Cardiol Young 2008; 18: 430–436
© Cambridge University Press
ISSN 1047-9511
doi:10.1017/S1047951108002357
First published online 25 June 2008

Original Article

The effects of surgically induced right bundle branch block on left ventricular function after closure of the ventricular septal defect

Thais A. L. Pedersen,[1] Niels H. Andersen,[2] Mette R. Knudsen,[1] Thomas D. Christensen,[1] Keld E. Sørensen,[2] Vibeke E. Hjortdal[1]

Departments of [1]Cardiothoracic and Vascular Surgery, and [2]Cardiology, Aarhus University Hospital, Skejby, Aarhus, Denmark

Abstract *Objective:* To determine the long-term significance of right bundle branch block on left ventricular systolic and diastolic function in children subsequent to surgical closure of ventricular septal defect. *Methods:* We studied 26 children who underwent surgical closure of a ventricular septal defect 11 ± 2 years postoperatively by use of conventional and tissue Doppler echocardiography, comparing the findings to those obtained from a control group. Of those having surgical correction 14 had postoperative right bundle branch block. *Results:* Irrespective of the presence of right bundle branch block, the peak systolic velocity of the mitral ring was lower in those undergoing surgical correction, with values of 5.2 ± 1.4 cm/s in those with right bundle branch block, 5.4 ± 1.2 cm/s in those without right bundle branch block after surgical correction, and 6.6 ± 1.0 cm/s in the control subjects ($p < 0.01$). In terms of diastolic function, the early septal velocity of transmitral inflow divided by the early diastolic mitral annular velocity was significantly higher in children with right bundle branch block, at 12 ± 3.0 cm/s compared to 8.4 ± 1.5 cm/s in the control subjects ($p < 0.01$), but not significantly higher in the children without right bundle branch block after correction compared to the control group. The fractional shortening percentage was similar in both patients and control subjects. The changes noted in left ventricular function were not significantly related to age at surgery, the period of follow-up, or the surgical method. *Conclusions:* Systolic long axis function is significantly reduced in children after surgical closure of ventricular septal defects, irrespective of the presence of right bundle branch block. Diastolic dysfunction, in contrast, was observed primarily in children with post-operative right bundle branch block.

Keywords: Congenital heart disease; echocardiography; surgery; post operative complications.

A VENTRICULAR SEPTAL DEFECT IS THE COMMONEST congenital cardiac malformation, and its surgical closure is the most frequent open-heart procedure performed in childhood.[1] Since parts of the cardiac conduction system in many cases are in close relation to the defect, injury can easily happen during repair.[2] While complete heart block currently only occurs postoperatively in less than 1% of children with ventricular septal defect,[3,4] right bundle branch block is seen in up to three-fifths of those operated through an atriotomy.[2] Whereas right bundle branch block in healthy subjects does not seem to carry any negative implications,[5] this apparently reassuring observation may not necessarily extend to children in whom this permanent abnormality of depolarization occurs in combination with other potential myocardial injuries, such as those caused by sutures and patches, perioperative myocardial damage, and perhaps longstanding preoperative ventricular volume

Correspondence to: Thais A. L. Pedersen, Department of Cardiothoracic and Vascular Surgery, Aarhus University Hospital, Skejby, DK-8200 Aarhus, Denmark. Tel: +45 8949 5481; Fax: +45 8949 6016; E-mail: thais.a.pedersen@ki.au.dk

Accepted for publication 27 February 2008

overload. Under these circumstances, left ventricular function may theoretically decline over time[6,7] analogous to the changes found in patients with left bundle branch block,[8] albeit not necessarily of similar clinical importance.

Echocardiography is the most widely used method for assessing cardiac function. Unfortunately, this technique has several limitations, particularly regarding the detection of early subclinical stages of left ventricular dysfunction.[9] Tissue Doppler imaging, however, not only provides quantitative assessment of regional myocardial function during the whole cardiac cycle, but also enhanced visual assessment of discrete abnormalities of regional wall motion.[10] It may, therefore, identify changes in cardiac performance that would otherwise remain undetected. We hypothesised that, over years, surgically induced right bundle branch block in children with ventricular septal defect would lead to subtle changes in left ventricular function detectable by sensitive modalities for imaging.

