

Full Length Research

Does the Blalock-Taussig-Thomas shunt cause catch-up growth of branch pulmonary arteries? No: A systematic review

Mark N. Awori, Jonathan A. Awori, Gilbert Langat and Kimberly Kipkoech

Department of Surgery, School of Medicine, University of Nairobi, P. O. Box 19676-00202, Nairobi, Kenya.

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To determine if the Blalock-Taussig-Thomas shunt (BTTS) causes sufficient growth of hypoplastic branch pulmonary arteries (BPAs) to facilitate total correction or definitive palliation of cardiac lesions, PUBMED (PM) and Google Scholar (GS) were systematically searched between January 1st, 1966, and March 31st, 2023. Search terms included: Blalock, Taussig, shunt, pulmonary artery and Tetralogy of Fallot. Only full-text papers that measured pre-BTTS and post-BTTS BPA sizes using the Nakata Index or z-scores were included. Twenty full-text articles (718 patients) were included in this review. When the pre-BTTS right pulmonary artery (RPA) z-score that corresponds to the mean pre-BTTS NI was considered for each article, nineteen out of 20 articles (717 patients) had normal-sized RPAs (that is, their z-scores fell within the normal distribution). Although BPAs were found to grow post BTTS, available evidence suggests that most of the growth (67%) was due to BPA distension. A longer duration of BTTS did not increase BPA growth. The evidence available suggests that the modified BTTS does not produce sufficient 'catch-up' growth of hypoplastic BPAs to facilitate total correction or definitive palliation via the Fontan pathway.

Key words: Blalock-Taussig-Thomas shunt, pulmonary artery, growth.

INTRODUCTION

Kirklin et al. (1977) noted that for 'some years,' surgeons had proposed that increasing pulmonary blood flow using a systemic arterial-to-pulmonary artery shunt (SAPAS) could increase the size of hypoplastic branch pulmonary arteries (BPAs) sufficiently to facilitate surgical correction of certain congenital cardiac defects. Using similar reasoning, others have queried if SAPAS could grow BPAs sufficiently to permit definitive palliation via the Fontan pathway. The practice of creating a SAPAS to 'grow' branch pulmonary arteries has become an

accepted indication for performing a Blalock-Taussig-Thomas shunt (BTTS) (Zhou et al., 2020). Data published by Nakata et al. demonstrated that successful correction of tetralogy of Fallot and successful definitive palliation via the Fontan pathway each requires normal-sized BPAs (Nakata et al., 1984). The growth of hypoplastic BPAs that would allow the attainment of either of these goals has been termed 'catch-up' growth. Catch-up growth refers to BPA growth that is faster than somatic growth (Godart et al., 1998). Despite it being widely accepted

*Corresponding author. E-mail: mawori@yahoo.com. Tel: +254707366336.

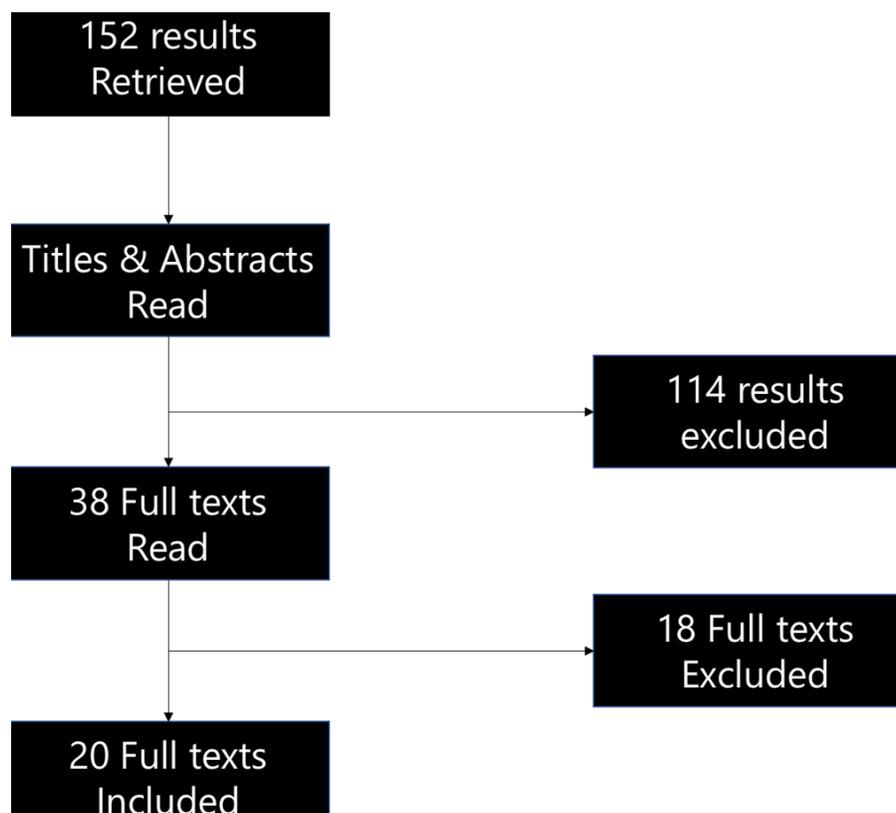
ORCID ID <https://orcid.org/0000-0003-1600-6004>.

