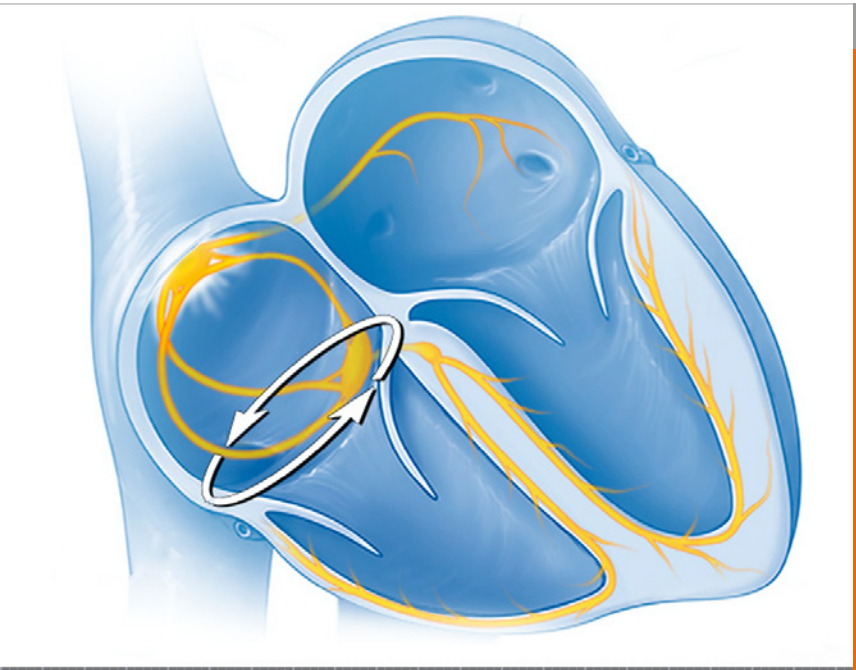
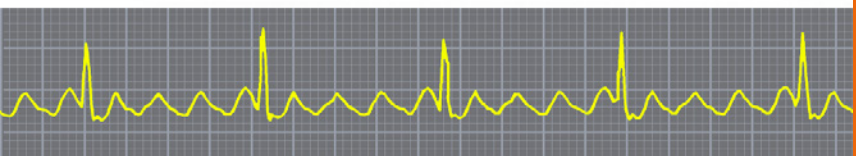
SWISS SOCIETY OF NEONATOLOGY

A rare cause of a racing heart





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Title figure:
Atrial flutter (adapted from www.enhum.com).

INTRODUCTION

Fetal arrhythmias are detected during second or third trimester routine obstetric ultrasound examinations and occur in up to 1–3% of all pregnancies (1,2). Most fetal arrhythmias are benign and transient; however, some of them, such as sustained tachycardia with more than 200 beats per minute (bpm), may result in low cardiac output, fetal hydrops or even fetal demise. Supraventricular tachycardia (SVT) is the most common type of tachycardia and a frequent cause of non-immune hydrops (3). As the neonate can achieve sinus rates upwards of 230 bpm, the ability to differentiate normal from abnormal rhythms via electrocardiogram can sometimes be challenging without further provocative maneuvers.

This case report describes a female infant born at 37 0/7 weeks of gestation to a healthy 31-year-old P1/G1 in a peripheral hospital. Birth weight was 3320 g (P50 – 90), length 47 cm (P10 – 50) and head circumference of 32.3 cm (P10 – 50). On routine cardiotocography (CTG), the fetus was noted to be very tachycardic, and fetal ultrasound revealed markedly impaired ventricular contractions. A decision was made to deliver the baby by emergency Cesarean section. Pregnancy had otherwise been unremarkable with no history of any infectious diseases and no anomalies on prenatal ultrasounds.