Materials and methods

During the period from 1990 to 1995, 123 children underwent surgical closure of a ventricular septal defect at our institution. In the 66 patients with a normal electrocardiogram at admission, who had their ventricular septal defect closed through a right atriotomy, 26 (39%) accepted our invitation to become involved in this follow-up study. The population consisted of 15 males and 11 females. Surgery was generally contemplated on the basis of medical history, physical findings, and echocardiographic examination. Cardiac catheterisations were only used occasionally in decision-making.

We reviewed the 12 lead electrocardiogram obtained at discharge in all patients to identify any postoperative occurrence of right bundle branch block, defined as an rSR′ pattern in lead V1 or V2, with a wide slurred S wave in V5 or V6, and a duration of the QRS complex exceeding 0.12 seconds. We recruited 26 age and gender matched healthy children with a normal electrocardiogram as controls. The study was approved by our local Ethical Committee, and the parents of all patients and healthy controls gave their informed consent.

Surgery

All patients had undergone surgery through a median sternotomy, and put on cardiopulmonary bypass with the aorta cross-clamped. Moderate hypothermia and crystalloid cardioplegia was used throughout the whole period. The cardiac defect was approached through a right atrial incision. In 12 children, a small ventricular septal defect had been closed by direct suture, whereas the remaining 14 patients required insertion of a Dacron patch.

Echocardiography

All echocardiographic examinations were performed by the same two observers, and all off-line analyses after the event was performed randomly and blindly with respect to clinical data, including the electrocardiogram. Echocardiograms were performed on a GE Vivid Seven (GE Healthcare, Horten, Norway) using a 2.5 MHz transducer. Images were obtained from the parasternal and apical views. Left ventricular dimensions and mural thicknesses were obtained from standard parasternal M-mode recordings, and based on 5 consecutive cardiac cycles.[11]

Systolic function

Radial left ventricular function was assessed by conventional fractional shortening derived from the parasternal view.[11] Left ventricular longitudinal function was examined from tissue Doppler based assessment of the basal-apical displacement, from peak systolic myocardial velocities and from tissue tracking with the tissue Doppler sample volumes, measuring 6 by 6 millimetres, placed in the lateral and medial part of the mitral ring.[12] In all cases, frames per second were above 140. The peak systolic velocity was defined as the highest value between the R-wave on the electrocardiogram and the timing of closure of the aortic valve, whereas tissue tracking, which displays the distance of motion during systole along the Doppler axis,[13] was defined as the peak displacement, expressed in millimetres, during the same time span. Timing of closure of the aortic valve was defined from a curved anatomical M-mode recording placed through the aortic valvar leaflets in the apical long axis view. Tissue Doppler values are presented as the mean from 3 consecutive heart cycles.

Longitudinal motion has been previously validated in children[14] and the intra-observer variability of tissue tracking and velocity recordings is known to be below 10%.[12]

Diastolic function

Pulsed Doppler measurements of left ventricular filling were obtained in the apical four-chamber view with the Doppler beam aligned perpendicularly to the plane of the mitral annulus, and the sample volume placed between the tips of the mitral valvar leaflets. We acquired 5 consecutive beats obtained during quiet respiration for calculating mean early and late diastolic mitral annular velocities, as well as for determining the early diastolic mitral deceleration time.

https://doi.org/10.1017/S1047951108002357 Published online by Cambridge University Press

Left ventricular myocardial diastolic properties were assessed using tissue Doppler imaging. Measurement of the peak diastolic early and late diastolic mitral annular velocities in the lateral and septal mitral annulus were performed in the four chamber view.[15,16] Results are presented as the mean of 3 consecutive heart cycles (Fig. 1).

