

# Alveolar recruitment manoeuvre is safe in children prone to pulmonary hypertensive crises following open heart surgery: a pilot study

Erica de Freitas Amorim<sup>a</sup>, Viviane Assuncao Guimaraes<sup>a</sup>, Fabio Carmona<sup>b</sup>,  
Ana Paula de Carvalho Panzeri Carlotti<sup>b</sup>, Paulo Henrique Manso<sup>b</sup>, Cesar Augusto Ferreira<sup>a</sup>,  
Jyrson Guilherme Klamt<sup>c</sup> and Walter Villela de Andrade Vicente<sup>a,\*</sup>

<sup>a</sup> Divisions of Thoracic and Cardiovascular Surgery, Hospital das Clinicas of Ribeirao Preto Medical School, University of São Paulo, São Paulo, Brazil

<sup>b</sup> Pediatric Intensive Care, Hospital das Clinicas of Ribeirao Preto Medical School, University of São Paulo, São Paulo, Brazil

<sup>c</sup> Department of Anesthesia, Hospital das Clinicas of Ribeirao Preto Medical School, University of São Paulo, São Paulo, Brazil

\* Corresponding author. Division of Thoracic and Cardiovascular Surgery, Ribeirao Preto Medical School, University of São Paulo, 1000, Bandeirantes Avenue, Monte Alegre District, 14048-900 Ribeirao Preto, São Paulo, Brazil. Tel: +55-16-36022497; fax: +55-16-36022497; e-mail: wvcent@fmrp.usp.br (W.V. de Andrade Vicente).

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## Abstract

**OBJECTIVES:** To test the tolerance and safety of an alveolar recruitment manoeuvre performed in the immediate postoperative period of corrective open heart surgery in children with congenital heart disease associated with excessive pulmonary blood flow and pulmonary arterial hypertension due to left-to-right shunt.

**METHODS:** Ten infants aged 1–24 months with congenital heart disease associated with excessive pulmonary blood flow and pulmonary artery hypertension (mean pulmonary artery pressure  $\geq 25$  mmHg) were evaluated. The alveolar recruitment manoeuvre was performed in the operating theatre right after skin closure, and consisted of three successive stages of 30 s each, intercalated by a 1-min interval of baseline ventilation. Positive end-expiratory pressure was set to 10 cmH<sub>2</sub>O in the first stage and to 15 cmH<sub>2</sub>O in the two last ones, while the peak inspiratory pressure was kept at to 30 cmH<sub>2</sub>O in the first stage and at 35 cmH<sub>2</sub>O in the latter ones. Haemodynamic and respiratory variables were recorded.

**RESULTS:** There was a slight but significant increase in mean pulmonary artery pressure from baseline to Stage 3 ( $P = 0.0009$ ), as well as between Stages 1 and 2 ( $P = 0.0001$ ), and 1 and 3 ( $P = 0.001$ ), with no significant difference between Stages 2 and 3 ( $P = 0.06$ ). Upon completion of the third stage, there were significant increases in arterial haemoglobin saturation as measured by pulse oximetry ( $P = 0.0009$ ), arterial blood partial pressure of oxygen ( $P = 0.04$ ), venous blood oxygen saturation of haemoglobin ( $P = 0.03$ ) and arterial oxygen partial pressure over inspired oxygen fraction ratio ( $P = 0.04$ ). A significant reduction in arterial blood partial pressure of carbon dioxide ( $P = 0.01$ ) and in end tidal carbon dioxide also occurred ( $P = 0.009$ ). The manoeuvre was well tolerated and besides a slight and transitory elevation in mean pulmonary artery, no other adverse haemodynamic or ventilatory effect was elicited.

**CONCLUSIONS:** The alveolar recruitment manoeuvre seemed to be safe and well tolerated immediately after open heart surgery in infants liable to pulmonary hypertensive crises.

**Keywords:** Open heart surgery • Congenital heart disease • Pulmonary arterial hypertension • Alveolar recruitment manoeuvre • Pulmonary hypertensive crises

## INTRODUCTION

Lung function impairment is commonly observed in the early postoperative period of open heart surgery for congenital heart diseases (CHD) associated with excessive pulmonary blood flow and pulmonary arterial hypertension (PAH). A combination of several trigger factors, including general anaesthesia [1], surgical trauma [2], cardiopulmonary bypass (CPB) [3, 4], pre-existing increased pulmonary vascular resistance (PVR), atelectasis and

hypoxaemia [5], has been implicated as a predisposing mechanism for postoperative lung dysfunction severe enough to preclude weaning from mechanical ventilation [6].