Apgar scores were 6, 9 and 10 at 1, 5 and 10 minutes, respectively and arterial cord pH value was 7.26. The neonate was clinically stable at birth, with a good transition to extra-uterine life, but was still noted to be tachycardic. The heart sounds were clear, and no murmur was heard. When no reliable pulse oximetry signal could be obtained, ECG electrodes were placed and narrow complex tachycardia with a heart rate of 280 bpm was noted. Due to suspected supraventricular tachycardia (SVT), intravenous adenosine with an initial dose of 0.1 mg/kg was administered via an umbilical venous catheter, after vagal maneuvers had been unsuccessful. Multiple trials with escalating doses of adenosine (max. 0.3 mg/kg) were unsuccessful in maintaining a stable sinus rhythm (video – part 1). It was noted that p-waves remained visible during brief interruption of AV conduction (suggestive of atrial

flutter), but no definitive diagnosis was made. Therefore, the infant was transferred to a tertiary pediatric hospital.

On arrival, the baby had a heart rate of 280 – 300 bpm and was still hemodynamically stable without clinical signs of congestive heart failure. Chest X-ray was unremarkable with the tip of the umbilical venous catheter clearly lying outside the cardiac silhouette. A twelve-lead electrocardiogram was performed, which demonstrated supraventricular tachycardia, suggestive of atrial flutter (Fig. 1). The diagnosis was confirmed after adenosine administration, when the typical "sawtooth" pattern, characteristic of atrial flutter, was observed during AV Block (Fig. 2, video – part 2).

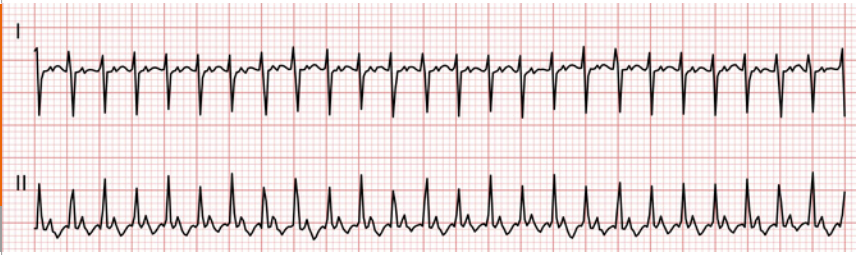

Fig. 1

An electrocardiogram showing atrial flutter with 2:1 atrioventricular conduction; ventricular rate is approximately 302 bpm.

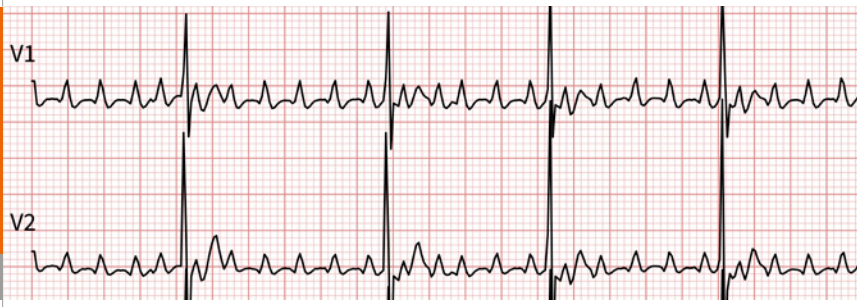

Fig. 2

ECG: atrial flutter was diagnosed after administration of adenosine: „sawtooth atrial flutter waves“ can easily be recognized.

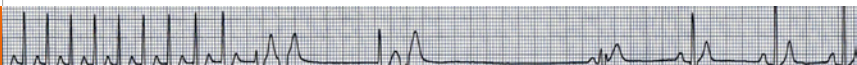

Fig. 3

ECG: SVT conversion with adenosine in a different patient with AV re-entry tachycardia.

After premedication with (S)-ketamine, electric synchronized cardioversion was performed with 1 joule/kg, which achieved conversion to a stable sinus rhythm (Fig. 4, video – part 3). Echocardiography performed on the day after cardioversion demonstrated a normal ventricular function and a moderate-sized (6 × 6 mm) atrial septal defect. No further drugs were given, atrial flutter did not reoccur, and the infant was discharged home in a good clinical condition on day 3 of life.

