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A Treatise on the Surgical Techniques used to close the Adult Persistent Arterial Duct

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Review Article

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Abstract

The morphologic and physiologic spectrums of late presenters of ductus arteriosus are many and varied. In this systematic review, we give an account of all the cases previously described as adult ductus, analyzing in detail those cases where an accurate antemortem and postmortem anatomical description was provided. We identified 70 investigations and reviewed the anatomic variations of adult ductus arteriosus, diagnostic modalities utilized for identification of ductal morphology and determination of operability, intraoperative findings and the surgical techniques used to close the adult ductus. We have analyzed different non-surgical and surgical approaches utilized for interruption of different types of ductus arteriosus, intraoperative adverse events encountered if any and their perioperative and postoperative course. We submit that an increased appreciation of various types of non-surgical and surgical strategies may contribute to improved patient management.

Keywords: Adult Ductus Arteriosus; Calcified Adult Ductus; Recanalized Ductus Arteriosus; Hypertensive Ductus Arteriosus; Window Ductus Arteriosus; Giant Ductus Arteriosus; Ductal Closure Under Cardiopulmonary Bypass

Introduction

Although PDA generally presents in infancy and childhood, a significant percentage of patients in developing countries do present in adolescence and adulthood with complications like congestive heart failure, pulmonary hypertension, and pulmonary vascular obstructive disease (PVOD), aneurysm, endarteritis, calcification and recanalization of the ductus arteriosus [1-6].

Congenital left-to-right shunts, such as late presenters with PDA, ventricular septal defect (VSD), atrial septal defect and aortopulmonary window are still important causes of PVOD in the developing world [7]. The natural history of large PDA shows that, if not closed in infancy, many patients develop pulmonary artery hypertension and progressive PVOD by the age of 2 years. Campbell, studying the natural history of disease, implied that 20% of patients with untreated persistent ductus died before they reached 30 years of age and only 10% of patients with untreated ductus lived beyond their 60s [5]. However, a small subset of patients does not develop PVOD even by 2nd or 3rd decade of life [6-8]. The oldest male patient recorded to survive without surgical repair was 68 years of age and the oldest female patient was of 74 years [9,10].

Chevers reported the first case of a PDA in an adult, with clinical and post-mortem observations in 1845 [11].

Gross and Hubbard reported the first successful ligation of a PDA in 1939 [12]. Kays and Shapiro from Minneapolis in 1942 published their observations during post-mortem examination of 57 adults (>17 years old), who had isolated PDA. They observed that PDA was predominant in women (75%) and was associated with an average reduction in life expectancy by about 25 years. In adults with isolated PDA, the causes of death were subacute bacterial endarteritis (40%), congestive cardiac failure (28%) and ruptured pulmonary aneurysm (3.3%). Ninety percent of patients had biventricular hypertrophy and 15% had pulmonary artery aneurysms. All the necropsied patients had short and wide ductus and. seventeen percent of the patients had difficult or inoperable conditions [13]. What explains the relative wellbeing and prolonged survival in older patients? Why is there gross variation of age at which disability develops and why does surgical treatment sometimes fail to arrest deterioration?

Methods

With these deficiencies in mind, we have analysed the published literature to identify the described instances of adult ductus, hypertensive ductus, giant ductus, short and wide ductus, window ductus, infective ductus, deformed ductus, recanalized / recurrent ductus, aneurysmal ductus, closed with or without utilizing cardiopulmonary bypass (CPB) or by employing video-assisted thoracoscopic ligation or endovascular stent grafting / device occluders. Subsequently we evaluated all clinical studies describing the principles, indications, techniques and outcomes.

The search engines employed were MEDLINE, Pubmed, Google scholar, Cochrane database and Embase. The search included literature in all languages. This strategy yielded 70 investigations that provided best answer to the diagnostic modalities utilized for identification of ductal morphology, determination of operability, the surgical techniques used for ductal interruption and its effects on postoperative hemodynamics. We have then synthesized all these features to outline the rationale, issues of concern, and potential future trends of various strategies of interruption of ductus arteriosus.

Relevant studies on adult ductus in the published literature are mostly small-scale in the form of case reports, and case series. Due to small sample sizes, heterogeneity of clinical status at the time of surgical intervention, difficulties in selection of appropriate cardiac quantifiable end-points, a meta-analysis is not possible. With respect to drawing conclusions from the sum total of the peer-reviewed published literature, we have attempted to interpret the results based on single case reports and case series.

Incidence

The incidence of PDA is approximately 1 in 2000 in full-term infants and consists of 5% to 10% of all congenital heart diseases (CHD) in children [2]. The late presenters with PDA may remain asymptomatic and detected during routine physical examination or echocardiography screening [2]. The exact incidence of adult ductus arteriosus is unknown. The incidence of aneurysm of the ductus arteriosus (DAA) is unknown. In the literature, 61% of patients were infants (n=65), 9 (9%) patients were children between 2 months and 15 years of age and 32 (30%) were adults. Out of 144 cases of DAA, 106 were of spontaneous and 38 patients were of postoperative variety [14].

Anatomical Variations and Morphological Classification

Normally the ductus arteriosus is a conical shaped conduit that connects the descending aorta just distal to the left subclavian artery to the left pulmonary artery near its origin. Literature documents considerable variation in ductal morphology and its relation to other intrathoracic structures. Krichenko et al, based on angiographic appearance, classified the persistent ductus into 5 types, which have a bearing on the surgical and transcatheter closure techniques (Figure 1) [15].