Since measurements of myocardial velocities using off-line tissue Doppler are lower than if acquired with spectral tissue Doppler technique,[17] the ratio of velocity of early transmitral inflow divided by the early diastolic mitral annular velocity is overestimated compared to studies using only spectral tissue Doppler.[16,18]

Statistics

Data were analyzed for normal distribution using the Shapiro-Wilk test and, if normal distributed, presented as means ± standard deviation. Ratios were compared using Fischer's exact test.

We compared the findings in the group of children found to have right bundle branch block after closure of the ventricular septal defect to those in the children not having this finding after surgical closure, and to the findings in the control subjects using one-way ANOVA. An un-paired t-test was used for comparisons between the groups, taking a value of p less than 0.05 when 2-tailed to be statistically significant. The statistic calculations were done with SPSS 14.0 (SPSS Inc., Illinois, US).

Results

Demographics and clinical data are displayed in Table 1. We found that 14 children (54%) had developed right bundle branch block postoperatively, with 12 (46%) having preserved intraventricular conduction. The state of conduction had not changed over the years. Associated cardiac malformations had existed in 8 of the patients, with

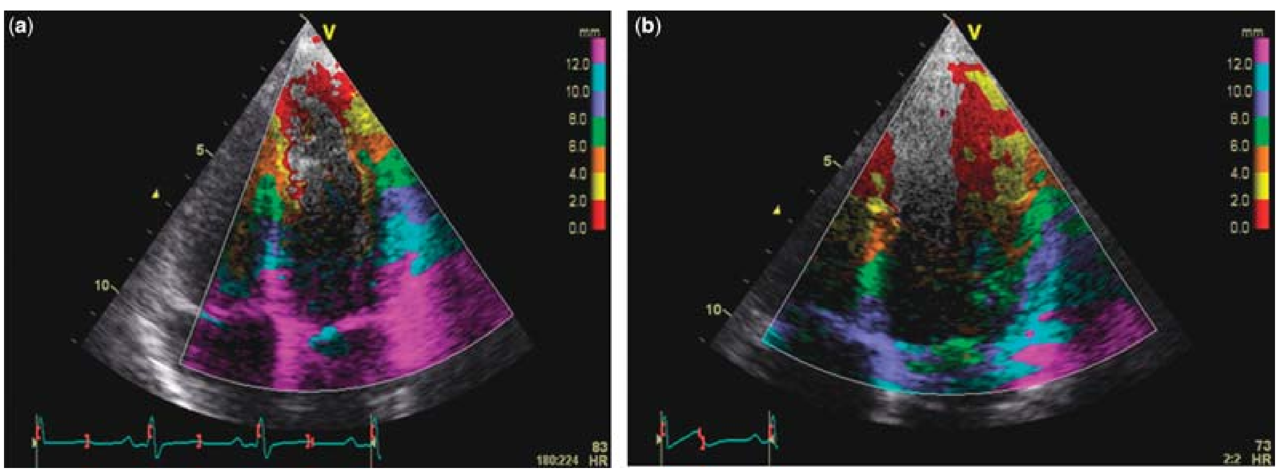

Figure 1.
*Tissue tracking images of the systolic function. Panel **a** illustrates a normal examination from a 12 year old boy with normal long axis function and 7 colour bands displayed with a peak systolic displacement of the mitral ring exceeding 12 millimetres. Panel **b** displays a similar recording from a 12 year old boy after surgical closure of a ventricular septal defect in infancy. Long axis displacement of the septum and septal part of the mitral ring are reduced with only 5 colour bands displayed. The lateral displacement is normal. VSD: ventricular septal defect.*

Table 1. Demographic and clinical data.

