ATRIAL FLUTTER*

CMDT D. P. MYBURGH,† SM, SAMC, *Military Medical Institute and 1 Military Hospital, Voortrekkerhoogte, Tvl*

The electrocardiographical entity of atrial flutter was first described by Jolly and Ritchie in 1910.[1] Over the past 60 years a vast amount of literature has accumulated with respect to its possible mechanism;[2] most of the argument concerned its electrophysiological mechanism, i.e. the ectopic focus theory[3-7] versus the circus movement theory of Lewis,[8] neither of which could be substantiated. In contrast to the possible mechanism of atrial flutter, comparatively little has appeared on its electrocardiographical manifestations. This communication deals with the electrocardiographic findings in 41 patients with atrial flutter.

MATERIAL AND METHOD

The electrocardiographs (ECG) used for analysis were obtained from patients admitted with a variety of disorders, the atrial flutter being in all cases coexistent with other serious disease.

The specific symptomatology of atrial flutter as such, apart from palpitations and precipitation or aggravation of congestive failure in some cases, was difficult to evaluate and no attempt will be made to pursue this matter. Frequent as well as long ECG strips were obtained from all patients.

A diagnosis of atrial flutter was made by the presence of rapid regular undulating atrial waves in one or more leads and often one or more of the following: presence of, and/or increase in atrioventricular (AV) block with carotid sinus massage, slowing of atrial rate with quinidine therapy, increasing and varying block with digitalis therapy, conversion to atrial fibrillation with digitalis therapy, and conversion to normal sinus rhythm with low voltage capacitator discharge.

RESULTS

Clinical Findings

Twenty-seven (66%) of this group of patients were males and 14 (34%) were females, and the average age was 55 years for both sexes. Serious diseases, most commonly cardiac, were present in all patients. Atherosclerotic cardiovascular disease was present in 23 patients, of whom 3 had hypertension, 2 were recovering from acute myocardial infarctions, 3 were admitted with pneumonia and 1 had complete AV block. Eight patients had acquired mitral insufficiency either as the dominant lesion or associated with mitral stenosis or aortic valvular disease. Alcoholic myocardiopathy was diagnosed in 4 patients, 2 of whom probably had associated atherosclerotic cardiovascular disease. Myocardiopathy of unknown aetiology was present in 1 patient and 2 patients had myotonia dystrophica. In 1 patient with an ostium primum defect, atrial flutter was precipitated by cardiac catheterization. Systemic lupus erythematosus with pericarditis was diagnosed in 2 patients and in 2 patients atrial flutter occurred after pneumonectomy. Thirty-two of the 41 patients had cardiac enlargement on chest X-ray, while 9 patients had normal cardiac size.

In the majority of patients no definite precipitating factor for the arrhythmia could be determined. Two patients developed the arrhythmia during an episode of pneumonia, 2 during the recovery period after an acute myocardial infarction, 2 following pneumonectomy, 1 during cardiac catheterization, 1 after a pulmonary embolus and 1 intermittently during periods of marked sinoatrial slowing.

Electrocardiographic Findings

Flutter rate (Fig. 1). The flutter rate varied between 225 and 475 per minute. Of the 12 patients with a rate

