

need to up-titrate tolvaptan to 30 mg/day to achieve normal sodium levels.

The available evidence on treatment with tolvaptan in patients with GBS and SIADH is strikingly scarce: to our knowledge, only one case has been published to date.

Sodium levels should be monitored in patients with GBS. Pseudohyponatraemia, water transport, and SIADH should be considered in the differential diagnosis of hyponatraemia. In our case, hyponatraemia may have played a role in the need for an additional cycle of Ig and the slow motor recovery.

References

1. Rumalla K, Reddy AY, Letchuman V, Mittal MK. Hyponatremia in Guillain-Barré syndrome. *J Clin Neuromuscul Dis*. 2017;18:207–17.
2. Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-Barré syndrome and SIADH. *Neurology*. 2011;76:701–4.
3. Sipilä JO, Kauko T, Soilu-Hänninen M. Admission sodium level and prognosis in adult Guillain-Barré syndrome. *Int J Neurosci*. 2017;127:344–9.
4. Hiew FL, Winer JB, Rajabally YA. Hyponatraemia in Guillain-Barré syndrome revisited. *Acta Neurol Scand*. 2016;133:295–301.
5. Wang Y, Liu J. Hyponatremia is a predictor for poor outcome in Guillain-Barré syndrome. *Neurol Res*. 2015;37:347–51.
6. Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, et al. Clinical practice guideline on diagnosis and treatment of hyponatraemia. *Nephrol Dial Transplant*. 2014;40:924.
7. Palevsky PM, Rendulic D, Diven WF. Maltose-induced hyponatremia. *Ann of Intern Med*. 1993;118:526–8.
8. Inoue M, Kojima Y, Shirakashi Y, Kanda M, Shibasaki H. A case of Guillain-Barré syndrome accompanied by syndrome of inappropriate secretion of antidiuretic hormone. *Rinsho Shinkeigaku*. 2010;50:710–3.
9. Çakırgöza MY, Duran E, Topuza C, Kara D, Turgut N, Türkmen Ü, et al. Syndrome of inappropriate antidiuretic hormone secretion related to Guillain-Barré syndrome after laparoscopic cholecystectomy. *Braz J Anesthesiol*. 2014;64:195–8.
10. Park SJ, Pai KS, Kim JH, Shin J. The role of interleukin 6 in the pathogenesis of hyponatremia associated with Guillain-Barré syndrome. *Nefrologia*. 2012;32:114.

J.E. Ternero Vega^{a,*}, R.G. León^a, D.A. Delgado^b, M.O. Baturone^a

^a Servicio de Medicina Interna, Hospital Universitario Virgen del Rocío, Sevilla, Spain

^b Servicio de Endocrinología y Nutrición, Hospital Universitario Virgen del Rocío, Sevilla, Spain

* Corresponding author.

E-mail address: jaraeloisa@hotmail.com

(J.E. Ternero Vega).

<https://doi.org/10.1016/j.nrleng.2017.12.011>
2173-5808/

© 2018 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Echocardiographic parameters of atrial cardiopathy and the detection of atrial fibrillation in patients with cryptogenic stroke[☆]



Parámetros ecocardiográficos de cardiopatía auricular y detección de fibrilación auricular en el ictus criptogénico

Dear Editor,

Cardioembolic stroke accounts for 20%-30% of all ischaemic strokes.¹ With the introduction of the term “embolic stroke of undetermined source” (ESUS), and given the considerable number of non-lacunar cryptogenic strokes, the hypothesis of an unknown embolic source has led to extensive study. This includes prolonged cardiac monitoring, which increases

sensitivity for identifying paroxysmal atrial fibrillation (AF).² Although the ability of these paroxysmal episodes to cause ischaemic events was previously unknown, studies into the pathophysiology of cardioembolic stroke have shown that paroxysmal AF, which usually lasts 5-6 minutes, increases the risk of stroke.^{3,4}

Given their short duration and the fact that they are often asymptomatic and may not co-occur with neurological symptoms, these episodes are classified under stroke of undetermined cause.^{3,5} The heterogeneity of this group, combined with the limited use of prolonged cardiac monitoring (whether due to patient intolerance or to the unavailability of monitoring equipment and differences between devices), has led researchers to investigate parameters correlated with greater incidence of AF,⁶ with a view to increasing the utility of the technique. These parameters include electrocardiographic, biochemical, and echocardiographic variables, which have been found to be associated not only with AF but also with ischaemic stroke and recurrent stroke.^{1,7}

We present the case of a patient with acute ischaemic stroke and morphological and functional signs of left atrial cardiopathy in an echocardiography study; these findings were correlated with paroxysmal AF.

