Percutaneous Left Ventricular Assist Devices During Cardiogenic Shock and High-Risk Percutaneous Coronary Interventions

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Left ventricular assist devices were developed to support the function of a failing left ventricle. Owing to recent technological improvements, ventricular assist devices can be placed by percutaneous implantation techniques, which offer the advantage of fast implantation in the setting of acute left ventricular failure. This article reviews the growing evidence supporting the clinical use of left ventricular assist devices. Specifically, we discuss the use of left ventricular assist devices in patients with cardiogenic shock, in patients with acute ST-elevation myocardial infarction without shock, and during high-risk percutaneous coronary interventions.

Introduction

Left ventricular assist devices are mechanical devices developed to substitute for, at least in part, the function of a failing left ventricle. Although the reasoning for left ventricular assist devices was introduced in the 1950s, their introduction into clinical practice was more recent. Further technological improvements with modification of pumps to develop a continuous rather than pulsatile blood flow allowed percutaneous placement of these devices. Percutaneous ventricular assist devices (pVADs), as opposed to surgical ventricular assist devices, offer greater availability, simplicity, and prompt installation in the setting of acute left ventricular failure. A growing body of evidence supports the use of pVADs in patients with acute left ventricular failure (cardiogenic shock), acute ST-elevation myocardial infarction (STEMI) without shock, and during high-risk percutaneous coronary interventions (PCIs).

At present, two pVADs are available for clinical use and have received Conformité Européenne (CE) and US Food and Drug Administration approval. The TandemHeart (Cardiac Assist, Pittsburgh, PA) [1] provides circulatory support by establishing a left-to-left shunt: oxygenated blood is harvested from the left atrium through the transseptal inflow cannula and injected through the outflow cannula into the abdominal aorta. The system consists of a 21F left atrial inflow cannula, an extracorporeal centrifugal pump rotating at up to 7500 rpm, a femoral outflow cannula (15F–17F) that extends into the iliac artery, and a microprocessor-based pump controller, which can provide blood flow up to 4 L/min. The tip of the atrial drainage cannula is positioned under fluoroscopic guidance into the left atrium following transseptal puncture.

The Impella Recover left percutaneous (LP) 2.5-L/min device (Abiomed-Impella Cardiosystems AG; Aachen, Germany) [2] is a catheter-mounted microaxial rotary pump with a nominal exterior diameter of 4.0 mm, which is retrogradely placed across the aortic valve into the left ventricle with the pump outlet and motor positioned in the aorta; oxygenated blood is harvested from the left ventricle, passes through the impeller, and is expelled into the ascending aorta. The microaxial pump consists of an impeller driven by an integrated microelectric motor on the distal end of a flexible catheter. At a maximum speed of 50,000 rpm, the pump provides a maximum output of 2.5 L/min. Abiomed also developed a more powerful but otherwise similar version of the LP 2.5 that can maintain an output of 5.0 L/min (Impella LP 5.0). In contrast to the smaller version, the larger device requires a femoral cutdown [3,4].

Technique for Implantation

Before implantation of either pVAD discussed above, angiography of the abdominal aorta, iliac, and femoral vessels is advised to delineate significant obstruction or excessive tortuosity of these vessels.

TandemHeart

Implantation

Following femoral venous access, transseptal puncture is performed using standard techniques. Then, the interatrial septum is dilated to accommodate the 21F left atrial drainage cannula. Using the Seldinger technique, a 15F to 17F femoral artery cannula is placed retrogradely into the femoral artery. Both cannulae are connected to the centrifugal pump under careful evacuation of any air within the tubing. Anticoagulation should be achieved with heparin at therapeutic levels (activated clotting time, 250 seconds during the intervention and > 200 seconds during the support phase). Attention should be paid to limb ischemia, as it may complicate TandemHeart implantation in up to 15% of patients owing to the diameter of the large outflow cannulae potentially impeding flow to the lower extremities [5].

Following stabilization during the acute phase, a stepwise weaning process is initiated. Weaning criteria comprise hemodynamic and clinical evidence of cardiogenic shock resolution. Hemodynamic criteria are usually met when cardiac index and mean arterial pressure exceed 2.0 L/min/m^2 and 60 mm Hg, respectively. Clinical criteria represent the absence of end-organ hypoperfusion and lack of inotropic support. Following successful weaning from circulatory support, the inflow and outflow cannulae can be removed. Hemostasis is usually achieved by manual compression but occasionally may necessitate surgical closure.