In this context, the application of an alveolar recruitment manoeuvre (ARM) could be useful due to its direct re-expanding effect on collapsed alveoli, thus improving functional residual capacity and alveolar oxygenation [7]. Unfortunately, the intrathoracic pressure elevation determined by the ARM may temporarily diminish both the cardiac output and the mean arterial pressure (MAP)

while simultaneously increasing the mean pulmonary artery pressure (MPAP) [8] and the PVR. These two adverse, albeit usually transient, haemodynamic effects are secondary to the direct functional alveoli-capillary blockage determined by the ARM [9].

For these reasons, a deterrent hypothesis that ARM might carry an unacceptable risk of inducing pulmonary hypertensive crises in children with CHD associated with excessive pulmonary blood flow and PAH was elaborated and has been consequently deterring the clinical investigation of ARM in these patients [6, 7]. Indeed, the ARM has been tested almost exclusively in adults [10], while investigations in children were virtually restricted to those with acute lung injury and acute respiratory distress syndrome [11]. So far, only one study [12] evaluated the ARM in the immediate postoperative period of paediatric patients undergoing cardiac surgery for CHD and provided strong evidence that an ARM based on a positive end-expiratory pressure (PEEP) of 8 cmH<sub>2</sub>O improves the end-expiratory lung volume, the dynamic lung compliance and oxygenation, and reduces the ventilation perfusion mismatch. In this study, however, case mix was a major confounding factor.

The aim of the present pilot study was to evaluate the clinical tolerance and benefit of an ARM protocol applied to a homogeneous group of children undergoing open repair of CHD associated with excessive pulmonary blood flow and PAH.

## MATERIALS AND METHODS

This is a pilot, prospective, non-controlled interventional study. After approval from the institutional review board and obtaining parental or legal guardian written informed consent, 10 children (6 males, aged 1–24 months) with congenital heart defects associated with excessive pulmonary blood flow and PAH due to left-to-right shunt who were submitted to open elective cardiac surgery under CPB were prospectively studied. The inclusion criteria included elective surgery, no previous cardiac operation, PAH (MPAP  $\geq$  25 mmHg) determined by echocardiography and/or angiocardiography, and confirmed intraoperatively by direct main pulmonary artery puncture right after opening the pericardium, before any other surgical manipulation.

The exclusion criteria were concomitant pulmonary disease other than PAH, postoperative haemodynamic instability (heart rate [HR]  $<$ 90 or  $>$ 160 bpm), cardiac pacing, cardiac arrhythmias, bronchospasm and inability to measure MPAP during ARM.

Before the induction of anaesthesia, all the patients were monitored with a five-lead, two-channel electrocardiogram, non-invasive blood pressure measurement and pulse oximetry. Following the insertion of a peripheral venous line, general anaesthesia was induced with intravenous midazolam<sup>®</sup> 0.1–0.3 mg/kg, fentanyl<sup>®</sup> 10  $\mu$ g/kg and vecuronium<sup>®</sup> 0.2 mg/kg. Anaesthesia was maintained with fentanyl 5  $\mu$ g/g/h, midazolam 0.1 mg/g/h and inhaled 0 a 1% isoflurane.

The patients were orally intubated with a cuffed ( $>$ 3 months old) or uncuffed ( $\leq$ 3 months old) tracheal tube. When an uncuffed cannula was employed, no detectable air leak was ensured in order not to compromise the ARM. The children were pressure controlled ventilated (Nikkei mechanical ventilator, Takaoka Co., São Paulo, Brazil) with a tidal volume (TV) of 7–10 ml/kg, a PEEP of 5 cmH<sub>2</sub>O and an inspiration/expiration ratio (I:E) of 1:2. The respiratory rate (RR) was adjusted to maintain the arterial blood partial pressure of carbon dioxide (PaCO<sub>2</sub>) between 35 and 45 mmHg. On line capnography (Dixtal 7100 monitor, São Paulo,

Brazil) was routinely employed. Catheters were placed in the radial or femoral artery and in the internal jugular vein. A Foley bladder catheter and a nasopharyngeal probe were inserted. Vital signs were continuously monitored (HP 1166A, Hewlett-Packard, Palo Alto, CA, USA).

