

# Fundamentals of Atrial flutter

**Concept:** Infrequent atrial tachyarrhythmia, nearly always present, accompanied by organic substrate, the electrophysiological mechanism of which in most cases, is a circular circuit of macro-reentry that encompasses all the RA; more rarely by unifocal or multifocal atrial focus with very high shock, or exceptionally by focal micro-reentry in the RA. Atrial flutter is a form of supraventricular tachycardia caused by a re-entry circuit within the right atrium. The length of the re-entry circuit corresponds to the size of the right atrium, resulting in a fairly predictable atrial rate of around 300 bpm (range 200-400). Ventricular rate is determined by the AV conduction ratio (“degree of AV block”). The most common AV ratio is 2:1, resulting in a ventricular rate of ~150 bpm. Higher-degree blocks can occur — usually due to medications or underlying heart disease — resulting in lower rates of ventricular conduction, e.g. 3:1 or 4:1 block. Atrial flutter with 1:1 conduction can occur due to sympathetic stimulation, or in the presence of an accessory pathway.

The administration of AV-nodal blocking agents to a patient with WPW can precipitate this. Atrial flutter with 1:1 conduction is associated with severe hemodynamic instability and progression to ventricular fibrillation.

NB. The term “AV block” in the context of atrial flutter is something of a misnomer. AV block is a physiological response to rapid atrial rates and implies a normally functioning AV node.

There are always intraatrial or interatrial dromotropic disturbances, with a minimal extension being necessary in the circular movement, refractoriness dispersion and variations in autonomic tone.

**ECG features of atrial flutter:** Narrow complex tachycardia, regular atrial activity at  $\approx 300$  bpm, “Saw-tooth” pattern of inverted flutter waves in leads II, III, aVF, upright flutter waves in V1 that may resemble P waves, loss of the isoelectric baseline and ventricular rate depends on AV conduction ratio:

- II) **Fixed AV conduction ratio** (“AV block”); Ventricular rate is a fraction of the atrial rate, e.g. 2:1 block = 150 bpm; 3:1 block = 100 bpm; 4:1 block = 75 bpm
- III) **Variable AV conduction ratio**: The ventricular response is irregular and may mimic AF. On closer inspection, there may be a pattern of alternating 2:1, 3:1 and 4:1 conduction ratios

ECG is characterized by the typical atrial “F” waves with aspect in “sawtooth” or “picket fence”, frequently better observed in II, III, aVF and V<sub>1</sub>, with average atrial HR of 250-350 bpm (HR of atypical flutter or type II is 350 to 450 bpm), characteristic absence of isoelectric line between F waves, variable degrees of AV block or rarely 1:1 conduction.

Ventricular HR is usually half of atrial HR (i.e. 150 beats/min). A significantly slower ventricular rate in absence of drugs, suggests normal AV conduction.

### **Etiology of atrial flutter**

- Prevalence: infrequent and less prevalent than AF: 10 to 1 in favor of the latter. Estimated in 0.0006% to 0.004% between 50,000 patients from a General Hospital.
- Gender: greater prevalence in males.
- Age: 25 to 35 y/o.: 2-3/1000; 55 to 64: 30-90/1000; 62 to 90: 50-90/1000
- Atrial flutter nearly always presents underlying organic heart disease. Exceptionally described in patients without heart disease, associated to preexcitation of the Wolff-Parkinson-White type.

### **Clinical causes of atrial flutter**

- Coronary artery disease: a) chronic b) AMI: 03% and 5.3% of cases.
- Hypertensive heart disease. 60% of flutters have heart failure or hypertensive heart disease as their cause.
- Chronic obstructive pulmonary disease (COPD).

- Rheumatic mitral and/or tricuspid valve disease.
- Severe aortic stenosis.
- Bronchopneumonia.
- Acute pulmonary embolism: transient.
- Bronchogenic carcinoma.
- Thyrotoxicosis: transient. Frequent rate of ventricular response 1:1. Ventricular HR of 300 bpm.
- Congenital heart diseases: E.g.: ASD not operated > 40 years old.
- Post-operative period of cardiac surgery. E.g.: Mustard surgery to correct TGA.
- Digitalis intoxication (exceptional)
- As part of the tachy-brady syndrome.
- Myocarditis and pericarditis.
- Patients that underwent surgical correction of congenital heart diseases.
- As part of the WPW syndrome (frequent 1:1 AV conduction and possible sudden cardiac death)
- Mitral valve prolapse syndrome.
- Alcoholism. 30% of atrial flutters do not have underlying structural heart disease.

