Chapter

45

Pericardial Effusion

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Case Scenario

A 3-year-old boy weighing 15 kg with acute lymphoblastic leukemia presents to the cardiac catheterization laboratory for drainage of a moderate to large pericardial effusion. He began induction chemotherapy with daunorubicin, vincristine, L-asparaginase, and prednisone 1 week earlier. He has a right tunneled subcutaneous internal jugular central line ("port-a-cath"). The medical response team was called to evaluate him this afternoon for down-trending blood pressure accompanied by worsening respiratory distress and admitted him to the pediatric intensive care unit for closer observation and evaluation. Recent vital signs are blood pressure 75/43 mm Hg, heart rate 144 beats/minute, respiratory rate 40 breaths/minute, SpO_2 94% on room air, and temperature 36.8°C. On physical examination, the boy has mild suprasternal and intercostal retractions and is sitting upright. Lung fields are clear bilaterally, but heart sounds are muffled. Chest radiograph shows no pulmonary disease.

Bedside echocardiogram demonstrates the following:

- *Moderate–large circumferential pericardial effusion*
- *Right atrial collapse during early systole*
- *Right ventricular collapse during early diastole*
- *Inspiratory "bounce" of the interventricular septum toward the left ventricle*

Key Objectives

- Recognize the physiologic implications of symptomatic pericardial effusion.
- Describe the preoperative assessment for a patient with symptomatic pericardial effusion.
- Describe anesthetic management strategies for a patient with a pericardial effusion.
- Describe potential perioperative complications.

Pathophysiology

What is a pericardial effusion and what common causes exist in children?

A pericardial effusion (PCE) is an abnormal accumulation of fluid within the pericardial sac. The pericardial sac is the space contained between the visceral and parietal layers of the pericardium, and normally it can contain up to 50 mL of fluid, usually plasma ultrafiltrate. Any accumulation of fluid beyond this amount is considered a PCE. The hemodynamic consequences of this will depend on the pressure–volume relationship of the pericardial sac. Common causes of PCE in children include infection (mostly viral and gram-positive bacteria), neoplasm, inflammatory or immune-mediated response (including post-pericardiotomy syndrome, occurring in up to 15%–30% of open-heart cases and in <2% of closed procedures). Additional causes include hemopericardium (secondary to trauma, postoperative bleeding or iatrogenic), uremia, chronic renal failure, hyper- or hypothyroidism, and radiation therapy.

How is cardiac tamponade defined and what are the hemodynamic consequences?

A PCE is usually asymptomatic unless it causes cardiac tamponade. In tamponade, the external compression of the heart results in equalization of pressure in all cardiac chambers, causing reduced atrioventricular blood flow during diastole and biventricular stroke volume during systole. This, in turn, reduces cardiac output (CO) for both sides of the heart, causing signs and symptoms related to hypoperfusion. The patient may present with right-sided symptoms of hepatic congestion, peripheral edema, and jugular venous distension as well as left-sided symptoms of orthopnea, hypotension, sinus tachycardia, and cool extremities with prolonged capillary refill.

It is important to note that cardiac tamponade may also occur in cases of open pericardium. This usually happens in patients in whom the pericardium was used as an autologous patch during the surgical correction of their congenital heart disease, and so the pericardial sac was left "open." In these cases, the pericardial fluid will fill the mediastinum and again, once the extrinsic pressure compromises cardiac compliance, tamponade physiology will be seen.

In addition to PCE, cardiac tamponade physiology is also seen in other instances that are outside the scope of

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this chapter, including pneumothorax, breath-stacking, mediastinal masses, pericarditis, and thoracoscopic insufflation.

How is cardiac tamponade diagnosed?

Cardiac tamponade is a clinical diagnosis that is supported by diagnostic studies. Classically, acute cardiac tamponade is defined by Beck's triad, which consists of the following:

- Jugular venous distension as a result of a noncompliant right ventricle (RV) with high filling pressures
- Muffled heart sounds secondary to the pericardial effusion or constriction
- Systemic hypotension due to diminished left ventricular filling

As all three findings occur simultaneously in only one-third of patients, the absence of any part of Beck's triad does not rule out tamponade. Other signs and symptoms may include right upper quadrant pain (due to hepatic congestion), orthopnea, dyspnea, tachypnea, and sinus tachycardia. Pulsus paradoxus, an abnormally large decrease in systolic blood pressure (>10 mm Hg) on inspiration, may also be present. It is important to note that patients may also have noncardiac symptoms related to the primary cause of the pericardial effusion.