In order to identify the entire spectrum of ductal anatomical variations and their postoperative outcomes, we have utilized the standardized definitions of ductal morphology as under:

Giant ductus arteriosus: Oldham HN defined giant persistent ductus as ductus with transverse dimension of 15 mm and above [16].

Large window ductus: Maurice Lev defined window ductus as a large ductus with virtually no length and on external inspection may not be visible. The left recurrent laryngeal nerve curls under the aortic arch from left-to-right distal to PDA and therefore act as a guide in identifying PDA [17,18].

Aneurysmal ductus arteriosus: An aneurysm of the ductus arteriosus (DAA) results from a local dilatation of the vessel, or from an enlargement of the remaining ductal tissue. Spontaneous DAA are true aneurysms and may be fusiform or saccular. Postoperative DAA following ligation of a ductus arteriosus are mostly

pseudoaneurysms. Dissecting DAA is a separate disease entity not associated with aortic dissection [14,19].

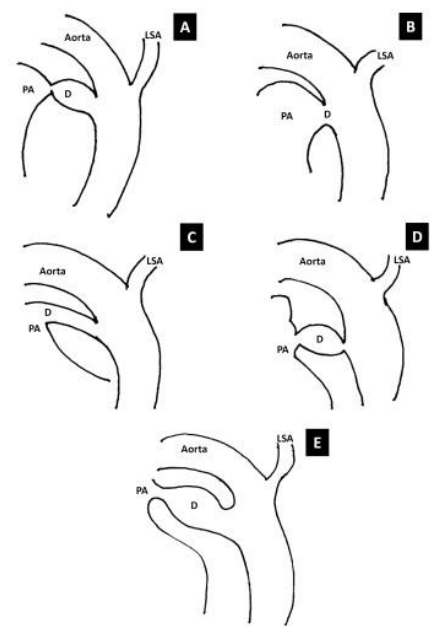

Figure 1: Diagrammatic representation of the variations in configuration of the patent arterial duct (D) as described by Krichenko, et al.
Type A: "Conical" ductus with well-defined aortic ampulla and constriction near the pulmonary arterial end.
Type B: Large "Window" ductus with very short length.
Type C: "Tubular" ductus without constrictions.
Type D: "Complex" ductus with multiple constrictions.
Type E: "Elongated" ductus with the narrowest area remote from the anterior tracheal air shadow.
[Legends: LSA= Left subclavian artery; PA= Pulmonary artery]

Ductus Phenotypes (Adult vs. Children, Impact of Ductal Circulation on Hemodynamics and Heart)

A large ductus frequently leads to heart failure in infancy. When surgical treatment is delayed into adulthood, it may not relieve heart failure or prevent death. Investigators have shown that a 35% left-to-right shunt through a PDA imposes a greater load on the left ventricle due to the combination of pressure and volume overload than a comparable shunt through a VSD [20,21].

"Coronary steal" from the aortic arch due to a large diastolic flow through the ductus, prolonged rise in left ventricular end-diastolic pressure and increased left ventricular mass have been variously incriminated as the causative factors for irreversible left ventricular dysfunction. Presence of subendocardial fibrosis demonstrated in these patients supports this theory [10,22-24]. Continued exertional dyspnoea, postoperative atrial fibrillation, persistence of heart failure, and postoperative death could all be explained by the irreversible nature of left ventricular dysfunction [22,25].

Because left ventricular remodelling is caused by significant left-to-right shunt through PDA, it is conceivable that left ventricular reverse remodelling occurs after closure of ductus arteriosus. However published literature documents immediate deterioration of left ventricular systolic function following PDA closure, which recovered within 6 months in children [22]. On the other hand, left ventricular ejection fraction (LVEF) remains low late after PDA closure compared with pre-closure state in adults [26].

Demographics and Preoperative Evaluation

Clinically, a PDA has a typical continuous murmur which can be heard at the higher left sternal edge and

may be associated with a wide pulse pressure due to the runoff to the pulmonary circulation. The electrocardiogram of patients with a large ductus in adulthood may show left atrial dilatation and left ventricular hypertrophy with strain. Atrial flutter and atrial fibrillation related to atrial distension may be present and contribute to worsening of heart failure. Depending on the degree of ductal shunting, the chest roentgenogram may demonstrate cardiomegaly mainly involving left atrium and left ventricle with increased pulmonary vascular markings. Echocardiography can establish the diagnosis, recognize volume overload, estimate pulmonary artery systolic pressure and identify associated cardiac pathology. Wiyono and colleagues proposed the following clinical severity grading in adults, which is depicted in Table 1 [27].

Type of the PDA	Murmur	Wide pulse pressure	Dilated left ventricle	Pulmonary hypertension
Silent	-	-	-	-
Small	Continuous	-	-	-
Moderate	Continuous	+	+	+
Large	Systolic + Diastolic ±	±	++	++
Eisenmenger	Ejection murmur	-	++	+++

Table 1: The clinical severity grading of the persistent ductus arteriosus in adults.

Computerized tomography and magnetic resonance imaging are the other imaging modalities useful in cases of adult ductus to assess the degree of calcification and define the anatomy in ductus with unusual morphology, aneurysms and associated abnormalities of the aortic arch. Examples include PDA associated with vascular ring, right aortic arch and with cervical arch [28,29].

