Left ventricular mass predicts left atrial appendage thrombus in persistent atrial fibrillation

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Aims
Atrial fibrillation (AF) can result in the development of left atrial appendage (LAA) thrombi. We sought to examine demographic and echocardiographic predictors of LAA thrombus in patients with persistent AF.

Methods and results
One hundred and sixty-five patients in persistent AF (36 with LAA thrombus and 129 without thrombus) were studied. Demographic and cardiovascular risk factors were retrospectively examined. Transthoracic (TTE) and transoesophageal echocardiography (TOE) were performed to assess the size and function of the left ventricle (LV), left atrium (LA), LAA, and spontaneous echo contrast (SEC) in the LA and right atrium (RA). Univariate demographic predictors of LA thrombus included systolic blood pressure, ischaemic heart disease and congestive heart failure. Indexed LV mass and septal E’ velocity on TTE and mean LAA emptying velocity and the presence of SEC in both the LA and RA on TOE were predictors of thrombus. In a multiple logistic regression analysis the only independent predictor of thrombus was indexed LV mass (\(P < 0.001\)). Receiver operator characteristic curve analysis also demonstrated that indexed LV mass had the highest area under the curve (AUC: 0.98).

Conclusion
In the present study, increased LV mass was the strongest predictor of LAA thrombus in persistent AF. LA SEC and RA SEC were univariate predictors of LAA thrombus but did not add predictive value to a multivariate model including LV mass. This study highlights the importance of diagnosing and treating LV hypertrophy associated with persistent AF, which may reduce the risk of LAA thrombus and thrombo-embolic stroke.

Keywords
thrombus • atrial fibrillation • hypertension

Introduction
Atrial fibrillation (AF) is the most common clinical arrhythmia, occurring in 0.4% of the population and increasing to 5% in patients over the age of 60 years.¹,² AF is associated with thrombo-embolic events, with thrombi most frequently being located in the left atrial appendage (LAA).³–⁶ Transoesophageal echocardiogram (TOE) has been used as the standard procedure to exclude thrombus in the LAA prior to cardioversion.⁷

The occurrence of spontaneous echo contrast (SEC) and decreased LAA velocities have been shown to be associated with thrombus formation and subsequent embolic events in previous studies.³,⁷,⁸ However, limited studies have investigated SEC in the right atrium (RA).⁹–¹¹ Furthermore it has been reported that age, hypertension, subtherapeutic international normalized ratio (INR), heart failure, valvular abnormalities (predominantly mitral stenosis), diabetes, an enlarged left atrium (LA), and left ventricular (LV) dysfunction are also associated with thrombus formation and the degree of SEC.⁵,⁷,¹²–¹⁵

The aim of this study was to determine predictors of thrombus in patients with persistent AF by assessing demographic, transthoracic (TTE), and TOE parameters. We hypothesized that a composite evaluation of these parameters may provide additional insights into predictors of LA thrombus, thereby identifying patient potentially at risk.
Methods

This was a retrospective study, from a single site, which analysed patients presenting for a TOE prior to a cardioversion procedure, identified from a departmental database, between August 1999 and April 2005. Patients with paroxysmal or permanent AF, with suboptimal images, with end-stage renal failure requiring maintenance dialysis and patients with secondary causes of AF were excluded from further analysis. Patients with any degree of valvular stenosis or greater than moderate valvular regurgitation were excluded. A total of 430 TOE procedures were performed during this period on patients with persistent AF as previously classified, and LAA thrombus was reported in 36 patients by the cardiologist performing the TOE. An expert reader, blinded to the original report, further verified the presence or absence of LA thrombus in all 430 patients, and confirmed thrombus with agreement between readers was present in 36 patients, resulting in a thrombus percentage of 8% (36 of 430). The ‘non-thrombus’ control group comprised the first 150 consecutive TOE examinations performed in patients with persistent AF (until June 2003), who were enrolled in research investigations, as this facilitated obtaining clinical and demographic details. Of these 150 TOE episodes, 12 patients underwent a repeat TOE and therefore only their first episode was included. Nine patients who did not have a baseline TTE were excluded from the final analysis. The final ‘non-thrombus’ control group comprised of 129 patients. The remaining non-thrombus patients who underwent TOE-guided cardioversion after June 2003 (n = 244) were not included in the present study as detailed analysis of this subsequent group was beyond the scope of the present study.