## **Electrophysiological mechanisms of atrial flutter**

### **A. Dromotropic mechanisms**

Macro-reentry around fixed or functional, anatomical or surgical barriers.  
Micro-reentry.

## B. Automatic mechanisms

Unifocal atrial focus.

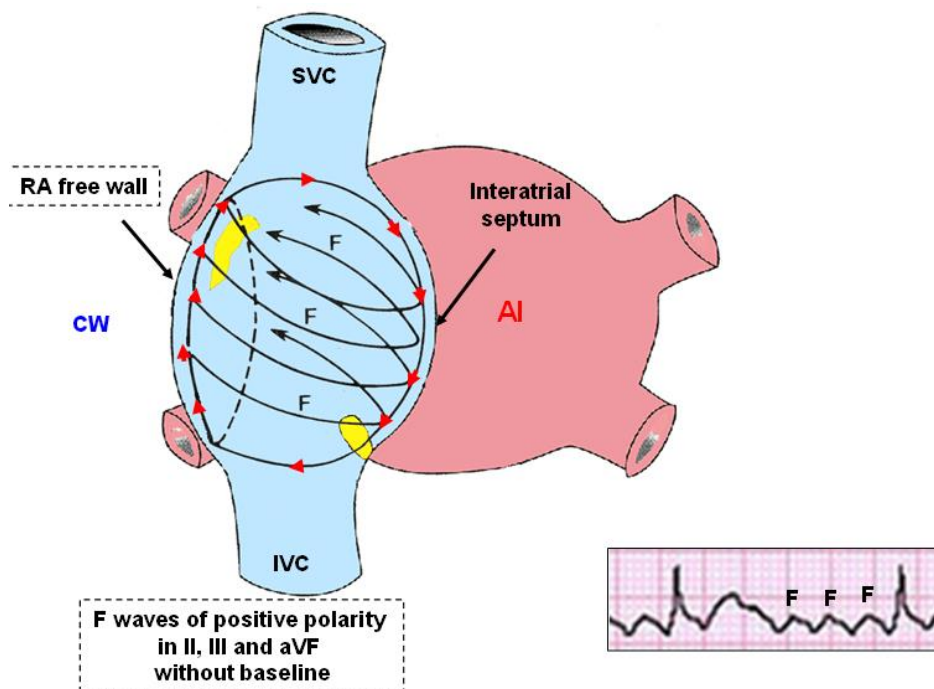
Multifocal atrial flutter with very high shock.

## Classification of atrial flutter

From the right atrium

- I. Isthmus dependent. HR between 240 to 339 bpm. Subtypes: with CCW and CW rotation.
  - Type I atrial flutter Common, with *Clockwise Reentry* CW rotation: In this case, F waves are positive in II, III and aVF: This uncommon variant produces the *opposite* pattern: Positive flutter waves in leads II, III, aVF, Broad, inverted flutter waves in V1 Figure

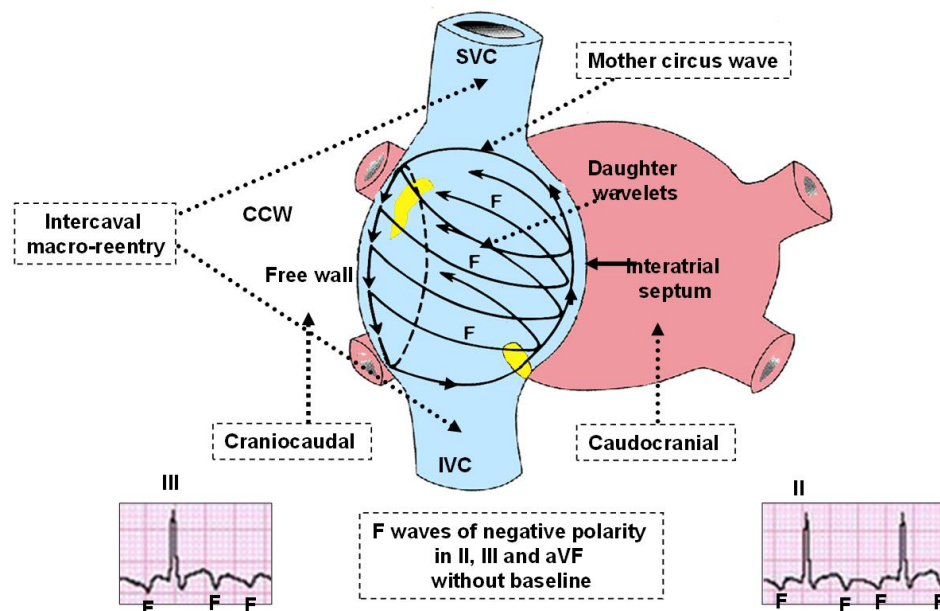
### Dromotropic mechanisms by macro-reentry in atrial flutter