	VSD + RBBB 14 patients	VSD – RBBB 12 patients	Control group 26 subjects
Females/males	6/8	5/7	11/15
Systolic pressure (mmHg)	114 ± 18	107 ± 14	110 ± 12
Diastolic pressure (mmHg)	68 ± 6	65 ± 7	62 ± 7
Heart rate (bpm)	64 ± 11	65 ± 13	66 ± 12
Weight (kg)	51 ± 15	53 ± 13	53 ± 13
Height (cm)	162 ± 13	162 ± 12	160 ± 12
Age at surgery (years)	2 ± 1	2 ± 1	–
Age at follow-up (years)	13 ± 2	13 ± 4	13 ± 3

VSD: ventricular septal defect; RBBB: right bundle brunch block; mmHg: millimetres of mercury; bpm: beats per minute; Kg: kilogram; cm: centimeters.

https://doi.org/10.1017/S1047951108002357 Published online by Cambridge University Press

coarctation of the aorta present in 4, atrial septal defect in 3, and patency of the arterial duct in 1. All 4 children with coarctation had undergone aortic repair prior to closure of the ventricular septal defect, while the other associated malformations were corrected at the time of closure of the ventricular septal defect. Blood pressure, heart rate, weight and height were similar in all three groups. Follow-up exceeded 10 years, and was similar in both surgical subgroups. All patients were asymptomatic and at school, with normal effort tolerance and without medical treatment. All had normal blood pressure and none had aortic or mitral regurgitation. Only one child with right bundle branch block had a tiny residual ventricular septal defect. There was no evidence of recoarctation in the four patients previously undergoing repair.

The conventional and tissue Doppler derived echocardiographic results are summarized in Table 2 and Figure 2. There were no differences in left ventricular dimensions or fractional shortening between patients with or without right bundle branch block and in controls.

The systolic long axis function, as reflected by the peak systolic myocardial velocity, was reduced to the same extent in both surgical groups compared to controls, irrespective of the presence of right bundle branch block (Table 2). Similarly, the medial displacement of the mitral ring as measured from the tissue tracking was significantly lower in both groups undergoing surgical closure, whereas the lateral displacement did not differ between the three subgroups (Fig. 2). The changes in systolic function were neither related to age at surgery, to follow-up period nor to whether the defect had been closed by insertion of a patch or direct suture.

The indexes of diastolic mitral inflow were similar in both groups undergoing surgical closure, and not different from the healthy subjects (Table 2). Lateral and septal peak early diastolic mitral annular velocities derived using tissue Doppler, however, were significantly lower in the patients, although not dissimilar according to the presence or absence of right bundle branch block (Table 2). The late diastolic mitral annular tissue velocity derived at the lateral part of the ring was also lower in both

Table 2. Echocardiography.

	VSD + RBBB 14 patients	VSD − RBBB 12 patients	Control group 26 subjects	ANOVA p-value
Dimensions				
Left ventricular diastolic diameter (mm)	46 ± 8	43 ± 7	46 ± 6	0.17
Left ventricular systolic diameter (mm)	31 ± 6	31 ± 5	31 ± 3	0.96
Septal wall thickness (mm)	9 ± 1	9 ± 1	8 ± 1	0.42
Posterior wall thickness (mm)	8 ± 2	8 ± 1	7 ± 2	0.20
Systolic function				
Septal peak systolic velocity (cm/s)	5.2 ± 1.4*	5.4 ± 1.2*	6.6 ± 1.0	<0.01
Lateral peak systolic velocity (cm/s)	6.0 ± 1.7*	6.0 ± 1.0*	8.5 ± 1.7	<0.01
Fractional shortening (percent)	32.3 ± 5.5	30.8 ± 5.2	33.1 ± 3.4	0.35
Diastolic function				
Mitral E-wave velocity (cm/s)	102 ± 17	95 ± 17	99 ± 12	0.69
Mitral A-wave velocity (cm/s)	47 ± 8	48 ± 9	51 ± 10	0.48
E/A ratio	2.2 ± 0.5	2.0 ± 0.4	2.1 ± 0.5	0.59
Deceleration time (ms)	140 ± 16	136 ± 20	150 ± 17	0.13
Septal E′ velocity (cm/s)	9.0 ± 1.7*	9.1 ± 1.2*	12.1 ± 1.4	<0.01
Septal A′ velocity (cm/s)	3.1 ± 1.2	3.1 ± 1.3	3.8 ± 1.2	0.21
Lateral E′ velocity (cm/s)	12.1 ± 3.0*	12.4 ± 3.0*	14.8 ± 2.0	<0.01
Lateral A′ velocity (cm/s)	1.9 ± 0.7*	2.0 ± 1.1*	3.3 ± 1.4	<0.01
Septal E/E′ ratio	12 ± 3.0*	10.6 ± 4.0†	8.4 ± 1.5	<0.01
Lateral E/E′ ratio	9.4 ± 4.0*	7.5 ± 3.8	6.8 ± 1.1	<0.01