Impella Recover LP 2.5

Implantation

Implantation of the 12F Impella Recover LP 2.5 system requires femoral arterial, but no venous, access. Using the Seldinger technique, a 13F peel-away sheath is placed retrogradely into the femoral artery. Then, a 5F pigtail or right Judkins catheter is used to pass the aortic valve and gain access to the left ventricle. This catheter is then exchanged over a 0.014-inch guidewire for the 12F Impella Recover LP 2.5 catheter. Circulatory support is initiated and can be adjusted at nine different levels (performance level: P1–P9; maximal flow ~ 2.5 L/min). Because the catheter remains positioned across the aortic valve, it has been suggested that at low speed, the pump just compensates the aortic regurgitation induced by the 12F catheter [6,7].

Anticoagulation

Anticoagulation should be achieved with heparin at therapeutic levels (activated clotting time, 250 seconds during the intervention and > 200 seconds during the prolonged support phase). Attention should be paid to hemolysis, which typically occurs within the first 24 hours after implantation of the high-speed rotary blood pump.

Following stabilization during the acute phase, stepwise weaning is attempted by slow reduction of the pump output. Following successful weaning of the patient from circulatory support, the Impella Recover LP 2.5 catheter is withdrawn from the left ventricle into the abdominal aorta, and then the catheter and intra-arterial sheath are removed together, allowing for some back bleeding. Hemostasis is achieved by manual compression.

Clinical Indications

pVADs during cardiogenic shock

Cardiogenic shock is encountered in 7% of ST-segment elevation and 2.5% of non–ST-segment elevation myocardial infarctions [8]. Its mortality remains high (50% to 70%) despite advances in reperfusion therapy [9–11.] In about half of these cases, cardiogenic shock is already overt at the time of hospital admission (early shock), whereas it evolves during the hospital stay in the remaining patients [12].

Cardiogenic shock results from pump failure that results in insufficient systemic perfusion pressure (low cardiac output state). The initial pump failure is further aggravated by a systemic inflammatory reaction with activation of the complement system and expression of cytokines and inducible nitric oxide synthase, which may result in inappropriate vasodilatation and myocardial depression, and thus perpetuates the vicious cycle (Fig. 1) [13].

The principal benefit of pVADs is compensation for the loss of myocardial pump function, normalizing cardiac output, and thus allowing physiologic perfusion of vital organs. Both commercially available pVADs are effective at rapidly providing hemodynamic support in patients with cardiogenic shock. Thiele et al. [14] reported that cardiac output and mean arterial blood pressure increased by 37% ($P < 0.001$) and 27% ($P < 0.001$), respectively, and pulmonary capillary wedge pressure and mean pulmonary arterial pressure decreased by 50% and 35% ($P < 0.001$), respectively, directly after implantation of the TandemHeart. Meyns et al. [15] described the acute hemodynamic effects of the Impella LP 5.0 in 16 patients presenting with cardiogenic shock. Cardiac output increased by 43% ($P = 0.01$) and mean arterial blood pressure by 51% ($P = 0.004$), whereas preload, as assessed by pulmonary capillary wedge pressure, decreased by 38% ($P < 0.001$).

In cardiogenic shock, pVAD support may attenuate the inflammatory reaction triggered by vital organ hypoperfusion, which in turn results in fewer circulating cytokines and less oxidative stress. Finally, pVADs may have beneficial effects on long-term outcome by reducing ventricular strain and improving remodeling. In animals, use of left ventricular assist devices was associated with reduced infarct size and improved microcirculatory perfusion compared with control animals [16,17]. In humans, failing ventricles harvested at the time of heart transplantation demonstrated reduced left ventricular size and myocyte diameter after left ventricular assist device implantation compared with unassisted hearts [18]. Of note, the beneficial effect was dependent on the duration