Decision-Making in Late Presenters: Determination of Operability, Role of Preoperative Hemodynamic Assessment and Lung Biopsy

Since the response of the pulmonary vasculature to high pulmonary blood flow in patients with patent arterial duct is variable and unpredictable, cardiac catheterization is particularly important in adult ductus [30]. In the “borderline” patients, the current protocol is to measure pulmonary artery pressure in room air and after administering a pulmonary vasodilator such as 100% oxygen or oxygen, nitric oxide (20 ppm).

Temporary test occlusion with a balloon catheter or an occluder device has been described to decide regarding the advisability of closure [31]. With the availability of various devices, the interventional cardiologists can now close most PDAs in the catheterisation laboratory [31,32]. Therefore the guidelines are not strictly adhered to and even if there is a drop of only 15 to 20% in PAP instead of 25% and with borderline haemodynamics, the decision would often be “to close” rather not to close. This is supported by our results showing response in the long run after duct occlusion, despite of variable responses to oxygen inhalation and balloon occlusion [30,32]. However Michael Rigby has pointed out that this method has its own flaws and the management of patients with hypertensive ductus should be based on accurate measurement of pulmonary vascular resistance (PVR) [33].

In 1973, Dushane and Kirklin reported that a pulmonary vascular resistance index (PVRI) of 14 Wood units/m^2 or less was an indication for surgery and in 1976 Kirklin and colleagues reported that a value of 10 or less to be an operative indication among older children [34]. More recently, Momma and colleagues have reported a pulmonary vascular resistance index of 8 units/m^2 or less to be an operative indication [35].

At present, patients with a PVR more than 6 Wood units when breathing 100% oxygen and a pulmonary vascular resistance / systemic vascular resistance >0.4 are considered unsuitable for repair of congenital heart defects with left-to-right shunts because they have higher perioperative morbidity, mortality, and likelihood of progressive PVR despite repair of the defect [32].

The assessment of the PVRI can also be flawed for various reasons [8]. Even if all efforts are made to ensure no sampling or measurement errors, the PVRI is often calculated by assuming oxygen consumption, which is less accurate. For patients breathing oxygen, it is crucial to include values for dissolved oxygen in the calculation of resistance to avoid underestimation of the resistance [32]. In addition to the absolute value; response to pulmonary vasodilators has been considered an important clue to assess reactivity of pulmonary circulation.

Secondly, one cannot assess pulmonary blood flow accurately in the absence of a fully mixed blood by either

Fick or thermodilution. Thirdly, calculation of PVR documented in the literature are mostly based on isolated VSD or combined VSD and PDA lesions and not on isolated PDA.

The efforts to differentiate reversible and irreversible pulmonary arterial hypertension in these patients have been going on for long. The Heath-Edwards classification, histopathologic criteria in widespread clinical use, can provide only qualitative information about plexogenic pulmonary arteriopathy and consequently has limitations for use in determining operability [36]. Available information, however, has been based on single case reports with little long-term follow-up. A morphometric approach was proposed, which quantifies and grades PVOD from A to C [37,38]. The results of the morphometric analysis of lung biopsy may be predictive of late outcome, but lung biopsy is not practical. Moreover, the correlation between lung morphology, PVR, and outcome in children with CHD is also not always linear [32].

Yamaki and colleagues, based on results of 23 autopsies and 26 lung biopsies of patients with ventricular septal defect and PDA operated upon, demonstrated that regardless of patients' age, when PVR of 8 Wood units/m^2 or more were obtained, operative indications should be determined on the basis of lung biopsies. They developed an index of pulmonary vascular disease (IPVD), a composite and quantitative evaluation of the severity of pulmonary vascular disease to determine the operability. An IPVD of 2.1 without Down's syndrome was considered as the upper permissible limits for a surgical intervention [39].

The role of arterial desaturation at rest and on exercise may well be useful but its role in patients with PDA has not been investigated. The role of pulmonary vasodilators in patients with secondary pulmonary arterial hypertension is being investigated. Endothelin-receptor antagonist Bosentan and Sildenafil, a phosphodiesterase-5 inhibitor have shown some benefits in patients with Eisenmenger syndrome with improvement in pulmonary artery pressure, 6-minute walk distance, NYHA class, and oxygen saturation [40,41]. Despite symptomatic improvement, there are no data to support their role in long-term potential reversibility of pulmonary vascular occlusive disease.

In adults without development of significant PVOD, increasing heart size is often the first indication of deterioration, which can then be arrested by prompt treatment before the development of heart failure. When an older patient has neither symptoms nor cardiomegaly, the indication for operation is unclear. When atrial fibrillation and heart failure respond well to medical treatment, surgical treatment especially in patients over 70 years of age is not essential and can be avoided.

A comprehensive assessment considering the clinical evaluation, radiological and electrocardiographic findings, echocardiography, and exercise testing with pulse oximetry or arterial blood gas should be performed before cardiac catheterisation. It is evident from the compilation of published data that PVRI with oxygen ≤6WU/m^2, pulmonary artery systolic pressure with oxygen ≤75mmHg, and pulmonary artery mean pressure with oxygen ≤55mmHg have the strongest association with regression of pulmonary hypertension ($p<0.001$). These variables predicted 97.8% of patients with regressed pulmonary hypertension in most of the investigations [31-34,39].

