

Real-world experience of transoesophageal echocardiography for detection of clinically significant persistent foramen ovale

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Keywords

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Abstract

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Introduction: Transoesophageal echocardiography (TOE) is claimed to be the investigation of choice for detecting a persistent foramen ovale (PFO) with almost 100% diagnostic accuracy. If true, TOE would detect all large/clinically significant PFOs.

Methods: Retrospective analysis to determine the sensitivity of TOE for detection of clinically significant PFOs. Patients were from a consecutive series of 150 patients who had transcatheter closure of a PFO following events attributed to paradoxical embolism (decompression sickness or stroke). In each patient, transthoracic echocardiogram with bubble contrast showed a clinically significant atrial right-to-left shunt. The data reported are from the sub-group of the 150 patients with a clinically significant PFO who also had a TOE performed in other hospitals.

Results: Twenty seven of 150 consecutive patients had a total of 31 TOEs performed at 22 United Kingdom regional cardiac centres. TOE failed to detect a PFO in 17 of the 27 patients. Four patients had a TOE on two separate occasions and in each case both of the TOEs failed to show a PFO. TOE gave a false negative test in 21 of 31 investigations (sensitivity 32%). The mean PFO diameter was 9.4 mm (median 9 mm, range 5–16 mm) in the 21 patients in whom balloon sizing was performed and 9.8 mm (median 10 mm, range 5–16 mm) in the 13 patients in whom balloon sizing was performed and a TOE failed to show a PFO.

Conclusions: These findings demonstrate that the precision of TOE for detecting a PFO in real world clinical practice is considerably lower than generally believed.

Introduction

The law of the conservation of ignorance: A false conclusion once arrived at and widely accepted is not easily dislodged and the less it is understood, the more tenaciously it is held.

Georg Cantor

Persistent foramen ovale (PFO) are present in approximately one quarter of adults.¹ Most PFOs are small,¹ but large PFOs allow significant right-to-left shunting, which can facilitate paradoxical thromboembolism and stroke,² and paradoxical gas embolism and some types of decompression sickness.^{3–6} Therefore it is important that any test for a PFO accurately identifies those that are large enough to present a significant risk of paradoxical embolism of thrombus or bubbles.

Intuitively, transoesophageal echocardiography (TOE), which provides the best images of the atrial septum, might be anticipated to be superior for identifying a PFO than transthoracic echocardiography with bubble contrast or

transcranial Doppler with bubble contrast. The intuitive superiority of TOE has resulted in it being readily accepted by cardiologists as the clinical investigation of choice for diagnosis of a PFO.⁷

It is commonly claimed that TOE has almost 100% predictive accuracy for detecting a PFO. If that claim were true one would anticipate that in specific groups of patients TOE would identify PFOs at rates comparable to post mortem examinations or cardiac catheterisation in those groups. For example, one would expect that TOE would identify a PFO in approximately a quarter of healthy adults, which is approximately the prevalence reported by Hagen et al. in their large series of post mortem findings in people without heart disease.¹ Similarly in groups of individuals with a high clinical probability of paradoxical embolism, such as divers with associated forms of decompression sickness, it would be anticipated that TOE would detect a PFO in many more than 25% of the group. Other methods of detecting a PFO should be judged against the same criteria.

There are many publications that report comparison of the rates of diagnosis of PFO using TOE and transthoracic echocardiography with bubble contrast. Discordant results are invariably attributed to errors in the transthoracic studies without adequate justification.