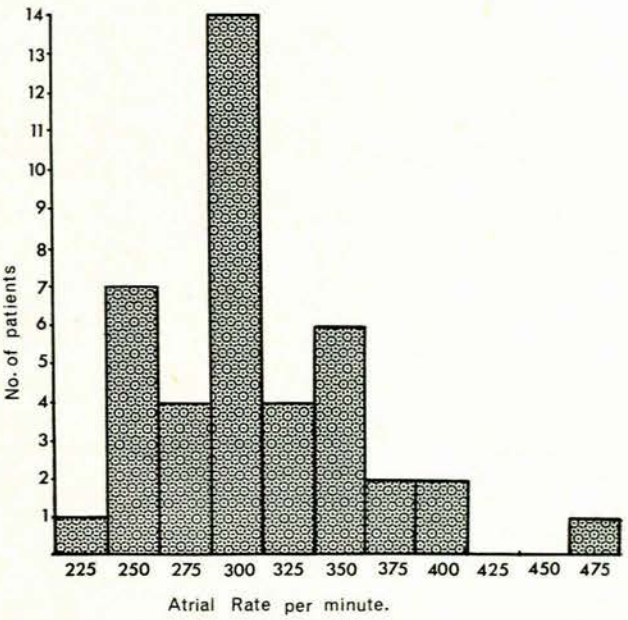


Fig. 1. Histogram depicting flutter rate per minute (value to the nearest 25) for 41 patients with atrial flutter.

less than 300 per minute, only 2 were on quinidine at the time of the initial ECG.

Regularity of flutter waves. In 16 patients there was slight irregularity of the flutter waves but occasionally as much as 0·05 seconds between FF intervals was recorded (Fig. 2). All patients with irregularity of flutter waves were taking either digitalis or quinidine.

PR interval during normal sinus rhythm. Tracings during normal sinus rhythm were available in 32 patients. The average PR interval in these patients was 0·18 seconds, 9 had a PR interval of 0·15 seconds or less while in 23 the PR interval measured 0·16 seconds or longer. The PR interval was longer than 0·20 seconds in 9 patients. The average degree of AV block in the group with PR intervals 0·15 seconds or less was 2·5:1, whereas in the group with PR intervals 0·16 or more the average degree of AV block was 2·6:1.

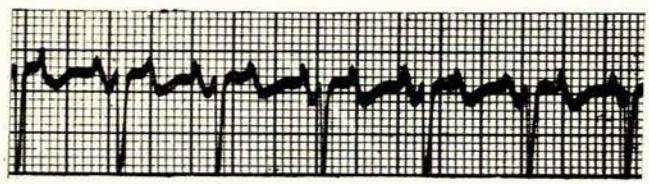
Ventricular response. The most common pattern of atrioventricular ratio was a regular 2:1 response seen in 14 patients and a regular 4:1 response seen in 4 patients.

*Date received: 26 August 1970.
†Formerly Assistant Professor of Medicine, Tulane School of Medicine, New Orleans, USA.

One patient converted from a regular 2:1 to a constant 3:1 AV ratio on digitalis therapy (Fig. 3). Patients with an irregular ventricular response could be divided into 2 groups. In the first group of 11 patients no distinct pattern was discernible. In the second group of 14 patients, one or more of the following patterns became obvious on measurement of sequential RR intervals:

1. Regular 2:1 alternating with 4:1 AV response in 3 patients.
2. Wenckebach type of ventricular response in 8 patients (Figs. 4 and 5).
3. Cyclical prolongation of RR intervals in 3 patients (Fig. 6).
4. Multiple interruptions of the basic mechanism, whether regular or irregular, by 1 or 2 or more consecutive periods of higher degree of AV block in 8 patients (Fig. 7).
5. Complete AV block in presence of atrial flutter in 2 patients.

Electrical axis of flutter waves. An attempt was made to calculate the approximate electrical axis in the frontal plane according to the usual hexaxial reference system. This was possible in 36 cases and was found to be superior and to the right in 13, superior and to the left in 9, inferior and to the right in 7 and inferior and to the left in 8 cases. In 3 patients of this group, spontaneous reversion of the polarity of the flutter waves was observed (Fig. 8). Unfortunately, in one patient only was it possible to obtain enough leads to calculate the resultant electrical axis during these reversions. In this patient the electrical axis changed from a direction



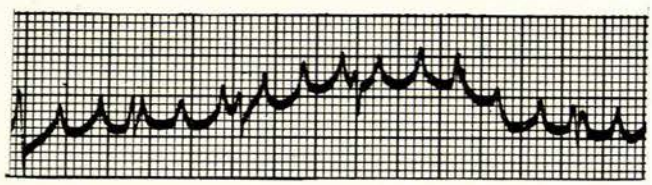


Fig. 2. Lead V1 of 2 patients with atrial flutter. Note variation in FF intervals.

