Acute myocardial infarction (AMI) is a common disease and one of the leading causes of death. 

**Background:** We studied the indications, complications, and course of patients admitted to a community hospital and needed temporary pacemaker. Iatrogenic cause of conduction disturbance is a frequent cause for need of temporary pacemaker.

**Purpose of Study:**

- To describe the characteristics of the patients who needed temporary pacemaker.
- To evaluate the role of temporary pacemaker as their permanent pacemaker generator had reached end of life.

**Methods:**

- All patients admitted to a community hospital and needed temporary pacemaker were included in the study.
- The patients were divided into: 1) those who were admitted with conduction disturbance as their permanent pacemaker generator had reached end of life and needed temporary pacemaker, and 2) those whose conduction disturbance was due to other causes.

**Results:**

- Ninety-eight percent of events occurred on Days 0–3. All 3 pts with later events also had cardiac complications, 9 had persistent conduction defect needing permanent pacemaker.
- Eighty-one (11%) patients had hyperkalemia (medication induced or renal failure) and 3 patients had digoxin toxicity as the cause of conduction disturbance. 6 patients needed temporary pacemaker as their permanent pacemaker generator had reached end of life.

**Conclusion:** Temporary support of conduction system prior to placement of permanent pacemaker was the commonest cause of temporary pacemaker in our study. Myocardial infarction leading temporary pacemaker patient with a high mortality.

The intracellular barrier properties of cultured endothelial cell monolayers are critical for the development of cardiovascular diseases.

**Hypothesis:** Extracellular adenosine triphosphate (ATP) has been known to protect endothelial barrier. Extracellular ATP may prevent thrombin-induced barrier disruption. In this study we defined the mechanisms of endothelial barrier enhancement caused by extracellular ATP.

**Methods:** Combination of pharmacological and molecular approaches is used in this study. Summary of Results: ATP and its non-hydrolyzed analogues enhanced barrier properties of cultured endothelial cell monolayers, caused remodeling of cell-cell junctions, and significantly attenuated thrombin-induced barrier disruption. Intracellular Ca²⁺ increase and EriK activation occurred by ATP were irrelevant to barrier enhancement. Inhibitory action and silencing RNA revealed the involvement of G proteins (specifically Gq, and Goα) as well as protein kinase A and its substrate VASP in ATP-induced barrier enhancement. Contractile state of endothelial cells governed by myosin light chain (MLC) phosphorylation underlies barrier properties. ATP treatment decreased MLC phosphorylation and specifically activated myosin-associated phosphatase. Depletion of Goα with siRNA prevented ATP-induced activation of specific PKC isoforms to the plasma membrane indicating their activation. PKC inhibitor Ro 31-8220 abolished EC-specific stimulation. A 1973 study revealed an increased minimal effect on permeability induced by 100 ng/mL VEGF. Collectively, these data suggest that low VEGF concentration may protect endothelial monolayer integrity in increase in CAMP production and activation of specific PKC isoforms.