**Figure.** Type I clockwise flutter: intercaval macro-reentry, with CW motion descending through the septum, going through the cavo-tricuspid isthmus and ascending through the RA free wall.

- Type I atrial flutter with CCW rotation **Anticlockwise Reentry**: Commonest form of atrial flutter (90% of cases). Retrograde atrial conduction produces: Inverted flutter waves in leads II, III, aVF Positive flutter waves in V1 – may resemble upright P waves. intercaval macro-reentry, CCW circular movement descending through the RA free wall, going through the cavo-tricuspid isthmus and ascending by the interatrial septum: mother circus wave. In both subtypes catheter ablation of the isthmus is the right procedure for treatment. The removal of conduction through the isthmus prevents the perpetuation of the circular motion, preventing atrial flutter recurrence.

### Dromotropic mechanisms by macro-reentry in atrial flutter



**Figure.** Type I counterclockwise, typical, common or classical flutter: intercaval macro-reentry: CCW circular motion descending by the RA free wall going through the cavo-tricuspid isthmus and ascending by the interatrial septum: mother circus wave.

- Double wave reentry.
- Lower loop reentry with single or multiple pauses.

### Atypical atrial flutter (Uncommon, or Type II Atrial Flutter)

Type II atrial flutter follows a reentry pathway significantly different from type I, and is typically faster, generally with heart rates between 340 to 440 bpm. Type II left atrial flutter is common after ablation procedures of the left atrium. Type II atrial flutter

is a particular entity that should be separated from other forms. Atrial flutter is high and may exceed 350 beats per minute.

Pacing techniques are not capable of removing the arrhythmia, which does not fit in an atrial macro-reentrant process. Using atrial cartography supports the role of the circular motion coexisting with atrial fibrillation areas. This atrial flutter variant is probably representing a transient moment in the transformation into sustained atrial fibrillation.

- Does not fulfill criteria for typical atrial flutter
- Often associated with higher atrial rates and rhythm instability
- Less amenable to treatment with ablation

Non-isthmus dependent

II. Upper loop reentry.

II.Scar reentry.

Critical flutter circuits.

- II) Surgical circuits: isthmus-dependent incisional scar and complex circuits.
- III) Left atrial circuits: they could be of the mitral annulus, related to scar and left membranous circuit.

From the left atrium (Jais, Shah et al. 2000)

Scar-dependent atrial flutter is a variant associated to history of congenital heart disease that has been subjected to repair surgery (mainly ASD, but also Fontan or Senning procedures for complex congenital diseases). The post-incisional scar provides a substrate for the subsequent development of reentry mechanism (Triedman, Saul et al. 1995). Right atrium dilatation may favor atrial flutter maintenance. Endocardial mapping in humans has confirmed the possibility of circular movement around atrial scars. However, common atrial flutter may also occur in this scenario. Careful electrophysiology studies are necessary to differentiate true scar-dependent flutter from common isthmus-dependent forms.