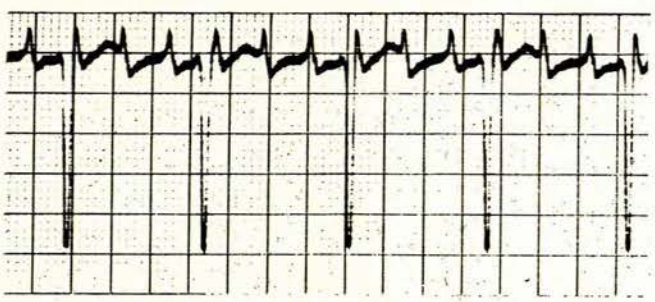
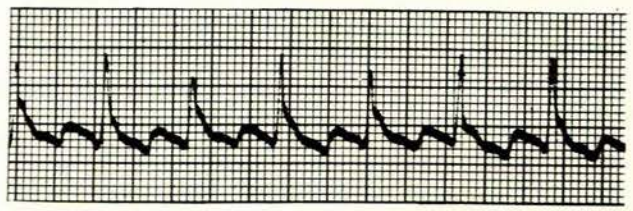
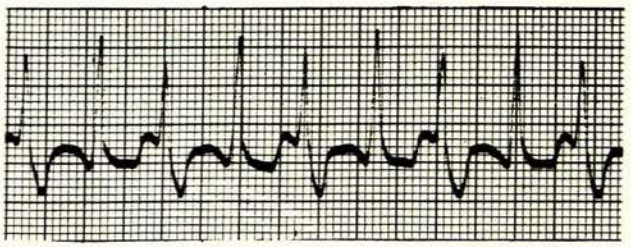


Fig. 3. Lead V1 from a patient with alcoholic myocardiopathy and probable associated atherosclerotic heart disease. The initial ECG showed a regular 2:1 AV ratio but reverted to a constant 3:1 AV ratio on digitalis therapy.





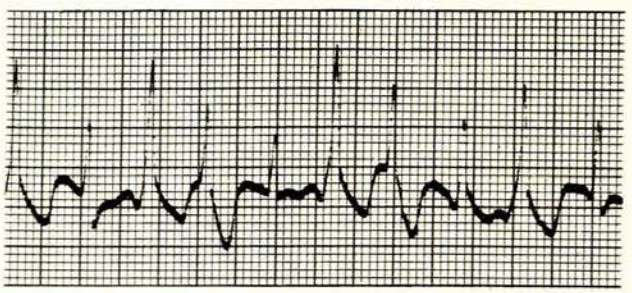


Fig. 4. Transverse bipolar chest lead corresponding to standard lead I recorded from a 57-year-old male with alcoholic myocardiopathy. The tracings are sequential but not continuous. A regular 2:1, a 3:2 and a predominant 4:3 AV response is depicted in the upper, middle and lower tracings respectively. In this specific lead the F waves are not clearly visible and the ventricular response in the middle tracing is also compatible with 2:1 AV ratio with concealed reduction of alternate 'blocked' atrial impulses.[9,10]

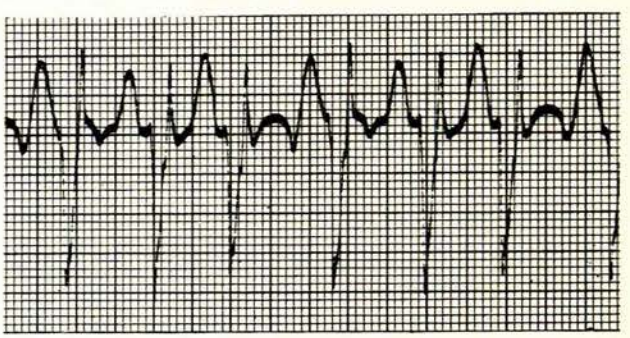


Fig. 5. Atrial flutter precipitated by cardiac catheterization in an 18-year-old male with an ostium primum defect. Although the ventricular response appears grossly irregular, measurement of the RR intervals reveals a consistent basic pattern, compatible with Wenckebach-type response.