Clinical Pearl

Symptomatic pericardial effusion usually involves systemic hypotension, tachycardia, and high systemic venous pressures.

What are the expected echocardiographic findings in a patient with cardiac tamponade?

Echocardiography is the gold standard for diagnosing PCE and detecting tamponade physiology, providing nearly 100% sensitivity. Furthermore, by allowing assessment of the site and characteristics of the effusion, it is a valuable tool to aid in determining and guiding the approach (percutaneous versus open) to drainage of the effusion.

In the case of PCE, echocardiographic images will show a hypoechoic fluid-filled pericardial sac. Effusions may be loculated or circumferential and are graded depending on the size of the hypoechoic space during diastole: small (<10 mm), moderate (10–20 mm), or severe (>20 mm).

In the case of cardiac tamponade, echocardiography will show collapse of cardiac chambers during various points in the cardiac cycle. Right atrial (RA) collapse during systole is the earliest and most sensitive sign of cardiac tamponade physiology, with increased diagnostic accuracy when it persists for more than one-third of the cardiac cycle. Additionally, inspiratory "bounce" of the interventricular septum toward the left ventricle (LV) is commonly observed during diastole. Finally, inferior vena cava (IVC) dilation ("IVC plethora") and respiratory variation of the mitral, aortic, or tricuspid valve inflow peak velocity of >25% may also be seen.

What hemodynamic monitoring findings may be suggestive of symptomatic PCE?

In addition to systemic hypotension and sinus tachycardia, other findings can be suggestive of symptomatic PCE. Abnormal ECG findings, including electrical alternans and arrhythmias, are common. If an arterial catheter is in place, beat-to-beat pulse pressure variation and pulsus paradoxus may be observed. Respiratory variation of the pulse oximetry waveform may be present as well. With central venous pressure monitoring, a diminished *y descent* may also be seen secondary to elevated diastolic filling pressures. Symptomatic PCE is also reflected by elevated RA ("central venous") and left atrial pressures.

It is important to note that PCE can result in decreased coronary perfusion pressures due to the combination of systemic hypotension and elevated LV end-diastolic pressure. Consequently, ST segment changes (either depression or elevation, depending on the degree of myocardial compromise) may also be observed.

What is electrical alternans and what is its significance? What other electrocardiographic findings may exist?

Electrical alternans is the appearance of beat-to-beat amplitude changes of the QRS complex and axis observed in any or all leads on an electrocardiogram (ECG) with no additional conduction changes. The amplitude and axis changes are significant because they represent the position of the heart "shifting" within a full pericardial sac with each contraction.

Other possible ECG findings include sinus tachycardia, low-voltage QRS waveform, ST-T wave changes, and possibly atrial or ventricular ectopy or arrythmias.

Clinical Pearl

Electrical alternans (amplitude changes of the QRS complex and axis) on ECG is suggestive of pericardial effusion.

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What are the cardiopulmonary interactions in symptomatic PCE?

Since the restrictive nature of PCE decreases diastolic filling and, in turn, stroke volume, CO becomes heart rate dependent.

$$CO = SV \times HR$$
$$SV = EDV - ESV$$

where CO = cardiac output; SV = stroke volume; HR = heart rate; EDV = end-diastolic volume; and ESV = end-systolic volume.

However, from these two equations, it may be inferred that another strategy to increase CO involves increasing EDV by increasing venous return and preload. This is important when considering the cardiopulmonary interactions with positive pressure ventilation (PPV), as PPV results in decreased venous return to the right heart and hence reduces CO. The change in intrathoracic pressure (from negative during spontaneous ventilation to positive during mechanical ventilation) decreases preload to a preload-dependent heart with restrictive physiology. For this reason, maintaining spontaneous (negative pressure) ventilation in this situation is of prime importance, particularly in a patient with a hemodynamically significant PCE.