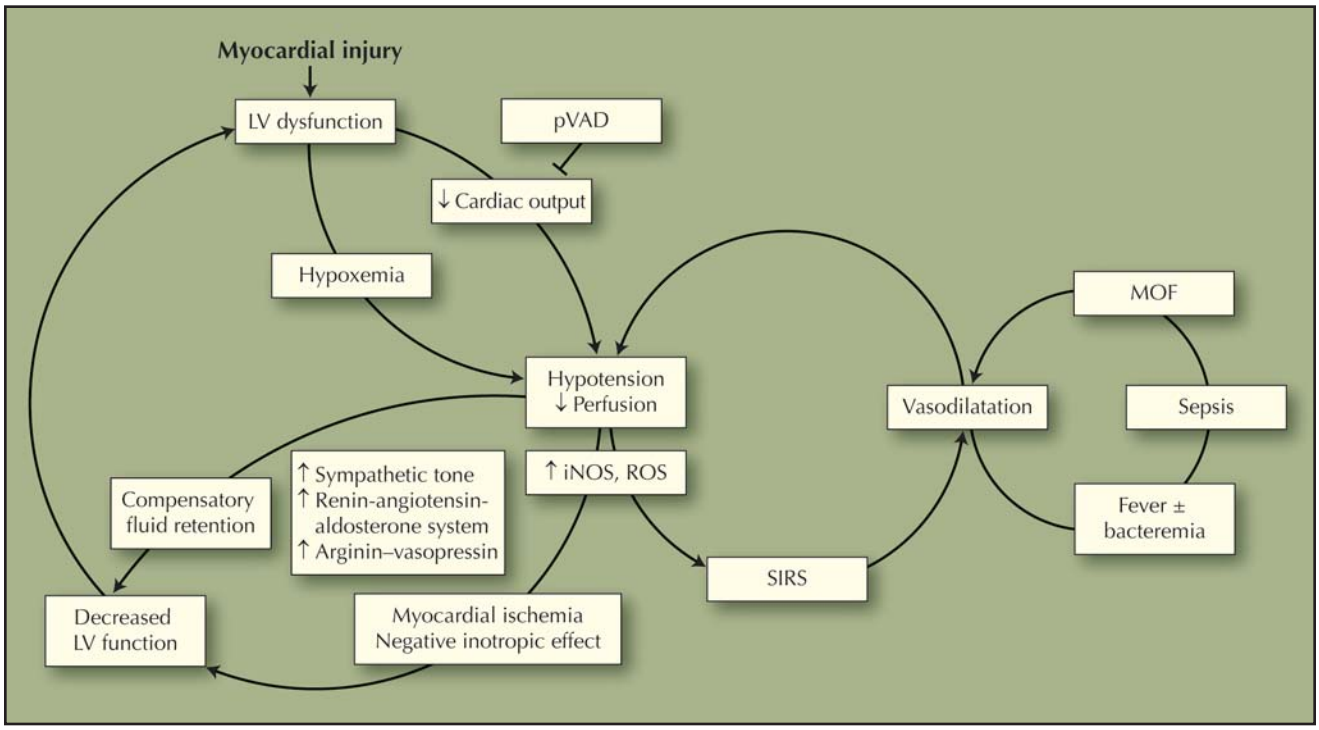

Figure 1. Cardiogenic shock results from pump failure. The primary left ventricular (LV) dysfunction leads to a decrease in cardiac output. On the systemic side, this results in insufficient systemic perfusion pressure (low cardiac output state). On the pulmonary side, pulmonary edema produces hypoxemia. The hypoxic hypoperfusion results in compensatory fluid retention and a negative inotropic effect, which result in a vicious cycle. Pump failure is further aggravated by a systemic inflammatory reaction (SIRS) with expression of cytokines and inducible nitric oxide synthase (iNOS) and generation of a large amount of nitric oxide and reactive oxygen species (ROS), which may result in inappropriate vasodilatation and further myocardial depression. Finally, activation of the complement system and formation of a "sepsis-like" reaction occasionally results in multiple organ failure (MOF) and death. The principal benefit of a percutaneous ventricular assist device (pVAD) is compensation for the loss of myocardial pump function, normalizing cardiac output and thus allowing physiologic perfusion of vital organs.

of left ventricular assist device support and was more pronounced if support was provided for more than 30 days.