The patient was a functionally independent 77-year-old woman who came to the emergency department due to right hemiparesis and mixed aphasia. She scored 1 on the NIHSS due to language impairment, which resolved com-

[☆] Please cite this article as: Lambea Gil Á, Tejada Meza H, López Perales CR, Artal Roy J, Marta Moreno J. Parámetros ecocardiográficos de cardiopatía auricular y detección de fibrilación auricular en el ictus criptogénico. *Neurología*. 2020;35:284–287.

Table 1 Transthoracic echocardiography findings: left atrium morphological and functional parameters, adjusted for body surface area and sex.

	Our patient	Normal results
AP diameter (mm) ⁸	40	LLN 38.0
Area in A4C view (cm ²) ⁹	25	LLN 20.0
AP diameter, indexed (mm/m ²) ⁸	24.1	LLN 23.0
Area in A4C view, indexed (cm ² /m ²) ⁸	15.1	LLN 11.0
Volume in A4C view, indexed (mL/m ²) ⁸	57.2	LLN 34.0
LAEF (%) ¹⁰	35.0	ULN 50.0
LAS in reservoir phase	20.0	ULN 25.8

A4C: apical 4-chamber; AP: anteroposterior; LAEF: left atrial ejection fraction; LAS: left atrial strain; LLN: lower limit of normal; ULN: upper limit of normal.

pletely 3 hours after symptom onset. A baseline head CT scan revealed no signs of haemorrhage or acute ischaemia; the patient scored 7 on the ABCD2 scale, and was admitted to the stroke unit for cardiac monitoring. She remained asymptomatic for 18 hours, after which she presented an episode of isolated mixed aphasia (NIHSS: 4). No changes were observed in CT images. Intravenous fibrinolysis achieved progressive recovery.

Our patient presented hypertension, dyslipidaemia, and type 2 diabetes mellitus; treatment adherence was good and blood analysis results were within normal ranges. In 2012, she underwent implantation of a DDDR pacemaker due to complete atrioventricular block associated with sinus dysfunction. An echocardiography performed in 2016 revealed mild left ventricular hypertrophy and mitral valve regurgitation, with no alterations in atrial morphology or other signs of structural heart disease. In view of the 2 episodes of sudden-onset focal neurological signs (with one episode characterised by isolated aphasia), we requested the pacemaker records from the cardiology department.

The patient continued under observation while we awaited the pacemaker records. Twenty-four-hour Holter monitoring revealed sinus rhythm and no arrhythmia; the patient was therefore transferred to the neurology ward. A Doppler ultrasound of the supra-aortic trunks revealed atheromatous plaques in the carotid territory bilaterally, without haemodynamic impairment; transcranial Doppler ultrasound did not detect stenosis. [Table 1](#) shows the findings from transthoracic echocardiography, adjusted for body surface area (1.657 m²). Atrial diameter and area were compatible with mild left atrial enlargement, whereas volume and such functional activity parameters as left atrial ejection fraction (LAEF) and left atrial strain (LAS) indicated more severe dysfunction of the cardiac chamber ([Fig. 1](#)). The pacemaker data revealed an episode of paroxysmal AF lasting 17 hours, which occurred 3 months previously, and another episode occurring 4 days after the cerebrovascular event; the patient presented sinus rhythm during neurological symptoms.

The patient was asymptomatic at discharge and was diagnosed with ischaemic stroke of cardioembolic origin secondary to paroxysmal AF. She started treatment with oral anticoagulants; her functional status at 3 months was similar to that before hospital admission (mRS: 1).