*p < 0.05 compared to control group.
†p = 0.13 compared to control group.
VSD: ventricular septal defect.
RBBB: right bundle brunch block.
mm = millimetres.
cm/s = centimetres per second.
ms = milliseconds.
mmHg: millimetres of mercury.
E/A ratio: transmitral inflow early velocity divided by late diastolic mitral annular velocity; E′ velocity: early diastolic mitral annular velocity.
A′ velocity: late diastolic mitral annular velocity.
E/E′ ratio: transmitral inflow early velocity divided by early diastolic mitral annular velocity.

https://doi.org/10.1017/S1047951108002357 Published online by Cambridge University Press

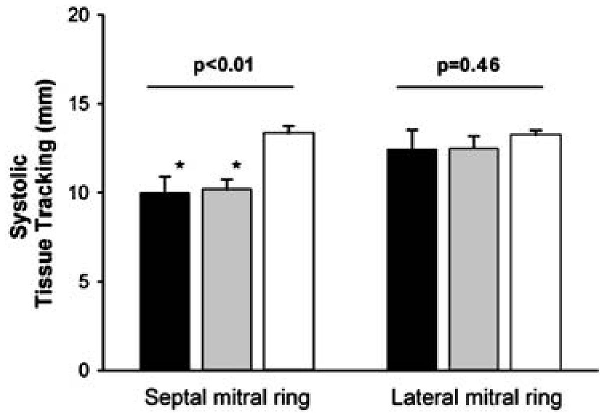

Figure 2.
Peak systolic tissue tracking derived from the lateral and septal parts of the mitral ring. The bars indicate mean values and standard errors of the mean. Black bars: patients with right bundle branch block after closure of the ventricular septal defect. Grey bars: patients without right bundle branch block after closure of the ventricular septal defect. White bars: Control group. *$p < 0.05$*: patients versus controls; RBBB: right bundle branch block; VSD: ventricular septal defect.*

sets of patients, but not particularly in the subgroup with right bundle branch block. The late diastolic mitral annular velocities derived at the septal aspect of the ring were also similar in all three groups.

Regarding the ratios of transmitral inflow early velocity divided by early diastolic mitral annular velocity, which combine the inter- and intramyocardial diastolic properties, the values derived both septally and laterally from the children with right bundle branch block were significantly higher than the control group. There was a similar tendency in the children without right bundle branch block, albeit that these values did not show statistical significance. Furthermore, there were no significant differences between the children with and without right bundle branch block after surgical closure (Table 2). The diastolic abnormalities were not related to age at surgery, the period of follow-up, nor whether the defect was closed by direct suture or insertion of a patch.

Discussion

Right bundle branch block is a frequent finding after surgical repair of congenitally malformed hearts,[3,4] and has in older reports been seen in more than half of children after closure of ventricular septal defects.[2,3] Our data, acquired in a more recent cohort, confirms the high prevalence of this abnormality in conduction, even when access to the defect was through the right atrium. Isolated occurrence of right bundle branch block in presumed healthy young subjects is only seen in 1%,[19] and has so far been considered a benign phenomenon even in the long term.[5]

Experience drawn from biventricular pacing studies in adults with congestive heart failure, nonetheless, suggests that alleviation of the abnormality in conduction, and the associated electro-mechanical dyssynchrony, can improve cardiac function, indicating that right bundle branch block is of clinical importance at least in patients with underlying cardiovascular problems, including those with tetralogy of Fallot.[6,20]