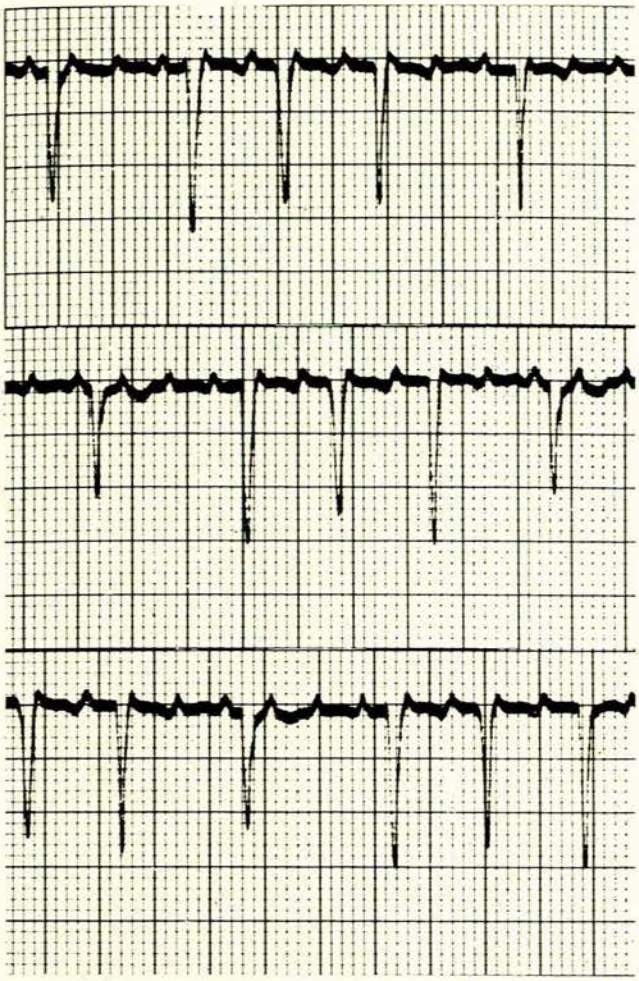


Fig. 6. The predominant pattern of ventricular response is that of progressive prolongation of RR intervals. The underlying mechanism probably also represents Wenckebach type of response as its counterpart during normal sinus rhythm has been shown to exist.[11] Frequent aberrant intraventricular conducted beats are present.

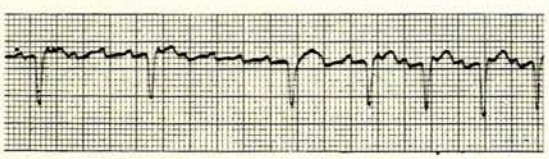


Fig. 7. A fairly regular 2:1 AV ratio is interrupted by cycles of prolonged RR intervals.

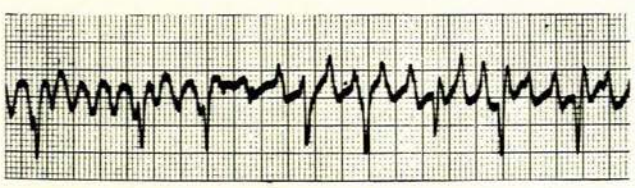


Fig. 8. Lead V1 of a 26-year-old male on digitalis therapy. Sudden reversal of impure flutter waves from an inverted to an upright direction is evident. Concomitant with the axial change there is also a fall in the flutter rate.

superior and to the right to a direction inferior and to the left. In 5 patients the flutter waves were clearly defined in lead V1 only, the so-called mimic type.[6]

ECG lead in which flutter waves were best seen. In 26 patients flutter waves were best seen in lead V1, while in 9 they were equally well seen in leads II, III aVF and V1. In only 6 patients were the flutter waves best seen in leads II, III and aVF.