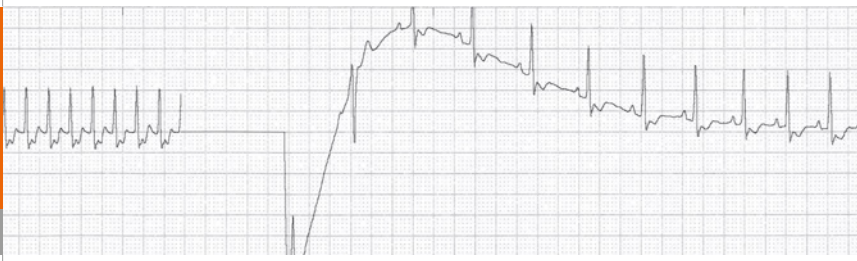

Fig. 4

ECG: synchronized direct current cardioversion with return to normal sinus rhythm.

DISCUSSION

When evaluating any infant with an arrhythmia, it is essential to simultaneously asses electrophysiology and hemodynamic status. The three broad categories for arrythmias in neonates are tachyarrhythmias, bradyarrhythmias and irregular rhythms. An algorithm for approaching the differential diagnosis of tachyarrhythmias can be consulted in most cases (Fig. 5).

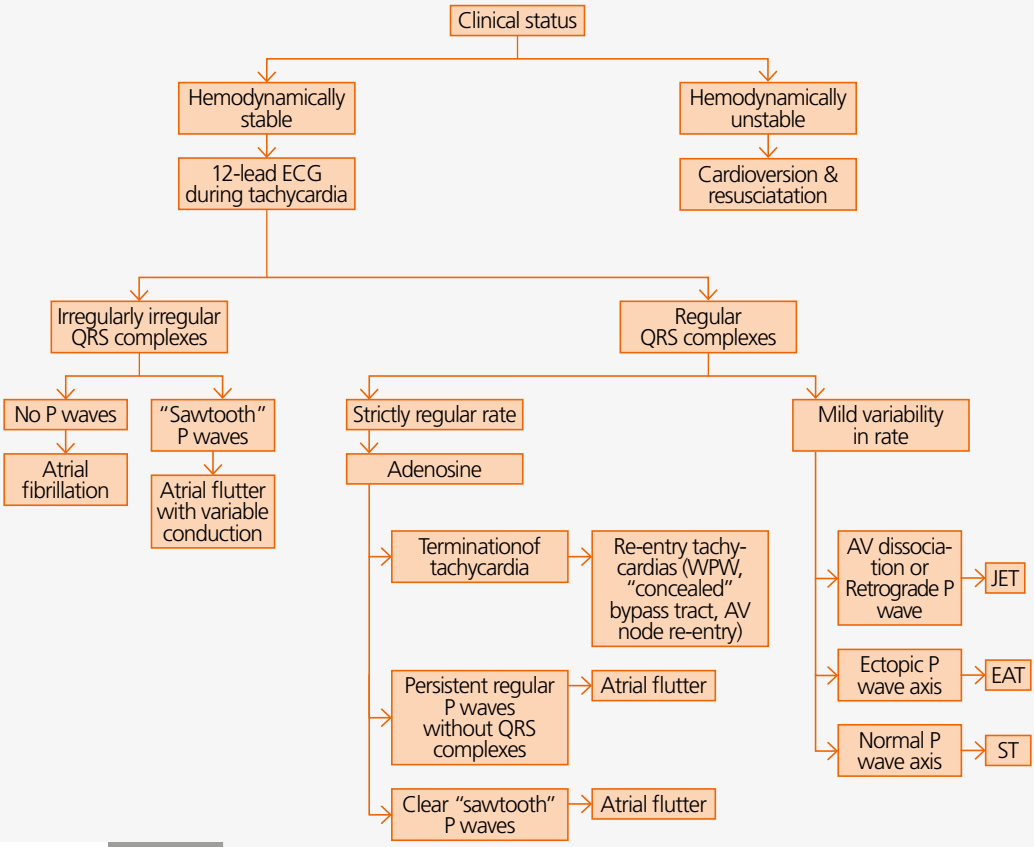

Fig. 5

Algorithm for bedside differential diagnosis of narrow complex tachycardias.