Although we hypothesized that postoperative right bundle branch block in children with ventricular septal defect would lead over time to changes in left ventricular function secondary to the altered right-sided depolarisation, the detailed echocardiographic evaluation only identified minor, albeit significant, abnormalities in the ratios of transmitral inflow early velocity divided by early diastolic mitral annular velocity in the patients with right bundle branch block. All other echocardiographic parameters, including the less robust velocities of mitral inflow, were similar in the subjects with and without right bundle branch block after closure of their septal defects. These observations are in accordance with data obtained in patients after repair of tetralogy of Fallot, in whom the presence of right bundle branch block also has been associated with some degree of left ventricular asynchrony, and secondary reductions in left ventricular diastolic function. It must be acknowledged, nonetheless, that the haemodynamics after repair of tetralogy of Fallot are far more complex, and often significantly influenced by severe pulmonary regurgitation and right ventricular overload and dilation.[6,21]

Since diastolic function usually is affected prior to systolic performance, it may well be that the changes attributable to the right bundle branch block are of minor importance. This is supported by the fact that the diastolic difference between the patients with and without right bundle branch block could only be detected with sensitive tissue Doppler techniques, but not on conventional measurements of mitral inflow. It was also the case that the numeric differences in the ratios obtained septally and laterally from the early velocities were small.

Although our study failed to identify major abnormalities specifically in the patients showing right bundle branch block after closure of their defects, the data surprisingly revealed that left ventricular long axis function as assessed by systolic displacement and displacement velocities was significantly reduced more than a decade after surgical correction. Circumferential shortening as indicated by the fractional shortening, in contrast, was not reduced, and ventricular dimensions not enlarged.

Abnormalities identified using tissue Doppler also included indexes of diastolic myocardial performance, whereas the parameters for mitral inflow were normal. Neither the systolic nor the diastolic abnormalities were related whether the defect was closed by insertion of a patch or by direct suture, nor to the age at surgery or the length of follow-up.

The abnormalities in left ventricular systolic and diastolic function detected with tissue Doppler imaging remain unexplained, but may have a multifactorial background. Although it might be intuitive to expect the changes observed to be induced by the surgical procedure, such as the myocardial protection or the surgical trauma itself, preoperative factors may also be involved. Thus, changes in turnover of collagen, and other intrinsic tissue abnormalities in the muscular ventricular septum related to the pathogenesis of the defect, could lead to functional impairment of myocardium adjacent to the defect. Such abnormalities have been described in children with tetralogy of Fallot and pulmonary atresia, in whom disarrayed bundles of collagen, and increased expression of collagen III, have been found in the septal region.[22] Postoperatively, a large patch will undoubtedly influence septal function negatively, and fibrosis either in the sutured area or around the patch could also lead to septal stiffness, influencing systolic and diastolic parameters sufficiently to make them detectable with advanced echocardiographic techniques.[6,23] The changes specifically seen in the ventricular septum could represent the persistence of the abnormalities of septal motion seen early postoperatively after cardiac surgery in children and adults.[24,25]

It is possible that the mechanisms for septal injury mentioned above also explain the reduction in the lateral displacement of the mitral ring, and thus the more global reduction in left ventricular function observed in the study, although it may seem surprising that left ventricular fractional shortening as an index of radial function remained normal. It is also possible that more advanced techniques would have disclosed minor changes in radial function. If the reduction in long axis function is secondary to the cumulative effect of the multiple perioperative myocardial risk factors acting immediately during or after the surgical trauma, the changes would be expected to be transient, rather than leading to a permanent reduction in ventricular function as indicated by our current observations. To our knowledge, this has neither been studied in detail nor prospectively, but only in cross-sectional designs and in studies with very short follow-up intervals, which make it impossible to evaluate the temporal evolution of these abnormalities.[24–26]

Interestingly, a study comparing closure of atrial septal defects surgically and by insertion of devices percutaneously has similarly demonstrated impaired diastolic function after surgical repair.[24] In the children who underwent percutaneous closure, diastolic filling seemed to improve after treatment, which suggests that open heart surgery directly may impair not only diastolic but also systolic function.[24–26]