There are only a few reports that validated the finding of a PFO by echocardiography against an objective standard, such as post mortem examination or cardiac catheterisation.⁸⁻¹¹ Those studies were performed decades ago when echocardiography imaging was poor compared with current equipment – one even used M-mode echocardiography.¹¹ Each study was small – the number of PFOs detected was between nine and 24. Few patients in these studies had imaging performed because of suspected paradoxical embolism. Instead, some studies consisted almost entirely of patients with structural/congenital heart disease (such as Ebstein's anomaly and pulmonary stenosis) and other diseases likely to increase right atrial pressure and therefore increase right-to-left shunting if a PFO was present.^{8,10}

Schneider and colleagues compared the findings of PFO detected by TOE in intensive care patients against post mortem examination of some of those who died by two pathologists.⁹ They reported that of 35 patients who died, a post mortem examination showed that nine had a PFO, although in one case the two pathologists initially disagreed about the presence of a PFO. The authors reported that TOE had 100% sensitivity and specificity for detecting a PFO, although in two of nine diagnosed as having a PFO only a single bubble was seen in the left heart after contrast injection and another had no left heart bubbles but colour flow Doppler showed a jet across the atrial septum. It is unclear whether post mortem examinations were performed blind to the result of TOE. The study was small and not typical of situations when tests for a PFO are performed because 18 patients, including five with a PFO, had their TOE when on mechanical ventilation, which increases shunting across a PFO.^{12,13}

In contrast, we report real-world experience of the ability of TOE to detect or confirm the presence of a clinically significant PFO in patients with paradoxical embolism (shunt-mediated decompression sickness and ischaemic stroke likely to be the result of paradoxical thromboembolism). In each patient, a clinically significant atrial right-to-left shunt was demonstrated by transthoracic echocardiography with bubble contrast and the presence of a PFO was confirmed during transcatheter closure.¹⁴

It is important that this is not a comparison of TOE and transthoracic echocardiogram with bubble contrast. The latter was simply the test used to detect the clinically significant PFOs. We are reporting the sensitivity of TOE in detecting a clinically significant PFO with the finding of a PFO at the closure procedure as the 'gold standard'.

Methods

The patients described are a sub-group of a consecutive series of 150 patients who had a PFO that was closed using a transcatheter device after our transthoracic echocardiogram with bubble contrast showed a significant atrial right-to-left shunt. The sub-group were all of those who had transoesophageal echocardiography at other hospitals in order to detect a PFO before we performed our transthoracic echocardiogram with bubble contrast or to confirm the presence of a PFO after we performed our test.

In patients thought to have had stroke or arterial thromboembolism as a result of paradoxical embolism and divers who had decompression sickness as a result of paradoxical gas embolism, we diagnosed atrial and pulmonary right-to-left shunts using transthoracic echocardiography with bubble contrast using a standard protocol.⁵ Based on this test alone, transcatheter closure of their shunt was one option we offered patients with a significant atrial shunt. We did not perform a TOE until the closure procedure, at which stage a TOE was used to differentiate an atrial septal defect from a PFO and used to ensure optimal positioning of the occlusion device. The TOE findings during the closure procedure are not included in this analysis because in those cases we were not performing a diagnostic test and because in many cases a guide wire was already positioned across the atrial defect using X-ray fluoroscopy before the TOE probe was positioned.

During the period in which we performed transcatheter closure of a PFO in the 150 consecutive patients in this report, ten additional patients were found to have an atrial right-to-left shunt on our transthoracic echocardiogram with bubble contrast, but at the time of their closure procedure an atrial septal defect was found using TOE. They are not included in the 150 patients.

The majority of the 150 patients with a PFO had decompression sickness and were seen in a specialist diving medicine clinic which took referrals from all parts of the United Kingdom. The clinic assessed divers that had decompression illness to determine whether they had shunt-mediated decompression sickness or another cause for dysbaric illness, such as arterial gas embolism secondary to pulmonary barotrauma. Each diver had investigations, including transthoracic echocardiography with bubble contrast, before the referral letter was read or a history was taken to avoid bias in interpretation of the results.^{5,6} The transthoracic echocardiography with bubble contrast was performed according to a set protocol.^{5,6} When a clinically significant right-to-left shunt (atrial or pulmonary) was detected, divers were counselled about the options for avoiding recurrence of decompression sickness – namely to stop diving, modify their dives or have closure of a PFO or atrial septal defect.¹⁵ Some of those found to have a clinically significant atrial shunt requested transcatheter closure.

