Eur J Echocardiography (2007) **8**, S2—S12

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EUROPEAN SOCIETY OF CARDIOLOGY®

REVIEW PAPER

The use of contrast echocardiography for the detection of cardiac shunts

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Available online 25 April 2007

KEYWORDS
Contrast echocardiography;
Shunts;
Paradoxical embolism;
Stroke;
Decompression sickness of divers;
Platypnea—orthodeoxia syndrome

Abstract Recently, debate has erupted about the clinical significance of cardiovascular shunts. Several major health problems such as stroke and migraine have been associated with patent foramen ovale (PFO) with right-to-left shunt (RLS). The nature of the relationship between these syndromes and PFO is not clearly understood. Technical advances have led to more therapeutic options including device closure of PFO, hence prevention of such a PFO-related stroke has become feasible. Therefore, optimal diagnosis of PFO has become of greater clinical importance. Contrast echocardiography with non-transpulmonary contrast agents has been the cornerstone in diagnosis of PFO with RLS for over four decades. Despite being a relatively invasive procedure, transesophageal echocardiography (TEE) is considered the gold standard for detection of RLS. Several other echocardiographic techniques such as transthoracic echocardiography (TTE) with second harmonic imaging and transcranial Doppler ultrasonography (TCD) have shown increased sensitivity and specificity compared to TEE for the detection of PFO with RLS. Moreover, improvement of skills and techniques used for detection of these shunts has led to greater detection of small and large sized RLS in the echocardiographic laboratory. This review gives and overview of the echocardiographic techniques, contrast agents and manoeuvres used for detection of the major cardiovascular shunts and their clinical relevance to major health problems.

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1525-2167/$32
doi:10.1016/j.euje.2007.03.006

Introduction

Although the clinical significance of cardiovascular shunts in relation to cerebrovascular accidents is still controversial,[1–4] detection and assessment of these shunts have become a routine diagnostic procedure in many echocardiographic laboratories especially in centers with an active cerebrovascular stroke unit. In the literature to-date, both the rate of shunt detection and the clinical interpretation of the shunts found have varied considerably. In some studies[5] a patent foramen ovale (PFO) with right-to-left-shunt (RLS) has been detected in about 10% of a cerebral stroke population, while others have reported an incidence of >50%.[6] This might be explained by differences in the patient population included, type of cerebral infarction (all cerebral infarction included or only cortical infarction which, is more likely to be caused by embolization than lacunar infarction), age of the patients, comorbidity (particularly hypertension and diabetes), and also by the skills and techniques used for detection of these shunts in the echocardiographic laboratories.

Since the first report in 1968 by Gramiak,[7] contrast echocardiography has become an indispensable tool for cardiovascular imaging. The primarily used aircontained contrast agents such as agitated saline were not able to pass the pulmonary circulation; in the normal circulation no contrast agent would appear in the left side of the heart, which implied that it was useless for the analysis of left-sided cardiac morphology. This property, however, forms the basis for visualization of right-to-left shunts (RLSs): if contrast appears in the left side of the heart, there must be a shunt. Currently, a variety of echocardiographic techniques are available for visualization of cardiac shunts, such as transthoracic (TTE) and transesophageal (TEE) echocardiography and transcranial Doppler ultrasonography (TCD). In this article, the value of contrast echocardiography for detection of shunts and its clinical implications are discussed. Emphasis will be on the four most common shunt types: PFO, atrial septal defect (ASD), ventricular septal defect (VSD) and pulmonary arterio-venous malformations (PAVMs).

Principles of contrast echocardiography

Microbubble generation

The established theory is that the contrast effect is based on microbubbles that are already present in the solution. However, the intensity of the contrast effect can be enhanced by rapid injection of contrast (see later).[8]

Echogenicity

The contrast effect of microbubbles depends on the difference in density at the interface between gas-contained microbubbles and the surrounding tissue, which is known as acoustic impedance. The higher the acoustic impedance, the more echogenic the interface, since gas is 100 000 times less dense than blood, gas-contained microbubbles are excellent contrast agents.[9]

Echo-contrast agents

The most commonly used contrast agent for the detection of shunts is agitated saline. However, several solutions (see Table 1) have the potential to be used for detection of shunts.