Anesthetic Implications

What past medical history should be sought prior to induction of anesthesia?

The patient's comorbidities should be reviewed, particularly those that could be a contributing factor to formation of the PCE. Any history of recent bacterial or viral respiratory infections, presence of a mediastinal mass, or history of recent cardiac or thoracic surgery are important and help tailor the anesthetic plan accordingly.

Special concerns exist for oncologic patients. Prior exposure to cardiotoxic chemotherapeutic agents (i.e., doxorubicin, daunorubicin, or fluorouracil) is concerning for ensuing cardiomyopathy or QTc prolongation. Additionally, a recent complete blood count with platelets, a coagulation panel to assess hematopoietic effects of chemotherapy, and an electrolyte panel are indicated. If high dose or chronic steroids are part of the oncologic regimen, then stress dose steroids may be warranted as part of the anesthetic plan.

What physical examination findings correlate with symptomatic PCE?

Physical examination findings in symptomatic PCE are reflective of right and/or left-sided cardiac congestion. A complete physical examination is required to determine the degree of cardiac involvement. As previously stated, right upper quadrant pain, abdominal distension, hepatomegaly, peripheral edema, and jugular venous distension are all signs of right-sided failure. Additionally, crackles, diminished breath sounds, intercostal muscle retractions with tachypnea and/or orthopnea, diminished peripheral pulses, and prolonged capillary refill all represent left heart compromise. All of these findings, associated with muffled heart sounds, hypotension, and sinus tachycardia, are supportive of cardiac tamponade.

What other information should be sought on physical examination?

Apart from the aforementioned findings, all indicative of symptomatic PCE, it is important for the anesthesia provider to evaluate any other systems affected by the patient's underlying condition. Additionally, a detailed assessment of the current vascular access lines (size, location, medications being delivered through them, and reliability), current invasive monitoring (presence of arterial and/or central venous catheters) and airway adjuncts or need for supplemental oxygen should be performed prior to going into the operating room or cardiac catheterization laboratory.

What are the fasting considerations for this case?

Fasting status is an important determinant in formulating the anesthetic plan to minimize the risk of pulmonary aspiration. If the patient is hemodynamically stable or the PCE is slowly progressing, the American Society of Anesthesiologists (ASA) preoperative fasting guidelines of 8 hours for a heavy meal, 6 hours for formula or a light meal, 4 hours for breast milk, and 2 hours for clear liquids may be followed. However, if the patient does not meet the preoperative fasting guidelines but is experiencing acute hemodynamic decompensation, one must weigh the risks of proceeding with the case assuming a risk for pulmonary aspiration against delaying the case with the risk of further circulatory collapse. Consideration should be given to performing the case with minimal sedation if possible, as performance of a rapid-sequence induction with initiation of PPV can be potentially life threatening.

What nonpharmacologic strategies are available to deal with separation anxiety?

A patient's developmental maturity and mental status are important factors when formulating a plan for child–parent separation. Most developmentally normal toddlers and

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small children experience extreme stress and fear near the time of separation. To the extent possible, nonpharmacologic interventions should be utilized first: age-appropriate videos or games on smart-devices, toys, and, if available, a child-life specialist or other staff member who can be solely dedicated to the patient's psychological needs.

What pharmacologic options for parental separation could be considered?

If pharmacologic anxiolysis is necessary, it is paramount to have ECG, blood pressure and oxygen saturation monitoring in place. Agents that avoid cardiac depressant effects and have minimal or no sympatholytic effects should be titrated in low doses. Intravenous (IV) midazolam is a reasonable choice and can be titrated in small aliquots (0.05 mg/kg or 0.5 mg boluses) to reach a desirable effect. Dexmedetomidine should be avoided as it predictably causes bradycardia, which can contribute to hemodynamic collapse as CO is primarily dependent on a fast heart rate. If a sedative/anxiolytic is given, exhibit patience while slowly titrating to clinical effect as CO is impaired.

Is it safe to use an existing in situ central venous catheter?