A small number of the 150 patients had an ischaemic stroke that was thought to be the result of paradoxical thromboembolism with other causes of stroke excluded. If transthoracic echocardiography with bubble contrast detected a significant atrial shunt they were offered transcatheter closure without a TOE being performed until the procedure.

Although we did not perform a TOE in patients with decompression sickness or suspected paradoxical thromboembolism until they had a closure procedure, 27 of the 150 patients who had transcatheter closure of their PFO did have a TOE at other hospitals – all United Kingdom regional cardiac centres. Four patients each had two TOEs. From correspondence we obtained the results of each TOE in each of the patients in so far as we knew whether or not the test had shown a PFO, but we did not obtain the details of protocols used in each case. Specifically, we do not know which transoesophageal echocardiograms included intravenous injection of bubble contrast.

Four divers had one or more TOEs in regional cardiac centres near their homes in order to see whether they had a PFO before they were referred to our clinic. In the remaining 23 patients the reason for the TOE was that the patients' local NHS commissioners (who authorise payments for out-of-area NHS treatments) had permitted us to see divers in the specialist clinic, but they insisted that if a significant shunt was detected, before a closure procedure could be performed, an opinion should be obtained from their local cardiologists. In every case the local cardiologist arranged for the diver to have a TOE. In each of the 23 patients the TOE operator was aware that we had previously demonstrated a significant atrial right-to-left shunt using transthoracic echocardiography with bubble contrast. A consultant cardiologist performed each TOE. Several of the cardiologists had taught on TOE courses and published research on TOE.

When a TOE confirmed a PFO the commissioners gave permission for transcatheter closure. When a TOE failed to confirm a PFO, we were able to use the recorded images from our transthoracic echocardiograms with bubble contrast to persuade the commissioners to permit cardiac catheterisation with a view to proceeding to a transcatheter closure if a large PFO was confirmed. Some of those closure procedures were by us and we measured the diameter of PFOs using balloon sizing.¹⁶ Some closure procedures were in other regional cardiac centres, and we obtained details of those procedures, but balloon sizing was not always performed.

Results

Of 150 consecutive patients who had a transcatheter closure of a PFO, 27 had a TOE at 22 regional cardiac centres. Each TOE was performed with the intention of determining whether there was an atrial right-to-left shunt because

paradoxical embolism was suspected. Twenty-five patients were divers with decompression sickness with characteristics (clinical manifestations and latencies) in keeping with shunt-mediated decompression sickness, but on a separate occasion one also had cerebral infarction 24 hours after a long-haul flight. Two other patients had a history of cerebral infarction only. The three patients with cerebral infarction had their stroke before age 45, had a predisposition to venous thrombosis and had no other medical condition that might predispose to stroke.

Table 1 shows the results of the echocardiographic investigations and cardiac catheterisation in the patients. Using transthoracic echocardiography with bubble contrast, we graded the shunt as large in 22 patients and medium in one patient during normal respiration. In the other four patients there was a large shunt with release of a Valsalva manoeuvre.

TOE failed to detect a PFO in 17 of 27 (63%) patients with a clinically significant PFO confirmed at the closure procedure. In those cases, the correspondence from the doctor who performed the TOE said that the TOE failed to demonstrate or confirm the presence of PFO.

Four of the patients (numbers 1, 18, 23 and 27) had negative TOE on two occasions. Two divers, who had decompression sickness, returned to diving after a TOE that was reported to show no PFO and they each had a second episode of decompression sickness, which resulted in them having a second TOE that was also negative, before our transthoracic echocardiogram with bubble contrast showed a significant right-to-left shunt. Two patients had a negative TOE before our transthoracic echocardiogram with bubble contrast showed an atrial shunt and that finding led to a repeat TOE that was also reported to be negative.