The operations were performed through median sternotomy with the patients in supine position with the neck extended by a soft roll. PAH was confirmed intraoperatively by direct puncture of the main pulmonary artery right after the pericardiectomy and pericardial cradling were performed. CPB was carried out at a nasopharyngeal temperature of 28°C. Aortic cross-clamping, intermittent antegrade blood hyperkalemic cardioplegia and pericardial ice sludge were employed. During CPB, ventilation was discontinued and the endotracheal tube was opened to atmosphere. Towards the end of the cardiac repair two catheters (BD Intracath 785901, 1.1 mm, Becton-Dickinson, NJ, USA) were inserted through the right atrium free wall. One of them was positioned into the pulmonary artery across the tricuspid valve while the other was introduced into the left atrium through the *foramen ovale*.

After rewarming, the lungs were re-expanded with three to five hand-bagging insufflations at a line pop-off pressure of 40 cmH<sub>2</sub>O, mechanical ventilation was resumed at the same settings as before CPB and the patient was weaned from CPB.

The ARM protocol was started when the skin closure was completed and before surgical dressing was applied, with the child haemodynamically stable, in sinus rhythm, under usual doses of milrinone<sup>®</sup> (0.375–0.75  $\mu$ g/kg/min) and norepinephrine (0.02  $\mu$ g/kg/min) according to our weaning protocol.

The ARM was performed by a respiratory therapist (E.F.A.) under the supervision of both the anaesthesiologist and the surgeon. Before it was initiated, ventilation was set to a baseline protocol for 10 min (an RR of 25 ipm, an inspired oxygen fraction (F<sub>i</sub>O<sub>2</sub>) of 100%, a PEEP of 5 cmH<sub>2</sub>O, an I:E ratio of 1:1.5 and a peak inspiratory pressure (PIP) to deliver a TV of 7–10 ml/kg).

The ARM protocol consisted of three successive stages of 30 s duration each. In Stage 1, the PIP and PEEP values were increased to 30 and 10 cmH<sub>2</sub>O, respectively. Stages 2 and 3 had both identical PIP and PEEP values of 35 and 15 cmH<sub>2</sub>O, respectively. A 1 min interstage interval was respected, during which baseline ventilation variables were resumed. Vital signs were closely screened during the ARM, and the MPAP was registered at the end of each stage.

Stage 3 and baseline, pre-ARM, arterial haemoglobin saturation as measured by pulse oximetry (SpO<sub>2</sub>), end tidal carbon dioxide pressure (ETCO<sub>2</sub>), HR, MAP, central venous pressure (CVP), left atrial pressure (LAP) and MPAP were compared. In these two moments, arterial and mixed venous blood samples were sent for blood gas analyses. The arterial oxygen partial pressure over inspired oxygen fraction ratio (PaO<sub>2</sub>/FiO<sub>2</sub>) for those two moments was calculated.

Red flags to promptly interrupt the ARM were tachy (HR  $>$  160 bpm) or bradycardia (HR  $<$  90 bpm), loss of sinus rhythm, MPAP rise over two-thirds of the MAP and any decrease in SpO<sub>2</sub> and/or ETCO<sub>2</sub>.

## Statistical analysis

Results are presented as mean ( $\pm$ SD) or median (and range). Comparisons between stage three and pre-ARM baseline values were carried out with the paired Student's *t*-test, while repeated-measures analysis of variance (ANOVA) with Bonferroni's *post hoc* test was employed for interstage and stage/baseline comparisons.

The level of significance was set at 5%. Statistical software packages SAS (version 9.1.3, SAS Institute, Inc., Cary, NC, USA) and Graphpad Prism (Graphpad Software, version 5.0, La Jolla, CA, USA) were used.

## RESULTS

Table 1 depicts the demographic and intraoperative data. The effects of the ARM on respiratory and haemodynamic variables are given in Table 2.

