with oxygen, then pumped back to the aorta. The aim is to relieve the work of the heart by reducing the volume of the load, so it is expected to help overcome cardiogenic shock.

The design and drawings of this tool have been registered with: DeutschesPatentamt.Nr.DE 2939048A1.A61M1/03. 27.9.1979-9.4.1981 for "Suction-Pump Aids in Cardiogenic Shock" by Lukman H.Makmun. ECMO must be more sophisticated.

VA ECMO provides mechanical circulatory support on the patients with cardiogenic schock, that inflow venous blood from the venen system and outflow arterial blood back into the arterial system. (Lewandowki K and Lewandowski M, 2006). Gibbon in 1937 has designed a system in which anticoagulant blood was directly exposed to oxygen.



ECMO devices for children made in Germany are still simpler.

Blood is drawn from the RA, dripped with Heparin, and entered into the oxygenator. Here, a mixture of gas: 02, CO2 and air after being mixed first. It was then flowed back into the Aorta, after being heated. The blood flow pressure was also measured.

The blood pump works with the following frequencies:

Rotation/minute x Stroke volume = cardiac output (approximately).



ECMO devices are for adults and appear more sophisticated. The working principle is similar to that for children.

There are two ECMO modalities:.venovenous (VV) and venoarterial (VA). VV ECMO offers gas exchange but n o direct cardiac support; instead, it removes blood from the venous system and reinfuses it. VA ECMO, on the other hand, provides whole cardiac support while draining blood from the venous system and reinfusing it into the arterial system(Cui et al., 2022)

References :

- Beeson Mc Dermott. Textbook of Med. 14th Ed.: pg 909
- Bussmann. Neue Aspekte zur Behandlung der li.insuff.: die Wirkung von Nitroglyerin. Med.Klin 1975 (70): 167-1706..

Enenkel W. Forthritte in der Therapie des Kardiogenen Schock. Wiener

- Klin.Wochenschrift 1976;88 (19); 631-636 Schroeder R. Medikamentoese Behandlung
 - haemodynamischer Komplikationen bei akuten Myokardinfark. Der Internist. 1972; 13 (9): 380-387.
- Siegenthaler W. Klinische Pathophysiology . 1973;: 593-604

Conroe. Die Lunge. 1973

:

Wolff. Die Kuenstliche Beatmung zur Intensive

Stationen. 1975

- Bartlett, R. ;H;. (2020). Physiology of Extracorporeal Gas Exchange. *Wiley Online Library*, 10(3).
- Cui, Y., Zhang, Y., Dou, J., Shi, J., Zhao, Z., Zhang, Z., Chen, Y., Cheng, C., Zhu, D., Quan, X., Zhu, X., & Huang, W. (2022).
 Venovenous vs. Venoarterial Extracorporeal Membrane Oxygenation in Infection-Associated Severe Pediatric Acute Respiratory Distress Syndrome: A Prospective Multicenter Cohort Study. *Frontiers in Pediatrics*, 10. https://doi.org/10.3389/fped.2022.832776
- Dias Claúdio, F., Piçarra, B., Neves, D., Trinca, M., & Dias Cláudio, F. (2023). Cardiogenic shock-Back to the basics. In *Crit Care Shock* (Vol. 26, Issue 4).
- Ficial, B., Vasques, F., Zhang, J., Whebell, S., Slattery, M., Lamas, T., Daly, K., Agnew, N., &Camporota, L. (2021). Physiological basis of extracorporeal membrane oxygenation and extracorporeal carbon dioxide removal in respiratory failure. *Membranes*, 11(3), NA. https://doi.org/10.3390/membranes11030225
- Ichinose, F., Roberts, J. D., &Zapol, W. M. (2004). Inhaled nitric oxide: a selective pulmonary vasodilator: current uses and therapeutic potential. In *Circulation* (Vol. 109, Issue 25, pp. 3106–3111). https://doi.org/10.1161/01.CIR.0000134595.8 0170.62
- Winiszewski, H., Guinot, P. G., Schmidt, M., Besch, G., Piton, G., Perrotti, A., Lorusso, R., Kimmoun, A., &Capellier, G. (2022).
 Optimizing PO2 during peripheral venoarterial ECMO: a narrative review. In *Critical Care* (Vol. 26, Issue 1). BioMed Central Ltd. <u>https://doi.org/10.1186/s13054-022-04102-0</u>
- William C.Wrisinger, Shaun L.Thompson. Basics of ECMO. Surg.Clin.N.Am. 102 (2022)23-35.
- Lewandowski K, Lewandowski M. Extre Corporeal Mbr. Gas Exchange. In Encyclopedia of Respiratoru Med. 2006.