Clinical evidence supports this conceptual framework of the benefits of pVADs in cardiogenic shock. For instance, analysis of 138 consecutive patients presenting with acute myocardial infarction complicated by cardiogenic shock during a 6-year period at the Cleveland Clinic revealed improved outcome in those who underwent aggressive intervention with use of early revascularization procedures, left ventricular assist device support, and heart transplantation compared with patients supported by only intra-aortic balloon pump (IABP) (54% vs 81%; $P < 0.002$) [19]. Furthermore, Chen et al. [20] demonstrated that the earlier the patient received pVAD support, the better the clinical outcome was. These investigators compared survival of 15 patients who received pVAD support within 2 weeks (early cohort) with 10 patients who were treated only after 2 weeks following severe myocardial infarction (late cohort). Mortality was substantially lower in the early cohort (26%) compared with the late cohort (40%). Similarly, Park et al. [21] reported seven patients presenting with refractory cardiogenic shock 3.9 ± 2.6 days (range, 1–9 days) after acute myocardial infarction who were treated with a left ventricular assist device as a bridge to heart transplantation. In this cohort, six patients (86%) underwent cardiac transplantation, and five (71%) were alive at 12-month follow-up.

Presence of biventricular pump failure carries a poorer prognosis even after successful pVAD implantation. Entwistle et al. [22] reported 17 patients suffering from cardiogenic shock after acute myocardial infarction who were supported by a pVAD. Eight of 11 patients (73%) who were suffering primarily from left ventricular dysfunction were successfully weaned from pVAD support, and six (54%) survived to hospital discharge. In contrast, none of the six patients with biventricular failure could be weaned from pVAD support, and only one (17%) patient survived [22]. Table 1 summarizes the in-hospital survival of patients with cardiogenic shock due to acute myocardial infarction treated with a surgical ventricular assist device or a pVAD.

To date, three clinical randomized controlled trials have compared pVADs with IABPs in patients presenting with cardiogenic shock due to acute myocardial infarction. Thiele et al. [14] reported the clinical outcomes of 41 patients assigned to the TandemHeart ($n = 21$) or IABP ($n = 20$). The mean support duration was 3.5 days in the pVAD group and 4 days in the IABP group. The primary end point of cardiac power index was more effectively improved by the TandemHeart (0.37 W/m^2) compared with IABP support (0.22 W/m^2; $P = 0.004$), and this was accompanied by a more rapid decrease in serum lactate and improved renal function. There were, however, no

Table 1. VADs for acute myocardial infarction complicated by cardiogenic shock

Study	Year	*N*	Device	30-day survival, *n (%)*
Zumbro et al. [36]	1987	4	Surgical VAD	1 (20)
Noda et al. [37]	1989	10	Surgical VAD	3 (30)
Farrar et al. [38]	1990	17	Surgical VAD	13 (76)
Frazier et al. [39]	1992	3	Surgical VAD	1 (33)
Moritz and Wolner [40]	1993	6	Surgical VAD	2 (33)
Körfer et al. [41]	1995	14	Surgical VAD	3 (21)
Keon and Olsen [42]	1996	8	Surgical VAD	3 (38)
Loisance et al. [43]	1996	3	Surgical VAD	2 (67)
Chen et al. [20]	1999	25	Surgical VAD	17 (68)
Leshnower et al. [44]	2006	49	Surgical VAD	33 (66)
Tayara et al. [19]	2006	18	Surgical VAD	14 (81)
Thiele et al. [14]	2005	21	Percutaneous VAD	12 (56)
Merhi et al. [45]	2005	1	Percutaneous VAD	1 (100)
Burkhoff et al. [23]	2006	13	Percutaneous VAD	7 (54)
Idelchik et al. [46]	2007	1	Percutaneous VAD	1 (100)
Patane et al. [47]	2007	1	Percutaneous VAD	1 (100)
Cook et al. [5]	2007	34	Percutaneous VAD	19 (56)
Gregoric et al. [48]	2008	1	Percutaneous VAD	1 (100)
Sassard et al. [3]	2008	2	Percutaneous VAD	2 (100)
Bruckner et al. [49]	2008	5	Percutaneous VAD	5 (100)
Idelchik et al. [50••]	2008	18	Percutaneous VAD	13 (73)
Lam et al. [4]	2009	5	Percutaneous VAD	3 (60)

VAD—ventricular assist device.