Therefore 21 of 31 (68%) TOEs gave a false negative test even though the TOEs were performed by experienced operators specifically looking for a cause of paradoxical embolism. In the majority of cases, we had demonstrated an atrial right-to-left shunt using transthoracic echocardiography with bubble contrast before the TOE and the operators were aware of the findings. The sensitivity of TOE for PFO detection was 32%.

Of the 10 patients in whom a PFO was detected by TOE, the size was considered to be medium or large in three, small in two and there was no comment about size in five.

In six patients the diameter of the PFO was not measured during the closure procedure, but a cardiac catheter passed easily across the PFO. The mean diameter of PFOs was 9.4 mm (median 9 mm, range 5–16 mm) in the 21 patients in whom balloon sizing was performed and 9.8 mm (median 10 mm, range 5–16 mm) in the 13 patients in whom balloon sizing was performed and a TOE failed to show a PFO.

Table 1
Echocardiographic and cardiac catheterisation findings in the patients; PFO – persistent (patent) foramen ovale

Patient	Transthoracic contrast echocardiography findings	Transoesophageal echocardiography findings	Diameter of PFO at closure
1	large shunt at rest	no atrial shunt x 2	11 mm
2	large shunt at rest	no atrial shunt	16 mm
3	medium shunt at rest	small PFO	9 mm
4	large shunt at rest	no atrial shunt	13 mm
5	large shunt at rest	no atrial shunt	easily crossed but not sized
6	large shunt with a Valsalva	medium-large PFO	10 mm
7	large shunt at rest	PFO – no comment on size	9 mm
8	large shunt at rest	no atrial shunt	7 mm
9	large shunt at rest	PFO – no comment on size	6 mm
10	large shunt at rest	PFO – no comment on size	11 mm
11	large shunt at rest	no atrial shunt	easily crossed but not sized
12	large shunt at rest	no atrial shunt	easily crossed but not sized
13	large shunt at rest	no atrial shunt	10 mm
14	large shunt at rest	PFO – no comment on size	easily crossed but not sized
15	large shunt at rest	no atrial shunt	easily crossed but not sized
16	large shunt at rest	no atrial shunt	13 mm
17	large shunt at rest	no atrial shunt	9 mm
18	large shunt at rest	no atrial shunt x 2	9 mm
19	large shunt with a Valsalva	no atrial shunt	8 mm
20	large shunt at rest	no atrial shunt	10 mm
21	large shunt at rest	large PFO	11mm
22	large shunt at rest	PFO – no comment on size	7 mm
23	large shunt with a Valsalva	no atrial shunt x 2	5 mm
24	large shunt at rest	small PFO	7 mm
25	large shunt with a Valsalva	no atrial shunt	6 mm
26	large shunt at rest	large PFO	easily crossed but not sized
27	large shunt at rest	no atrial shunt x 2	10 mm

Discussion

Each of the 27 patients described had a significant atrial right-to-left shunt on transthoracic echocardiography with bubble contrast and was found to have a large PFO which was closed using a transcatheter technique.

When measured (i.e., in 21 of 27 patients), PFO diameters (mean 9.4 mm and median 9 mm) were comparable to the diameters of atrial defects (mean 9.9 mm and median 10 mm) in a series of 200 divers (189 had a PFO and 11 had a secundum atrial septal defect), who had shunt-mediated decompression sickness and later had transcatheter closure.¹⁶

To put these diameter measurements in context, a large post-mortem study showed 27.3% of the normal population have a PFO with a median diameter of 5 mm and mean diameter 4.9 mm (SD 2.6).¹ A PFO diameter of 9 mm or greater was

present in 2.6% of normal adult hearts.¹ In contrast 14 of 21 (67%) of the patients we reported had a PFO diameter of 9 mm or greater. A PFO diameter of 10 mm or greater was present in 1.3% of normal adult hearts,¹ but was present in 10 of 21 (48%) of our patients.