## Clinical aspects of atrial flutter

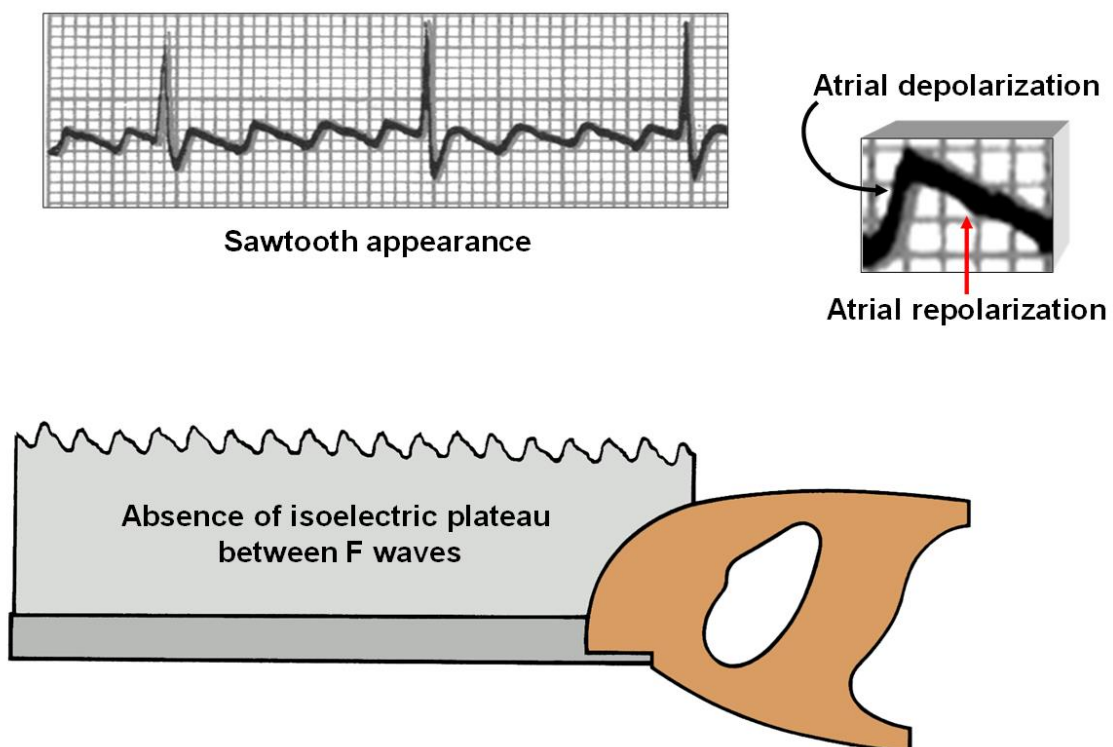
History: Palpitations, fatigue, poor tolerance to exercise, dyspnea, angina, dizziness and syncope.

Physical examination: heart rate approximately 150 bpm in most cases, regular pulse or mildly irregular, frequent hypertension, left CHF, syncope.

Jugular pulse: the frequent atrial contractions are greater in relation to those recorded in the arterial pulse and in the tip shock.

## Characteristics of atrial activation in atrial flutter

Waves with sawtooth or picket fence appearance called “F” waves, with rate between 250 and 350 bpm, better observed in the inferior wall and V<sub>1</sub> with slow descending ramp and rapid ascending ramp. These waves seem an inverted P, followed by ascending ramp: Tp waves.



**Figure.** Characteristics of atrial activation in atrial flutter.

## **Characteristics of atrial activation in atrial flutter**

In the ordinary form, F waves polarity is negative in inferior leads II, III and aVF: caudocephalic atrial activation: type I, common or classical flutter.

Lead II or V<sub>1</sub> should be recorded separately, in prolonged tracings (from 15 s to 20 s) to establish the relationship between F waves and QRS complexes.

During atrial flutter with 2:1 conduction, for example F waves could be masked in lead II and be quite visible in V<sub>1</sub>.

## **Causes of F waves of low rate flutter**

- Large mega-atria show atrial rate sometimes lower than 200 bpm.
- Use of group IA drugs: drugs of intermediate kinetics (quinidine, procainamide, disopyramide and ajmaline) reduce V<sub>max</sub> and extend action potential.
- IC: drugs of slow kinetics (propafenone, flecainide, encainide, moricizine and lorcinida).
- Class III drugs: K<sup>+</sup> channel block (action potential prolongation in phase 3) (amiodarone).

## **Causes of F waves of high rate flutter**

- Flutter in children: average HR of 300 bpm with 1:1 response.
- Flutter of ventricular preexcitation.
- Type II flutter of Wells: rate of F between 340 and 433 bpm: cannot be interrupted by pacing.