significant differences with respect to 30-day mortality (nine deaths in pVAD group vs nine deaths in IABP group; odds ratio, 0.92; 95% CI, 0.27–3.15; $P = 0.49$). Burkhoff et al. [23] randomly assigned 19 patients to TandemHeart and 14 patients to IABP support. The mean duration of support was 2.5 days. Compared with the IABP, the TandemHeart significantly improved cardiac output ($P < 0.05$) and decreased pulmonary capillary wedge pressure. However, there were no significant differences with respect to 30-day mortality (nine deaths in the pVAD group vs five deaths in IABP group; odds ratio, 1.52; 95% CI, 0.39–6.58; $P = 0.45$). In both studies using the TandemHeart as a pVAD device, complications—including limb ischemia and severe bleeding—were more frequent with the pVAD than the IABP.

To date, only one randomized controlled study has compared the Impella Recover LP 2.5 with IABP support in patients presenting with cardiogenic shock. Seyfarth et al. [24••] randomly assigned 26 patients to pVAD or IABP. The primary end point of cardiac output 1 hour after device implantation was more effectively improved by the Impella LP 2.5 (increased by 1.1 L/min) compared with IABP support (increased by 0.2 L/min), and this was accompanied by a more rapid decrease in serum lactate. As in the previous studies, there were no significant differences with respect to 30-day mortality between the two groups.

In the setting of cardiogenic shock, pVADs can be considered as a bridge to recovery in case of a potentially reversible cause of left ventricular failure, including acute myocarditis; drug overdose; hypothermia; PCI-related complications (no-reflow phenomenon, occlusive dissections, or air embolism); postcardiotomy syndrome; and incessant ventricular tachycardia or fibrillation. Moreover, pVADs may be considered as a bridge to a permanent (surgical) left ventricular assist device (bridge-to-bridge) or to heart transplantation (bridge-to-transplantation) in case of severe but irreversible left ventricular failure. In the latter group, advantages of percutaneous over surgical left ventricular assist devices include the speed of implantation, lower incidence of arrhythmias, and the additional time gained to perform diagnostic procedures to determine the suitability of patients for the financially and technically more demanding therapeutic options. Relative contraindications for pVADs are severe peripheral vascular disease prohibiting device implantation and severe bleeding diathesis.

In summary, pVADs are effective tools for rapid hemodynamic stabilization in patients presenting with

cardiogenic shock, and usually maintain vital organ perfusion even in case of cardiac arrest [25]. Although improvement of hemodynamic parameters by means of pVAD implantation are favorable compared with support by an IABP alone, it remains to be determined whether this benefit translates into improved clinical outcome.

Utility of ventricular assist devices in myocardial infarction without cardiogenic shock

The use of pVADS also has been considered in the setting of very large myocardial infarctions. The specific goals in this particular setting are as follows: 1) to prevent hemodynamic deterioration and 2) to prevent negative ventricular remodeling (left ventricular dilation) by prolonged mechanical unloading. The rationale is mainly based on animal models [26,27]. Experimental studies in humans subsequently demonstrated that support with a left ventricular assist device normalizes diastolic function and chamber size in dilated cardiomyopathy [28–30]. More recently, some studies also suggested that use of pVADs (Impella) in STEMI may decrease infarct size [31] and improve microcirculation in peripheral tissue [32]. The single-center, nonrandomized Academic Medical Center Mechanical Support for Acute Congestive Heart II trial [31] included 20 consecutive patients with acute anterior STEMI without cardiogenic shock undergoing primary PCI. Immediately after PCI, 10 patients received 3 days of Impella support (Impella group) and were compared with 10 patients treated according to current routine care (including IABP) therapy. The primary safety and feasibility end points included device-related complications and major adverse cardiac and cerebral events during support, and 4-month follow-up. With the exception of more frequent bleeding complications requiring transfusion in the Impella group (four patients vs two controls), no difference in primary outcome was observed between the two groups. The secondary end point was left ventricular recovery as assessed by transthoracic echocardiography. In the Impella group (n = 8), left ventricular ejection fraction improved from 28% at baseline to 37% ($P < 0.05$) and 41% ($P < 0.05$) after 3 days and 4 months, respectively, whereas in the control group (n = 9), no significant improvement was observed. Based on these initial data, the authors plan a head-to-head randomized comparison of IABP and Impella support in patients with large anterior STEMI.