There was a significant increase in the MPAP after both the second and the third ARM stages vs baseline and first-stage values. Furthermore, there was a significant decrease in MPAP after the ARM vs the third stage. The behaviour of the MPAP before, during and after the ARM protocol is depicted in Fig. 1, and ANOVA results are given in Table 3. The ARM protocol was well tolerated by all the children and did not significantly impair any haemodynamic or respiratory variables whether during or after its application, until admission to the Pediatric Intensive Care Unit (PICU). It was never necessary to interrupt the ARM, and no PAH crisis was detected.

Pleural cavity was left intact in 5 (50%) patients. All the patients had chest radiographs taken upon PICU arrival and all had their lungs well expanded and homogeneous, with no signs of atelectasis or pneumothorax. The patients were kept on mechanical ventilation for a median of 23 h (range of 5–192 h).

## DISCUSSION

The present study represents the first prospective pilot study of an ARM in the immediate postoperative period of open heart surgery in children presenting with CHD and PAH secondary to excessive pulmonary blood flow due to left-to-right shunt.

It is well established that open heart surgery for the repair of CHD associated with PAH and excessive pulmonary blood flow may be complicated by various degrees of pulmonary dysfunction generated, among other factors, by the development of atelectasis

in up to 80% of the patients [6]. Widespread atelectasis might determine an acute increase in PVR and trigger pulmonary hypertensive crises, risking death from acute right ventricle failure [13].

Manoeuvres and strategies of ventilatory support capable of preventing or reverse atelectasis are consequently indicated in order to improve hypoxaemia and lower the PVR [14]. Currently, the postoperative application of an ARM is deemed clinically useful and potentially capable in reducing the duration of mechanical ventilation although, so far, no survival benefit could be demonstrated. Curiously, the more efficient and less aggressive ARM is still a matter of controversy, and also, benefit evidences in children are missing [7]. Moreover, there are still limitations in terms of invasive haemodynamic monitoring in the paediatric population, hindering the establishment of the ARM in clinical practice.

As patients with PAH are considered at a higher risk of developing postoperative pulmonary hypertensive crises [13], and given the putative ARM adverse effects, this study was performed in the operating theatre, with the patients still under general anaesthesia and paralyzed, thus avoiding any discomfort from the manoeuvre, as suggested by Boriosi *et al.* [11]. Besides, the ARM was applied under stable haemodynamic and respiratory conditions and under respiratory and invasive haemodynamic monitoring. In addition, although the chest was already closed, the surgical field remained sterile, in case a circulatory or ventilatory deterioration demanded immediate chest reopening.

The patient age range was chosen to avoid the inclusion of neonates as well as children older than 2 years, once these two groups are considered at a higher risk of lung complications when presenting with elevated PVR [15]. Furthermore, as it is known that the haemodynamic response to ARM is directly related to the increase in PIP [16], our ARM protocol included intermittent PIP, as proposed by Celebi *et al.* [7], that is, the ARM was split into three successive stages to allow a short interval of interstage baseline ventilation aimed at reversing of any eventual PVR elevation secondary to ARM itself. As another safety measure, the Stage 1 ARM had lower pressures and the additive alveolar recruitment may occur during both the inspiratory and expiratory phases.

**Table 1:** Demographic and intraoperative data

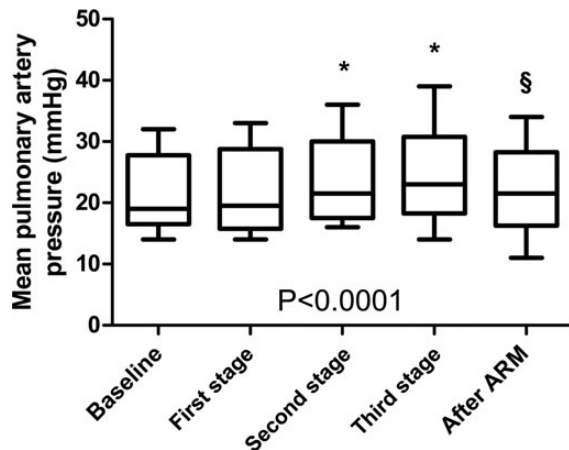
Age (months)	Sex	Weight (kg)	Height (cm)	Diagnoses	Operation	CPB (min)	X-clamp time (min)
1	M	3.4	54	TGA/ASD/VSD*	ASO	175	120
1	M	4	57	TGA/ASD/VSD*	ASO	165	113
1	F	4.1	45	TAPVC, supracardiac/PDA	Anastomosis of collecting chamber with LA, vertical vein ligation; ASD closure, PDA ligation	81	63
2	F	2.7	47	VSD	VSD closure	65	40
2	M	4.4	58	<i>Truncus arteriosus</i> (type I)	VSD closure, RV-PA conduit	165	115
3	M	5	59	VSD	VSD closure	45	30
6	F	4.4	56	CAVSD/PDA	VSD closure, ASD closure, PDA ligation, AV valve plasty	95	77
9	F	7	65	VSD	VSD closure	45	22
20	M	7	83	TAPVC, supracardiac	Anastomosis of collecting chamber with LA, vertical vein ligation, ASD closure	125	92
24	M	10.3	82	VSD	VSD closure	50	35