Surgical options for the adult ductus arteriosus: Despite the experience spanning over 80 years, there are no established guidelines in the published literature to decide the optimal approach for patients with PDA presenting in adulthood. Among the variety of available surgical options, the standard techniques of closed ligation and ductal division and suture are the ideal options. However, correction of PDA remains a surgical challenge in the subset of patients with PDA presenting in adulthood with diminished elasticity, vessel friability and/or calcification, previous infection causing ductal endarteritis and unusual anatomic features as previously described [1-10,40,42-60].

Dissection and isolation of the ductus arteriosus in these clinical scenarios poses a risk of intraoperative hemorrhage, injury to the left recurrent laryngeal nerve, phrenic nerve and chylothorax. Lung dysfunction and adhesions due to previous thoracotomy present additional technical challenge in reoperative cases [17,40].

In an attempt to decrease or eliminate these dreaded complications, diverse surgical techniques have been advanced. Published literature documents the following techniques for different kinds of complicated PDA (Table 2).

S.No.	Authors	Year	Age/Sex	No. of patients	Diagnosis	Treatment	Results
1	Gurcun U, et al. [55]	2005	47 years female	1	Adult ductus, diameter 5mm, PAP (systolic 50mmHg), Qp:Qs= 1.8	CPB, circulatory arrest, nasopharyngeal temperature 20°C, 2 pledgeted 4-0 polypropylene mattress suture, circulatory arrest time = 5 min, bypass time=67 min	Follow-up at 4½ yearsà NYHA I, no residual shunt
2	Taira A, Akita H [56]	1975	22 years male	1	Adult calcified ductus	Normothermic CPB; patch mounted on a Fogarty catheter	Survived
3	Toda R, et al. [57]	2000	Mean 55 years (range 35-74 years)	3 males, 6 females	Adult calcified ductus on CT and chest x-ray (n=4), atrial fibrillation (n=2), NYHA II, Qp:Qs= 2.4±0.95 (mean 1.44-4.18), PP/PS= 0.42±0.23 (range 0.18-0.91), SPAP= 56±26.4 (range 27-109 mmHg)	Direct closure (n=5), patch mounted on Foley catheter (n=4)	Follow-up at 6 months, systolic PAP 35.3±6.6 mmHg, follow-up at 55 monthsà no recanalization, no pseudoaneurysm
4	Erdman S, et al. [42]	1978	>40 years	4	Calcified ductus (n=3)	Two tapes around the aorta above and below the ductus. Two mattress sutures 2-0 silk through 2 Teflon pledgets, controlled systemic hypotension 80-90 mmHg, closure is accomplished by gradually tightening the two mattress sutures	Survived
5	Morrow Ag and Clark D [49]	1965	44 years female	1	Adult ductus, atrial fibrillation, NYHA IV- 7 years, PAP 132/56 mmHg, left to right shunt, calcified ductus 1.5 cm diameter	Normotheramic CPB, arterial cannula, LSA, LFA; MPA, aorta proximal and distal to PDA isolated; aortotomyà Dacron patch closure on the aortic end	Died of low cardiac outcome syndrome one week postoperatively
6	Arbatli H, et al. [51]	2003	43 years female	1	Recurrent ductus, PDA ligation 17 years back, Electron beam tomography, recurrent ductus 6mm diameter, Qp:Qs= 2.2	CPB, profound hypothermia 20°C, balloon occlusion, pledgeted 4=0 polypropylene sutures	Follow-up at 7 months, NYHA I, no residual shunt, no pseudoaneurysm formation

7	Omari BO, et al. [52]	1998	17 years female	4	Short wide hypertensive ductus [Trans-pulmonary balloon occlusion, 16F Foley catheter]	CPB, primary closure, CPB time-30+23 min	All discharged home, no residual shunt
			19 years female			CPB, primary closure, CPB time-35 min	
			26 years female			CPB, Dacron patch closure, CPB time-77 min	
			63 years female			CPB, primary closure, CPB time-43 min	
8	Wiyono SA, et al. [27]		28 years female	2	Adult ductus, sinus rhythm,	Percutaneous closure 5mm coil (Flipper, Cook Medical, Ireland)	No residual shunt
			37 years female		Adult ductus, atrial fibrillation, LVEDD 82 mm, LVESD 65 mm, systolic PAP 70 mmHg, Qp:Qs= 3:1, duct diameter 11 mm	Amplatzer ductus occluder 16-14mm (ADO, AGA Medical Corporation, Minnesota, USA), residual shunt, LVEDD decreased 64mm, LVESD 57mm	Residual shunt, SBE prophylaxis
9	Grunenfelder J, et al. [16]	1998	18 months, female	1	Large window ductus, cor-triatriatum, persistent LSVC, unroofed coronary sinus, OS ASD, Large window type, ill defined PDA, PAH	LSVC rerouting with Goretex tunnel, Patch closure of ASD, Enlargement of Coronary Sinus with bovine pericardium clipping of intra pericardial PDA. Autopsy: oval window ductus, transverse diameter 15mm, circumference 30mm	Died day 1, with acute renal and hepatic failure, intestinal ischemia and absent lower limb pulses.
10	O' Donovan TG and Beck W [25]	1977	55 years female (1) 3 adults of unspecified age	4	Adult ductus, severe PAH	Femoral artery./ Asc Aorta, RA cannulation. Low flow CPB (200mL/min), PA opened, interrupted mattress sutures	1 patient died after 4 years, 3 patients surviving
11	Varma PK, et al. [18]	2004 (1976-2002)	Initial surgery (mean age 13.7±8.2) Aneurysm surgery (mean age 16.9±8.8yrs)	13		Initial ligation/ Division 10/3 Lateral thoracotomy, Femoro-femoral bypass and circulatory arrest (3) Sternotomy and circulatory arrest (10) (Sternotomy: median =9, transverse=1	Thoracotomy (3) died due to rupture during dissection and bleeding Sternotomy (1) died due to bleeding from friable aorta Follow up 9.6±5.3 years
12	Bell Thompson J,	1979	68 years female	1	Shortness of breath 8 years Qp/Qs: 2.8:1 PAP:	Division and suture with cross-clamps above below and over pulmonary end of	Survived

