mality and its incidence was 0.6% in the Coronary Artery Surgery Study (CASS) registry [12]. It was demonstrated that atrial fibrillation is a predictor of survival. Interatrial conduction delays have been shown to be implicated in initiating and maintaining AF [13-15].

Another mechanism for increased PD may be the increase in collagen fiber deposition in the cardiac interstitium. It was reported that PD was associated with inhomogeneus and discontinuous propogation of sinus impulses [16]. Electrocardiographic markers of abnormal atrial conduction, such as PD, P maximum, and P minumum, may be influenced by myocardial ischemia. Atrial fibrosis due to myocardial ischemia may prolong PD [17-20]. Previous studies have demonstrated that atrial ischemia is implicated in the pathogenesis of AF [21, 22].

Dilaveris et al. reported that myocardial ischemia prolongs PD in 95 patients with documented CAD and Özmen et al. confirmed this feature in patients with angioplasty induced myocardial ischemia [23,24]. PD has also been found to be associated with carotid atherosclerosis [8]. In addition, it has been shown that P-wave dispersion is increased in coronary slow- flow phenomenon [25].

Ischemia- induced inhomogeneous and discontinuous atrial conduction may be related to increased P maximum and PD [26]. Reduced blood flow due to coronary atherosclerosis may contribute to the development of tissue injury and fibrosis [27]. Another explanation for this is that ischemia causes renin angiotensin system activation [28, 29]. The regional fibrosis in the atrial wall, due to chronic ischemia could cause different atrial conductions leading to increased PD in surface ECGs.

Another pathophysiological explanation for increased P-wave duration and dispersion in CAD may be autonomic tone associated with CAD. Tükek et al.[30] reported that the autonomic tone changes may prolong PD. Increased serum catecholamine levels may cause atrial fibrosis and heterogeneus conduction properties.

It was reported that PD was significantly associated with LV diastolic dysfunction [31]. Ischemic left ventriculer dysfunction may increase left atrial pressure, and might another fundamental causes of increased P wave duration and PD in patients with CAD compared to control subjects [32]. Atrial strain, which is a sugnificant factor in the pathophysiology of AF together with ischemia- induced hetergeneous atrial conduction, may results an increase in P wave duration and PD. Yilmaz et al. [9] found no significant association between P min and coronary artery disease severity. Similarly, in our study there was no significant association between P min and Gensini and vessel scores.

There were some limitations in our study. The major limitation of our study is the small number of patients included in the study. For evaluation of ECG results we did not use the high-resolution computer software program. Previous studies have found a low error of the measurement of PD on paper printed ECGs, contrarily other studies reported that manual PD measurement on paper printed ECGs obtained at a standard signal size may effect the accuracy and reproducibility of the results [33,34].

In conclusion, our results suggest that there is a considerable association between increased PD and the severity of CAD.

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