We were presented with the case of a patient with no signs of significant intra- or extracranial atherosclerosis, dis-

playing sinus rhythm during 24-hour Holter monitoring. In view of the strong association between isolated aphasia and cardioembolic arrhythmia,¹² we performed a comprehensive transthoracic echocardiography study to evaluate parameters that may support prolonged cardiac monitoring, in case the pacemaker records showed no relevant data, considering the potential for episodes of AF not to co-occur with stroke onset.⁵

The echocardiographic parameters most frequently used in our hospital to characterise the left atrium are area and anteroposterior diameter; whenever possible, these are adjusted for body surface area to avoid sex-related variability. However, these parameters tend to underestimate the true size of the atrium (as compared to volumetry) since linear measurements do not reflect differences in deformation on different planes.⁸ Left atrial volume is more accurate but has the limitation that it is difficult to measure. Technical advances have made it easier to calculate, making this the morphological parameter of choice in clinical practice.^{8,9} Another potentially useful structural parameter is the analysis of left atrial appendage morphology with transoesophageal echocardiography, due to the potential association between certain morphological patterns and increased risk of thromboembolism.¹³

Left atrial function parameters are less frequently used, although they are also reported to be correlated with increased risk of AF and ischaemic stroke regardless of the morphology of this chamber.^{10,11} Even in patients with normal left atria, this association represents a shift from the traditional classification of atrial damage based exclusively on chamber enlargement. Two useful left atrial function parameters are LAEF and LAS. The latter measures myocardial deformation using speckle tracking echocardiography; this technique analyses atrial contractility, which has been found to be associated with structural degeneration.¹¹

Based on the data shown in [Table 1](#) and the considerations discussed with regard to different echocardiographic parameters, the transthoracic echocardiography study typically performed at our hospital (with the left atrium assessed by measuring anteroposterior diameter and area) would only have shown mild left atrial enlargement; however, volumetric data indicated severe atrial enlargement ([Table 2](#)). This finding, which would have gone unnoticed had we not gathered volumetric data, points to an association between left atrial enlargement and both higher incidence of AF and

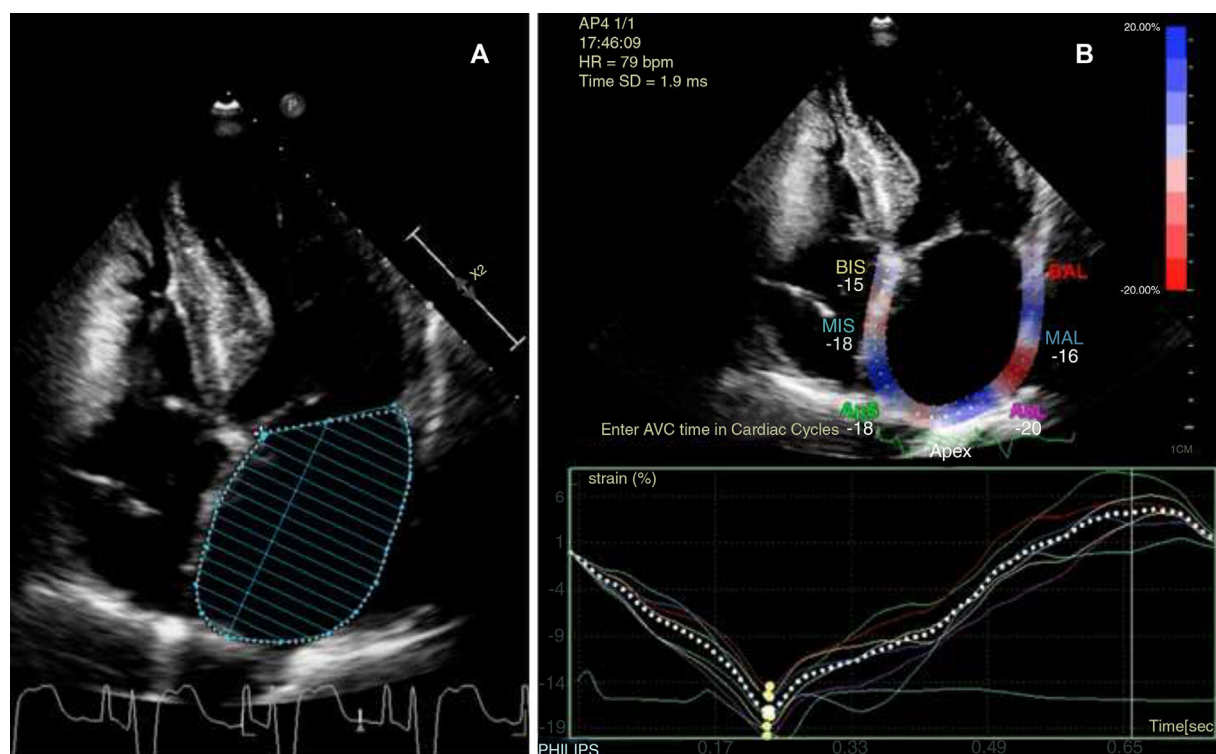


Figure 1 Two-dimensional transthoracic echocardiography; apical 4-chamber view. Measurement of left atrial volume with the biplane Simpson method (A) and longitudinal left atrial strain with speckle tracking echocardiography (B).

