

# A Rare Case of Second Degree Mobitz Type II AV Block Associated with Cocaine Use

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**Abstract** Cocaine is a commonly abused illicit drug in the United States. The complex effects of cocaine on the conduction system of the human heart has not been completely understood. Cocaine acts as a sympathomimetic by inhibition of reuptake of neuronal catecholamines, leading mostly to tachyarrhythmias on presentation. However, cocaine also exerts other effects on the conduction system including sympathomimetic, sino-bradycardic as well as local anesthetic properties. While Multiple cases of atrioventricular (AV) conduction blocks including first degree AV block, Mobitz type I and third degree AV blocks have been previously reported, we hereby present the first case report of cocaine- induced Mobitz type II second degree AV block. This case occurred in a 55 year old woman who presented with retrosternal chest pressure and tested positive for cocaine abuse as documented by urine toxicology test. Patient spontaneously converted to normal sinus rhythm the following day post admission to the hospital. Cocaine is known to inhibit sodium channels and thus has been known to decrease SA node automaticity and conduction via AV node. Electrophysiology studies have previously confirmed cocaine mediated delay in impulse conduction and repolarization. Though rare, physicians should be aware of the possibility of bradyarrhythmias associated with cocaine abuse in order to apply standard therapy such as pacemaker in the event of non-resolution of this serious arrhythmia.

**Keywords:** cocaine, second degree mobitz type II AV block

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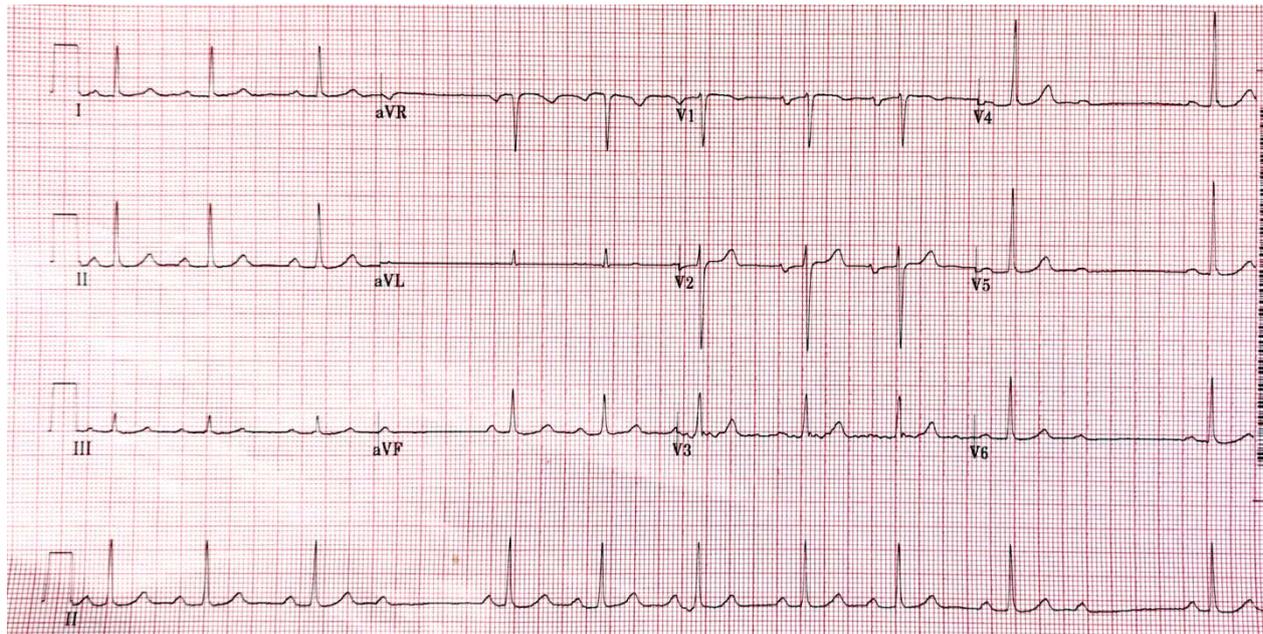
## 1. Introduction

Cocaine is a widely used drug of abuse [1]. Multiple cardiovascular complications such as myocardial infarction and arterial dissection have been reported with cocaine abuse [2,3,4]. Net effect of cocaine use is related to its sympathomimetic property, sino-bradycardic effect and local anesthetic property [2,5,6]. Rare isolated case reports of AV conduction blocks associated with cocaine use have been reported. To the best of our knowledge, we hereby present the first case of Mobitz type II second degree AV block associated with cocaine use.

