Myocardial Electrophysiology

ANATOMY OF CONDUCTION

SINOATRIAL NODE: Tiny little 15mm long oval area on the back wall of the right atrium, just under the entrance of the superior vena cava. These cells are not contractile. They conduct action potentials directly to the atrial wall.

INTERNODAL PATHWAYS: Most textbooks will rant on thin bands of atrial wall muscle fibers which conduct faster than the rest (1m/s vs. 30cm/s). Our wise elders in cardiology will tell you that in actual fact, the impulse simply picks the thickest part of the atrium (the one with the fastest conduction) and reach the AV node that way.

ATRIOVENTRICULAR NODE: Another little non-contractile mass of modified muscle tissue designed to delay conduction so that the atria have time to empty into the ventricles. The delay is only about 0.16 seconds; its accomplished in a brutally stupid way, by decreasing the number of gap junctions and thereby slowing the rate of ion movement- so each subsequent cell takes longer to reach its threshold and get excited.

ATRIOVENTRICULAR BUNDLE: 15 mm bundle of Purkinje fibers, which conduct at a ridiculous velocity of about 4 meters per second. The speed increase is accomplished by increasing the number of gap junctions, i.e. the reverse of the way the atrioventricular node slows down its conduction. These fibers have practically no myofibrils in them, so they don’t really contract at all. Additionally, the AV bundle DOES NOT PERMIT BACKWARD CONDUCTION: its just one-way to the ventricles.

PURKINJE FIBERS: LEFT AND RIGHT BUNDLE BRANCHES sub-endocardial bands traveling on either side of the septum; their ends penetrate about a third of the way into the ventricular muscle, and then merge with the muscle fibers there. It takes 0.03 seconds for an impulse to travel from the AV node to the ends of the Purkinje fibers.

VENTRICULAR MUSCLE conducts at a lazy pace of 30 to 50 centimeters per second. The fibers are wrapped in a spiral fashion around the heart, with fibrous septa between them; and so it takes a little longer for the impulse to reach the outside surface.

AUTOMATIC DEPOLARISATION: SINOATRIAL PACEMAKER

Unlike a proper myocardial muscle cell, the SA node cells rest at a less negative baseline of about -55 mV. Because of this, the fast sodium channels remain inactive- they require a voltage of -60 or more to activate. Therefore, the upstroke is not as fast as in a myocardial muscle cell; the channels responsible for the upstroke are the sluggish calcium channels. DURING PHASE 4: instead of staying stable at rest, the potential difference gradually becomes less negative. This is due to an inward sodium leak, which slowly brings positive charge. The sympathetic nervous system increases the rate of sodium leakage, and so brings these cells to threshold sooner. As the negativity reaches -40mV, voltage-gated calcium channels open and depolarize the cell at a leisurely pace. Thereafter, just like in any myocyte, the calcium current drops off and the potassium current repolarises the cell.