## **Types of atrioventricular conduction in atrial flutter**

### **A) Regular:**

2:1 – <sup>1</sup>/<sub>2</sub>



1:1 – it suggests ventricular pre-excitation (anomalous accessory pathway). In these cases, regular and wide QRS complexes without apparent atrial activity may lead to the mistaken diagnosis of ventricular tachycardia, with atrial and ventricular rate close to 250 bpm.

3:1 –  $1/3$  – rare and it means 3 F waves per each QRS

4:1 –  $1/4$

6:1 – observed in cases with marked AV block. The differential diagnosis of conduction with a high rate of ventricular response should be done with complete AV block. A constant ventricular rate with FR intervals is present in the first case with fixed FR, contrary to the third degree or complete block with constant RR intervals accompanied by FR interval variations.

## **B) Irregular**

**C) Absent:** with complete AV block – ventricular rate is usually low and independent from atrial rate.

**Commentaries:** in flutter, ventricular rhythm could be regular or irregular, unlike AF where ventricular rhythm is nearly always irregular.

## **Regular atrial flutter with 1:1 AV conduction**



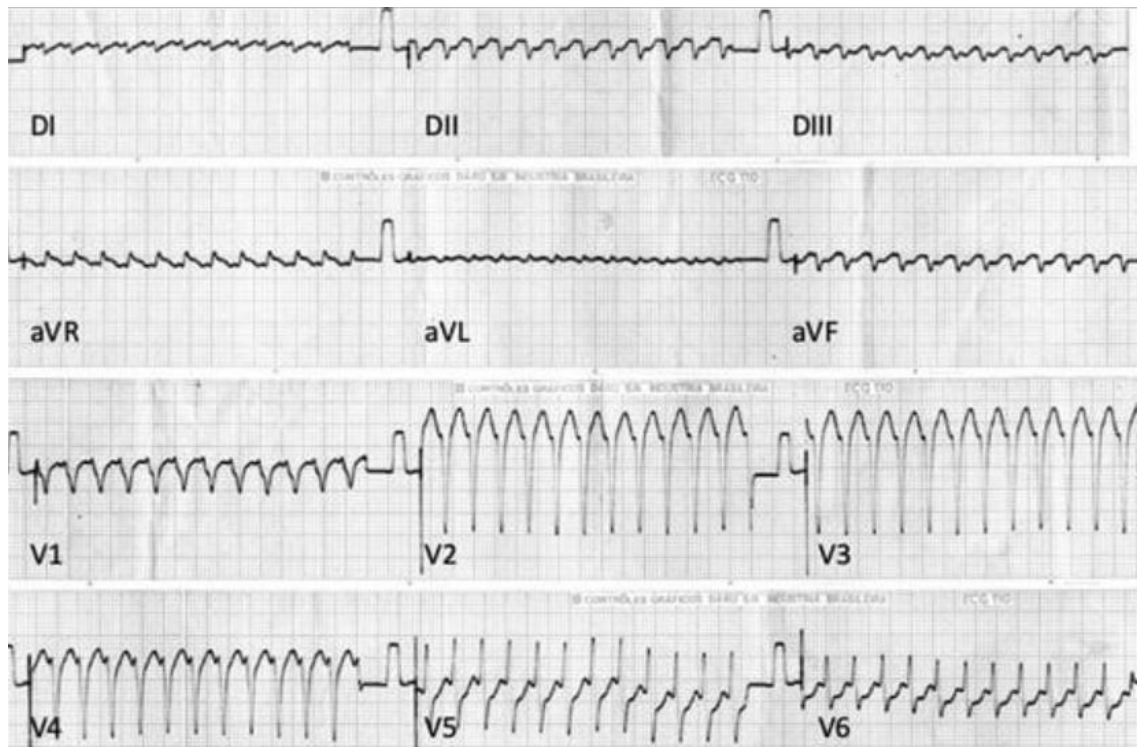
**Figure.** Regular atrial flutter with 1:1 AV conduction.

1:1 AV conduction (rare) is a medical emergency. Ventricular rate close to 300 bpm should be treated immediately. 1:1 AV conduction could be found in the following scenarios:

- A) Preexcitation of the WPW type because the stimulus is conducted in anterograde fashion by the anomalous pathway;
- B) Atrial flutter secondary to hyperthyroidism;
- C) Pediatric group flutter;
- D) Subsequent to the initial use of class IA drugs (quinidine, procainamide or disopyramide) by atrial slowing and by vagolytic anti-cholinergic action in the AV junction that this set of drugs causes, especially if the drugs were used without previously administering digoxin, calcium antagonists or  $\beta$ -blockers with the aim of controlling the rate of ventricular response.