M: male; F: female; TGA: transposition of the great arteries; ASD: atrial septal defect; VSD: ventricular septal defect; TAPVC: total anomalous pulmonary venous connection; PDA: patent *ductus arteriosus*; CAVSD: complete atrioventricular septal defect; CPB: cardiopulmonary bypass; X-clamp time: aortic cross-clamp time; ASO: arterial switch operation; RV-PA: right ventricle to pulmonary artery; AV: atrioventricular; LA: left atrium.

**Table 2:** Respiratory and haemodynamic variables obtained before and after the third stage of the alveolar recruitment manoeuvre

Variable	Mean ( $\pm$ SD)		P-value
	Before ARM	After ARM	
SaO <sub>2</sub> (%)	94.9 $\pm$ 9.3	97.9 $\pm$ 4.9	0.31
SpO <sub>2</sub> (%)	96.5 $\pm$ 4.3	98.5 $\pm$ 2.7	0.009
SvO <sub>2</sub> (%)	76.7 $\pm$ 18.5	82.4 $\pm$ 2.0	0.03
PaO <sub>2</sub> /FiO <sub>2</sub>	123 $\pm$ 223.7	273.9 $\pm$ 117.4	0.04
PaO <sub>2</sub> (mmHg)	183.7 $\pm$ 107.2	232.9 $\pm$ 118.6	0.04
ETCO <sub>2</sub> (mmHg)	36.3 $\pm$ 10.2	30.9 $\pm$ 10	0.0009
PaCO <sub>2</sub> (mmHg)	43.7 $\pm$ 6.2	37 $\pm$ 9	0.01
RAP (mmHg)	10.8 $\pm$ 3.2	10.1 $\pm$ 2.5	0.39
LAP (mmHg)	10.6 $\pm$ 3.2	9.9 $\pm$ 2.5	0.54
HR (bpm)	137.6 $\pm$ 19.5	141.6 $\pm$ 18.7	0.38
PAP (mmHg)	21.3 $\pm$ 6.4	22.2 $\pm$ 7.1	0.29
MAP (mmHg)	50.2 $\pm$ 10.9	54.9 $\pm$ 13.5	0.21

SD: standard deviation; ARM: alveolar recruitment manoeuvre; SaO<sub>2</sub>: arterial blood oxygen saturation of haemoglobin; SpO<sub>2</sub>: arterial haemoglobin saturation as measured by pulse oximetry; SvO<sub>2</sub>: venous blood oxygen saturation of haemoglobin; PaO<sub>2</sub>/FiO<sub>2</sub>: arterial oxygen partial pressure over inspired oxygen fraction ratio; PaO<sub>2</sub>: arterial blood partial pressure of oxygen; ETCO<sub>2</sub>: end tidal carbon dioxide partial pressure; PaCO<sub>2</sub>: arterial blood partial pressure of carbon dioxide; RAP: right atrial pressure; LAP: left atrial pressure; HR: heart rate; PAP: pulmonary artery pressure; MAP: mean arterial pressure.



**Figure 1:** The mean pulmonary artery pressure behaviour before, during and after the alveolar recruitment manoeuvre (ARM). \*significantly different from both baseline and first-stage values; \$: significantly different from third-stage values.

There are few studies with similar methodology in the paediatric population in the literature. Scohy *et al.* [12] tested an ARM in the immediate postoperative period of open heart surgery in 20 children with several different CHD. A preoperative diagnosis of PAH was not mentioned, and the investigation took place in the PICU. The ARM consisted of five consecutive respiratory cycles, under a 1:1 I:E ratio, with a higher PIP (40 cmH<sub>2</sub>O) and lower PEEP (8 cmH<sub>2</sub>O) when compared with our study. The pulmonary gas exchange, dynamic compliance of the respiratory system and end-expiratory lung volume all improved.