Table 2 Left atrial morphology data obtained with transthoracic echocardiography and classification by severity.

	Our patient	Normal range	Mild enlargement	Moderate enlargement	Severe enlargement
AP diameter (mm) ⁹	40	27-38	39-42	43-46	≥ 47
Area in A4C view (cm ²) ⁹	25	≤ 20	20-30	30-40	> 40
Volume in A4C view, indexed (mL/m ²) ⁸	57.2	16-34	35-41	42-48	> 48

A4C: apical 4-chamber; AP: anteroposterior.

recurrent stroke.¹⁴ An LAEF below 50% and an LAS below 25.8% in the reservoir phase also point to underlying paroxysmal AF.^{10,11}

The aetiological study of stroke includes echocardiographic parameters and other biochemical and electrocardiographic markers (proBNP levels, P terminal force in the V1 lead, etc) to identify probable emboligenic arrhythmia; the inclusion of these parameters is based on the traditional idea of AF as a cause of cardiac remodelling, venous stasis, and subsequent thromboembolism. However, research into the association between these parameters and AF incidence suggests that many of them correlate directly with the incidence and recurrence of ischaemic stroke, regardless of whether the patient has AF.^{1,7} Consequently, the pathophysiology of cardioembolic stroke is thought to be more complex: the idea of atrial cardiopathy as a dysfunction capable in itself of increasing the risk of embolism is becoming more widespread; AF is still considered the cause, aggravating factor, or consequence, depending on the case, but is no longer regarded as a necessary condition for cardioembolic stroke.^{1,7,15}

However, regardless of these new hypotheses about the pathophysiology of thrombus formation, AF continues to be a major source of cardioembolism; detection of AF is closely related to the type and duration of cardiac monitoring to be performed.¹⁵ The case presented constitutes a good example of the usefulness of certain echocardiographic parameters due to their association with higher incidence of underlying AF. Furthermore, in our patient these parameters were the only factors associated with emboligenic arrhythmia; this is particularly interesting considering the increasing importance of focused cardiac ultrasound in neurology training.

In conclusion, different tools may be used to determine whether ischaemic stroke of undetermined aetiology is due to emboligenic factors. When the most frequent emboligenic causes are ruled out, left atrial volumetry or assessment of left atrial function (LAEF and LAS) may help us to determine whether patients are eligible for prolonged cardiac monitoring to screen for AF with a view to choosing the most suitable treatment.