### ***Case Report***

A 47-year-old female patient seeks the emergency room due to worsening of functional class (class IV) for 1.5 hours. She has a history of non-ischemic dilated cardiomyopathy (LVEF 42%) and NYHA functional class II. It comes in therapy with carvedilol, enalapril, furosemide and spironolactone. Physical examination of the service demonstrates: conscious, oriented, acyanotic, BP 98/62mmHg, HR 230bpm, AC regular rhythm 2 beats, AP MV symmetrically distributed and crackling rales in bases. After installing oxygen therapy, venoclysis and monitoring of cardiac and vital signs (SpO<sub>2</sub>, BP, HR), the patient presented clinical stabilization and the ECG shown in the figure was performed.



### **Atrial flutter 1:1 treated with - anticoagulation and electrical cardioversion**

#### ***Discussion***

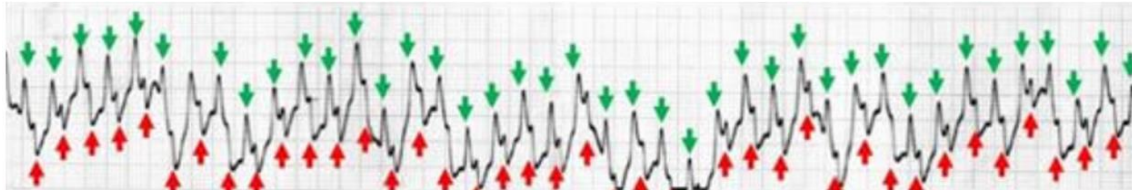
*In consideration of a tachycardia with a regular narrow QRS complex (120ms), differentiation between supraventricular rhythms is necessary. A less frequent option is a high septal ventricular tachycardia with activation of the conduction system, which is a rare form of fascicular tachycardia.*

*After clinical stabilization, it was possible to record the atrial activity using an esophageal electrode that demonstrated a tachyarrhythmia with 1:1 atrioventricular conduction (figure 2). Due to the history of dilated cardiomyopathy and the uncertain history of the onset of tachyarrhythmia, it was decided to control HR with slow administration of IV metoprolol. However, the patient presented shortness of breath and increased pulmonary congestion, requiring IV sedation and synchronized electrical cardioversion. IV heparin anticoagulation was also administered.*

*By analyzing the ECG, the differential diagnosis is difficult as it is a rapid tachycardia and atrial activity is difficult to visualize. The esophageal electrode with P wave recording allowed the demonstration of a tachycardia with a narrow QRS complex and a 1:1 P-QRS ratio, making the diagnosis of ventricular tachycardia unlikely. Very high HR (>200bpm) makes atrioventricular reentry tachycardia less likely. Although AF*

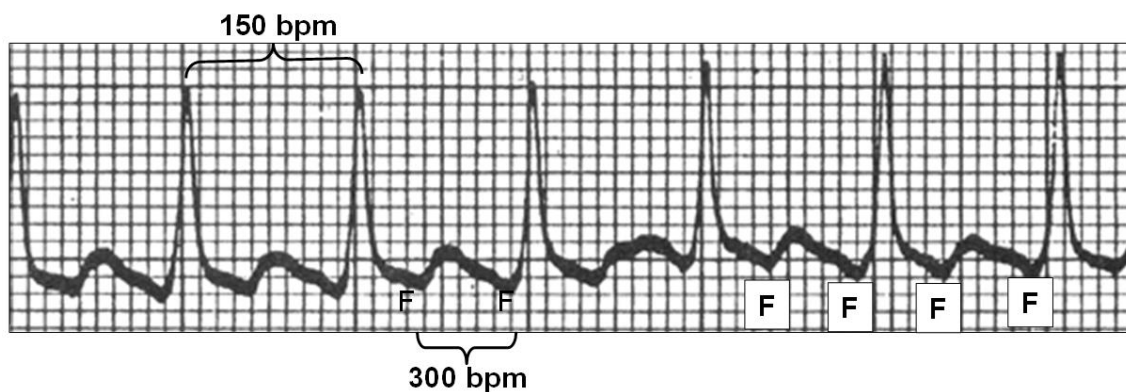
*with very fast HR may present little variation in the RR interval, it is noted on the ECG that tachycardia presents an RR interval without variation, which rules out the hypothesis of atrial fibrillation. Upon careful analysis, a negative F wave can be seen in the inferior wall that deforms the ST segment. The most likely diagnosis is a typical atrial flutter with 1:1 atrioventricular conduction.*