	et al. [9]				70/42 mmHg PCWP 26 mmHg Cardiomegaly on Chest X-ray	ductus	
13	Gonclaves Estella A, et al. [54]	1975	24 years female	1	Large PDA PAP (S/D) 57/37 mmHg Aorta 113/70 mmHg Qp:Qs-7.2:1 PR/SR: 0.1	Median sternotomy, Femoro-femoral bypass, Right Brachiocephalic artery, Left Carotid artery dissected, circulatory arrest at 20oC, Root on suction, PA opened and PDA closed, 3 Teflon felted mattress sutures, (same technique as closure for Potts shunt)	Survived
14	Bhati B S, et al. [3]	1971	9 years female	1	ASD+ PDA PAP 110/55 mmHg, Qp/Qs: 3.4, PVR 11.3	Fogarty balloon occlusion + Intracardiac repair	All survived
			6 years female		TOF+ PDA PAP 120/70 mmHg, Qp/Qs: 1.2, PVR 10		
			17 years female	1	VSD+ PDA PAP 120/75 mmHg, Qp/Qs: 3.4, PVR 11.3		
15	Katsumato K, et al. [59]	1993	67 years female	1	Giant hypertensive PDA, Cardiomegaly on Chest-Xray PAP 30/10 mmHg Qp/Qs:2.0	Aorta clamped above and below the ductus, PDA clamped at PA end, transductal silicon sponge inserted and sutured.	Survived
16	Djukanovic BP, et al. [40]	2014	23-60 years (Median 50 years)	7	Short, wide tortuous ductus, severe calcification (n=2), severe AS (n=1), Ascending Aorta aneurysm (n=2), Qp/Qs=2.27±0.49 (1.73-3.12) PAP systolic 52.2±15.1 mmHg unsuitable for device	Normothermic CPB, Balloon occlusion (n=4), Deep hypothermic short circulatory arrest (n=1) Deep hypothermic short circulatory arrest and aortic reconstruction (n=2)	Mean follow up 48 months, No residual shunt, Asymptomatic
17	Sadiq M, et al. [70]	2017	2-27 years, (Median 10 years)	45	Hypertensive ductus	Amplatzer duct occluder, amplatzer muscular VSD occluder Systolic PAP mmHg Preclosure: (systolic-79, mean-59) Postclosure: (systolic-67, mean-50)	Follow-up median 80 (41-151 months), persisting severe PAH 9.7% (n=4) patients

18	Jeong YH, et al. [26]	2007	40±13 years	45	Hypertensive ductus	Surgical PDA closure (17)- 34±11 years Interventional PDA closure (28)- 40±23 years	Follow-up 18±16 months (6-45 months), 11.1% persistently low LVEF
19	Liang CD, et al. [64]	2003	6 months-55 years (mean 5.5 years)	75	Mean PDA length 7.2±3.5 mm, mean PDA diameter 2.1±1 mm, Mean angle of PDA and aortic arch 18.5±14.9°	Gianturco coil embolization	All vocal cord paralysis occurred in patients <1 year old. 3 of 75 patients had VCP post Gianturco coil
20	Sankhyan LK, et al. [50]	2018	26-35 years (28.2±6 years)	18	Adult ductus (3), giant ductus (3), short and wide ductus (2), window ductus (2), calcified ductus (6), infected ductus (1), recanalized ductus (1) PAP 62±18.4 (47-126 mmHg) PVR 4-8 Woods unit/m2 Qp:Qs- 2.2±0.89 (1.48-4.26)	CPB, transpulmonary normothermic balloon occlusion with cardioplegia, no circulatory arrest	All survived, follow-up 12.6±5.8 years (12 months-20 years) No residual shunt CT angio (14) Complete ductal interruption
21	Morrow AG, Clark D [49]	1965	44 and 52 years females	2	Adult calcified hypertensive ductus PAP (132/56, 104/46 mmHg respectively) Qp:Qs- 1.4:1	CPB, arterial cannulas-one into LSA, 2nd into LFA, venous into RVOT, aortic cross clamp above and below the juxta ductal aorta & MPA; aortotomy- Teflon patch closure of ductus	One died one week later- LCOS Postoperative PAP- 108 mmHg Post-mortem: calcified aortic and pulmonary end of ductus, no thrombus. Second patient survived, postoperative PAP (systolic)- 60mmHg
22	Roques F, et al. [65]	2001	65 years female	1	Large PDA CT- heavily calcified DTA, 3.5 cm aneurysmal PDA, systolic PAP 65 mmHg, Qp: Qs- 2.8:1, SaO2- 93%, coronary angio- non-surgical TVD, severe COPD, aortography PDA origin 2.5 cm distal to LSA	General anaesthesia, fluoroscopic guidance, custom made "Talent" stent graft in front of entry of the PDA	Discharged- day 3, follow-up (3 months), thrombosed aneurysm-PDA, no ductal flow
23	Wiyono SA, et al. [27]	2008	28 and 37 years females	2	Patient 1: Tiny PDA (not catheterized) Patient 2: Systolic PAP 70 mmHg,	Patient 1: 5mm coil (Flipper® Cook Medical, Ireland) Patient 2: percutaneous PDA closure, amplatzer device	Both survivedPatient 2: follow-up 18 months, LVEDD 64 mm, LVESD 57 mm, not much improved