Aberrant ventricular conduction. Aberrant ventricular conduction diagnosed according to the criteria of Marriott and Sandler[12] was seen in 17 patients and usually appeared after digitalis therapy was started.

DISCUSSION

Males predominated in this group of patients and the average of the whole group was 55 years. Most of the patients were in the older age-group, the average age being brought down by a relatively small number of young patients with rheumatic heart disease. Atrial flutter occurred in a wide variety of diseases and in no patient could the diagnosis of 'benign' atrial flutter be made. This reaffirms the impression[13] that atrial flutter is almost always associated with serious cardiac disease; in fact, 7 patients of this series died while in the hospital.

The actual rate in flutter has not been clearly defined, but both very slow[14] and very high rates have been reported.[15,16] In this study the flutter rate was mostly in the range of 300 per minute.

In contrast to the notion of a high degree of regularity of flutter waves,[17] several patients were noted to have slight to marked irregularity of atrial flutter waves (Fig. 2). However, all patients that revealed irregularity of their flutter waves were taking quinidine or digitalis or both drugs at the time of recording of the tracings. Both quinidine[14] and digitalis exhibit effects on impulse conduction and could account for this phenomenon.

The AV ratio has been suggested as an indication of the severity of AV conduction tissue disease;[18] the higher the degree of anterograde block, the longer the PR interval during normal sinus rhythm. No direct correlation could be found for this in the present series.

In the majority of cases of untreated atrial flutter, the ventricular response is regular and usually the AV ratio is 2:1 and less often 4:1. This almost monotonous pattern is easy to recognize and has been reaffirmed with this study. However, commonly after digitalis and quinidine therapy and less often without any drugs this pattern is disturbed and a confusing erratic ventricular response may be seen, at which time the diagnosis of the arrhythmia is sometimes misinterpreted. This is even more liable to occur in the presence of frequent aberrant intraventricular conduction (Fig. 4).

Measurement of sequential RR intervals will often suggest a basic mechanism in that certain groupings of RR intervals reappear cyclically. Frequently the behaviour of RR intervals shows progressive shortening before a long pause (Figs. 4 and 5) and is identical with the Wenckebach phenomenon seen during normal sinus rhythm. This feature of atrial flutter has been recognized by Lewis[19] and it was assumed to be the most probable mechanism in 50% of cases of atrial flutter with an irregular ventricular response.[18] To explain the Wenckebach type of

ventricular response 2 levels of block in the AV conducting tissue[20] were assumed, i.e. a higher level functional block at least halving the number of incoming impulses, and a lower level block that might delay or block conduction of whatever impulses are let through by the higher level. This proposition also satisfactorily explains the occurrence of regular 4:1, 2:1 alternating with 4:1 and the rarity of regular 3:1 AV ratio.[20] Conceivably in a complex system such as the AV transmission, no preference should be given to the above mechanism for any one or multiple mechanisms, including concealed conduction[9,21-23] longitudinal dissociation[24] and decremental conduction[25] which might equally well account for irregular ventricular response in a regular atrial mechanism.

Contrary to popular belief, standard lead II was not the best lead for recognizing atrial flutter. Flutter waves were readily visualized in lead V1 in 35 patients and among the routine 12 leads this proved to be by far the most reliable lead for recognizing atrial flutter. In doubtful situations carotid sinus massage with resultant increase in AV block proved to be still the most effective procedure for substantiating the diagnosis.

The so-called caudal type of atrial flutter, in which the waves supposedly originate caudally in the atria, was present in 54%, whereas the cranial type was seen in 36% of cases in contrast to 69% and 15% reported by Prinzmetal *et al.*[6] In 2 out of 3 patients in which spontaneous reversion of the direction of flutter waves occurred this reversion was present only for very short periods of time in an otherwise stable type. The mimic type of atrial flutter[6] in which atrial activity is visible only in the precordial leads was also seen in 10% of cases.