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Table 1. Search strategy.

S/N	Searches and terms
1	Blalock, Taussig, shunt, pulmonary, artery, growth (GS, PM)
2.	Blalock, Taussig, shunt, tetralogy (GS)

GS = Google scholar, PM= Pubmed.

**Figure 1.** Search flow on the study.

that 'catch-up' growth occurs after a BTTS, there is evidence to the contrary (Godart et al., 1998). We performed a systematic review of the literature to determine if BTTSs result in sufficient BPA growth to facilitate total correction of cardiac lesions or allow definitive palliation via the Fontan pathway.

MATERIALS AND METHODS

PUBMED (PM) and Google Scholar (GS) were systematically searched between January 1st, 1966, and March 31st, 2023. The search details are shown in Table 1. GS searches used the "all in title" function, and PM searches used the "title/abstract" function. The Boolean operator "AND" was used for all searches. Titles, abstracts, and, where appropriate, full-text articles were examined if the abstract suggested that there was a possibility that the size of BPAs was examined. Only full-text articles that measured pre-BTTS and post-BTTS BPA sizes using the Nakata Index (Nakata et al.,

1984) or z-scores (Pettersen et al., 2008) were included. As there is evidence that patients with CHD are generally similar in size-for-age to normal patients (Santoro et al., 2009), (Guo et al., 2023), we estimated patient weight-for-age and height-for-age using standard charts (WHO: Child Growth Standards, 2023). We then calculated the BSA using the Haycock formula (Awori et al., 2011) and used it to determine the right pulmonary artery (RPA) z-score (Pettersen et al., 2008) that corresponded to the mean pre-BTTS and mean post-BTTS NI (Nakata et al., 1984) for each article. To calculate the RPA z-score, we considered the RPA and left pulmonary artery (LPA) to be the same size. Patient age was rounded to the nearest month. Non-English language papers were excluded. The search flow is shown in Figure 1.

RESULTS

The search yielded 152 results; after accounting for duplicate articles, 38 full-texts were examined, and 18

Table 2. Key details of articles.