Echocardiographic techniques

Several echocardiographic techniques can be used for detection of shunts (including TTE, TEE and TCD), because of superior resolution TEE is the most commonly used technique for detection of shunts especially in the current era if PFO closure is considered.[10,11] However, a good Valsalva manoeuvre (see later) is often more difficult to obtain from a patient during TEE study, especially if he/or she is heavily sedated than can do a patient during TTE study. The most relevant contrast studies that compared two or more of the echocardiographic techniques for the detection of shunts are summarized in Table 2.

Table 1 Echo-contrast agents commonly used for shunt detection

Agents	Recommended dose (ml)
Saline/blood/air	9 ml 0.9% saline/1 ml blood + 0.1 ml air
Dextrose 5% water	10
D-galactose microparticle solution (Echovist®)	5–10
Urea-linked gelatin (Haemaccel®)[a]	10
Oxypolygelatine (Gelifundol®)[22]	10

[a] Main agent in our echo-lab used for shunt detection.

Table 2 Contrast echocardiography for detection of right-to-left shunts (transesophageal echocardiography used as gold standard)

Authors	Year	Patients	Echocardiographic technique	Contrast agent	Patients with RLS at TEE	Sensitivity	Specificity
Di Tullio[70]	1993	49	TCD	Saline	19	68	100
Job[71]	1994	137	TCD	Gelifundol®	64	89	92
Klötzsch[72]	1994	111	TCD	Echovist®	50	91	94
Devuyst[73]	1997	37	TCD	Saline	24	100	100
Hamann[17]	1998	44	TCD	Echovist®	22	75	100
Stendel[11]	2000	92	TCD	Echovist®	24	92	97
Droste[74]	2000	64	TCD	Echovist®	20	100	65
Di Tullio[70]	1993	49	TTE-FI	Saline	19	47	100
Kuhl[75]	1999	111	TTE-FI	Gelifundol®	51	62	100
			TTE-HI	Gelifundol®	51	92	100
Stendel[11]	2000	92	TTE-FI	Echovist®	24	42	83
Ha[16]	2000	136	TTE-FI	Saline	40	22	100
			TTE-HI	Saline	40	63	100
Van Camp[76]	2000	109	TTE-FI	Saline	24	46	100
			TTE-HI	Saline	24	100	100
Daniels[77]	2004	256	TTE-HI	Saline	53	91	97

FI = fundamental imaging, HI = harmonic imaging, TCD = transcranial Doppler ultrasonography, TTE = transthoracic echocardiography.

Preparation and safety of contrast agents

A 20-Gauge or more cannula is placed in either an antecubital or femoral vein and 0.9% saline with blood from the patient is agitated between two 10 ml syringes connected to a three-way stopcock. A range of 5—20 ml saline/blood mixed with 0.2—1 ml air has been used. Alternatively, 5—10 ml Echovist®, a galactose-based contrast agent, can be used. All non-transpulmonary agents are safe when injection of large microbubbles is prevented.[12]

Provocative tests

The majority of RLSs cannot be seen during conventional TEE and TCD.[13,14] Therefore, provocative manoeuvres such as Valsalva or coughing are used to disclose transient RLSs. Coughing or releasing a sustained Valsalva manoeuvre results in increased filling of the right atrium and therefore a right-to-left atrial pressure gradient develops with opening of the foramen ovale (Fig. 1).[15] Abdominal compression may be used instead if the patient is deeply sedated during TEE, however it seems not sensitive as a good Valsalva manoeuvre. Kronik et al.[13] were the first to demonstrate the effect of the Valsalva manoeuvre for the detection of RLSs. Later on, Lynch et al.[14] demonstrated that in healthy volunteers the detection of PFO increased from 5% to 18% using the Valsalva manoeuvre. Likewise, a 3—4 times increase in sensitivity for shunt detection was shown for TEE.[16]

Femoral versus antecubital vein

In some reports[17—19] it was shown that the sensitivity of TCD for detection of PFO was markedly increased when a femoral vein is used for contrast injection rather than a antecubital vein. This is probably explained by different inflow patterns into the right atrium: inferior vena caval flow is directed to the atrial septum, whereas superior vena caval flow is directed to the tricuspid valve.