Aberrant ventricular conduction was commonly seen in patients on digitalis therapy but also occurred in patients not receiving any therapy. In both treated and untreated patients, aberrant ventricular conduction occurred only in the presence of irregular ventricular response.

Finally, 11 patients were subjected to electrical cardioversion for control of their arrhythmia. Normal sinus rhythm was obtained in 10 patients with 25 and in 1 patient with 50 watt-seconds.

SUMMARY

A clinical electrocardiographic study was performed on 41 patients with atrial flutter. In all patients an underlying cardiac disorder was present or suspected.

An atrial flutter rate of 300 per minute was commonly seen; slight irregularity of flutter waves does occur. The most common regular AV ratio is 2:1. Often certain patterns can be recognized when the ventricular response is irregular, while in others the response is completely erratic.

In most cases the electrical axis of the flutter waves is orientated superiorly. Lead V1 is the most reliable lead for recognizing the arrhythmia. Carotid sinus massage with resultant increase in AV block is the most reliable procedure for substantiating the diagnosis. Aberrant intraventricular conduction occurs commonly. Normal sinus rhythm can be obtained with low energy capacitator discharge.

REFERENCES

1. Jolly, W. A. and Ritchie, W. T. (1910 - 1911): Heart, **2**, 177.
2. Scherf, D. (1966): Amer. Heart J., **71**, 273.
3. Rothberger, C. J. (1922): Klin. Wschr., **1**, 82.
4. *Idem* (1923): *Ibid.*, **2**, 1407.
5. Scherf, D., Romano, F. J. and Terranova, R. (1948): Amer. Heart J., **36**, 241.
6. Prinzmetal, M., Corday, E., Brill, I. C., Oblath, R. W. and Kruger, H. E. (1952): *The Auricular Arrhythmias*. Springfield, Ill.: Charles C. Thomas.
7. Scherf, D., Schaffer, I. A. and Blumenfeld, S. (1953): Arch. Intern. Med., **91**, 333.
8. Lewis, T., Feil, H. S. and Stroud, W. D. (1918 - 1920): Heart, **7**, 191.
9. Kaufman, R. and Rothberger, C. J. (1927): Z. exp. Med., **57**, 600.
10. Langendorf, R. and Pick, A. *in* Dreifus, L. S. and Likoff, W., eds. (1966): *Mechanisms and Therapy of Cardiac Arrhythmias*. New York: Grune & Stratton.
11. Watanabe, Y. and Dreifus, L. S. *in* Dreifus, L. S. and Likoff, W., eds. (1966): *Ibid.*
12. Marriott, H. J. L. and Sandler, A. (1966): Progr. Cardiovasc. Dis., **9**, 18.
13. Gillman, H. and Preiss, H. (1963): Z. Kreisl.-Forsch., **52**, 139.
14. Korns, H. M. (1923): Arch. Intern. Med., **31**, 36.
15. Corday, E. and Irving, D. (1962): *Disturbance of Heart Rate, Rhythm and Conduction*. Philadelphia: W. B. Saunders.
16. Doliopoulus, T. H. and Marousis, S. (1968): Cardiologia (Basel), **52**, 113.
17. Lewis, T. (1918 - 1920): Heart, **7**, 127.
18. Besoain-Santander, M., Pick, A. and Langendorf, R. (1950): Circulation, **2**, 604.
19. Lewis, T. (1925): *Clinical Disorders of the Heart Beat*. Chicago: Chicago Medical Book Co.
20. Ashman, R. and Hull, E. (1941): *Essentials of Electrocardiography*. New York: MacMillan.
21. Ashman, R. (1925): Amer. J. Physiol., **74**, 121.
22. Söderström, N. (1950): Amer. Heart J., **40**, 212.
23. Langendorf, R. and Pick, A. (1956): Circulation, **13**, 381.
24. Moe, C. K., Preston, T. B. and Burlington, H. (1956): Circulat. Res., **4**, 357.
25. Watanabe, Y. and Dreifus, L. S. (1965): Amer. Heart J., **70**, 505