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# Electrical alternans in cardiac tamponade

ANDREAS P. NIARCHOS

*Royal Southern Hospital, Caryl Street, Liverpool 8*

**Niarchos, A. P. (1975).** *Thorax*, **30**, 228–233. **Electrical alternans in cardiac tamponade.** Of nine patients with pericardial effusion due to various causes, four developed cardiac tamponade. Electrical alternans was present in all four, being total in three and ventricular in one. The alternans corresponded very well with the clinical diagnosis of cardiac tamponade and the radiological signs of a large pericardial effusion. In two patients alternans was present even with heart rates below 100 per minute. Apart from the exact (1 : 1) type of electrical alternans, three new types are described, a 2 : 1, 3 : 1, and a varying type. It is concluded that (*a*) electrical alternans associated with pericardial effusion is strongly suggestive of impending or established cardiac tamponade, and (*b*) electrical alternans is produced when the heart is oscillating within the pericardial sac distended by fluid with a frequency equal to one-half (exact alternans), one-third (2 : 1 alternans), and one-quarter (3 : 1 alternans) of the heart rate.

The aetiology and mechanism of electrical alternans are discussed.

Electrical alternans has been defined as an alternation of the configuration of the electrocardiographic complexes arising from the same pacemaker and being independent of periodic extracardiac phenomena (Spodick, 1962). This electrocardiographic abnormality was initially observed in the laboratory by Herring in 1909 (Brody *et al.*, 1973), and first reported clinically the year after by Lewis (1910). Other early reports were those of Hamburger, Katz, and Saphir (1936) and of Brody and Rossman (1937). The literature on the subject up to 1955 has been reviewed by McGregor and Baskind (1955).

Any electrocardiographic complex can exhibit alternation, the P wave rarely (Kisch, 1949; Bernreiter, 1956), the QRS complex (McGregor and Baskind, 1955), the T wave alone (Doherty and Hara, 1961; Kimura and Yoshida, 1963; Littmann, 1963; Dolara and Pozzi, 1971), or the U wave (Eyer, 1974). Finally, all main complexes (P–QRS–T) may alternate simultaneously when the phenomenon is called total electrical alternans (Littmann and Spodick, 1958; Brody *et al.*, 1973). Although ventricular and total electrical alternans are not very common they are of clinical interest because they occur primarily in association with massive pericardial effusion and cardiac tamponade (McGregor and Baskind, 1955; Colvin, 1958; Littmann and Spodick, 1958; Spodick, 1962; Bashour and Cochran, 1963; Lawrence and Cronin, 1963; Gabor, Winsberg, and Bloom, 1971; Usher and Popp, 1972; Brody *et al.*, 1973).

Two theories have been suggested to explain the mechanism of electrical alternans. One attributes the alternation of the electrocardiographic complexes to alternating conduction within the myocardium (Spodick, 1962). The other maintains that the conduction within the myocardium during alternans remains unchanged, but that a cyclic motion of the heart within the distended pericardial sac accounts for the alternating electrocardiographic pattern (Feigenbaum, Zaky, and Grabhorn, 1966; Price and Dennis, 1969; Usher and Popp, 1972; Brody *et al.*, 1973). It is the purpose of this paper to report the clinical, radiological, and electrocardiographic findings in patients with pericardial effusion and tamponade associated with total and ventricular electrical alternans in whom the confirmation of the mechanical theory of the genesis of alternans is based on electrocardiographic data.

## PATIENTS AND METHODS

Nine patients were studied, eight males and one female; their ages ranged from 36 to 72 years. Pericardial effusion with tamponade was diagnosed in four and pericardial effusion without tamponade in five. Clinical data are presented in Table I. All but two patients were treated in an intensive therapy unit under continuous electrocardiographic monitoring

