Fig. 2 : Second rhythm strip of ECG (initial 10 beats)

dissociation with interference-Isorhythmic arrhythmia.

AV dissociation occurs when atria and ventricle are under control of separate pacemakers[1]. The atria is under control of either sinus node, ectopic atrial or AV junctional rhythm. Incomplete AV dissociation is indicated when a single pacemaker establishes control of both atria and ventricle for either one beat (capture) or series of beats (sinus rhythm, AV junctional rhythm with retrograde atrial capture [2]. Our ECG reveals both the mechanism.

AV dissociation can occur by slowing of dominant pacemaker, acceleration of latent pacemaker, complete AV blocks and combination of above[2]. In our case it was due to combination of slowing of dominant pacemaker (second strip) and acceleration of latent

pacemaker (first strip). AV dissociation can develop with an AV junctional rhythm in response to severe sinus bradycardia; when the sinus rate and the escape rate are similar it is called isorhythmic arrhythmia[1]. It can occur in response to beta-blocker[1] which is probable cause in our case who was given tab atenolol.

REFERENCES

1. Josephson ME, Buxton RE, Marchlins FE. The tachyarrhythmias. In Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL eds. Harrison's Principle of Internal Medicine. Vol 1. 13th edition. New York : McGraw Hill Inc. 1994; 1019-1036.
2. Zipes PD. Specific arrhythmia : Diagnosis and Treatment In ed Braunwald E,ed. Braunwald heart disease. Vol-1, 5th edition. India Bangalore : Prism Books Pvt Ltd, 1997;640-704.

Maj MK GARG^{*}, Lt Col JS DUGAL⁺, Col AP SINGH[#].

^{*} Classified Specialist (Medicine) & Endocrinologist, ⁺ Classified Specialist (Medicine) & Cardiologist, [#] Senior Adviser (Medicine & Neurology), Department of Medicine, Command Hospital (Southern Command), Pune-411040.

ATRIAL FIBRILLATION IN THYROTOXICOSIS

Dear Editor,

DPR, 45-year-old male, presented to peripheral hospital with signs and symptoms of thyrotoxicosis of one-month duration on 3rd March '97. He was not on any medication. His pulse was 140/minute irregularly irregular. Electrocardiogram confirmed presence of atrial fibrillation. there was no cardiomegaly on X-ray. There was no facility for thyroid function test, hence he was put on tab Neomercazole 10 mg tid and tab propranolol 40 mg tid on clinical ground. AF reverted to sinus rhythm on 7th day. He was referred to this centre for further evaluation on 27 March '97. His symptoms had improved but he was still clinically toxic and had sinus rhythm. His drugs were stopped to confirm diagnosis and evaluate for hyperthyroid state. While awaiting report of thyroid function test he developed palpitation and clinical examination and ECG revealed recurrence of AF. He was sent for thyroid scan, which showed diffuse thyromegaly with increased thyroid uptake-10%(normal 1-4%). His thyroid function confirmed thyrotoxicosis (Table 1). He was placed on tab Neomercazole 10-mg tid and tab propranolol 40-mg tid. Sinus rhythm was documented on third day. he was followed up for three months. He is presently euthyroid and maintained sinus rhythm.

TABLE 1
Thyroid hormone profile

Hormone	April 97	May 97	June 97	July 97
T3 (0.8 - 2.1 ng/ml)	2.95	2.61	2.56	2.19
T4 (4.5 - 13.5 ug/dl)	17.77	14.97	14.39	11.66
TSH (0.5 - 6.5 mIU/L)	0.13	0.12	0.18	0.32

Hyperthyroidism is major cause of paroxysmal AF. It is reported in about 9-22% patients of hyperthyroidism, occult or manifest [1]. The AF in thyrotoxicosis reverts spontaneously to sinus rhythm with

control of hyperthyroidism in 75% of patients within 13 weeks. The shortest duration of AF is reported for one-week [2]. Antithyroid drugs usually takes 2-6 weeks for achieving euthyroid status depending upon preformed and stored hormone level, and iodine content of the thyroid gland [3]. In present case AF reverted to sinus rhythm within 7 and 2 days after starting antithyroid drugs and beta-blocker. Euthyroid status was achieved after 12 weeks (Table-1), hence antithyroid drugs are unlikely to contribute to early reversion of AF to sinus rhythm. Beta-blockers are drug of choice for thyrotoxicosis induced arrhythmias. These drugs by reducing hyperadrenergic drive contribute largely to reversion of AF. The longer AF continues, more difficult it becomes to treat as it is said that AF begets AF [2]. It is therefore advised to treat AF early with beta-blockers in suspected cases of hyperthyroidism pending hormonal evaluation. Beta-blocker has effect on atrial action potential, increases its refractory period and acts as membrane stabilising agent, therefore is an agent of choice in atrial arrhythmias in hyperthyroidism.

REFERENCES

1. Nakazawa HK, Sakurari K, Hamada N, Nomotani N, Ito K. Management of atrial fibrillation in the post thyrotoxic state. Am J Med 1982;72:903-6.
2. Ciaccheri M, Cecchi F, Arcangeli C, Dolara A, Zuppirolli A, Pieroni C. Occult thyrotoxicosis in patients with chronic and paroxysmal isolated atrial fibrillation. Clin Cardiol 1984;7:413-6.
3. Larsen PR, Ingbar SH. the thyroid gland. In Williams Textbook of Endocrinology Editors: Wilson JD and Foster DW, 8th Edition, WB Saunders Company. p 429-36,1992.

Maj MK GARG^{*}, Maj KM HASSAN⁺, Lt Col JS DUGAL[#], Brig JS SAINI^{*}.

^{*} Classified specialist (Medicine) & Endocrinologist, ⁺ Trainee Medicine, [#] Classified Specialist (Medicine) & Cardiologist, Consultant (Medicine & Endocrinology), Department of Medicine, Command Hospital (SC), Pune-40