Accordingly, pVADs may play a beneficial role in selected patients suffering from large (anterior) acute myocardial infarction without cardiogenic shock by unloading the stunned left ventricle and preventing further hemodynamic deterioration. The clinical benefit over aggressive standard therapy remains to be determined.

Utility of ventricular assist devices during high-risk PCIs

PCIs are deemed to be at increased risk when the intervention is performed in vessels subtending a large ischemic myocardium at risk (ie, last remaining vessel) and in patients with poor left ventricular ejection fraction. Before pVADs were available, attempts to protect the area at risk included the use of IABP counterpulsation or cardiopulmonary bypass. Valgimigli et al. [6] reported the hemodynamic effect of the Impella Recover LP 2.5 device in 10 patients undergoing high-risk PCI. Using pressure–volume loop analysis and intracardiac echocardiography, the investigators observed an acute aortic regurgitation with increase in left ventricular volume immediately after device insertion into the left ventricle in all patients that tended to persist even at maximal pump speed. The net effect resulted in no significant unloading of the left ventricle. However, in a similar experiment using hyperemia-induced intracoronary pressure and flow measurements, Remmelink et al. [33] demonstrated positive effects on aortic pressure and coronary perfusion pressure in 11 patients undergoing high-risk PCI. Using incremental support of Impella LP 2.5 (P1, P3, P6, and P9), mean aortic pressure increased by up to 13% ($P = 0.001$) and coronary flow velocity reserve up to 18% ($P < 0.001$).

Several small registers show low rates of device-related complications and lower-than-expected procedural and in-hospital mortality. These studies also report similar or lower-than-predicted mortality at 1-month follow-up (Table 2) [34,35]. However, to date, no randomized trial has assessed the clinical benefit of pVAD support in the setting of high-risk PCI. It is important to carefully weigh the benefit of pVAD support in this setting against the potential risk associated with the procedure.

In summary, pVADs may be considered in selected patients undergoing high-risk PCI, owing to their ability to protect the ischemic area at risk during the procedure and to insure hemodynamic stability. However, the clinical benefit of pVAD support in this setting requires further study.

Conclusions

Acute left ventricular failure is encountered during spontaneous myocardial infarction or high-risk PCI. It remains a life-threatening medical condition due to primary cardiac pump failure with the risk of secondary vital organ hypoperfusion that results in a downward spiral leading to multiple organ failure and potentially death. Preventive or bailout pVAD implantation can restore or maintain hemodynamic stability and represents an important advance in the management of patients with acute left ventricular dysfunction or cardiogenic shock. Although improvement of hemodynamic parameters by pVADs appears promising compared with IABP support alone, it remains to be determined whether this benefit translates into improved clinical outcome.

Disclosure

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Table 2. Percutaneous ventricular assist devices during high-risk percutaneous coronary intervention

Study	Year	*N*	Device	30-day survival, *n (%)*
Vranckx et al. [51]	2003	3	TH	3 (100)
Aragon et al. [52]	2005	8	TH	7 (87)
Giombolini et al. [53]	2006	3	TH	3 (100)
Kar et al. [54]	2006	7	TH	6 (86)
Kar et al. [55]	2006	5	TH	4 (80)
Vranckx et al. [56]	2008	23	TH	14 (61*)
Rajdev et al. [57]	2008	20	TH	19 (95)
Al-Husami et al. [58]	2008	6	TH	5 (83)
Gimelli and Wolff [34]	2008	10	TH	10 (100)
Vranckx et al. [59]	2009	9	TH	8 (89)
Cook et al. [60]	2007	19	TH/IP	15 (79*)
Henriques et al. [61••]	2006	19	IP	11(89%)
Remmelink et al. [62]	2007	11	IP	11 (100)
Thomopoulou et al. [35]	2008	3	IP	2 (67)
Valgimigli et al. [6]	2006	10	IP	—
Burzotta et al. [63]	2008	10	IP	10 (100)
Vecchio et al. [64]	2008	7	IP	7 (100)
Eichhöfer et al. [65]	2008	2	IP	2 (100)
Dixon et al. [66]	2009	20	IP	18 (90)

*Denotes 6-month rather than 30-day survival.
IP—Impella Recover left percutaneous 2.5; TH—TandemHeart.

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