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Confusion in connection with pulmonary hypertension due to premature ventricular complexes requires diagnostic work-up

We read with particular interest the comments of an experienced letter's author[1] regarding our case report on a 76-year-old patient with pulmonary hypertension and severe hypoxaemia caused by a significant mitral regurgitation during premature ventricular complexes.[2] First and foremost, we would like to emphasize that the narrative title of our report was deliberately chosen, so that the pathophysiology of this rare case would not be given away right at the beginning. Our patient had a clear cardiopulmonary aetiology of the severe generalized hypoxaemia, which caused confusion among other symptoms. The treatment of the acute decompensated heart failure with correction of hypoxaemia eliminated the confusion in this case. If the patient or her relatives after correction of hypoxaemia and treatment of the underlying cause had complained of ongoing, permanent, or intermittent/recurrent focal or diffuse neurological signs and symptoms, further investigations to determine neurological sequelae of her hypoxaemia or other differential diagnoses would of course have been initiated. This could have included among others the suggested cerebral magnetic resonance imaging to rule out focal or generalized hypoxaemic lesions, callosal lesions, microbleeds, and other structural damage such as stroke; an electroencephalogram for suspected ictual phenomena or cerebrospinal fluid analysis; and finally extensive neuropsychological testing. In our case, this was neither efficient or necessary, nor constructive or effective, and by no means mandatory.

We are happy to explain to the letter's author that brain-type natriuretic peptide (BNP) is released in response to myocardial stretch from ventricular cardiomyocytes[3,4] and its levels are not necessarily related to the systolic function of either ventricle. Previous studies have illustrated sufficiently that BNP levels (or levels of the inactive N-terminal fragment of proBNP = NTproBNP) are markedly elevated in patients with mitral regurgitation[5] or pulmonary hypertension.[6]

We would like to remind the letter's author that we have provided the relevant information from the Holter-ECG in our report. Episodes of atrial fibrillation were not recorded during Holter-ECG or telemetry, nor was paroxysmal atrial fibrillation known in the anamnesis.

Finally, we would like to thank the letter's author for sharing his concerns from a neurological point of view and the moderately helpful comments with regard to our case.

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Data availability
The data underlying this article will be shared upon reasonable request to the corresponding author.

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