## 2. Case Presentation

A 55 year old female with a past medical history of asthma, schizoaffective disorder, presented with a chief complaint of retrosternal chest pain after cocaine use. The

patient described the pain as 6/10 in intensity, non-radiating, not related to breathing or changes in position. Pain was no reproducible on chest palpation. The last cocaine use was 10 hours ago. On presentation patient was afebrile, bradycardic at 54 beats per minute, respiratory rate was 16 per minute and blood pressure was 123/58 mm of Hg. Physical examination was benign. The patient was not on any AV nodal blocking agents. Investigations including complete blood count, comprehensive metabolic panel, urine toxicology, thyroid function tests, troponin, brain natriuretic peptide, chest X-ray (CXR) and electrocardiograph (EKG) were obtained. EKG revealed mobitz type II heart block (Image 1). Urine toxicology was positive for cocaine. Thyroid function tests were within normal limits. Troponin levels were also within normal limit. CXR was without any acute abnormalities. Acute coronary syndrome was ruled out. Transthoracic echocardiogram showed normal ejection fraction with no wall motion abnormalities. An outpatient stress test was planned. Cardiac monitoring subsequently revealed normal sinus rhythm on the second day of hospitalization.



**Image 1.** EKG showing Mobitz type II AV block (fixed PR interval with drop in beats) associated with cocaine use in the patient

### 3. Discussion

About 1.5 million people reported cocaine use in the past month in 2014. Adolescents in the age group of 18 to 25 years of age reported a prevalence of use of 1.4% [1]. Myocardial infarction, arrhythmias, aortic dissection and cardiomyopathy are among the cardiovascular complications reported with cocaine use [2,3,4]. Cocaine is a neuronal norepinephrine and dopamine reuptake inhibitor and thus acts as a sympathomimetic agent [2]. Net effect of cocaine abuse is dependent on sympathomimetic action [2], sino-bradycardic effect [5] and its direct anesthetic properties [6]. Multiple studies have reported increase in mean heart rate in cocaine users [7,8,9]. Studies have reported bradyarrhythmia in patients who presented with chest pain after acute cocaine use [10]. A study on asymptomatic chronic cocaine users reported sinus bradycardia in 21% of the cases [11]. First degree AV block [12,13,14,15,16], mobitz type I second degree AV block [17,18], and third degree AV block [19,20,21] has been reported with cocaine use. Cocaine in healthy hearts has been shown to reduce the ventricular effective refractory period however with no change in the intraventricular conduction or spontaneous or induced ventricular arrhythmias [22]. Cocaine is also known to cause increase in intra-atrial conduction time, atrioventricular conduction time and atrial effective refractory period [23]. Electrophysiology studies have demonstrated cocaine induced prolongation of atrium to the His bundle (AH) as well as His bundle to ventricular (HV) duration [24]. Decrease in sinus node automaticity and AV nodal blockade has been reported with cocaine use [25]. Despite the experimental evidence that cocaine causes AH and HV conduction delays, limited cases have reported these findings [12-21]. Marked increase effective refractory period in cocaine users is either due to premature stimulation or minimum pacing intervals [25].

The mechanism of cocaine induced conduction abnormality is not entirely understood. Literature review reveals contradictory results of the effect of cocaine on

AV conduction as measure by PR interval. Studies have reported cocaine related increase [26], decrease [27] and no effect [28,29] on PR interval have been reported. Cocaine, when inhaled can cause activation of parasympathetic fibers via nasal vagal neuronal terminals [19], however this mechanism may not provide an explanation to AH and HV conduction delay when cocaine is abused via intravenous routes. The conduction abnormalities associated with cocaine are largely explained by the inhibitory action of cocaine on cardiac sodium channels (INa). Cocaine acts similar to Class I antiarrhythmic drugs which act by inhibiting INa channels. Such inhibition results in prolongation of HV interval and thus QRS duration [24,25]. The increase in action potential duration and thus QT prolongation is secondary to cocaine mediated inhibition of potassium channels (IKr). Depressed repolarization secondary to inhibition of IKr has been noted in SA node and right atrium [25]. Bradycardia associated with Mobitz type II in our patient may be explained by delayed HV conduction secondary to inhibition of INa channels.

### 4. Conclusion

Although Cocaine abuse is often associated with tachyarrhythmias, accumulating evidence through multiple case reports indicate that bradyarrhythmias due to cocaine use is not infrequent. Our case report underscores the possibility of AV conduction blocks associated with cocaine use that is likely due to the net effect of cocaine on conduction system of the heart that include sympathomimetic action and inhibitory action on INa and IKr currents.

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