One potential complication of central venous cannulation is intrapericardial placement or migration, which can itself result in PCE. This uncommon yet dangerous complication should be ruled out prior to using an in situ central venous catheter, as further volume administration into the pericardial space will further worsen the clinical scenario. For this reason, it is advisable to carefully review the chart for evidence of difficulty with central venous line placement, as well as any recent chest imaging studies showing the catheter trajectory and tip position.

Is an invasive arterial blood pressure monitor indicated?

The need for invasive blood pressure monitoring depends on the degree of the patient's hemodynamic compromise, as well as on the type of procedure that will be performed. In the case of a symptomatic though hemodynamically stable patient in which a percutaneous ultrasound-guided drainage will be performed with sedation, analgesia, and local anesthesia, an arterial catheter is probably not necessary.

However, in a patient with an acute effusion and significant hemodynamic compromise, or recurrent pericardial effusions in which a pericardial window is planned, an arterial catheter would be useful as it would allow beat-to-beat blood pressure monitoring and rapid recognition of hemodynamic changes both during induction of anesthesia and during the procedure itself. In this instance, multiple providers can be useful to help prepare the patient and expedite the procedure without delaying therapy. If there is no arterial line present, and the patient is in extremis, then it would not be advisable to delay definitive life-saving intervention for the placement of this monitor.

Additionally, placement of an arterial line in a young child without sedation or prior to induction of anesthesia is challenging and delays definitive treatment. In this scenario, other measures of perfusion (distal pulses, peripheral perfusion, capnography, and pulse oximetry waveform) can be closely monitored and treated to avoid delaying drainage of the PCE. Ultimately, the decision to place an arterial line is dependent on the entirety of the clinical scenario and should be made on a case-by-case basis.

What is the preferred anesthetic plan?

The ideal anesthetic plan depends on the patient's current status, as well as the type of drainage planned. When a percutaneous ultrasound-guided drainage will be performed, moderate sedation, analgesia, and local anesthesia should be sufficient and have minimal hemodynamic effects.

With either an acute severe effusion, or a recurrent pericardial effusion in which a pericardial window is planned, it may be beneficial to resuscitate the patient with IV fluids and start vasoconstrictor and inotropic agents prior to beginning the procedure. On the other hand, if the patient is unstable due to significant tamponade physiology and there is no time for preoperative optimization, a "staged" anesthetic and surgical plan can be proposed. While keeping the patient spontaneously breathing, moderate sedation, analgesia, and local anesthesia can be initially administered, allowing percutaneous drainage of the effusion and improving cardiac reserve before proceeding to induction of general anesthesia.

What precautions should be taken prior to initiation of sedation or general anesthesia?

Once in the procedure suite the patient is generally positioned with the head of the bed elevated at 30–45 degrees to alleviate orthopnea. With ECG, blood pressure, and oxygenation monitors in place, and after applying supplementary oxygen, the chest should be prepped and draped, and the procedural/surgical team should be scrubbed and ready to proceed before sedation or anesthesia is induced. Adequate communication and coordination between the various teams is of utmost importance. If sedation is planned, soft hand restraints should be applied to keep the patient from reflexively reaching during the intervention.

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The induction of general anesthesia can precipitate circulatory collapse. For this reason, if general anesthesia is chosen, all ASA monitors should be applied, and emergency medications and airway resuscitation equipment should be ready prior to administration of any anesthetic medications. All team members should be available, including the sonographer and a cardiologist capable of quickly decompressing the PCE. It may be beneficial to increase preload with IV fluids and/or to initiate vasoconstrictor and inotropic agents prior to inducing anesthesia in order to ameliorate hemodynamic effects which can include loss of vascular tone and decreased preload. Additionally, it may be advisable to have packed red blood cells, a surgical team and in some cases availability of extracorporeal membrane oxygenation backup prior to starting the procedure.

What are the preferred sedative agents?

The ideal sedative/anesthetic agent is one that does not cause cardiac depression or decrease sympathetic outflow, while allowing the patient to remain spontaneously breathing. If sedation is the plan of choice, IV midazolam should be titrated (0.05 mg/kg boluses or 0.5 mg boluses), and after injection of local anesthetic, low-dose fentanyl may be given if needed (0.5 mcg/kg, slowly titrated as it may cause bradycardia).