The clinical impact of the abnormalities in myocardial function seen in the children undergoing surgery in our study remains speculative. A similar study performed on children after repair of tetralogy of Fallot revealed left ventricular asynchrony in over half of the patients with right bundle branch block, which was associated with reduced regional and global left ventricular function.[6] Moreover, the abnormal ratio of transmitral inflow early velocity divided by early diastolic mitral annular velocity, which was observed in the subset with right bundle branch block, correlates well with elevated pulmonary capillary wedge[16,18] and left ventricular end-diastolic pressure.[16,18,27] Such higher ratios may reflect stiffness and reduced compliance of the left ventricle in this subset, and could theoretically influence their exercise capacity.[28] Similarly, the altered functional properties of the left ventricle may theoretically increase the arrhythmogenic potential, and thus the prevalence of atrial fibrillation as has been described after surgical closure of ventricular septal defect.[29,30]

Our present study, of course, is limited by the relatively small number of subjects studied. The cross-sectional design also means that we cannot determine the order of events that eventually lead to subclinical systolic and diastolic dysfunction noted in our children after surgical closure of their ventricular septal defects.

In conclusion, systolic long axis function is significantly reduced after surgical closure of ventricular septal defects in children, irrespective of the presence of right bundle branch block. Diastolic dysfunction, in contrast, seemed primarily related to the post-operative presence of right bundle branch block.

Acknowledgments

We appreciate greatly the skilled assistance of Bente Mortensen with the echocardiographic and tissue Doppler measurements. We acknowledge also the financial support provided by the research foundation at Aarhus University Hospital, Skejby. There are no competing interests to report.
Conflicts of interest: None