Authors	n	Age at BTTS: months (mn/md)	Duration BTTS in situ (mn/md)	Mean NI (Pre-op/post-op)	RPA z-score (Pre- op/post-op)	Most frequent shunt: diameter mm)	Type of CHD
Guo (Guo et al., 2023)	9	15.8(mn)	10.8(mn)	160/146	-2.85/-1.18	6	TET
Kotani (Kotani et al., 2021)	130	1(md)	24(md)	126/170	-0.95/0.07	4	MCHD
Kim (Kim et al., 2021)	30	1(mn)	9.6(mn)	120/189	-1.18/-0.11	-	TET-PA
Lenoir (Lenoir et al., 2021)	38	1(mn)	10.9(mn)	101/219	-2.27/-0.43	4	MCHD
Zhou (Zhou et al., 2020)	90	6(md)	33.7(md)	150/286	-0.75/3.46	4	MCHD
Lenoir (Lenoir et al., 2020)	70	1(mn)	10(md)	112/243	-1.22/0.61	4	MCHD
Bigdelian (Bigdelian et al., 2018)	8	3(mn)	-	113/146	-2.80/-1.1	3.5	MCHD
Quandt (Quandt et al., 2017)	28	2(md)	14.6(md)	84/145	-2.28/-1.13	3.5	TET
Ross (Ross et al., 2015)	31	1(md)	7.1(md)	69/101	-2.75/-1.97	-	TET
Nakashima (Nakashima et al., 2014)	21	1(mn)	6.7(mn)	95/237	-1.22/0.68	3.5	MCHD
Zheng (Zheng et al., 2014)	18	32(md)	24(mn)	90/192	-2.78/-0.60	-	TET-PA
Santoro (Santoro et al., 2009)	14	1(mn)	9(md)	151/295	-0.20/1.31	-	MCHD
Amark (Amark et al., 2006)	43	1(md)	24(md)	157/250	-0.20/1.20	-	TET-PA
Ishikawa (Ishikawa et al., 2001)	12	12(mn)	32 (mn)	139/297	-1.06/0.81	Classic	MCHD
Sabri (Sabri et al., 1999)	35	4(md)	11.9(mn)	121/184	-1.34/-0.31	5	TET
Godart (Godart et al., 1998)	78	12(mn)	51(mn)	166/257	-0.78/0.28	5	MCHD
Cotrufo (Cotrufo et al., 1989)	26	3.2(mn)	14.9(mn)	81/134	-2.43/-1.31	5	TET-PA
Ullom (Ullom et al., 1987)	16	2.4(mn)	29(mn)	144/431	-0.61/2.14	5	MCHD
Brandt (Brandt et al., 1986)	14	12*	25.2(mn)	127/286	-1.46/0.81	Classic	MCHD
Kirklin (Kirklin et al., 1977)	1	50	50	9.1/49	-9.84/-4.67	Classic	TET-PA

BPA = Branch pulmonary artery; BTTS = Blalock-Taussig-Thomas Shunt; CHD = congenital heart disease; MCHD = multiple types of congenital heart disease; NI = Nakata index; RPA = right pulmonary artery; TET = Tetralogy of Fallot; TET- PA = Tetralogy of Fallot with pulmonary atresia; *= all patients were less than 12 months old.

were rejected. Twenty full-text articles representing 718 patients were included in this study; the key details of these articles are shown in Table 2.

DISCUSSION

Traditionally, the main indications for performing a BTTS are: to grow hypoplastic branch pulmonary arteries sufficiently to facilitate total correction; the first step on the Fontan Pathway and to improve oxygenation where total correction or cavo-

pulmonary connection is not possible. The current study reviewed the available evidence supporting the first indication. In nineteen out of 20 articles (96%), the mean Nakata index (NI) was reported to have increased after a Blalock-Taussig-Thomas shunt (BTTS). This implies that there is a possibility that the branch pulmonary arteries (BPAs) grew in 717 out of 718 (99%) patients. According to Nakata et al., a normal NI is 330 +/- 30 mm²/M² (Nakata et al., 1984); the mean NI in all the studies included was less than this. In nine out of 20 studies (247 patients), the mean NI was

less than half of the normal value quoted by Nakata et al. In seven out of 20 studies (182 patients), the NI doubled after the BTTS. One of these seven studies examined 4 patients but only provided sufficient data on one patient; the preoperative NI in this patient was 9.1 and the postoperative NI was 49 (Kirklin et al., 1977). This was the oldest study and also reported the lowest preoperative NI of all the studies included. Only one study (Ullom et al., 1987) reported that the mean post-BTTS Nakata index was greater than the normal value as defined by Nakata et al.

(1984).