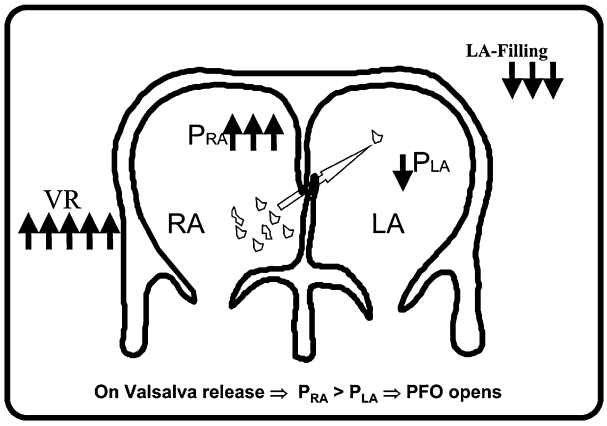

Figure 1 Mechanism of unrevealing right-to-left shunt by Valsalva manoeuvre, an arrow with head up means increase and head down is decrease, number of arrows represent how much is the change. Abbreviations: LA = left atrium, RA = right atrium, P_{LA} = left atrial pressure, P_{RA} = right atrial pressure and VR = venous return.

Practical disadvantages are the more vascular complications such as arterio-venous fistula when the femoral vein is used; this explains why this is hardly used in clinical practice.

Diagnosis of intra-cardiac shunts by contrast echocardiography

Normally, right atrial cardiac pressure is lower than the left atrial pressure. However, during isovolumic contraction and early ventricular diastole transient periods with a positive right-to-left atrial pressure gradient can occur with opening of the foramen ovale.[20] This fact explains why the use of colour-Doppler echocardiography is mainly applicable for detection of left-to-right shunts (LRSs) but not for transient RLSs.

Positive contrast effect

Since contrast microbubbles with a diameter ≥ 9 μm do not pass the pulmonary capillary circulation, any appearance of intravenously injected microbubbles in the left side of the heart is considered positive for an RLS. However, timing of contrast appearance in the left atrium and in particular the exact amount of contrast to define a positive test is still controversial.[21]

A negative contrast effect

A negative contrast effect is a sharply delineated washout phenomenon appearing on the right atrial side of the inter-atrial septum in continuity with the contrast-free left atrium and indicates inter-atrial LRS.[22] A negative contrast effect is consistent with ASD, with a considerably wide range in sensitivity.[22] However, with the current use of colour-Doppler imaging the use of contrast for detection of ASD is less important. More importantly, in our practice we use the contrast after closure of ASD to detect any residual shunt after closure (see later). Pitfalls of the positive and negative contrast effects are summarized in Table 3.

Semi-quantification of shunt

The principle of semi-quantification is based on counting the number of microbubbles crossing from the right to the left atrium within the first three cardiac cycles from right-sided contrast opacification. However, there is no uniform definition in the literature about the number of microbubbles that should appear in the left atrium; either 1,[23] 3[2,24] or 5[25] microbubbles was considered positive for PFO. Small, moderate, and large PFOs may be defined as less than 10, more than 10, and full opacification of the left atrium with microbubbles, respectively.[26] With TCD a ''curtain'' or ''shower'' pattern (indicating a large RLS) may be associated with the highest risk of cryptogenic stroke.[27] The number of microbubbles passing the PFO is often dependent on the quality of the Valsalva manoeuvre. In case of absent (no microbubbles) or small (few microbubbles) appeared in the left atrium, the study should be repeated using optimal Valsalva manoeuvres at least once. With varying results in one

Table 3 Pitfalls of contrast echocardiography for shunt detection

False test results	Possible mechanism and solution
False positive contrast effect	
Valsalva effect	Stagnation of blood in the pulmonary veins during strain phase of Valsalva, upon release a rouleaux formation from stagnant blood flowing into left atrium.[78] Repeat the Valsalva manoeuvre without contrast.
Large Eustachian valve	A fibro-muscular crescentic fold located at the junction of right atrium and inferior vena cava that can be mistaken for inter-atrial septum. Injected contrast into antecubital vein is streamed along the fold to the right ventricle directly. Use femoral venous injection.[17,18]
False negative contrast effect	
Inadequate contrast injection	Inadequate opacification of right atrium. Increase the dose or optimize machine settings.
Inadequate Valsalva manoeuvre	Failure of the Valsalva manoeuvre to increase the right atrial pressure above left atrial pressure. Either the patient ineffectively performs Valsalva manoeuvre or due to a persistently elevated left atrial pressure. The left-sided cardiac pathology is probably seen clearly by echocardiography. RLSs could not be excluded.
Large Eustachian valve	As mentioned before.