Despite the large diameters of the PFOs in our patients who had paradoxical embolism, the sensitivity of TOE for PFO detection in was only 32%. It is likely that TOE would have a lower sensitivity for detecting smaller PFOs. These findings suggest that contrary to what is commonly claimed, in real-world clinical practice TOE is not an accurate test for PFO detection even when performed by experienced operators.

The TOEs were performed in 22 regional cardiac centres by a number of consultant cardiologists who used their individual protocols in order to detect or confirm the presence of a PFO in patients who had paradoxical embolism. As far as

we are aware the TOEs were not performed using a standard protocol.

We do not know why the sensitivity of TOE for demonstrating a PFO was low, but we postulate that the widely held belief in the infallibility of TOE may have resulted in poor attention to technique. We do not know whether bubble contrast was injected and if it was, we do not know the quality of the opacification obtained. Inability to perform provocative manoeuvres, such as Valsalva manoeuvres, as a result of sedation is unlikely to be a factor, because 23 of 27 of the patients had significant right-to-left shunts during transthoracic echocardiography with bubble contrast when breathing normally.

We do not know the sensitivity or specificity of our transthoracic echocardiography with bubble contrast protocol for detecting an atrial right-to-left shunt because we do not perform right heart catheterisation in patients other than those having a closure procedure. Nor did we assess sensitivity of transthoracic echocardiography with bubble contrast in detection of PFOs that were identified using transoesophageal echocardiography and confirmed during a closure procedure. We emphasise that we were not comparing the diagnostic accuracy of the two echocardiographic tests.

However, when performing transthoracic echocardiography with bubble contrast for detection of a right-to-left shunt, we insist on a rigorous protocol and perform the test blind to history.⁴⁻⁶ As a result, we consistently reported prevalence rates of PFOs in normal control subjects of 24–27.6%.⁴⁻⁶ This is similar to the 27.3% incidence in normal hearts at post-mortem.¹ Using our protocol in normal control subjects, only 4.9% had a large shunt with normal respiration and a further 2.4% had a large shunt with multiple contrast injections and release of a Valsalva manoeuvre, whereas 4.9% had a medium shunt and 15.4% had a small shunt.⁶

In addition, using our protocol in divers with decompression illness, we detected all those in whom a significant right-to-left shunt is likely to account for the symptoms with provocative dive profiles or pulmonary barotrauma explaining other episodes.^{5,6} As a result in divers with cutaneous and neurological decompression sickness the prevalence of large right-to-left shunts was between seven and ten times the prevalence in control divers.^{5,6}

During 30 years, in which hundreds of patients judged to have significant atrial shunts using our protocol of transthoracic echocardiography with bubble contrast went forward to a closure procedure without a TOE, only two were found to be unsuitable for the procedure because they did not have either a PFO or secundum atrial septal defect. Both had right-to-left shunts, but one had a sinus venosus defect that was subsequently corrected surgically. The other, who was the fifth patient we investigated, had a large pulmonary arteriovenous malformation with high flow so that left

atrial opacification was seen in one beat after right heart opacification. The defect was occluded because of its size.

Our experience cannot be extrapolated to the real-world accuracy of transthoracic echocardiography with bubble contrast for detecting a PFO when operators do not adhere to a protocol with confirmed accuracy.

LIMITATIONS

We assessed the sensitivity of transoesophageal echocardiography for detecting clinically significant PFOs that were proven to be present during a closure procedure as the 'gold standard'. Transthoracic echocardiograms used to detect clinically significant PFOs were performed according to a clear protocol, but we suspect that there was no uniformity in performance of transoesophageal echocardiograms and we do not know how many incorporated intravenous injection of bubble contrast. An alternative study that selected patients who had a large PFO detected by transoesophageal echocardiography would probably find that transthoracic echocardiography with bubble contrast performed without a set protocol by several different cardiologists had sensitivity less than 100%.

Nevertheless we conclude that these findings demonstrate that the precision of TOE for detecting a PFO in real world clinical practice is considerably lower than generally believed.

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