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Impact of Patent Ductus Arteriosus Closure on Linear Growth and Weight Gain in Children: A Prospective Cohort Study

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ABSTRACT

Background: Patent ductus arteriosus (PDA) can cause pulmonary circulatory volume overload and hemodynamic disturbances that result in feeding difficulties, recurrent infections, and growth retardation. PDA closure intervention is expected to improve physiological conditions, thereby supporting improvements in linear growth and weight gain.

Subject and Methods: This prospective cohort study was conducted at Dr. Moewardi General Hospital, Surakarta, Central Java, Indonesia, from January 2024 to July 2025. The target population was children diagnosed with patent ductus arteriosus (PDA). Subject was all of pediatric PDA patients at Dr. Moewardi General Hospital Surakarta who underwent PDA closure procedures (total sampling). Subjects with complete anthropometric data such as body weight and height at PDA pre-procedure, with 3 and 6 months after PDA procedure (post-procedure) were selected. The dependent variables were changes in height (cm) and body weight (kg), measured using standard anthropometric methods. Data were analyzed using the Friedman test for repeated measures, followed by paired Wilcoxon tests with Bonferroni correction for post-hoc comparisons.

Results: Total there were 48 children who underwent PDA closure with complete anthropometric data at PDA pre- and 3 to 6 months post-procedure. Mean height significantly increased 4.5% from 83.60 (SD= 26.23) at PDA pre-procedure to 87.34 cm (SD= 24.87) at 3 months post-procedure, and increased 8.1% to 90.39 cm (SD= 23.64) at 6 months post-procedure ($p<0.001$). Mean body weight also significantly increased 9.3% from 11.49 kg (SD= 12.21) at PDA pre-procedure to 12.56 (SD= 11.93) at 3 months post-procedure then increased 16.1% to 13.34 (SD= 11.88) at 6 months post-procedure ($p<0.001$). The height and weight of children with PDA closure increased over the observation period.

Conclusion: PDA closure is associated with a significant increase in linear growth and weight gain up to 6 months post-procedure.

Keywords: Patent ductus arteriosus; PDA closure; linear growth; body weight; prospective cohort

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BACKGROUND

Patent ductus arteriosus (PDA) is a congenital heart defect that can cause a left-to-right shunt, pulmonary over-circulation, and left heart volume overload. Its clinical consequences can include tachypnea, easy fatigability during breastfeeding or eating, impaired intake tolerance, and growth retardation. Growth retardation in children with congenital heart defects occurs through multifactorial mechanisms, including increased energy requirements, decreased intake due to fatigue or dyspnea, fluid restriction, impaired absorption, and systemic inflammation affecting metabolism (Herridge et al., 2021; Lee et al., 2025; Salvatori et al., 2022).

The incidence of PDA varies by gestational age, occurring in at least 10% of congenital heart disorders (Gillam-Krakauer & Mahajan, 2023). In a study of 11,845 neonatus admitted to Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) South Africa from January 2013 to December 2020 reported that PDA occurred in 4.52% of the population (Shulman et al., 2023).

Research at RSCM Jakarta in August to October 2003 reported the incidence of PDA in premature infants was 14% (Deselina et al., 2004). Research at Prof. Dr. R.D. Kandou Manado General Hospital in September 2022-August 2023 reported PDA of 11.7% (Baksh et al., 2024). In the Taiwanese population from 2005-2014, the prevalence of PDA was reported to be 1 in 2000 births in full-term pregnancies, and 20-60% in preterm neonates (Hsu et al., 2021). A study at Yamanashi Prefectural Central Hospital Japan reported of PDA cases in preterm infants as much as 42.4% (Kikuchi et al., 2024).