					Qp:Qs- 3:1			
24	Erdman S, et al. [42]	1978	More than 40 years old	4	Adult hypertensive ductus, calcified ductus-1	Controlled hypotension, two Teflon felt pledgets are sutured on the superior and inferior surfaces of the ductus		All survived
	Thoracic-Endovascular aortic repair, tapered stent grafts, Grikin Advanced Materials Co. Ltd., Beijing, China (4 patients)							
				Case 1	**Case 2**	**Case 3**	**Case 4**	**Results**
25	Lai YQ, et al. [67]	2008	Age (years)	17	37	30	42	Follow up 3-18 months, TTE, CT- no ductal flow, LVEDD – Pre: 64.3±6.9 mm, LVEDD Post: 56.5±6.1 mm (p<0.05)
			Sex	Female	Female	Female	Female	
			Aortic opening diameter of PDA (mm)	30	28	28	29	
			Pulmonary opening diameter of PDA (mm)	16	12	22	14	
			Stent graft (mm)					
			Proximal diameter	30	36	34	34,32	
			Length	100	120	120	85, 100	
			Distal diameter	26	30	28	30, 28	
			Preoperative MPAP	75	45	60	83	
			Postoperative MPAP	50	20	38	54	
			Follow up (months)	18	15	6	3	

Table 2: Summary of the published investigations documenting the diagnosis and management of the adult ductus.

Legends: AS= Aortic stenosis, ASD= Atrial septal defect. COPD= Chronic obstructive pulmonary disease, CPB= Cardiopulmonary bypass, CT-angio= Computerized tomographic angiography, DTA= Descending thoracic aorta, LCOS= Low cardiac output syndrome, LFA= Left femoral artery, LSA= Left subclavian artery, LSVC= Left superior vena-cava, LVEDD= Left ventricular end-diastolic diameter, LVESD= Left ventricular end-systolic diameter, MPAP= Mean pulmonary arterial pressure, MPA= Main pulmonary artery, NYHA= New York Heart Association, PA= Pulmonary artery, PAH= Pulmonary artery hypertension, PAP= Pulmonary artery pressure, PCWP= Pulmonary capillary wedge pressure, PDA= Patent ductus arteriosus, Pp/Ps= Pulmonary-to-systemic resistance ratio,PVR= Pulmonary vasculat resistance, Qp:Qs= Pulmonary-t-systemic flow ratio, RA= Right atrium, RVOTO= Right ventricular outflow tract, SaO2= Systemic arterial oxygen saturation, SPAP= Systemic pulmonary arterial pressure, SBE= Subacute bacterial endocarditis, TOF= Tetralogy of fallot, TVD= Triple vessel coronary disease, VCP= Vocal cord paralysis, VSD= Ventricular septal defect