Examining the BPA size in terms of the NI, as defined by Nakata et al. (1984), gives the impression that all the studies included examined the effect of BTTSs in patients with hypoplastic BPAs. The fact that post-BTTS BPA growth is reported in all the studies, even doubling the NI in almost 200 patients, suggests that a BTTS could cause significant growth of hypoplastic BPAs. However, when the size of the BPAs is examined in terms of z-scores, as recommended by Awori et al. (2011), it is evident that the pre-BTTS RPA z-score that corresponds to the mean NI for each article was larger than -3 in 19 out of 20 articles. Only one article reported a pre-BTTS RPA z-score of less than -3 (-9.84); this was the oldest study, and it only examined one patient (Kirklin et al., 1977). In this study, the post-BTTS RPA z-score was -4.67. Ten out of 20 studies (488 patients) reported a post-BTTS RPA z-score increase to greater than zero. The post-BTTS RPA score increased to greater than 3 (3.46) in only one of these ten studies (Zhou et al., 2020). All of these ten studies had pre-BTTS RPA z-scores of greater than -2. Considering the preoperative BPA size in terms of z-scores reveals that 19 out of 20 studies (717 out of 718 patients) actually examined preoperative BPAs that were normal in size (that is, their z-scores fell within the normal distribution). This discrepancy between the NI and z-scores has been reported previously by Awori et al. (2020). When the NI is calculated using a more robust dataset for normal dimensions for cardiac structures in children (Pettersen et al., 2008), the normal NI is significantly smaller than that reported by Nakata et al., and it varies with age. Using this more robust dataset, Awori et al. (2020) found that the lowest normal NI was 155; this was found in neonates. The highest normal NI was 204; this was found at the age of 5 years. When the size of the BPAs is examined with these normal values for the NI, the discrepancy between the NI and z-score essentially disappears.

The oldest study included in this review was undertaken to determine whether a systemic-arterial-to-pulmonary-artery-shunt (SAPAS) could result in sufficient growth of hypoplastic BPAs to permit successful correction of Tetralogy of Fallot (Kirklin et al., 1977). Only one out of 4 patients in that study had sufficient data to comment on BTTS-related BPA growth: the pre-BTTS RPA z-score was -9.84, and the post-BTTS z-score was -4.67. This patient went on to have successful correction of Tetralogy of Fallot. From this single case, the authors concluded that a BTTS could promote sufficient BPA growth to facilitate total correction of Tetralogy of Fallot. Although both the post-BTTS mean NI and corresponding RPA z-score increased in 19 out of 20 studies included, there is evidence that most (67%) of this increase was probably due to distension of the BPAs as a result of the pressure caused by increased BPA blood flow through the BTTS (Sabri et al., 1999). As the rationale for performing a BTTS is: 'use the BTTS to cause sufficient

BPA growth to facilitate total correction or definitive palliation,' the concept of 'Catch-up' growth was introduced. Catch-up growth refers to BTTS-induced BPA growth that is faster than somatic growth. For BTTS-induced BPA growth to increase the post-BTTS BPA size sufficiently (that is, make it normal) to facilitate total correction or definitive palliation, the BPA would have to grow faster than the rest of the body. Only one study was specifically designed to examine BTTS-related catch-up growth (Godart et al., 1998). They examined 78 patients and concluded that BTTSs do not result in catch-up growth of BPAs.

It would be reasonable to argue that: 'the longer a functioning BTTS remains in situ, the more the BPAs would grow.' Only one study examined the effect of the duration of the BTTS in situ. They found that a longer time in situ was not associated with greater catch-up BPA growth (Ross et al., 2015). Similarly, one could posit that the size of BTTS could affect the amount of BPA growth: smaller shunts would be expected to result in less growth than larger shunts. The optimal BTTS size has been suggested (Singh et al., 2014), and when we compared these suggestions with the shunts used in the studies included in our review, we found that it was likely that the ideal shunt size had been used in the majority of the studies. The modified BTTS (use of a prosthetic conduit) was introduced in 1981; the classical BTTS is rarely if ever used in contemporary practice (de Leval et al., 1981). As opposed to the classical BTTS, the modified BTTS has no growth potential as it is a prosthetic tube. Although the early rise in systemic arterial oxygen saturation (SaO₂) is essentially the same after classical and modified BTTSs (Awori et al., 2024), there is evidence that the SaO₂ difference increases with time after the classical BTTS (Bauersfeld et al., 1956). This late increase in SaO₂ rise may be as a result of classical BTTS growth, and this could theoretically result in catch-up growth of branch pulmonary arteries.

