In Fig.S1, the system hardware of the detection device included EFP conditioning module, CI conditioning module and a DAQ card (National Instruments). The EFP conditioning module was used to record and modulate the extracellular field potential (EFP) signal of HL-1 cardiomyocytes, including a two-order amplification circuit and the notch filter was applied to eliminate the 10 k Hz influence because of the excitation voltage in CI conditioning module. The CI conditioning module was a current-to-voltage amplification circuit to measure the cell impedance (CI), high-pass filter was used to exclude the drift baseline and get the formed 10 k Hz signal. DAQ card was used both to generate the 10 k Hz excitation voltage for cell impedance detection and acquire EFP and CI signal in this system.



Fig.S1. The workflow of the platform hardware

<u>CCK-8</u> methods was a control experiment for the comparison with the result of cell viability result through our CI monitoring method. A micro-titer plate reader SpectraMax Paradigm (Molecular Devices, USA) was used to measure the optical density (OD) and the value of its detection result ranged from 0 to 4. Fig.S2 showed the result of the scanning wavelength spectrum and the best detection sensitivity could be seen at 450 nm, which was the same as the kit's instruction. It also left much space in the detection range of SpectraMax Paradigm because the result of the OD at 450 nm was 1.



Fig.S2. The distribution of optical density (OD)-wavelength spectrum

The two drug-induced optical results using live/dead assays were provided in Fig.S3 (Vinblastine) and Fig.S4 (Nifedipine). The figures for the cell status were present with 10x sight at start, 6 hours, 12 hours and till the end of our experiment, 24h. First, HL-1 cardiomyocytes covered the whole surface of the electrode both in Vinblastine or Nifedipine-induced groups. In Vinblastine-induced experiment, the results of 0.01 µM and 0.1 µM groups showed that Vinblastine at low-dose did little harm to the cell viability as live ones became much more crowed over the surface of the electrodes, and the same trend could also be seen in our cell impedance monitoring and CCK-8 results that the curves of these two groups saw the same growing tendency as the control group. However, in the 1  $\mu$ M Vinblastine group, the proliferation of HL-1 cardiomyocytes was partly inhibited that there were many holes could be seen in the images where the HL-1 cells could not grow over there. And this phenomena became more obvious in the 10 µM Vinblastine group that with higher Vinblastine dose, more serious proliferation inhibition even apoptosis cells leaving the surface of the electrodes due to cytoskeletal toxicity could be observed. Our cell viability monitoring and CCK-8 result also showed a downwards curve of 10 µM Vinblastine group. However, unlike Vinblastine, Nifedipine showed little harm to the HL-1 cardiomyocytes, with most cells in live status through the images. Likewise, these similar tendencies held the same results as our cell viability curves, which showed a minimum 86.2% viability compared to the control group, and CCK-8 results with 83.3% survival rate at least.



Fig.S3. Vinblastine-induced live/dead assays result.



Fig.S4. Nifedipine-induced live/dead assays result.

Fig.S5 presented the statistical results of different durations of the extracted 5 inner-period feature points in a single period. It could be seen from the 0.001  $\mu$ M and 0.01  $\mu$ M group that t<sub>1</sub> and t<sub>4</sub> saw a decrease, t<sub>2</sub> increased a little, while t<sub>3</sub> remain nearly unchanged since 10 min to 30 min. t<sub>QT</sub> represented the total duration of t<sub>1</sub> to t<sub>4</sub>, which decreased obviously due to the changes of each component. t<sub>5</sub> represented the duration between each signal period and it saw a huge increase from ~100 milliseconds to more than 1 second, indicating a harder depolarization cardiac side-effects induced by Nifedipine.



**Fig.S5.** Statistical results of inner-period intervals. a-d) Line graph of  $t_1$  to  $t_4$  respectively. e) Overall QT intervals (the sum from  $t_1$  to  $t_4$ ). f) Line graph of the period-to-period interval  $t_5$