1. **Double ligation of the adult ductus arteriosus**: This technique involves suturing two Teflon felt pledgets on the superior and inferior surfaces of the ductus and gradual tightening of the two mattress sutures under controlled hypotension [42,43].
2. **Division and suture without cardiopulmonary bypass:** Bell-Thomson and colleagues described a technique in which the aorta was cross-clamped above and below the PDA [9]. A third vascular clamp was applied at the ductus itself. The ductus was divided at the aortic end including a small rim of the aortic wall. Both the aortic and pulmonary ends were sutured using 4-0 polyproplene suture [9]. Thomas and associates described a technique wherein a Potts-Smith Clamp was applied to the aorta. A curved vascular clamp was applied to the pulmonary artery and the ductus was divided and sutured with felt supported double-layered continuous polypropylene suture [44]. Other investigators including ourselves have employed a C-shaped vascular clamp at the aortic end of the ductus and a right angled vascular clamp at the pulmonary arterial end under controlled hypotension using sodium nitroprusside and division is performed through the aortic tissues which allow for a secure closure of both sides without undue risk of bleeding [45]. In case aorta is cross clamped, it is recommended by Crafoord that the cross clamp not be applied longer than 15 min [46].
3. **Median sternotomy, normothermic cardiopulmonary bypass:** Kirklin and Silver were the first to describe a technique of ductal closure using No.2 ductus silk under CPB that was later popularized by McGoon [47,48]. However there is a high chance of inadvertent tearing during the manipulation of a tense, wide ductus with high intraluminal pressure.
4. **Transaortic patch closure of calcified ductus arteriosus without utilizing cardiopulmonary bypass:** A shunt is placed between the left subclavian artery and descending thoracic aorta after low dose heparinisation. The aorta is clamped proximal and distal to the PDA. A Satinsky clamp is placed over the pulmonary artery about 0.5 cm from the ductal orifice. The aorta is opened longitudinally. The aortic end of the ductus is closed using a Dacron patch [45].
5. **Transaortic Teflon patch closure under cardiopulmonary bypass:** Morrow and Clarke described a technique of ductal interruption on two patients for a large, calcified ductus under CPB [49]. Two arterial cannulas were placed, one into the left subclavian artery and the other into the left femoral artery and a single venous drainage cannula into the right ventricular outflow tract. Under CPB, the main pulmonary artery and the aorta proximal and distal to the ductus were occluded. An incision was made in the lateral wall of the aorta, an area free of calcification. The calcified aortic end of the ductus was closed using a Teflon patch by a series of interrupted mattress sutures in the aorta beyond the calcified areas [49].
6. **Cardiopulmonary bypass, mild hypothermia, transpulmonary Fogarty/Foley balloon occlusion:** Bhati and colleagues described a technique of ductal interruption under CPB from inside the pulmonary artery after the aortic orifice had been occluded with the help of a venous Fogarty catheter [3]. We have published our observations on transpulmonary balloon occlusion of the ductus under normothermic CPB without circulatory arrest in cases of adult, giant, recanalized and calcified ductus [50].
7. **Cardiopulmonary bypass, profound hypothermia, transpulmonary balloon occlusion:** Arbatli and colleagues described this technique of ductal occlusion via transpulmonary route, under CPB, profound hypothermia to 20°C, balloon occlusion and transient low flow state without circulatory arrest in a case of adult recurrent ductus. There was no recurrence or recurrent laryngeal nerve injury [51]. Omari and colleagues described successful ductal interruption using transpulmonary balloon occlusion technique on four adult patients [52].
8. **Profound hypothermia and circulatory arrest:** This technique has been advocated in cases of short, wide ductus in which balloon catheter occlusion had a danger of balloon rupture during suture placement [53-55]. The procedure is analogous to the technique described by Kirklin and Devloo for closure of Potts anastomosis. However, adoption of such a policy is time consuming with the added risk of air embolism, flooding of the pulmonary circulation, cardiac distension and is therefore abandoned for patients undergoing ductal interruption.
9. **Profound hypothermia, low flow cardiopulmonary bypass and transpulmonary ductal closure without balloon occlusion:** In 1978, O'Donovan and Beck described a method of closure of calcified ductus under low flow CPB and profound hypothermia without balloon occlusion [25]. Arterial inflow was obtained either via transfemoral route (n=2) or ascending aorta (n=2). The neck vessels were not isolated. They successfully used this technique on four patients.
10. **Normothermic cardiopulmonary bypass, transpulmonary patch mounted on either a Fogarty catheter or Foley catheter:** In this technique, following insertion of the catheter tip into

the aorta through the ductus, the balloon is inflated in the aorta. The catheter is then pulled up gently to plug the ductal orifice and may be slanted in any direction convenient for the operative procedure without obscuring the vision. After completion of the procedure, the catheter is withdrawn and the purse string suture is tied [56,57].

11. **Transaortic transductus repair without utilizing cardiopulmonary bypass:** Wernly and colleagues, Katsumoto and associates described this novel surgical technique in which the juxta-ductal aorta above and below the ductus and the pulmonary arterial end of the ductus are cross-clamped. The ductus is opened longitudinally in between stay sutures and an appropriate sized silicone sponge is inserted into the ductus. The ductus is subsequently sutured and an additional anchor suture with felts is applied over the ductus [58,59].
12. **Left heart bypass**: Laustela and associates used a Teflon pledgeted suture ligation technique with left heart bypass [60].
13. **Percutaneous transcatheter closure of ductus using coil or ductus occluder device:** Interventional percutaneous PDA closure (PTDC) is presented as an alternative with a high level of reproducibility. However, intermediate (1 year) residual shunt was present in 5% of patients enrolled in the European Registry [61]. Immediate residual patency however was 41% and it is unknown in how much time complete occlusion occurs. Consequently, concerns about effective complete closure, repeat examinations, duration of follow up, and necessity of long-term antibiotic prophylaxis exist [61]. A PDA "neck" is required for effective PTDC, and large PDAs, window PDAs may lack such a neck. Moreover, in large PDAs, which require multiple coils or bigger devices for closure, PTDC can lead to left pulmonary artery stenosis or aortic coarctation and embolization of the coils into the pulmonary circulation. Consequently, PTDC may not be safe for closing ducts that are more than 4 mm in diameter in adults. Despite the use of new technologies**,** transcatheter PDA closure techniques maintain the disadvantages and potential intraprocedural risks such as peripheral vascular injury, device migration, obstruction of the pulmonary arteries or thoracic aorta, late embolization, flow disturbance in the left pulmonary artery or descending thoracic aorta from a protruding device, hemolysis from high velocity shunting, distal embolization, thrombosis of the vascular access, infection, severe residual shunting and recanalization. Large PDAs are more likely to have a residual shunt that causes hemolysis or endocarditis [40,61,62].
14. **Video assisted thoracoscopic surgery:** Laborde and colleagues introduced the VATS approach for PDA ligation in 1993 [63]. The VATS technique is safely applicable to cohorts of patients at a young age with ductus diameter <9mm. Calcified ducts, severe pleural scarring, and short, wide, window-like ducts are considered contraindications to the VATS approach [40]. Use of VATS for PDA closure also carries the risk of uncontrolled hemorrhage and recurrent laryngeal nerve injury [64].
15. **Endovascular treatment with a stent-graft:** Roques and associates first described the use of endovascular occlusion of an adult ductus. Subsequently, there are three isolated case reports by other investigators [65-67]. Although the use of self-expansive prosthesis for the endovascular treatment of PDA is promising, the technology used to insert the prosthesis requires adequate access route, adequate landing zone (arch diameter < 35mm), and the distance between the ductus arteriosus and the left subclavian artery must be a minimum of 15mm to assure efficiency and safety [66,67]. Anomalies of the arch vessel origin, lack of proper proximal landing zone, endoleak, delayed cessation of ductus patency and residual shunt limit the use of this technique to a few selective patients who have comorbidities that make them a high-risk candidate for surgery [65-67].