It is crucial to keep in mind that given the low cardiac output state of the patient, the onset of action of the medications administered will be delayed. For this reason, the agents should be slowly titrated in order to avoid oversedation, drastic hemodynamic changes, and the potential need for PPV.

What if sedation is unsuccessful?

In cases in which additional deep sedation/anesthesia is required prior to drainage of the effusion, if sedatives and analgesics have already been administered, IV ketamine (0.2–1 mg/kg, in divided doses) may be cautiously titrated. Caution is indicated as ketamine has direct myocardial depressant properties which may be profound in patients who are catecholamine depleted or at maximal sympathetic drive. In most patients, ketamine will preserve sympathetic tone and spontaneous ventilation. Neuromuscular blockade should be avoided as the patient should be kept spontaneously breathing. If airway obstruction is an issue, placement of a laryngeal mask airway to maintain spontaneous ventilation may be an option.

If it is necessary to progress to a general endotracheal anesthetic, etomidate may be utilized for induction and low-dose sevoflurane may be used for maintenance. At this point, hypotension may be treated with volume expansion or medications as indicated. If necessary, the use of epinephrine, which has both α- and β-receptor effects, is preferred over phenylephrine, which has purely α-receptor effects. To help minimize the effects of PPV, increasing the expiratory time and utilizing minimal or no positive end-expiratory pressure should be considered to maximize venous return.

Clinical Pearl

Positive pressure ventilation will reduce preload and hence CO. Maintenance of spontaneous ventilation is preferred until the PCE has been drained.

What is the primary hemodynamic goal during induction of anesthesia?

The hemodynamic goal during induction of anesthesia is to maintain CO via the following:

- Avoid cardiac depression.
- Maintain sympathetic outflow: preserve vascular tone and an elevated heart rate.
- Avoid decreased preload either due to vasodilation and/or PPV.

Caution is advisable with cardiac depressant medications. Although ketamine preserves sympathetic tone, heart rate, and spontaneous ventilation, it also has direct cardiac depressant properties and can precipitate hemodynamic collapse. Similarly, although opioids have minimal cardiodepressant effects, they can decrease central sympathetic outflow and result in bradycardia. Maintaining normovolemia and spontaneous ventilation can help minimize hemodynamic changes.

Clinical Pearl

The primary hemodynamic goal is to preserve CO by maintaining an elevated heart rate, high preload, and spontaneous ventilation.

How is pericardiocentesis generally performed?

Percutaneous catheter drainage, or pericardiocentesis, is the most common method currently used for drainage of pericardial effusions. This is usually done under echocardiographic or fluoroscopic guidance in the cardiac catheterization laboratory. Echocardiographic imaging prior to starting the drainage allows the proceduralist to approximate the amount of fluid needing to be drained, as well as determining the most accurate location for the puncture site.

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Common access points are apical (needle directed parallel to the LV long axis toward the aortic valve), parasternal (needle inserted 1 cm lateral to the left sternal border; >1 cm lateral will risk injuring the internal thoracic/mammary vessels), or subxiphoid (needle inserted 1 cm inferior to the left xiphoid-costal angle, aiming toward the left mid-clavicle).

Once the pericardial sac is accessed and fluid obtained, the Seldinger technique is utilized to insert a catheter connected to a three-way stopcock; this will be used for drainage, intrapericardial pressure monitoring and reinfusion if indicated. According to the 2015 European Society of Cardiology guidelines, fluid should be drained in small sequential aliquots to prevent acute RV dilation. If the aspirate is nonclotting blood, it can be reinfused directly to the patient to aid with resuscitation (peripheral or central access line connected to pericardial catheter via the three-way stopcock). The procedure is usually continued until the intrapericardial pressure is <5 mm Hg during inspiration. Figure 45.1 shows echocardiographic images of a pericardial effusion pre- and post-pericardiocentesis.

What acute complications can occur during pericardiocentesis?

Pericardiocentesis is considered a safe and effective procedure, with an incidence of major complications of 1.2%–1.6% in experienced hands. However, it is still important to be aware of the possible complications given the high morbidity and mortality associated with them.