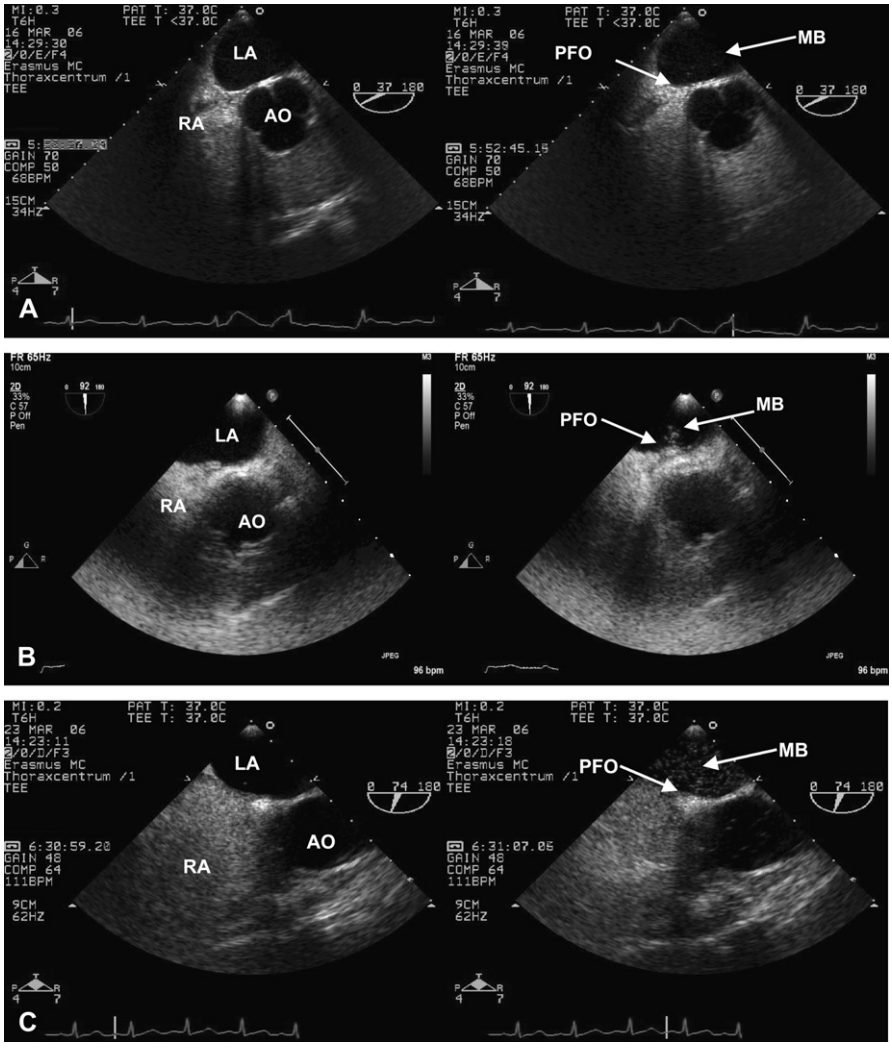

Figure 2 Transoesophageal echocardiography using Haemaccel® (contrast agent), showing: (A) small-sized (less than 10 microbubbles), (B) small-to-moderate (more than 10 microbubbles), and (C) large right-to-left shunt. Abbreviations: AO = aorta, LA = left atrium, MB = microbubbles, PFO = patent foramen ovale and RA = right atrium.

patient, the largest number of microbubbles decides the size of shunt. RLSs of different sizes are shown in Fig. 2.

Patent foramen ovale

General overview

Persistent PFO is associated with stroke, decompression sickness (DCS), and other disorders related to paradoxical embolism (Table 4). However, it is still not clear whether this association is causal, due to predisposition, or have an innocent coincidence. Most evidence for a causal relation exists for young patients with cryptogenic stroke.[1,28] Also, PFO may induce hypoxemia that predispose to the platypnea–orthodeoxia syndrome (see later).[29] In the USA of the 700 000 non-hemorrhagic strokes per year 80% are ischemic and 20% are cryptogenic.[30] PFO and/or ASA with or without