Complications following interruption of adult ductus arteriosus

The intraoperative complication most feared during interruption of adult ductus arteriosus is bleeding from the friable pulmonary or aortic stump related to the presence of calcification, endarteritis and aneurysms. Analysis of the published literature suggest that an elective institution of CPB or left heart bypass and hypothermic circulatory support may be useful in this subset of patients to avoid the occurrence of the dreaded bleeding complication (Table 2).

Multiple centres have demonstrated a high incidence of residual shunting (42%) after transcatheter occlusion of ductus arteriosus [61,62]. The reported incidence of residual leaks following surgical closure of ductus arteriosus ranges between 0% to 23% [62,68].

With standard ductus arteriosus ligation, literature documents 2.8% to 8.8% incidence of transient recurrent laryngeal nerve injury probably induced by traction or by electrocautery [64,69]. A long ductus length (> 12mm.) and a small ductus diameter (< 1mm) are associated with an increased risk of vocal cord paralysis after Gianturco

coil embolization due to tense stretching and angulation causing compression injury of the ductus [64]. Several investigators found no residual shunt or recurrent laryngeal nerve injury in children and adolescents undergoing video-thoracoscopic ligation of the ductus arteriosus [63,64].

Influence of ductal interruption on pulmonary artery pressure, left ventricular size and function

The detailed published investigations on the surgical management of adult ductus arteriosus, the results in the short- and long-term have been tabulated in (Table 2). Yan and associates (2007) reported on 29 adult patients aged between 18 and 58 years (mean 31.1 ±11.4) with PDA undergoing transcatheter closure. Twenty of 29 patients had successful occlusion and nine patients had failed occlusion. In patients with successful occlusion, PAP decreased markedly from 78 (±19.3), range 50-125 mmHg before occlusion to 41 (±13.8), range 23-77 mmHg after occlusion. The group with failed occlusion had increased PAP and desaturation on trial occlusion. In 2006, Eerola and associates have demonstrated normalization of LV volume and function after percutaneous closure in children [22].

In 2007, Jeong and associates demonstrated persistent long-term deterioration of LVEF in 11.1% of adult ductus treated either by surgery or Amplatzer occlusion [26]. Sadiq and associates evaluated the results of device closure in patients with large ductus aged between 2-27 (median 10 years). Device closure was successful in 96% of patients; severe pulmonary hypertension persisted in 9.7% patients [70].

In our previous investigation, we evaluated the results of transpulmonary closure of adult ductus under normothermic CPB with cardioplegia in 18 patients, aged between 26 to 35 (mean 28.2±6) years. Indications were adult ductus arteriosus (n=3), calcified ductus (n=6), infected ductus (n=1), window ductus (n=2), short and wide ductus (n=2), giant ductus (n=3) and recanalized ductus (n=1). All patients survived the operation. There was no phrenic or left recurrent laryngeal nerve damage, chylothorax, massive bleeding or recanalization. At a mean follow-up of 12.6±5.8 years, the PAP decreased significantly from 62.0±18.4 mmHg to 33.6 ±6.4 mmHg ($p<0.01$) [50]. Computerized tomographic angiography (n=14) revealed complete ductal interruption with no residual shunt or ductal aneurysms [50].

Conclusions

On the basis of the published literature including ours, enunciated in the manuscript, we conclude the following:

1. Hemodynamically, systolic and mean pulmonary artery pressure and pulmonary vascular resistance index on oxygen are the most important prognostic variables associated with regression of pulmonary artery hypertension.
2. Despite the advent and usefulness of interventional catheterization, its usage may not be possible or feasible in cases of short, giant, calcified ductus, window type ductus, aneurysmal ductus, deformed ductus, ductus associated with vascular rings and concomitant aortic and other cardiac diseases requiring surgery.
3. In non-calcified large ductus, vascular clamps supported by Teflon bars, controlled systemic hypotension, division of ductus and felt-supported sutures, may be used. Circulatory arrest provides an optimal exposure and may be the management of choice in cases of postoperative ductal aneurysm.
4. The most feared intraoperative complication of surgery for adult ductus is bleeding from the pulmonary or aortic stump related to the presence of calcification or infection causing endarteritis. In such cases, the preferred surgical approach may be the ones involving cardiopulmonary bypass or left heart bypass or circulatory arrest to avoid bleeding complications.
5. An appropriate surgical management of adult patients with giant, calcified, hypertensive, window type ductus should consist of occlusion with patch or pledgeted sutures on cardiopulmonary bypass via transpulmonary approach using temporary balloon-occlusion. This method is safe, expedient, obviates the need for descending aortic cross-clamping, internal shunting, profound hypothermia, and circulatory arrest. It avoids dissection in the presence of ductal wall calcification, adhesions, thereby avoiding perioperative injury to the tense pulmonary artery, recurrent laryngeal nerve and large lymphatics.
6. Knowledge of these approaches should contribute to the armamentarium of the cardiac surgeon faced with such complex congenital anomalies.

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