**Figure Esophageal electrode register demonstrates a 1:1 atrioventricular relationship**



**Green arrows record ventricular depolarization Red arrows record ventricular repolarization**

**Regular atrial flutter with 2:1 AV conduction**

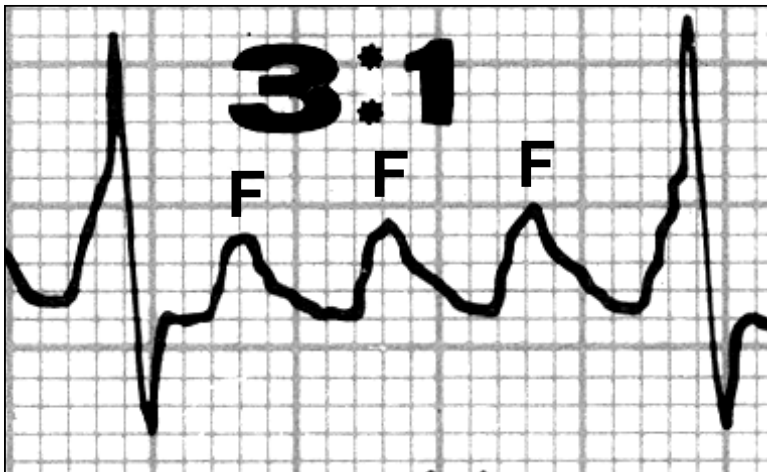


**Figure. ECG.**

The most frequent ratio in untreated patients is 2:1 with atrial and ventricular rate of 300/150 bpm respectively. This ratio is due to physiological interference in the junction. If the ventricular response rate is regular and constant (E.g. always 2:1) the FR interval will also be so, varying between 260 ms to 460 ms.

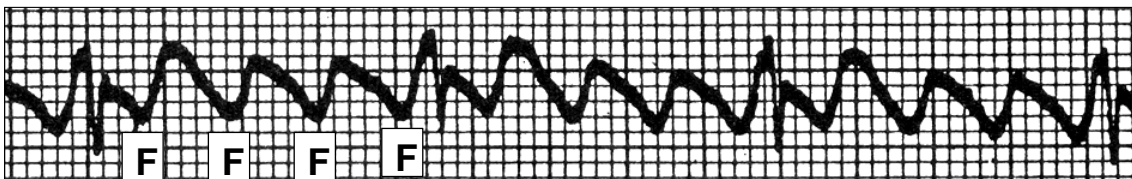
If the rate of F is 240 bpm, in 2:1 flutter, ventricular rate will be 120 ppm in the arterial pulse.

### Regular atrial flutter with 3:1 AV conduction



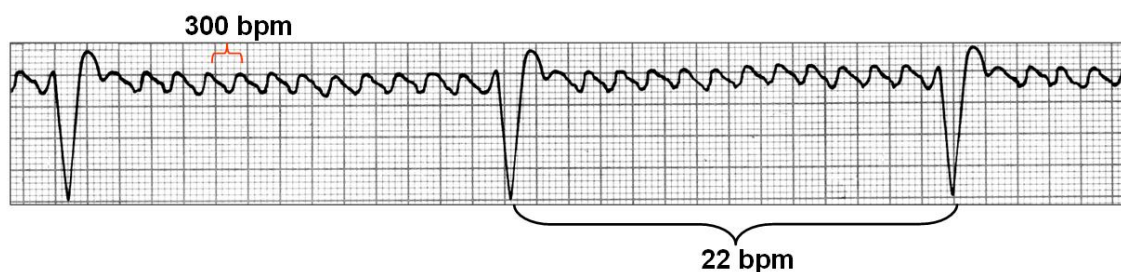
**Figure.** If the rate of F is 240 bpm, in 3:1 flutter, ventricular rate will be 80 ppm in arterial pulse, i.e. within what is considered a normal HR.