References

1. Roos-Hesselink JW, Meijboom FJ, Spitaels SE, et al. Outcome of patients after surgical closure of ventricular septal defect at young age: longitudinal follow-up of 22–34 years. Eur Heart J 2004; 25: 1057–1062.
2. van Lier TA, Harinck E, Hitchcock JF, et al. Complete right bundle branch block after surgical closure of perimembranous ventricular septal defect. Relation to type of ventriculotomy. Eur Heart J 1985; 6: 959–962.
3. Andersen HO, de Leval MR, Tsang VT, et al. Is complete heart block after surgical closure of ventricular septum defects still an issue? Ann Thorac Surg 2006; 82: 948–956.
4. Smerup M, Hjertholm T, Johnsen SP, et al. Pacemaker implantation after congenital heart surgery: risk and prognosis in a population-based follow-up study. Eur J Cardiothorac Surg 2005; 28: 61–68.
5. Fleg JL, Das DN, Lakatta EG. Right bundle branch block: long-term prognosis in apparently healthy men. J Am Coll Cardiol 1983; 1: 887–892.
6. Abd El Rahman MY, Hui W, Yigitbasi M, et al. Detection of left ventricular asynchrony in patients with right bundle branch block after repair of tetralogy of Fallot using tissue-Doppler imaging-derived strain. J Am Coll Cardiol 2005; 45: 915–921.
7. Zannad F, Huvelle E, Dickstein K, et al. Left bundle branch block as a risk factor for progression to heart failure. Eur J Heart Fail 2007; 9: 7–14.
8. Bax JJ, Bleeker GB, Marwick TH, et al. Left ventricular dyssynchrony predicts response and prognosis after cardiac resynchronization therapy. J Am Coll Cardiol 2004; 44: 1834–1840.
9. Ho CY, Solomon SD. A clinician's guide to tissue Doppler imaging. Circulation 2006; 113: e396–e398.
10. Swaminathan S, Ferrer PL, Wolff GS, et al. Usefulness of tissue Doppler echocardiography for evaluating ventricular function in children without heart disease. Am J Cardiol 2003; 91: 570–574.
11. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. Eur J Echocardiogr 2006; 7: 79–108.
12. Andersen NH, Poulsen SH. Evaluation of the longitudinal contraction of the left ventricle in normal subjects by Doppler tissue tracking and strain rate. J Am Soc Echocardiogr 2003; 16: 716–723.
13. Pan C, Hoffmann R, Kuhl H, et al. Tissue tracking allows rapid and accurate visual evaluation of left ventricular function. Eur J Echocardiogr 2001; 2: 197–202.
14. Arce OX, Knudson OA, Ellison MC, et al. Longitudinal motion of the atrioventricular annuli in children: reference values, growth related changes, and effects of right ventricular volume and pressure overload. J Am Soc Echocardiogr 2002; 15: 906–916.
15. Nagueh SF, Mikati I, Kopelen HA, et al. Doppler estimation of left ventricular filling pressure in sinus tachycardia. A new application of tissue doppler imaging. Circulation 1998; 98: 1644–1650.
16. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation 2000; 102: 1788–1794.
17. McCulloch M, Zoghbi WA, Davis R, et al. Color tissue Doppler myocardial velocities consistently underestimate spectral tissue Doppler velocities: impact on calculation peak transmitral pulsed Doppler velocity/early diastolic tissue Doppler velocity (E/Ea). J Am Soc Echocardiogr 2006; 19: 744–748.
18. Nagueh SF, Middleton KJ, Kopelen HA, et al. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol 1997; 30: 1527–1533.
19. Pelliccia A, Culasso F, Di Paolo FM, et al. Prevalence of abnormal electrocardiograms in a large, unselected population undergoing pre-participation cardiovascular screening. Eur Heart J 2007; 28: 2006–2010.
20. Garrigue S, Reuter S, Labeque JN, et al. Usefulness of biventricular pacing in patients with congestive heart failure and right bundle branch block. Am J Cardiol 2001; 88: 1436–1441, A8.
21. D'Andrea A, Caso P, Sarubbi B, et al. Right ventricular myocardial activation delay in adult patients with right bundle branch block late after repair of Tetralogy of Fallot. Eur J Echocardiogr 2004; 5: 123–131.
22. Peters TH, de Jong PL, Klompe L, et al. Right ventricular collagen and fibronectin levels in patients with pulmonary atresia and ventricular septal defect. Mol Cell Biochem 2003; 251: 27–32.
23. Brili S, Alexopoulos N, Latsios G, et al. Tissue Doppler imaging and brain natriuretic peptide levels in adults with repaired tetralogy of Fallot. J Am Soc Echocardiogr 2005; 18: 1149–1154.
24. Hanseus KC, Bjorkhem GE, Brodin LA, et al. Analysis of atrioventricular plane movements by Doppler tissue imaging and M-mode in children with atrial septal defects before and after surgical and device closure. Pediatr Cardiol 2002; 23: 152–159.
25. Waggoner AD, Shah AA, Schuessler JS, et al. Effect of cardiac surgery on ventricular septal motion: assessment by intraoperative echocardiography and cross-sectional two-dimensional echocardiography. Am Heart J 1982; 104: 1271–1278.
26. Lehmann KG, Lee FA, McKenzie WB, et al. Onset of altered interventricular septal motion during cardiac surgery. Assessment by continuous intraoperative transesophageal echocardiography. Circulation 1990; 82: 1325–1334.
27. Liang HY, Cauduro SA, Pellikka PA, et al. Comparison of usefulness of echocardiographic Doppler variables to left ventricular end-diastolic pressure in predicting future heart failure events. Am J Cardiol 2006; 97: 866–871.
28. Meijboom F, Szatmari A, Utens E, et al. Long-term follow-up after surgical closure of ventricular septal defect in infancy and childhood. J Am Coll Cardiol 1994; 24: 1358–1364.
29. Bol RG, Meijboom FJ, Kappetein AP, et al. Long-term follow-up and quality of life after closure of ventricular septal defect in adults. Eur J Cardiothorac Surg 2007; 32: 215–219.
30. Li W, Somerville J. Atrial flutter in grown-up congenital heart (GUCH) patients. Clinical characteristics of affected population. Int J Cardiol 2000; 75: 129–137.