All the patients undergoing TOE consented to participate in a departmental AF study that had human research ethics committee approval (Westmead Hospital). All AF patients were anticoagulated with warfarin with a view to maintaining their INR between 2.0 and 3.0. There was no specific hospital anticoagulation clinic that patients with warfarin with a view to maintaining their INR between 2.0 and 3.0. There was no specific hospital anticoagulation clinic that patients attended to monitor their warfarin therapy; anticoagulation was managed in the community by the patient’s general physician.

Demographic parameters

Detailed examination of patient’s records was performed to determine cardiovascular risk factors including smoking, history of hypertension, hypercholesterolaemia, diabetes, ischaemic heart disease, coronary artery bypass graft surgery, congestive heart failure, prosthetic valve and significant valvular regurgitation, presence of pacemaker or cardiac defibrillator, presence of patent foramen ovale or atrial septal defect and previous cerebrovascular accident, including strokes and transient ischaemic attacks. Furthermore, the use of cardioactive medications, such as warfarin, acetylsalicylic acid, clopidogrel, class III antiarrhythmics, digoxin, calcium channel, and beta-blockers, were also documented.

The CHADS2 scoring scheme to quantify risk of stroke (congestive heart failure = 1 point, hypertension = 1 point, age > 75 years = 1 point, diabetes mellitus = 1 point and prior Stroke or transient ischaemic attack = 2 points) was calculated for each patient.

Echocardiographic parameters

Standard TTE and TOE were performed on all patients according to established clinical laboratory practice using the GE Vivid 5 system using a 2.5 MHz variable frequency transducer for TTE. TOE images were acquired using a 5 MHz variable frequency phased array probe. Measurements were performed offline using GE Echopac 6.1.0.

Transthoracic echocardiogram

LV volumes were determined from apical four- and two-chamber views using the modified Simpson’s biplane method of discs, and LV ejection fraction was calculated. LV mass was measured using the area length method. Biplane LA maximum volume was measured just before mitral valve opening from apical four- and two-chamber views. LA total emptying fraction was estimated as:

\[
\text{LA maximum volume} - \text{LA minimum volume} \times 100 / \text{LA maximum volume}
\]

Peak systolic (S’) and early diastolic (E’) tissue velocities were measured using pulsed wave Doppler tissue imaging at the septal mitral annulus. An average of three beats was used for all measurements.

Transoesophageal echocardiogram

TOE was performed as per standard clinical practice. In particular, the presence of thrombus was determined by evaluation of the LA and LAA in multiple zoomed imaging planes. The zoomed LAA was viewed in at least four different angles, 30°—40° apart at the midoesophageal level, to evaluate the presence of thrombus. The angle that maximized the size of the LAA was used to trace the area. Pectinate muscles, LAA thrombus and the superior ridge of the LAA were excluded from the tracing. Mean and peak LAA emptying and filling velocities were measured, SEC, in both the LA and RA, was assessed and the severity graded by an observer blinded to the patients group (Grade 1: minimal and transient SEC not present throughout the cardiac cycle; Grade 2: SEC present throughout the cardiac cycle in part of the atria; Grade 3: moderate density SEC in the majority of the atria; Grade 4: severe density SEC throughout the entire atria). Inter and intra-observer agreement in grading LA and RA SEC was performed in 20 randomly selected studies by the expert reader and an independent reader.