Table 4 Conditions associated with PFO and inter-atrial septal abnormalities

Cryptogenic stroke
Transient ischemic attacks
Migraine
Peripheral ischemia
Platypnea–orthodeoxia syndrome
Decompression sickness of the divers
Un-explained dementia[79]
Un-explained syncope
''Economy-class'' stroke syndrome[80]
Obstructive sleep apnea[81]
Post-total knee arthroplasty cerebral microembolism[82]

Table 5 Relationship between PFO and stroke or transient ischemic attack from prospective studies (incidence of stroke or transient ischemic attack among patients with PFO)

Author	Year	Follow-up (yrs)	Echo	Patients	Stroke or TIA	Event rate (%)		
						PFO	PFO + ASA	No PFO
Negative studies								
Homma[26]	2002	2	TEE	630	203 (34%)[a]	14	16	15
Meissner[3]	2006	5	TEE	585	41 (7%)[b]	9	19	7
Positive studies								
Mas[2]	2001	4	TEE	581	24 (4%)[a]	2	15	4

a = recurrent; b = new-onset; ASA = atrial septal aneurysm; PFO = patent foramen ovale; TEE = transesophageal echocardiography; TIA = transient ischemic attack. All hazard ratios were non-significant.

PFO are present in approximately half of the patients with cryptogenic stroke and in approximately a quarter of healthy individuals.[30] In a meta-analysis by Overell et al.[31] cryptogenic stroke and transient ischemic attacks were significantly associated with PFO and/or ASA. In contrast, in a recently published population-based prospective study from the Mayo Clinic[4] only ASA, but not PFO, was an independent predictor of stroke or transient ischemic attacks, confirming other negative studies. However, essential differences in the Mayo Clinic study should be taken into account when interpretation of the results is applied; the relatively higher age (mean 66.9 ± 13.3 years) of the studied population, other risk factors (hypertension and diabetes mellitus) for stroke are often present. Moreover, it should be noticed that in all (negative and positive) PFO studies conclusions are based on a relatively low number of events. A summary of the results of some important studies is seen in Tables 5 and 6.

PFO and recurrence of cerebrovascular complications

The annual stroke recurrence rate in patients with cryptogenic stroke and PFO with or without ASA varies widely from 1.5% to 14%, depending on the study population. Mas et al.[2] reported a cumulative 4-year recurrence rate of 14% among patients with PFO and ASA. It is still controversial whether PFO and/or ASA are associated with an increased risk of stroke recurrence. In a meta-analysis of 10 studies of PFO device closure and 6 studies of medical therapy for PFO, overall, the 1-year rate of recurrent neurological thromboembolism with transcatheter intervention was 0%—4.9% compared to 3.8%—12.0% on medical management[32] (Table 7).

When does PFO need treatment?

In the recently published AHA/ACC/AAN guideline for the management of stroke it is recommended

Table 6 Observational and cross-sectional studies (prevalence of PFO among patients with stroke)

Author	Year	Echo	Patients	Cryptogenic stroke		Non-cryptogenic stroke		Control	
				PFO/stroke (*n*)	%	PFO/stroke (*n*)	%	PFO/controls (*n*)	%
Negative studies									
Hausmann[83]	1992	TEE	238	16/74	22	10/48	21	25/116	22
Jones[84]	1994	TEE	422	14/71	20	21/149	14	31/202	15
Fisher[85]	1995	TEE	1000	39/391	10	NA	NA	391/609	9
Positive studies									
Lechat[1]	1988	TTE	160	14/26	54	4/19	21	10/100	10
Webster[86]	1988	TTE	80	20/40	50	NA	NA	6/40	15
De Belder[5]	1992	TEE	198	9/35	26	10/69	14	3/94	3
De Tullio[87]	1992	TEE	146	22/45	48	4/101	4	NA	NA
Cabanes[6]	1993	TEE	150	56/100	56	NA	NA	9/50	18
Job[71]	1994	TCD	137	27/41	66	11/33	33	27/63	43
Van Camp[88]	1994	TEE	57	6/29	21	NA	NA	0/28	0

Abbreviations as in Table 2. NA = Not applicable.