The ductus that fails to close allowing blood to flow from the high-pressure descending aorta to the low-pressure pulmonary artery (pulmonary overflow). That condition leads pulmonary edema, reduced lung compliance and respiratory failure, also systemic hypoperfusion. This hemodynamic disruption is especially in preterm infants due to an immaure cardiovascular system (Backes et al., 2022). A hemodinamically significant PDA causes nutritional disruption primarily by stealing blood flow from the sytemic circulation, especially the mesenteric arteries, leading to intestinal ischemia and poor nutrient absorption (Babla et al., 2021), that's why children with PDA needs enteral nutrition higher than children without PDA (Kikuchi et al., 2024).

The chronic reduction in systemic flow also often leads to a rise in serum lactate and persistent acidosis or unexplained metabolic acidosis. PDA cause a decline in overal energy metabolism such as decreased levels of ATP, oxygen, and glucose which prevents the ductus itself from remodelling (Hsu et al., 2021). Failure to close a significant PDA is often linked to chronic malnutrition, stunting, and reduced head growth due to persistent hemodynamic consequences (Mullaly et al., 2025).

The risk of malnutrition in children with PDA will affect the level of morbidity and mortality, so that PDA closure efforts are needed (Hartaty et al., 2016). PDA closure is expected to improve hemodynamics, reduce volume load and pulmonary over-circulation, and ultimately improve nutritional status and growth. Several center reports indicate that percutaneous PDA closure yields good short-term outcomes in pediatric populations with varying body weights (Suwitri et al., 2020).

Service-based prospective data in Indonesia monitoring changes in height and weight up to 6 months post-closure remain limited. This study aims to assess changes in linear growth and body weight in children

following PDA closure at Dr. Moewardi General Hospital.

SUBJECTS AND METHOD

1. Study Design

This study used a prospective cohort design conducted at Dr. Moewardi General Hospital from January 2024 to July 2025.

2. Population and Sample

The target population was children with patent ductus arteriosus (PDA). The source (accessible) population consisted of children aged 0-18 years diagnosed with PDA who underwent a PDA closure procedure at the study site. The sampling technique was total sampling of eligible subjects who had complete anthropometric data at three measurement time points: pre-procedure, 3 months, and 6 months post-procedure. The sample size included all subjects meeting these criteria during the study period. A search of medical records during the study period revealed 49 pediatric PDA cases undergoing PDA closure. Of these, one subject was excluded from the analysis due to lack of anthropometric data at 6 months post-procedure, resulting in a total of 48 subjects.

3. Study Variables

The dependent variables were height (cm) and body weight (kg). The independent variable was time of measurement (pre-procedure, 3 months post-procedure, and 6 months post-procedure).

4. Operational Definition of Variables

Height was defined as the child's body length/height measured in centimeters using a calibrated measuring instrument according to hospital standards. Body weight was defined as the child's weight measured in kilograms using a calibrated scale. Measurements were taken at three time points: before the PDA closure procedure (baseline), 3 months after the procedure, and 6 months after the procedure. Data were presented as mean ± standard deviation. Differences among the three repeated measurements were analyzed using the Friedman test. Post-hoc analysis between paired time points (pre vs 3 months, pre vs 6 months, and 3 vs 6 months) was performed using the paired Wilcoxon test with Bonferroni correction ($\alpha = 0.05/3$).

5. Study Instruments

Anthropometric data were collected using standardized and calibrated instruments. Body weight was measured using a digital weighing scale, while height was measured using a stadiometer, both in accordance with hospital service standards at Dr. Moewardi General Hospital. Data on patient diagnosis, procedure, and measurement time points were obtained from medical records.

6. Data analysis

Data were presented as mean ± standard deviation. Differences in height and body weight across three repeated measurements (pre-procedure, 3 months, and 6 months post-procedure) were analyzed using the Friedman test. Post-hoc pairwise comparisons (pre vs 3 months, pre vs 6 months, and 3 vs 6 months) were conducted using the paired Wilcoxon test with Bonferroni correction ($\alpha = 0.05/3$).