Statistical analysis

All values are expressed as a mean ± SD. Two sample t-test and \( \chi^2 \)-test as appropriate examined differences between the two groups. Spearman’s rank correlation was performed between systolic blood pressure and reported hypertension for each group. Univariate analysis was performed with student t-test. Multiple logistic regression analysis with backward likelihood ratio was used to determine the independent predictors of thrombus in a best-fit model. Receiver operator characteristic (ROC) curves were plotted for univariate predictors of thrombus with \( P < 0.01 \) and the area under the curve (AUC) was calculated. To address the issue of a small sample size for determining predictors from amongst multiple parameters, only the four most significant \( (P < 0.01) \) univariate predictors of thrombus were entered into the multivariate and ROC curve analysis. Inter-observer and intra-observer variability for grading of LA SEC and RA SEC were examined by Spearman’s rank correlation. Data were analysed using SPSS (version 15.0, Chicago, IL, USA).

Results

Demographic parameters

The demographic variables, cardiovascular risk factors, and cardioactive medications for the two groups are listed in Table 1. Mean arterial and systolic blood pressures were significantly higher in the thrombus group. Interestingly there was no correlation between systolic blood pressure and the incidence of
reported hypertension in the thrombus group ($R = 0.03, P = 0.86$). Nine patients in the thrombus group had a systolic blood pressure of $>140$ mmHg, whilst four of these patients reported no history of hypertension. However, in the non-thrombus group, there was a strong correlation between systolic blood pressure and reported hypertension ($R = 0.47, P = 0.001$). The incidence of ischaemic heart disease and congestive heart failure was significantly higher in the thrombus group. There was no difference in the CHADS2 score between the groups ($P = 0.82$). Additionally, the percentage of patients with a CHADS2 score $\geq 2$ did not vary between thrombus and non-thrombus groups ($P = 0.23$).

### Transthoracic echocardiogram parameters

TTE parameters are listed in Table 2. There was a significantly higher indexed LV mass in the thrombus group compared with non-thrombus group. Indexed LV mass weakly correlated with systolic blood pressure ($R = 0.19, P = 0.04$). LV diastolic function, as measured by $E'$ velocity, was significantly lower in the thrombus group. However, LV systolic function, as measured by ejection fraction and $S'$ velocity, did not differ. Twelve patients (33%) in the thrombus group had LVEF $<40\%$, whilst no patients in the non-thrombus group had LVEF $<40\%$ (AUC: 0.50). There was no difference in indexed LA volume between patients with thrombus and those without thrombus.

### Transoesophageal echocardiogram parameters

TOE parameters are listed in Table 3. In 37% of patients with a thrombus, the thrombus was not seen in a zoomed view of the LAA in one or more of the angles imaged. The LAA area was significantly larger in the thrombus group, whilst the mean emptying velocity was reduced (Table 3). The incidence of moderate to severe SEC in both the LA and RA was higher in patients with thrombus compared with non-thrombus.

### Table I  Demographic variables, cardiovascular risk factors and medications for non-thrombus and thrombus atrial fibrillation groups