Unlike Halbertsma and Hoeven [17] and Scohy *et al.* [12], who tried to obtain alveolar recruitment with a PIP of up to 40 cmH<sub>2</sub>O,

**Table 3:** Interstage MPAP comparisons

MPAP comparisons	Mean difference	95% CI difference
Baseline vs first stage	-0.5	-2.390 to 1.390
Baseline vs second stage	-2.5	-4.390 to -0.6097 <sup>a</sup>
Baseline vs third stage	-3.5	-5.390 to -1.610 <sup>a</sup>
Baseline vs after ARM	-0.9	-2.790 to 0.9903
First stage vs second stage	-2.0	-3.890 to -0.1097 <sup>a</sup>
First stage vs third stage	-3.0	-4.890 to -1.110 <sup>a</sup>
First stage vs after ARM	-0.4	-2.290 to 1.490
Second stage vs third stage	-1.0	-2.890 to 0.8903
Second stage vs after ARM	1.6	-0.2903 to 3.490
Third stage vs after ARM	2.6	0.7097-4.490 <sup>a</sup>

<sup>a</sup>ANOVA with Bonferroni's *post hoc* test,  $P < 0.0001$ .

MPAP: mean pulmonary artery pressure; CI: confidence interval; ARM: alveolar recruitment manoeuvre.

in the present study the PIP was limited to 35 cmH<sub>2</sub>O in order to prevent lung barotrauma and reduce the transitory alveolo-capillary blockage [15, 18].

In our study, the significant increase in MPAP between Stages 1 and 2, and 1 and 3 probably resulted from pulmonary microvasculature cross-sectional area compressive reduction determined by the alveolar distension, all hallmark of the ARM [19, 20]. The similarity of the MPAP values between Stages 2 and 3 was expected to occur once they both shared the same PIP and PEEP. The return of MPAP to baseline upon completion of the ARM third stage demonstrates that the ARM did not cause any permanent alteration in PVR.

The lack of significant changes in blood pressure, HR, LAP and CVP can be related to the short duration of the ARM as well as to the inclusion of interstage intervals in the study protocol. Accordingly, Scohy *et al.* [12] also found no significant changes in HR, MAP and CVP in their investigation.

The effectiveness of the manoeuvre in terms of improvement in arterial oxygenation and lung gas exchange was characterized by the significant increase in the PaO<sub>2</sub>, a well as in the PaO<sub>2</sub>/FiO<sub>2</sub> ratio and in the SvO<sub>2</sub> concomitantly with a significant decrease in ETCO<sub>2</sub> and PaCO<sub>2</sub>. As SvO<sub>2</sub> is considered an important indicator of tissue oxygenation and adequate cardiac output [21], it seems that our ARM protocol did not adversely affect the ventilatory/haemodynamic interplay.

The aforementioned results are clinically relevant and may reflect that the re-expansion of collapsed alveolar unit could have increased the gas exchange alveolar surface area thus improving the alveolar ventilation and reducing the pulmonary shunt [4, 12, 22].

This study has some limitations. First, as this study was confined to the very short period encompassing the ARM, it remains to be determined for how long the ventilatory benefits may extend over the postoperative period, since different mechanisms of alveolar collapse may ensue [10, 17, 22]. Secondly, due to logistic restraints, our investigation protocol did not include more sophisticated methods that can directly document lung re-expansion and alveolar recruitment [12]. Finally, although children with complex CHD were included in this research, the ARM was not prolonged and the patient cohort was small. For these reasons, a larger and more extended investigation is necessary to confirm ARM efficacy and safety in similar patients. A new study should include a control group to evaluate the duration of mechanical ventilation, the

incidence of pneumothorax, length of oxygen use and perhaps include chest computed tomography after 1 month to check whether high pressures may lead to a chronic pulmonary disease or ventilator-induced lung injury.

In conclusion, the ARM protocol tested in this study was safe. Although a transient and minor MPAP elevation was noticed, no other adverse clinical effect was detected.

**Conflict of interest:** none declared.

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