7. Research Ethics

This study involved human participants and adhered to ethical principles in medical research. Ethical clearance was obtained from the authorized Health Research Ethics Committee of Dr. Moewardi General Hospital. All data were handled confidentially, and patient anonymity was maintained throughout the study.

RESULTS

1. Sample Characteristics

There were 49 children who underwent PDA closure during the study period, and

48 children had complete anthropometric data up to 6 months (1 child incomplete at 6-month evaluation). Subject characteristics (n=48, complete data): median age at procedure 13.0 months (IQR 9.9–53.5), range 1.1–190.0 months; female 32 (66.7%); median PDA diameter (n=45) 4 (IQR 2–5), range 0.3–10; most frequent comorbidities (may overlap) included ASD (≈22,9%), PAH/pulmonary hypertension (≈20,8%), Down syndrome (≈12,5%), VSD (≈10,4%), dan hypothyroidism (≈8,3%). (see Table 1).

Table 1. Baseline characteristics of subjects (n=48, complete data)

Characteristics	Category	Frequency	Percentage
Sex	Male	16	33.3
	Female	32	66.7
Age at procedure (months)	Median (IQR)	13.0 (9.9–53.5)	-
PDA diameter (mm)	Median (IQR)	4.0 (2.0–5.0)	-
Comorbidities	ASD	11	22.9
	PAH	10	20.8
	Down syndrome	6	12.5
	VSD	5	10.4
	Hypothyroidism	4	8.3

Note: ASD = atrial septal defect, PAH = pulmonary arterial hypertension, VSD = ventricular septal defect

2. Bivariate Analysis

Mean height increased from 83.60 cm (SD= 26.23) (pre) to 87.34 (SD= 24.87) (3 months) and 90.39 (SD= 23.64) (6 months). The Friedman test indicated a significant difference (p<0,001). Mean body weight increased from 11.49 kg (SD= 12.21) (pre) to 12.56 kg (SD= 11.93) (3 months) and 13.34 kg (SD= 11.88) (6 months). The Friedman test indicated a significant difference (p<0,001). Further analysis of the comparison of anthropometric data between two observation points using the Wilcoxon rank test obtained significant results for both height and weight. Height and weight increased over the observation period. Mean height significantly increased 4.5% from 83.60 cm (SD= 26.23) at PDA pre-procedure to 87.34 (SD= 24.87) at 3 months post-procedure, and increased 8.1% to 90.39 (SD= 23.64) at 6 months post-procedure. Mean body weight significantly increased 9.3% from 11.49 kg (SD= 12.21) at PDA pre-procedure to 12.56 kg (SD= 11.93) at 3 months post-procedure then increase 16.1% to 13.34 kg (SD= 11.88) at 6 months post-procedure (see Table 2).

Table 2. Anthropometric changes pre, 3 months, and 6 months (Friedman test)

Independent Variables	Pre-procedure		3 Months		6 Months		p
	Mean	SD	Mean	SD	Mean	SD	
Height (cm)	83.60	26.23	87.34	24.87	90.39	23.64	<0.001
Body weight (kg)	11.49	12.21	12.56	11.93	13.34	11.88	<0.001

DISCUSSION

In significant PDA, pulmonary over-circulation and volume load can lead to tachypnea, increased work of breathing, easy fatigability during feeding, and a risk of recurrent respiratory infections; these conditions reduce energy intake and increase energy requirements. Following hemodynamic improvement, energy requirements may decrease and intake may improve, such that body weight generally improves more rapidly. However, improvement in linear height often requires a longer duration as it relates to chronic deficits and endocrine–metabolic adaptation (Herridge et al., 2021; Lee et al., 2025; Salvatori et al., 2022).

Recent research emphasizes that growth failure in children with congenital heart defects is multifactorial, involving clinical factors (feeding intolerance, infection), hemodynamic factors, as well as nutritional and social factors. Therefore, PDA closure in indicated cases needs to be accompanied by structured nutritional monitoring during the recovery period (Herridge et al., 2021; Lee et al., 2025; Salvatori et al., 2022).