<table>
<thead>
<tr>
<th></th>
<th>Non-thrombus ($n = 129$)</th>
<th>Thrombus ($n = 36$)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>$66 \pm 11$</td>
<td>$67 \pm 11$</td>
<td>0.7</td>
</tr>
<tr>
<td>Gender (% male)</td>
<td>72</td>
<td>78</td>
<td>0.5</td>
</tr>
<tr>
<td>Body surface area (m$^2$)</td>
<td>$1.9 \pm 0.2$</td>
<td>$2.0 \pm 0.3$</td>
<td>0.09</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>$124 \pm 15$</td>
<td>$132 \pm 17^{*}$</td>
<td>0.01</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>$73 \pm 10$</td>
<td>$76 \pm 12$</td>
<td>0.2</td>
</tr>
<tr>
<td>Mean arterial blood pressure (mmHg)</td>
<td>$90 \pm 11$</td>
<td>$95 \pm 13$</td>
<td>0.05</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>$82 \pm 16$</td>
<td>$84 \pm 19$</td>
<td>0.4</td>
</tr>
<tr>
<td>Duration of AF (months)</td>
<td>$24 \pm 36$</td>
<td>$14 \pm 19$</td>
<td>0.2</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>$2.4 \pm 1.0$</td>
<td>$2.1 \pm 1.0$</td>
<td>0.05</td>
</tr>
<tr>
<td>CHADS2 score $\geq 2$ (%)</td>
<td>40</td>
<td>51</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Cardiovascular risk factors (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td>42</td>
<td>31</td>
<td>0.3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>54</td>
<td>63</td>
<td>0.3</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>35</td>
<td>34</td>
<td>1.0</td>
</tr>
<tr>
<td>Diabetes</td>
<td>16</td>
<td>23</td>
<td>0.4</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>28</td>
<td>54$^{*}$</td>
<td>0.04</td>
</tr>
<tr>
<td>Coronary artery bypass graft surgery</td>
<td>15</td>
<td>23</td>
<td>0.3</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>14</td>
<td>31$^{*}$</td>
<td>0.02</td>
</tr>
<tr>
<td>Permanent pacemaker/defibrillator</td>
<td>4</td>
<td>8</td>
<td>0.4</td>
</tr>
<tr>
<td>Cerebral vascular accident</td>
<td>13</td>
<td>14</td>
<td>0.9</td>
</tr>
<tr>
<td><strong>Cardioactive medications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>5</td>
<td>6</td>
<td>0.9</td>
</tr>
<tr>
<td>Class III antiarrhythmics</td>
<td>65</td>
<td>52</td>
<td>0.2</td>
</tr>
<tr>
<td>Digoxin</td>
<td>58</td>
<td>49</td>
<td>0.4</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>17</td>
<td>12</td>
<td>0.5</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>32</td>
<td>18</td>
<td>0.1</td>
</tr>
</tbody>
</table>

$^{*}P < 0.05$ compared with non-thrombus group.
presence of thrombus in the group studied.

indexed LV mass was the most significant determinant for the parameter predictive of thrombus in this multivariate model was

univariate analysis had a
curve analysis was performed only for the four parameters that on

analysed were as follows: ischaemic heart disease: 0.63; LA SEC:

LA SEC (%)

1. Minimum 6 0 0.001

2. Mild 48 17 0.001

3. Moderate 43 50 0.001

4. Severe 4 3 1

LAA mean emptying velocity (cm/s)

44 ± 18 35 ± 15* 0.009

P- value,

LA SEC (%)

0. Absence 3 0 <0.001

1. Minimum 31 0 0.001

2. Mild 54 63 0.001

3. Moderate 13 34 0.001

4. Severe 0 3 0.001

RA SEC (%)

0. Absence 0 0 <0.001

1. Minimum 6 0 0.001

2. Mild 48 17 0.001

3. Moderate 43 50 0.001

4. Severe 4 3 1

LAA, left atrial appendage; LA, left atrium; SEC, spontaneous echo contrast; RA, right atrium.

*P < 0.05 compared with non-thrombus group.

Univariate predictors of thrombus in persistent atrial fibrillation patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non-thrombus (n = 129)</th>
<th>Thrombus (n = 36)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Mass (g)</td>
<td>133 ± 34</td>
<td>258 ± 69*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV Mass indexed (g/m²)</td>
<td>68 ± 17</td>
<td>132 ± 40*</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>42 ± 13</td>
<td>43 ± 15</td>
<td>0.7</td>
</tr>
<tr>
<td>systolic velocity (cm/s)</td>
<td>5.0 ± 1.1</td>
<td>4.6 ± 1.5</td>
<td>0.09</td>
</tr>
<tr>
<td>LA peak filling velocity (cm/s)</td>
<td>8.3 ± 2.2</td>
<td>7.3 ± 2.2*</td>
<td>0.02</td>
</tr>
<tr>
<td>LA maximum volume (mL)</td>
<td>80.4 ± 21.2</td>
<td>86.1 ± 28.4</td>
<td>0.2</td>
</tr>
<tr>
<td>LA maximum volume indexed (mL/m²)</td>
<td>41.7 ± 11.3</td>
<td>43.5 ± 16.6</td>
<td>0.5</td>
</tr>
<tr>
<td>LA total emptying fraction (%)</td>
<td>27 ± 7</td>
<td>28 ± 10</td>
<td>0.6</td>
</tr>
</tbody>
</table>

LV, left ventricle; LA, left atrium; SEC, spontaneous echo contrast.