It is thought that antegrade blood flow across the right ventricular outflow tract (RVOT) could affect BPA growth in patients with a BTTS. Only one of the studies included examined this and found that anterograde flow did not alter BPA growth compared to patients who did not have antegrade flow (Lenoir et al., 2021). It has been suggested that the age at which the BTTS is performed has an effect on the amount of BPA growth. Three studies commented on how age at BTTS relates to BPA growth: Zhou et al. (2020) examined 90 patients and reported no BPA growth in patients who received a BTTS after 24 months of age. Ishikawa et al. (2001) examined 12 patients and reported less growth in patients who received a BTTS after 12 months of age. Sabri et al. (1999) examined 35 patients and found that age at BTTS has no effect on the amount of BPA growth. Based on these three studies, the evidence for age at BTTS affecting BPA growth is equivocal. The initial size of BPAs may be another factor that affects the BPA growth

after a BTTS; two studies made comments in this regard. Sabri et al. (1999) examined 35 patients and found that the initial size of the BPAs did not affect the amount of BPA growth after BTTS. Cotrufo et al. (1989) examined 26 patients and reported that less growth was seen after BTTS in patients with smaller BPAs. Available evidence of whether the initial size of BPA affects the amount of post-BTTS growth is also equivocal. Ullom et al. (1987) reported a BTTS operative mortality of 28% in patients with smaller BPAs. The question as to whether leaving a patent ductus arteriosus (PDA) open affects BPA growth after a BTTS has been raised. The fate of PDAs is not mentioned in 19 out of 20 articles (692 patients). Cotrufo reported that they found a large PDA in 10 out of 26 patients (38.5%); they left these PDAs open when they performed the BTTS. Although Lenoir et al. (2020) did not indicate how many PDAs they encountered or left open, they did report that they only closed a PDA if they performed a BTTS using a sternotomy for access; they used a sternotomy in 44 out of 77 patients (53.2%). Using this information, it would not be unreasonable to suggest that they left about half of the PDAs open. Although the numbers of patients with comments about the status of the PDA post-BTTS is small, the evidence suggests that the presence of a PDA does not affect BPA growth post-BTTS. One large study reported that PDA stenting resulted in significantly greater BPA growth in comparison to a BTTS; post-procedure NI of 158 and 131 respectively (Glatz et al., 2018). This difference may not be clinically significant but further study is justified. Lenoir et al. (2020) found that transannular patch-augmentation of the right ventricular outflow tract (RVOT) resulted in significantly more BPA growth than a BTTS; postoperative NI of 365 and 206 respectively. Similarly, this may not be clinically significant but warrants further study. By extension, RVOT stenting may have a similar effect to transannular patch augmentation and warrants further study too (Bigdelian et al., 2018).

Limitations of our study are as follows: it is a review of non-randomized retrospective studies; as such, there is the potential for bias and confounding. Of particular interest in this regard are: the size of BTTS, age at BTTS, pre-operative size of BPAs, and the duration of BTTS in situ. All these factors were examined to some degree, and it could be reasonably concluded that any bias or confounding would not be significant. The skill of the surgeon was not examined and this remains a potential source of bias or confounding. However, the nature of the surgical discipline of pediatric cardiac surgery makes it likely that most surgeons had adequate skill to execute the procedure correctly.

Conclusion

Available evidence, as outlined in this review, suggests that the modified BTTS does not produce 'catch-up'

growth of hypoplastic BPAs. Although BPA growth do occur post-BTTS, most of this growth appears to be due to distension of the BPAs. It seems unlikely that a modified BTTS can cause sufficient growth of truly hypoplastic BPAs to facilitate total correction or definitive palliation via the Fontan pathway. However, there remains the possibility that a classical BTTS may cause enough catch-up growth of branch PAs to allow total correction or the next stage of palliative surgery. If this is indeed the case, discussions about 'antegrade' RVOT flow, age at BTTS, or the pre-BTTS size of the BPA essentially become redundant; particularly with respect to modified BTTSs. Based on available evidence, we would not recommend the practice of performing a modified BTTS to grow hypoplastic BPAs to facilitate total correction or definitive palliation via the Fontan pathway. This is a significant departure from the traditional recommendations and implies that the main indications for a modified BTTS are: the first step on the Fontan Pathway and to improve oxygenation when total correction or cavo-pulmonary connection is not possible. However, there remains the possibility that a classical BTTS can be considered for catch-up growth of branch PAs to allow next palliative or corrective surgeries. Further study on PDA stenting, RVOT transannular-patch augmentation, and RVOT stenting, as means to induce BPA growth, are warranted.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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