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• 英文摘要	3.1. The [Ca2+]i transient and Calcium stores in PV and Atrial Cardiomyocytes Figure 1A shows the tracings of the [Ca2+]i transient in the PV and LA cardiomyocytes. The PV cardiomyocytes with pacemaker activity (n = 15) had a larger amplitude of the [Ca2+]i transient (R410／485, 0.36 +- 0.04, 0.25 +- 0.02, 0.24 +- 0.02, P < 0.05), and higher peak systolic (R410／485, 1.16 +- 0.05, 0.92 +- 0.03, 0.96 +- 0.02, P < 0.01) and diastolic [Ca2+]i concentration (R410／485, 0.79 +- 0.02, 0.67 +- 0.02, 0.73 +- 0.01, P < 0.01) than did the PV cardiomyocytes without pacemaker activity (n = 12) 8 and LA cardiomyocytes (n = 12). In addition, the PV cardiomyocytes with pacemaker activity had a longer time to the peak of the [Ca2+]i transient than did the PV cardiomyocytes without pacemaker activity and LA cardiomyocytes (46 +- 3, 35 +- 3, 34 +- 3, P < 0.05). The tauCa in the PV cardiomyocytes with pacemaker activity was shorter than that in the PV cardiomyocytes without pacemaker activity and LA cardiomyocytes (64 +- 5, 117 +- 13, 119 +- 9 ms, P<0.01). However, the amplitudes of the [Ca2+]i transient, peak systolic [Ca2+]i, diastolic [Ca2+]i, time to the peak of the [Ca2+]i transient, and tauCa were not significantly different between the LA cardiomyocytes and PV cardiomyocytes without pacemaker activity. The administration of caffeine induced a rapid rise in the [Ca2+]i in the PV and LA cardiomyocytes (Figure 1B). The PV cardiomyocytes with pacemaker activity (n = 8) had a larger SR Ca2+ store than did the PV cardiomyocytes		

without pacemaker activity (n = 16) and LA cardiomyocytes (n = 13, R410／480, 0.61 +- 0.03, 0.42 +- 0.02, 0.40 +- 0.02, P < 0.05). However, there was a similar SR Ca2+ store between the PV cardiomyocytes without pacemaker activity and the LA cardiomyocytes. The time constant of the caffeine-induced [Ca2+] decay was similar among the LA and PV cardiomyocytes with and without pacemaker activity. 3.2. Effect of magnesium on the [Ca2+]i transients In the PV cardiomyocytes without pacemaker and LA cardiomyocytes, magnesium (1.8 and 5.4 mM) reduced the amplitude of the [Ca2+]i transient and peak systolic [Ca2+]i, in a concentration-dependent manner but did not change the diastolic [Ca2+]i. In contrast, only magnesium with a high concentration (5.4 mM) reduced the [Ca2+]i transient and peak systolic [Ca2+]i in the PV cardiomyocytes with pacemaker activity (Figure 2A and 2B). Moreover, as the examples show in figure 2C, magnesium (5.4 mM) could also reduce the spontaneous beating rates in the PVcardiomyocytes from 1.5 +- 0.4 Hz to 0.9 +- 0.3 Hz (n = 3, P < 0.05).