LA SEC (R = 0.64, P = 0.002, 70% concordance) and RA SEC (R = 0.68, P = 0.001, 75% concordance).

Multiple logistic regression

Univariate predictors of thrombus are listed in Table 4. To address the issue of small sample numbers, multivariate analysis and ROC curve analysis was performed only for the four parameters that on univariate analysis had a P-value < 0.01. These included ischaemic heart disease, LA SEC, RA SEC, and indexed LV mass. The only parameter predictive of thrombus in this multivariate model was indexed LV mass (P < 0.001). The AUC for the parameters analysed were as follows: ischaemic heart disease: 0.63; LA SEC: 0.73; RA SEC: 0.75, and indexed LV mass: 0.98 (Figure 1). Thus, indexed LV mass was the most significant determinant for the presence of thrombus in the group studied.

Discussion

We examined clinical and echocardiographic predictors of LA thrombus in patients with persistent AF, recruited from a single site. The incidence of thrombus in patients with AF prior to cardioversion, despite anticoagulation, is reported at ~7% (13), in the current study the incidence of thrombus was similar at 8% (36/430). In 37% of patients in the thrombus group, the thrombus was not visualized in a zoomed view of the LAA in one or more of the angles. This highlights the importance of careful examination of the LAA in multiple imaging planes before excluding a thrombus, as the LAA can be a complex multi-lobed structure.
Demographic predictors of thrombus

Hypertension is the most prevalent condition associated with the development of AF in the community.\textsuperscript{23,24} This was emphasized in the recent position paper of the Working Group ‘Hypertension, Arrhythmias and Thrombosis’ of the European Society of Hypertension, which stated that hypertension is the primary risk factor for developing AF and that their coexistence significantly increases cardiovascular events.\textsuperscript{24} In AF patients, hypertension has been associated with increased SEC and thrombus formation.\textsuperscript{5,14,25,26} In the current study although a reported history of hypertension was not associated with thrombus, systolic blood pressure was a univariate predictor of thrombus. This may suggest a higher incidence of undiagnosed hypertension in the group studied. Ischaemic heart disease with LV dysfunction has also been associated with a higher risk of stroke, independent of blood pressure and other risk factors.\textsuperscript{38} Cujec et al.\textsuperscript{39} have previously reported that LV hypertrophy was associated with cardiac source of embolus; however, others have demonstrated no significant effect of LV mass.\textsuperscript{30,41} These studies, however, included patients in sinus rhythm with normal LV function. LV mass can easily be obtained and should perhaps be measured routinely in patients with persistent AF both to identify patients in whom antihypertensive treatment needs to be improved and to carefully screen those at an increased risk of LA and LAA thrombus. This consideration may be of special importance when cardioversion is being performed without a prior TOE (i.e. AF of short duration or anticoagulation for 4 weeks).

LV diastolic dysfunction results in LA enlargement and dysfunction, and both may precipitate AF.\textsuperscript{42} The $E'$ velocity was reduced in the thrombus group. LA enlargement has been shown to be predictive of LA SEC and thrombus in patients with chronic\textsuperscript{7,12,14,40} and paroxysmal AF.\textsuperscript{39} Both groups in the current study had LA enlargement and indexed LA volumes were similar due to the selective evaluation of patients with persistent AF.

Transthoracic echocardiogram predictors of thrombus

We have demonstrated that LV mass is the strongest predictor of thrombus in patients with persistent AF, by limited multivariate analysis. A recent literature review analysis has reported that the prevalence of LV hypertrophy is between 36 and 41% in hypertensive populations.\textsuperscript{33} LV hypertrophy has been weakly correlated with systolic, diastolic, and mean blood pressure,\textsuperscript{44,33} which was observed in our study. LV hypertrophy is not present in all hypertensive patients and this discrepancy may be due to genetic factors, suggesting that hypertensive patients with the LV hypertrophy phenotype may be at increased risk of LAA thrombus and subsequent stroke. The increased LV mass may also be a consequence of undiagnosed or poorly treated hypertension, resulting in LV diastolic dysfunction with resultant LA enlargement and altered LA haemodynamics\textsuperscript{24} which may predispose to thrombus formation.

