A 69-year-old woman who had never evidenced heart disease was admitted to our hospital for the treatment of an aneurysm at the tip of the basilar artery. The aneurysm was treated by endovascular treatment using Guglielmi detachable coils. However, the aneurysm ruptured during the procedure, resulting in massive subarachnoid hemorrhage (SAH). The patient had convulsions and was orally intubated to control her respiration. Finally, the SAH was stopped by coil embolization. ECGs were serially recorded during the procedure (Figure). The tracing, which was normal before SAH, showed sinus tachycardia with frequent ventricular ectopics after 15 seconds, wide QRS tachycardia indicating either transient ventricular tachycardia or sinus tachycardia with intraventricular conduction delay after 5 minutes, and sinus tachycardia with marked ST-segment elevation in almost all leads after 7 minutes. These ECG changes mimicked those observed in acute myocardial ischemia. Thereafter, the ST-segment elevation gradually improved, and the QT interval was prolonged. The following day, the ECG showed an inverted T wave, and echocardiography showed global hypokinesis except for the basal segment of the left ventricle. These findings were similar to takotsubo (am-pulla) cardiomyopathy. Serial measurements of creatine phosphokinase showed an elevation to a level of 2890 IU/L; however, the MB isozyme accounted for only 2% of this value. The patient died as a result of brain damage 5 days after the procedure, and it could not be confirmed whether the myocardial dysfunction was reversed. Takotsubo cardiomyopathy has been reported frequently in Japan; however, it is relatively unknown in the United States. Most patients with this cardiomyopathy experience symptoms suggestive of either acute myocardial infarction or heart failure after emotional distress or physical stress, including cerebrovascular accidents. The pathophysiology of this disease is still unknown; however, multiple vasospastic angina, microvascular spasm, mid-ventricular obstruction, and enhanced sympathetic activities have been implicated as potential causes. Conversely, myocardial damage after SAH was studied intensively after the 1960s, and the central nervous system is considered to affect the myocardium in 2 ways. First, an indirect effect can be mediated through the release of humoral substances such as epinephrine and norepinephrine. Second, direct control occurs through efferent and afferent connections with the 2 main divisions of the autonomic nervous system: the parasympathetic, and the sympathetic nervous systems. The findings in this patient suggest that both takotsubo cardiomyopathy and myocardial damage after SAH are induced by the same mechanism. Furthermore, direct sympathetic control through efferent neural connections resulted in myocardial damage at least in the initial stage because the changes in the ECG findings of this patient were too fast to be caused by an elevation in neurohumoral factors.

References
The serial changes in the ECG and the plot of ST level in ECG, heart rate (HR), and systolic blood pressure (SBP) versus time after the onset of SAH. Open circles show the ST level during wide QRS tachycardia, and open circles with crosses show the ST level during intraventricular conduction delay.
Serial Changes of the Electrocardiogram During the Progression of Subarachnoidal Hemorrhage

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