Interaction Between Brain and Heart
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In 1990, Sato et al first reported Japanese patients presenting with chest symptoms, ECG changes and a reversible regional wall motion abnormality in the apical to mid segments of left ventricle (LV) extending beyond the territory of a single coronary artery. It was given the name of takotsubo cardiomyopathy, and is now recognized as an established clinical entity. A large number of studies from other countries as well as Japan have shown that takotsubo cardiomyopathy is often preceded by exposure to emotional or physical stress, and occurs predominantly in elderly women. Sympathetic hyperexcitation, coronary vasospasm and microcirculatory disorder have been regarded as the most likely factors, but our understanding of the pathologic mechanism remains limited at present. Typically, the regional wall motion abnormality is observed in the LV apical to mid segments, and compensatory hypercontractility is present in the basal segment. There has been no single universally accepted diagnostic definition of takotsubo cardiomyopathy. In 2004, Mayo Clinic criteria were first proposed for the diagnosis of takotsubo cardiomyopathy. The initial Mayo Clinic criteria required the exclusion of head trauma or intracranial bleeding, but it was pointed out that takotsubo cardiomyopathy and subarachnoid hemorrhage (SAH)-induced acute cardiac dysfunction (ACD) may share a similar mechanism of a massive catecholamine release. In 2008, the Mayo Clinic criteria were modified, and it became no longer required to exclude head trauma or intracranial bleeding, but to consider atypical types of takotsubo cardiomyopathy in terms of the involvement of LV segments other than the apical segment. At present, the revised Mayo Clinic criteria are most widely used in clinical practice and research. For this reason, recent studies in the population of takotsubo cardiomyopathy include some cases of ACD associated with acute brain hemorrhage as mentioned as stroke, central nervous conditions or acute neurogenic disorder in each report. According to these studies, the apical type and mid-ventricular type are found in 82–84% and 15–17% of the patients, respectively, and other types such as basal or focal type are rare.

SAH-induced ECG changes, pulmonary edema or ACD has been well recognized since Burch et al first described them. In 1994, Kono et al evaluated coronary angiograms of patients with SAH and ST-segment elevation, and first demonstrated that patients with SAH may have ACD in the absence of angiographic evidence of obstructive coronary artery disease. In the clinical setting, various types of acute brain hemorrhage other than SAH are often found.

In this issue of the Journal, Lee et al report that ACD occurred in 7% with even sex distribution in a population of patients with acute brain hemorrhage. Furthermore, they show that apical-sparing types were observed more frequently rather than the apical type, and point out that the distribution of the LV type was in contrast to that of takotsubo cardiomyopathy. So far, it remains unclear what regulates the location of the regional wall motion abnormality. Infrequently, takotsubo cardiomyopathy presents as the apical type on initial presentation and an apical-sparing type during a recurrence. It is a matter of great interest whether young age and potent catecholamine surge represented by acute brain hemorrhage are major determinants of the apical-sparing types. It should be tested by further studies, and this may give a clue to the pathologic mechanism of takotsubo cardiomyopathy. The present study adds further new insights to an important clinical issue of acute brain hemorrhage. Lee et al demonstrate that ACD as well as Glasgow coma scale was a major predictor of in-hospital death; 8 of the 9 patients with ACD and in-hospital death had poor neurogenic condition. This finding indicates that ACD would be important as a surrogate marker of poor neurogenic condition rather than the cause of death in the setting of acute brain hemorrhage. Nonetheless, ACD has potential cardiac complications such as heart failure, arrhythmias, cardiogenic shock, intracardiac thrombosis and acute cardiac dysfunction.

Figure. Potential implications of acute cardiac dysfunction in the setting of acute brain hemorrhage.

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shock or intracardiac thrombosis. These can lead to the complicated management of acute brain hemorrhage; for example, prolonged intubation, vasospasm, hemodynamic instability or unacceptable anticoagulation (Figure). The potential implications of ACD are substantial for neurologists primarily responsible for the care of patients with acute brain hemorrhage. Early detection and appropriate management of ACD based on collaboration between neurologists and cardiologists are required to prevent downward spiral, and will contribute to the improvement of prognosis.

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**References**