This prospective cohort study demonstrates that PDA closure is associated with a significant increase in height and body weight up to 6 months post-procedure. This finding is consistent with the physiological concept that correcting hemodynamic abnormalities improves energy balance and intake tolerance, thereby supporting catch-up growth (Lee et al., 2025).

Similar research findings were also demonstrated by Hartaty et al. (2016) in children aged <5 years with transcatheter PDA closure. Body growth was assessed by Z-score weight for age before and at 1, 3, 6, and 12 months after the procedure. The results obtained were an improvement in the mean z-score weight/ age before and at 1, 3, 6, and 12 months after PDA closure procedure (-2.63 vs. -2.41, -2.14, -1.92 and -1.56; $p<0.05$). A study by Leo et al. (2019 also found that PDA closure in children 2 years of age or below resulted in body weight improvement at 1, 3, and 6 months after procedure.

This finding is also in line with that shown by Mullaly et al. (2025) that the PDA closure procedure is associated with changes in anthropometric measurements as assessed by the occipitofrontal circumference (OfC) score. PDA closure positively affect OFC z-score trajectories ($\beta = 0.2$; $p= 0.01$). Optimal OFC growth also can represent the body growth (weight and height) is also going well (Watanabe et al., 2018).

Contributions of PDA closure on body growth can occur through eliminates a chronic left-to-right shunt that diverts blood away from systemic circulation and causes pulmonary overload, and relieves cardiac strain allowing blood to flow properly to the intestines (gut perfusion) (Backes et al., 2022). That circulation improvement can increase the children's ability to tolerate enteral feeds and absorbs nutrients (Babla et al., 2021). Closing the shunt can reduce the high-calorie expenditure due to pulmonary overload, allowing children to use their energy for weight gain and linear growth than cardiac effort (Leo e al., 2019).

In addition to classic hemodynamic mechanisms, PDA also has the potential to affect growth through neuroendocrine pathways regulating appetite (Hassan et al., 2020). In children with congenital heart defects, research indicates differences in leptin levels in malnourished groups compared to those with normal nutritional status, as well as changes in appetite-related hormones following the correction of certain heart defects. This supports the

hypothesis that hemodynamic improvement may indirectly modulate leptin signaling, such that appetite and weight gain may improve post-PDA closure (Hassan et al., 2020; Taşcı et al., 2023). However, the mediating role of hormones in the relationship between PDA closure procedures and improvement of Anthropometric results is unknown in this study so that it requires further related research.

The clinical implications of this study are a linear increase in height and weight could be a key clinical indicator of a successful PDA closure in children. But this study sill has several limitations, such as: assessing body growth from height and weight does not adequately represent the child's nutritional status. Then, unknown factors that may influence the success of the PDA closure procedure.

According to research by Okadharma et al. (2022), in children with PDA treated at Udayana Sanglah University Hospital, Denpasar, earlier age at PDA closure and normal body weight increased the chances of successful PDA closure (PR= 7.7; 95% CI= 1.2 to 47.7; p = 0.035 and PR= 13.3; 95% CI = 2.4 – 72.4; p= 0.001). Even, a study of Irfan et al. (2021) could proof that PDA closure significantly improve nutritional status over the 12 months post-procedure compares to pre-procedure. Others limitation of this study is the absence of a control group, variations in age and comorbidities that may affect growth patterns. Future studies with WHO z-score measurements and analysis of confounding factors, also prolonging the observation period are suggested.

AUTHOR CONTRIBUTIONS

Sri Lilijanti Widjaja is the main author who conducted the research, conducted data analysis and wrote the manuscript. Bagus Artiko and Masayu Lubna Anniazi examined the background and discussion of the research, while Mylco Trisaputra Ahmadwirawan examined the research framework

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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