During needle insertion, myocardial and coronary artery laceration may cause acute myocardial ischemia, generating ECG ST-segment changes, echocardiographic regional wall motion abnormalities, or a delayed presentation as persistent hemopericardium despite needle/catheter aspiration. Other vascular injuries to the intercostal or internal thoracic/mammary arteries may have a similar delayed presentation. Adequate and prompt communication with the procedural team is paramount as escalation to surgical intervention may be necessary.

Additionally, needle insertion can cause cardiac arrhythmias (supraventricular or ventricular) which usually resolve with retraction or redirection of the needle/catheter. Other potential complications include air embolism, pneumothorax (evidenced by worsening dyspnea), and intraabdominal organ injury, most commonly to the liver, by transperitoneal needle insertion.

The amount of pericardial fluid drained and the rate at which it is removed is also important. As referenced earlier, the 2015 European Society of Cardiology guidelines recommend that fluid should be drained in small sequential aliquots, as up to 25% of patients may have a vasovagal response secondary to pericardial decompression. This is evidenced by a sudden decrease in blood pressure and heart rate associated with a "fainting" sensation referred to by the patient (if conscious). Additionally, rapid pericardial decompression may cause acute left or right ventricular dilation and subsequent dysfunction, referred to as "pericardial decompression syndrome" (PDS). This has been reported in up to 5% of patients, and results in worsening hemodynamic instability, as well as pulmonary edema, following drainage. The best way to avoid PDS is to limit total initial drainage to <500 mL, continuing to drain the remaining fluid during the next

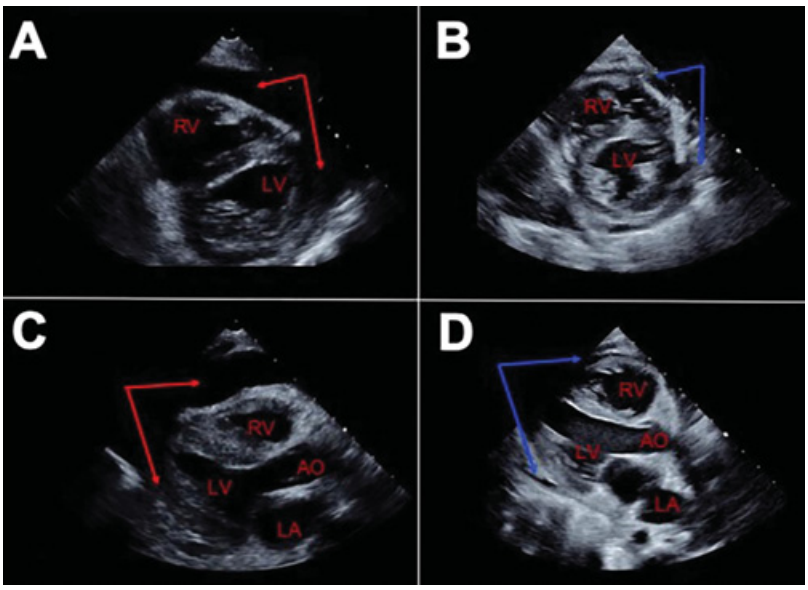

Figure 45.1 Echocardiographic images before and after pericardiocentesis. (A) Parasternal short axis view with large pericardial effusion (red arrows). (B) Parasternal short axis view post-pericardiocentesis showing resolution of pericardial effusion (blue arrows). (C) Parasternal long axis view with large pericardial effusion (red arrows). (D) Parasternal long axis view post-pericardiocentesis showing resolution of pericardial effusion (blue arrows). AO, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

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24–48 hours by leaving the catheter in place. The treatment for PDS is mainly supportive.

What additional perioperative complications need to be considered?

Other complications that may present in the postoperative period include the following:

- Cardiopulmonary edema secondary to aggressive fluid resuscitation, potentially worsened by the potential occurrence of PDS
- Pericardial decompression syndrome may manifest hours after the procedure
- Pneumopericardium secondary to a pleuro-pericardial fistula, causing tamponade physiology without pericardial effusion
- Persistent PCE, due to loculated effusions which may have been only partially drained
- Hemopericardium secondary to an initially "silent" vessel or myocardial injury
- Pericardial drain occlusion
- Infection

With this in mind, the postoperative disposition of these patients should be to a critical care unit.

Suggested Reading

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