Hypertension has been shown to coexist in 70% of stroke patients, highlighting the strong association between hypertension, AF, and cardiovascular events.\textsuperscript{37} Similar to the results of this study, LV hypertrophy has been associated with a higher risk of stroke, independent of blood pressure and other risk factors.\textsuperscript{38} The increased LV mass may additionally be attributed to other factors independent of hypertension, such as obstructive sleep apnoea syndrome.\textsuperscript{36}

Transthoracic echocardiogram predictors of thrombus

SEC, due to blood stasis, is common in AF and is associated with LA and LAA dysfunction and thrombus formation.\textsuperscript{3,5} Non-valvular AF causes global atrial dysfunction and consequently SEC formation should be similar for both the LA and the RA.\textsuperscript{9} Few reports have investigated RA SEC; although it has been closely associated with AF aetiology\textsuperscript{10} and pulmonary embolism.\textsuperscript{11} We have demonstrated that moderate-to-severe SEC in the LA (37%) and RA (33%) were univariate predictors, however, they failed to independently predict thrombus on multivariate analysis. LAA mean emptying velocity was significantly reduced and LAA area was
larger in patients with thrombus in our study, as has been described previously.\textsuperscript{3,8,12,40,43}

**Study limitations**

This study was a retrospective analysis, dependent on the medical records and echocardiographic images acquired previously. Secondly, patients had their anticoagulation managed by their general practitioners in the community aiming to maintain INR between 2.0 and 3.0. There was no central monitoring of the INR and the INR was not specifically checked on the day of admission, but was checked in all patients 1–2 days prior to admission.

The present study was conducted on relatively small numbers of patients, which may have led to beta-errors, concealing true predictive abilities. However, patients were recruited from a single site and over a significant time (August 1999 and April 2005) to obtain the presented numbers. Despite the relatively small population studied, the incidence of LAA thrombus formation in our study is similar to previous reports.\textsuperscript{13} The advantage of a single site study is that performance of TTE and TOE’s, as well as the measurements performed, utilized the same protocol. To address the issue of a small sample size being assessed with multiple parameters, we performed multivariate as well as ROC curve analyses using only the four most significant univariate predictors.

We do not have data available regarding thrombo-embolic events that occurred following the presentation for cardioversion. Renal function analysis, by either creatinine or eGFR, was not available in this cohort. It was beyond the scope of our present study to perform detailed measurements on all subsequent non-thrombus patients (i.e. the remaining 244 patients) presenting for TOE; however, as the controls were recruited consecutively it is unlikely to cause a systematic bias.

There is no standard view in which the LAA is imaged on TOE, although the two-chamber long-axis and the horizontal short-axis at the level of the aortic valve, as well as appendage views at varying angles were utilized.\textsuperscript{3,44} The view that best optimised the LAA size was used to obtain the area measurement. Finally, varying angles were utilized.\textsuperscript{3,44} The view that best optimised the LA site is that performance of TTE and TOE’s, as well as the measurements performed, utilized the same protocol. To address the issue of a small sample size being assessed with multiple parameters, we performed multivariate as well as ROC curve analyses using only the four most significant univariate predictors.

**Conclusions**

The strongest predictor of LAA thrombus in the current study was increased LV mass, which may be due to genetic factors or undiagnosed or inadequately treated hypertension. LA SEC and RA SEC were univariate predictors of LAA thrombus but did not add predictive value to a multivariate model including LV mass. This study highlights the importance of diagnosing and treating LV hypertrophy associated with persistent AF, which may reduce the risk of LAA thrombus and thrombo-embolic stroke.

**Acknowledgements**

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**Conflict of interest:** none declared.

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