References

- Kamel H, Healey J. Cardioembolic stroke. *Circ Res*. 2017;120:514–26.
- Hart RG, Diener HC, Coutts SB, Easton JD, Granger CB, O'Donnell MJ, et al. Embolic strokes of undetermined source: the case for a new clinical construct. *Lancet Neurol*. 2014;13:429–38.
- Healey JS, Connolly SJ, Gold MR, Israel CW, van Gelder IC, Capucci A, et al. Subclinical auricular fibrillation and the risk of stroke. *N Engl J Med*. 2012;366:120–9.
- Glutzer TV, Daoud EG, Wyse DG, Singer DE, Ezekowitz MD, Hilker C, et al. The relationship between daily auricular tachyarrhythmia burden from implantable device diagnostics and stroke risk: the TRENDS study. *Circ Arrhythm Electrophysiol*. 2009;2:474–80.
- Brambatti M, Connolly SJ, Gold MR, Morillo CA, Capucci A, Muto C, et al. Temporal relationship between subclinical auricular fibrillation and embolic events. *Circulation*. 2014;129:2094–9.
- Kamel H, Okin PM, Elkind MS, Iadecola C. Atrial fibrillation and mechanisms of stroke. *Stroke*. 2016;47:895–900.
- Kamel H, Okin PM, Longstreth WT, Elkind MS, Soliman EZ. Atrial cardiopathy: a broadened concept of left auricular thromboembolism beyond auricular fibrillation. *Future Cardiol*. 2015;11:323–31.
- Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;28:e14.
- Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification. *Eur J Echocardiogr*. 2006;7:79–108.
- Sanchis L, Montserrat S, Obach V, Cervera Á, Chamorro Á, Vidal B, et al. La función de la aurícula izquierda está alterada en algunos pacientes con ictus criptogénico: potenciales implicaciones en su evaluación y tratamiento. *Rev Esp Cardiol*. 2016;69:650–6.
- Pagola J, González-Alujas T, Flores A, Muchada M, Rodríguez-Luna D, Seró L, et al. Left atrial strain is a surrogate marker for detection of atrial fibrillation in cryptogenic strokes. *Stroke*. 2014;45:e164–6.
- Fennis TFM, Compter A, van den Broek MW, Koudstaal PJ, Algra A, Koeheler PJ. Is isolated aphasia a typical presentation of presumed cardioembolic transient ischemic attack or stroke? *Cerebrovasc Dis*. 2013;35:337–40.
- Korhonen M, Muuronen A, Arponen O, Mustonen P, Hedman M, Jakala P, et al. Left atrial appendage morphology in patients with suspected cardiogenic stroke without known atrial fibrillation. *PLoS ONE*. 2015;10, e0118822.
- Yaghi S, Moon YP, Mora-McLaughlin C, Willey JZ, Cheung K, Di Tullio MR. Left atrial enlargement and stroke recurrence: the Northern Manhattan Stroke Study. *Stroke*. 2015;46:1488–93.
- Nouh A, Hussain M, Mehta T, Yaghi S. Embolic strokes of unknown source and cryptogenic stroke: implications in clinical practice. *Front Neurol*. 2016;7:37.

Á. Lambea Gil^{a,*}, H. Tejada Meza^{b,c}, C.R. López Perales^d, J. Artal Roy^b, J. Marta Moreno^b

^a Servicio de Neurología, Hospital Universitario Miguel Servet, Zaragoza, Spain

^b Unidad de Ictus, Servicio de Neurología, Hospital Universitario Miguel Servet, Zaragoza, Spain

^c Instituto de Investigación Sanitaria (IIS) Aragón, Zaragoza, Spain

^d Servicio de Cardiología, Hospital Universitario Miguel Servet, Zaragoza, Spain

*Corresponding author.

E-mail address: alvarolambea@gmail.com (Á. Lambea Gil).

<https://doi.org/10.1016/j.nrleng.2017.12.013>
2173-5808/

© 2018 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Transient contrast-induced encephalopathy after internal carotid artery embolisation prior to surgery for nasopharyngeal carcinoma[☆]

Encefalopatía transitoria por contraste tras la embolización de la arteria carótida interna previa a la cirugía de carcinoma nasofaríngeo

Dear Editor,

Angiography is a technique used in the diagnosis, surgical planning, and treatment of tumours. Presurgical embolisation of the internal carotid artery is a common adjuvant



therapy to the surgical resection of head and neck cancers.¹ The technique is made possible by the flow compensation provided by the circle of Willis through the intracranial communicating arteries.

Contrast-induced encephalopathy is a rare but reversible complication that appears after the administration of an iodinated contrast agent; it was first described in 1970 in a patient who presented cortical blindness following cardiac catheterisation.²

We present the case of a patient with transient contrast-induced encephalopathy after embolisation of the left internal carotid artery before surgery for nasopharyngeal carcinoma.

Our patient was a 41-year-old man with a history of locally relapsing squamous cell carcinoma of the nasopharynx; he had previously been treated with surgery, chemotherapy, and radiotherapy, and was admitted electively to our centre for surgical reintervention. Three months before surgery, the patient underwent an angiography-based balloon test occlusion of the carotid artery (200 mL of non-ionic iodinated contrast agent were administered), which confirmed that there was sufficient flow compensation by the circle of Willis and the absence

[☆] Please cite this article as: Montejo C, Rodríguez A, Pascual-Vicente M, Renú A. Encefalopatía transitoria por contraste tras la embolización de la arteria carótida interna previa a la cirugía de carcinoma nasofaríngeo. *Neurología*. 2020;35:287–289.