Table 7 Recurrence of neurological complications in patients with patent foramen ovale

Author	Year	Follow-up (yrs)	Patients	PFO closure		Medical therapy		
				Patients	Recurrence rate	Patients	Recurrence rate	
							Aspirin	Warfarin
Khairy[32]	2003	>1	2250	1355	0–4.9[a]	895	3.8–12[a]	3.8–12[a]
Windecker[89]	2004	2.4	308	150	8.5	158	28.3	13.3
Schuchlenz[90]	2005	2.6	280	167	0.6	113	13.0	5.6

[a] Range of studies included in the meta-analysis by Khairy et al.

that any patient, who suffered ischemic stroke or a transient ischemic attack with a PFO, may receive antiplatelets or warfarin therapy to minimize stroke recurrence (Class IIa, level of Evidence C) while device or surgical closure of PFO is not supported (Class IIb, level of Evidence C).[30] Ongoing research in this field will provide definite answers in the near future. Our practice is that only patients <55 years who suffer stroke or transient ischemic attack are screened for the presence of PFO. If a PFO is diagnosed with an RLS and there is no other risk factor present, patients are informed about the therapeutic choices, medical treatment or device closure with advantages and disadvantages of both management regimens. If both a PFO and an ASA are present, we recommend PFO device closure because of the huge recurrence rate of stroke under medical treatment alone. If a patient with a PFO but without other risk factors had a recurrent stroke despite medical treatment, we advise device closure. If other risk factors are not present we advice medical treatment.

PFO and migraine

Recent evidence supports an association between PFO and migraine headache, particularly if combined with aura. In a series of case-controlled studies[33–36] approximately half of migraine patients had a PFO. However, compared to controls the prevalence of PFO was only increased in migraine patients with aura (54% vs. 17%).[33] PFOs in migraineurs are moderately large but rather less than that associated with stroke patients.[35] The nature of the relation between PFO and migraine is still poorly understood. Autosomal dominant inheritance,[37] small venous thrombus or platelets aggregate, or vaso-active substances[38] entering the cerebral circulation through the PFO are possible mechanisms. In several PFO closure device studies a significant reduction in the frequency of migraine was described, particularly in patients with aura (Table 8).[39–42] However, in a double-blinded, prospective placebo and sham procedure controlled study, the results were not as convincing as in retrospective studies. Other trials are currently in progress.[36]

PFO and DCS

Nitrogen is an inert gas normally stored throughout the human body such as tissues and fluids, in physical solution. When the body is exposed to decreased pressures, such as during a scuba ascent through water, the nitrogen dissolved in the body comes out of the solution. These nitrogen bubbles are mostly trapped in the lung capillaries because of their larger size than the tiny diameter of the capillaries. Once trapped, the bubbles break up and the nitrogen gas is exhaled. A PFO allows bubbles to pass from right-to-left circulation,

Table 8 Improvement of migraine after PFO closure

		Migraine		Migraine with aura		Migraine without aura	
		Patients	%	Patients	%	Patients	%
Wilmshurst[41]	2000	18/21	86	15/16	94	3/5	60
Morandi[40]	2003	15/17	88	NA	NA	NA	NA
Schwerzmann[91]	2004	35/43	81	26/33	79	9/10	90
Azarbal[42]	2005	24/30	80	16/20	80	8/10	80
Reisman[92]	2005	35/50	70	NA	NA	NA	NA
Composite		127/161	79	57/69	83	20/25	80

bypassing the screening effects of the pulmonary circulation. However, about 30% of divers have a PFO and the prevalence of serious DCS is very low (1%)[43]; a causative role for DCS of PFO is outsized entity and therefore, routine screening of divers for PFO is not recommended. Moreover, in the analysis of diving incidents with serious DCS with high spinal cord and cerebral affection, a PFO with a RLS was present much more often than in a control population of divers without DCS.[44,45] This suggests that a PFO with the possibility of intra-cardiac RLS may play a role, albeit small, in DCS. Because of a possible elevated risk for DCS for patients with a PFO, it might be advisable to screen scuba professional divers who will make significantly more dives than amateur/sport/recreational divers for the presence of a PFO with RLS.[46] If a PFO with RLS is present they should be advised to stop diving or at least change diving habits, minimising the amount of nitrogen load on the tissue.[46] Theoretically, percutaneous PFO closure by a device could be efficacious in lowering the risk of DCS in divers, but this hypothesis has not been properly tested yet.