Abbreviations: ECG: electrocardiogram, AV: atrioventricular, JET: junctional ectopic tachycardia, EAT: ectopic atrial tachycardia, ST: Sinus tachycardia, WPW: Wolff-Parkinson-White syndrome.

(adapted from Cloherty and Stark's Manual of Neonatal Care, 8th Edition, Chapter 41)

Atrial flutter is a relatively uncommon form of SVT in the neonatal period (2, 4, 5). The term flutter first appeared a century ago in 1887, when Mc William described the visual phenomenon resulting from 'faradic stimulation of the auricles which sets them into a rapid flutter'. Since its first description, our understanding of atrial flutter has evolved from a unique electrocardiographic pattern corresponding to right atrial macro-reentry, to a variety of atrial tachycardias originating from the right atrium as well as the left atrium (7).

During childhood, atrial flutter is mostly seen secondary to cardiac surgical procedures. In contrast, if atrial flutter manifests during the perinatal period, it is usually not associated with congenital heart disease. Important factors for the clinical course of atrial flutter depend upon the time of onset, duration of symptoms and the ventricular response rate (8, 9). The ventricular response rate is variable, but 2:1 and 3:1 AV blocks are common. If the ventricular rate exceeds 250 bpm, congestive heart failure or – if atrial flutter manifests during the fetal period – fetal hydrops with severe hemodynamic compromise may occur (4).

In utero, SVT may be suspected when a very rapid heart rate is noted during prenatal care. Prenatally, echocardiography can be used to assess the fetal cardiac rhythm. Atrial flutter may be diagnosed by simultaneous atrial and ventricular M-mode tracing,

showing a more rapid rate of atrial contractions than ventricular contractions (1, 8, 9).

For treatment in utero, antiarrhythmic medication is recommended for all sustained SVTs, with the goal of conversion to sinus rhythm or reduction of the ventricular rate to tolerable levels to prevent or even reverse fetal hydrops (6, 8, 9). Failure to control fetal SVT in the presence of fetal hydrops is an indication for delivery.

Postnatally, the diagnosis of atrial flutter is made by surface electrocardiogram showing the typical "sawtooth" or "picket fence" pattern, best seen in leads II, III and aVF. This may be difficult to identify in the presence of 2:1 AV block (Fig. 1). A definitive diagnosis can be made by inducing AV block transiently, for example by administering adenosine (Fig. 2). However, conversion to sinus rhythm will only occur in case of SVT due to re-entry tachycardia (Fig. 3). Although adenosine is sometimes helpful in establishing the diagnosis, it does not convert atrial flutter to sinus rhythm because the reentrant circuit does not involve the AV node. In otherwise healthy neonates, atrial flutter can be converted to sinus rhythm by use of direct synchronized cardioversion or overdrive pacing (1, 7). If this treatment is unsuccessful, ventricular rate control with medication (digoxin) or with temporary atrial pacing to create a 2:1 atrioventricular block must be achieved (9).

Newborn infants with atrial flutter and structurally normal hearts generally have an excellent prognosis once in sinus rhythm with a low risk of recurrence, and long-term antiarrhythmic therapy is rarely necessary (8 – 10). Nevertheless, regular follow-up of these patients is recommended to prevent possible complications.

CONCLUSIONS

In general, atrial flutter in infants is a well-tolerated arrhythmia, despite the often high atrial and ventricular rates. Nevertheless, cardiac compromise with development of congestive heart failure can occur. Echocardiography should always be performed to rule out congenital heart disease, ventricular dysfunction or intracardiac clots (12). Atrial flutter may be difficult to distinguish from other forms of SVT, unless the atrioventricular block is > 2:1. Administration of adenosine may increase the block and unmask an obvious sawtooth wave pattern characteristic of atrial flutter. Once atrial flutter in the newborn period is converted to sinus rhythm, recurrence is uncommon, and infants have an excellent prognosis.

Acknowledgment

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