### Example of regular atrial flutter with 4:1 AV conduction



**Figure.** If the rate of F is 240 bpm, in 4:1 flutter, ventricular rate will be 60 ppm in arterial pulse; i.e. within what is considered a normal HR.

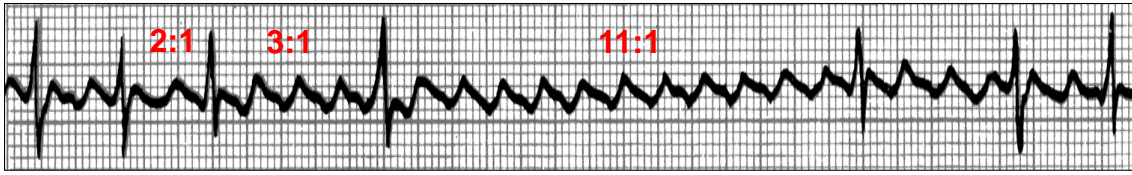
### Example fo Atrial flutter with complete AV block



**Figure.** Atrial heart rate of 300 bpm. The stimulus does not conduct to the ventricles. Very low, regular ventricular heart rate, regardless of atrial activity.

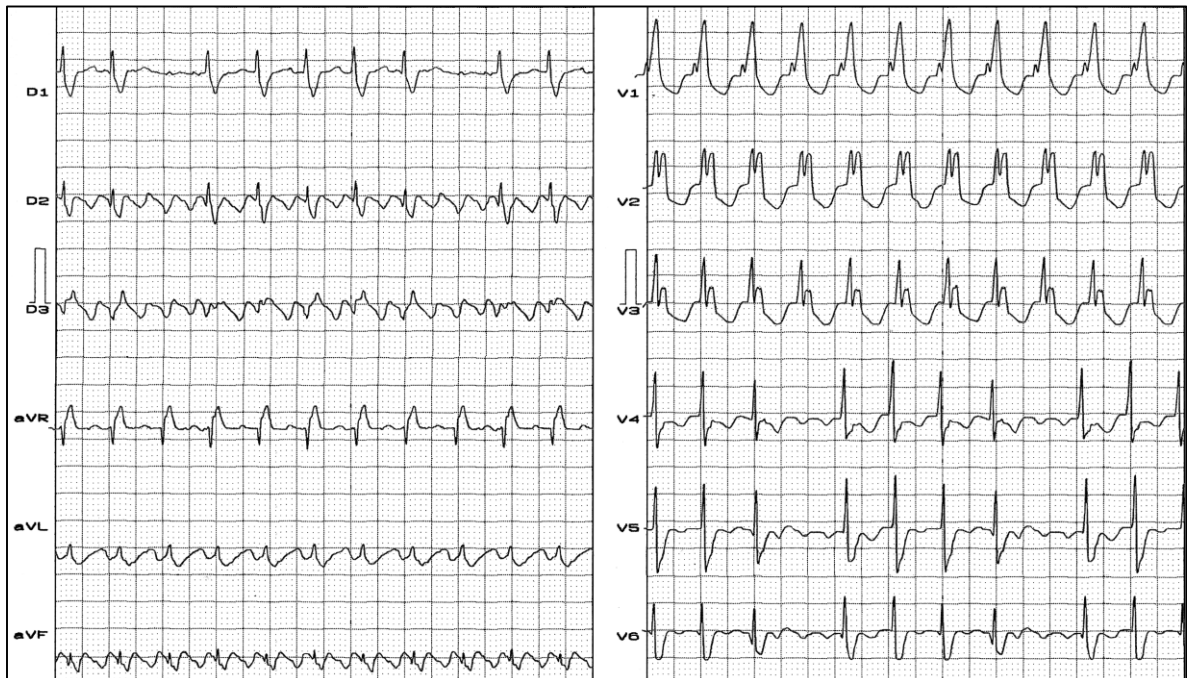


### Example of Atrial flutter with irregular AV conduction



**Figure.** ECG.

### Example of Atrial flutter with irregular AV conduction



**Figure.** Atrial flutter with 2:1 and 4:1 AV conduction. Atrial rate of 330 bpm. QRS complexes with complete right bundle branch block pattern.

