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# Intrapulmonary Shunts and Its Implications for Assessment in Stroke and Divers with Type II Decompression Illness

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The recent article "Intrapulmonary Shunt Is a Potentially Unrecognized Cause of Ischemic Stroke and Transient Ischemic Attack" (TIA) by Abushora et al.[1] raises several questions in our daily clinical evaluation of patients with stroke, and also divers with unexplained Type II decompression sickness (DCS).

A cohort of patients (stroke group) with either nonhemorrhagic stroke (CVA) or TIA (n = 321) was compared with age- and gender-matched control patients (n = 321). A subgroup of patients with CVA or TIA had what was defined as cryptogenic stroke (n = 71). These patients underwent transesophageal echocardiography, and were evaluated for visualization of saline contrast bubbles within the left atrium (LA) via either the pulmonary veins or through a patent foramen ovale (PFO). An intrapulmonary shunt was defined as direct visualization of bubbles within one or more pulmonic veins entering the LA. A PFO was defined as direct visualization of contrast crossing the foramen ovale, or visualization of contrast with the LA within three cardiac cycles of its appearance within the right atrium.

Studies were performed with normal respirations, with no provocative maneuvers for detection of PFO. The authors mention that the study was performed without provocative maneuvers, to allow for a definitive differentiation between shunting via a pulmonary vein or PFO.

For both detected intrapulmonary shunts and also PFOs, severity of shunting was graded based on the amount of contrast in a pulmonary vein or that entering via a PFO. Mild, intermediate, or large shunts were defined as 1–4, 5–20, or >20 visualized within any single image frame.

Intrapulmonary shunts occurred more often in the stroke group than controls (72 of 321 or 22% vs. 32 of 321 or 10%). The severity of shunting was similar in both groups, mostly mild or intermediate. The number of pulmonary veins demonstrating shunting appeared to occur in 1, 2, or all 4 veins for both groups. The stroke group with detected intrapulmonary shunting did have a tendency toward contrast appearing in all 4 veins (38% incidence) more often than in controls (28%).

Patent foramen ovale detection was higher (73 of 321 or 23%) for the stroke group versus controls (54 of 321 or 17%), but did not achieve statistical significance. The stroke group tended toward larger shunts compared to controls.

Patent foramen ovale and/or an intrapulmonary shunt were noted more often in the stroke group (136 of 321 or 42%) than in controls (54 of 321 or 17%). The incidence of both an intrapulmonary shunt and a PFO in the same patient was not mentioned.

From the stroke group a subgroup of patients was identified as having either a cryptogenic CVA (n = 50) or cryptogenic TIA (n = 21). This cryptogenic stroke group (n = 71) was then compared with an age- and gender-matched control group (n = 71). In this cohort, intrapulmonary shunting occurred more often (25 of 71 or 35%) than in controls (5 of 71 or 7%). Shunting in the cryptogenic group occurred in 1 or 2 veins 2/3 of the time and 1/4 of the time in all 4 veins, whereas in the control group, shunting occurred exclusively in 1 or 2 veins. Severity of shunting

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was mild or intermediate most often in both groups. PFO detection was not mentioned for this cohort of patients with cryptogenic stroke. From this study, the following statements may be made:

For control patients:

1 Incidence of a resting PFO detected was 17%.
2 Incidence of a pulmonary vein shunt was 10%, and 7% for a smaller control cohort that had been matched to patients with cryptogenic stroke.

For the stroke group:

1 A shunt occurred 2.2 times more frequently in stroke patients versus controls.
2 Shunting in all 4 pulmonary veins occurred more often in the stroke group than in controls. Severity of shunting was not higher in stroke versus control patients.

For the subgroup of stroke patients with cryptogenic stroke:

1 Incidence of intrapulmonary shunt was 5 times more frequent than in controls.
2 Shunting was mild or intermediate in both groups, but had a tendency to occur more often in multiple pulmonary veins in the cryptogenic stroke group versus controls.

The detection rate for PFO was similar for stroke patients versus controls (23% vs. 17%). As no provocative maneuvers were used for detection, this would be defined as the detection rate of a "resting PFO."[2] In this study, the rate of detection of a resting PFO in controls appears to be somewhat higher than generally reported in the literature. In a study of functional characteristics of the fossa ovalis in stroke, of those found to have a PFO, a resting PFO was noted in 61% of stroke patients and 31% of controls.[3]

For stroke patients, it is evident that a resting PFO appears to have more clinical significance than that found only during provocative maneuvers.[2] This may not hold for divers with unexplained DCS, in which a Valsalva maneuver is essentially performed upon completion of a prolonged dive.[4]

As occurs with many good studies, it appears that more questions are raised than are answered.

1 It would be interesting to note the incidence of coexistent PFO and pulmonary shunting in both stroke patients and controls. This might shed some light as to why some patients have persistent shunting after percutaneous closure of a PFO.
2 Could the relatively high incidence of pulmonary shunts found in controls and stroke patients explain why transcranial Doppler detection of shunts generally is higher than that detected by echo methods?
3 Are shunts detected via the pulmonary veins dynamic? That is, do clinically significant shunts "open and close" over time? It appears that pulmonary shunts may functionally change, as evidenced by the fact that patients with liver disease "develop" pulmonary shunting. Also, patients with surgically created inferior vena cava to right pulmonary artery shunts will develop multiple pulmonary arteriovenous shunts within the left lung.

As related specifically to divers with DCS:

1 Is there a clinical association with pulmonary shunting? Through the years, we have identified divers with DCS who do not have a PFO, but have shunting through pulmonary veins. Whether there exists a cause and effect is unknown.
2 Could divers, because of changes in pressure and oxygen saturation have pulmonary shunts at one point in time, and not demonstrate shunting at another? Divers have presented with clinical DCS patterns that are characteristic for what is found in divers with documented shunts, but have no shunt identified.
3 The size of pulmonary vessels involved in shunting may take on a special significance in regard to DCS. As the inert gas comes out of solution the bubble will "grow" and coalesce with other bubbles. This could theoretically allow a greater sized bubble to be present on the outflow side than what originated on the inflow, and hence not be restricted by the diameter of the shunting vessel.

This paper certainly answers questions, but for patients with cryptogenic stroke and for divers, the water becomes muddied even more.

## References

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