PFO and the platypnea–orthodeoxia syndrome

Platypnea–orthodeoxia is an uncommon syndrome of dyspnea (platypnea) and hypoxemia (orthodeoxia) induced by upright posture, which is subsequently relieved by recumbence. Traditionally, this condition has been reported in association with pulmonary, hepatic and cardiac diseases. The occurrence of the syndrome mandates anatomical and functional abnormalities.[47] The anatomical abnormality is an RLS either intra-cardiac (PFO, ASD, fenestrated ASA) or extra-cardiac such as PAVM. Mechanisms explaining postural hypoxemia are RLSs resulting from either elevation of right atrial pressure or redirection of the inferior vena cava flow towards the atrial septum[48] due to the presence of an associated persistent Eustachian valve. The definitive treatment in such cases is closure of the defect.[49] It should be noted that a PFO with RLS could be only seen in upright position in such patients.

Pulmonary arterio-venous malformations

PAVMs represent a direct communication between one or more pulmonary arteries and one or more pulmonary veins and often associated with hereditary hemorrhagic teleangiectasia (Osler–Weber–Rendu syndrome).[50,51] PAVMs are commonly ($>$70%) single sacks ranging from 1 to $>$10 cm in diameter, with a single feeding artery and a single draining vein.[52] The main complications of PAVMs are thought to relate to the RLS of thrombotic particles with eventually stroke and/or brain abscess, which may occur in up to one half of patients if left untreated.[53,54] Several diagnostic techniques were investigated for screening of PAVMS including contrast echocardiography, oxygen shunt test (oxygen saturation after breathing 100% oxygen), pulmonary angiography, computed tomography and magnetic resonance imaging.[55] In comparison to other techniques such as pulmonary angiography [56,57] and oxygen shunt test[58] contrast echocardiography was the most sensitive but least specific non-invasive technique for detection of PAVMs.[54,56,57] However, it should be noticed that radioisotope evaluation of PAVMs in a non-comparative study resulted in a similar sensitivity to contrast echocardiography but had a higher specificity.[59] Appearance of microbubbles in the left atrium after 3 beats from opacification of the right atrium is suggestive of PAVMs.[60] However, microbubbles appearing earlier do not exclude PAVMs, and may be caused by a combined inter-atrial RLS plus PAVMs or PAVMs connecting to a lower pulmonary vein.[61] Thus, a comprehensive TEE examination of the lower pulmonary veins during contrast echocardiography is important to exclude PAVM. The 3-cardiac cycle conventional rule of differentiating extra-cardiac from intra-cardiac RLSs through a PFO by noting the appearance of contrast echoes in the left atrium 3 or more beats after first appearance in the right atrium has been found to be unreliable.[62] Treatment is recommended for all PAVMs with single feeding vessels of $\geq$3 mm, in order to reduce the risk of paradoxical embolization, however, if multiple feeding vessels are present it may be difficult or impossible to treat.[63]

Atrial septal defects

ASDs are the most common congenital heart abnormalities in adults after bicuspid aortic valve. The most common anatomical types are ostium primum, secundum and sinus venosus defects. The use of contrast echocardiography for the diagnosis of ASD is limited; the combination of 2D (TTE and TEE) and Doppler techniques (especially colour-Doppler) almost always leads to the diagnosis. In case of doubt or incomplete diagnosis contrast echocardiography can be useful. Contrast echocardiography is very useful and is used very often for the assessment of residual shunt after device closure of an ASD.[64–66]

Because of the high echogenicity of the closure device, the ultrasound wave reflections make the conventional echocardiographic techniques (including Doppler) unreliable. A residual LRS can be seen as a negative contrast effect in contrast-filled right atrium. Residual RLSs can be seen as bubbles passing the inter-atrial septum to the left atrium which mandates the use of optimal Valsalva manoeuvre.[64]

Ventricular septal defect

A VSD may be an isolated single congenital defect, part of a complex congenital cardiac anomaly or acquired after an acute myocardial infarction. It may be situated in the membranous or muscular part of the septum. In the past contrast echocardiography has been used successfully in clinical practice for the detection of VSD.[67] Since the introduction of colour-Doppler the use of contrast for detection of VSD is almost not seen in clinical practice. However, contrast could be used for the detection of residual shunt after VSD closure. At the Thoraxcenter, both colour flow mapping and contrast echocardiography are performed after VSD closure before closure of the chest for the immediate exclusion of residual shunting.[68,69]

Conclusion

Contrast echocardiography is a safe, simple, non-invasive, feasible and reproducible imaging technique with a proven accuracy for the detection of cardiovascular shunts.

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