TABLE I
DIAGNOSIS AND OUTCOME IN THE NINE PATIENTS

| Case | Sex/Age | Clinical Diagnosis | Type of Fluid and Means of Confirmation | Electrical Alternans | Primary Disease | Outcome |
|---|---|---|---|---|---|---|
| 1 | M 65 | Tamponade | Haemorrhagic; pericardiocentesis | Total | Acute myocardial infarction | Survived |
| 2 | M 56 | Tamponade | Haemorrhagic; pericardiocentesis and necropsy | Total | Carcinoma L bronchus invading pericardium | Died |
| 3 | F 62 | Tamponade | Haemorrhagic; pericardiocentesis and necropsy | Total | Secondary carcinomatous pericarditis, primary unknown | Died |
| 4 | M 36 | Large effusion | Hydropericardium; albumin/globulin ratio = 0·6/1 (see text) | Ventricular | Malabsorption due to coeliac disease | Survived |
| 5 | M 53 | Effusion | Not known | Absent | Benign pericarditis | Survived |
| 6 | M 72 | Effusion | Haemorrhagic; necropsy | Absent | Acute myocardial infarction | Died |
| 7 | M 63 | Effusion | Haemorrhagic; necropsy | Absent | Acute myocardial infarction | Died |
| 8 | M 37 | Effusion | Not known | Absent | Uraemia | Survived |
| 9 | M 62 | Effusion | Hydropericardium; low albumin/ globulin ratio | Absent | Extensive burns | Survived |

All patients had serial electrocardiograms and chest radiographs. The cardiothoracic ratio and cardiac silhouette volume were estimated from data obtained from radiographs (taken from a distance of 6 feet (1·8 m) with the patient standing), as described by Glover, Baxley, and Dodge (1973). In addition the diagnosis of a large pericardial effusion was confirmed by radioisotope heart scans in two patients (Fig. 1), cardiac catheterization in one (case 4), and by pericardiocentesis in three patients. Necropsy was performed in four cases. The incidence of clinical, electrocardiographic, and radiological findings was compared between the tamponade and effusion groups. The radiological findings were statistically analysed using Student's *t* test. Two patients from this series (cases 6 and 7) have been briefly described previously (Niarchos and McKendrick, 1973).

## RESULTS

CLINICAL FINDINGS The main clinical findings in both groups are listed in Table II. Pericardial paracentesis relieved the symptoms in cases 1, 2, and 3. Frusemide was given to the first patient after the pericardiocentesis and his condition improved gradually. The second and third patients, although showing initial improvement, died later from recurrent cardiac tamponade. The patient with coeliac disease was treated from the start with frusemide with considerable improvement. Cardiac catheterization after one week's treatment showed cardiac restriction with raised end-diastolic pressures in all cardiac chambers. He

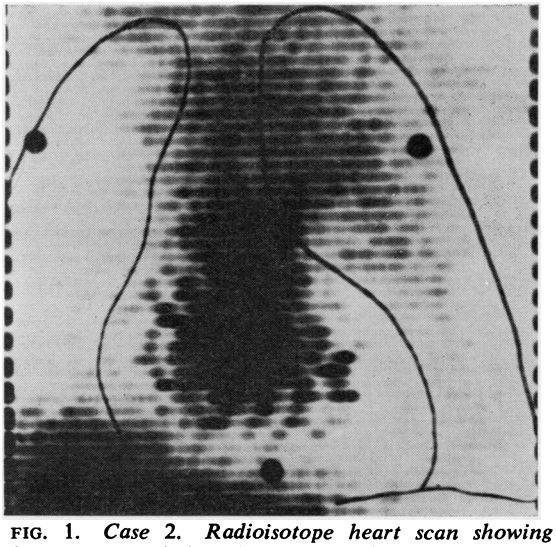

FIG. 1. *Case 2. Radioisotope heart scan showing large pericardial effusion mainly anteriorly; it coincided with total electrical alternans.*

TABLE II
MAIN CLINICAL FINDINGS IN THE NINE PATIENTS

| Findings | Number of Patients | |
|---|---|---|
| | Tamponade | Effusion |
| Pulsus paradoxus | 2 | Nil |
| Sinus arrhythmia | 4 | 4 |
| Sinus tachycardia (heart rate > 100/min) | 2 | 4 |
| Raised JVP | 4 | 4 |
| Hypotension (systolic BP < 100 mmHg) | 4 | 1 |
| Dyspnoea | 4 | 1 |
| Absent heart sounds | 2 | Nil |
| Faint heart sounds | 2 | 1 |
| Pericardial friction rub | 1 | 3 |
| Absent apex beat | 4 | — |

was given a gluten-free diet and the pericardial effusion disappeared radiographically when his plasma proteins returned to normal.

ELECTROCARDIOGRAPHIC FINDINGS Sinus arrhythmia and generalized low voltage (that is, the amplitude of the R wave was less than 3·7 to 16·6 mm in the various electrocardiographic leads as described by Burch and Winsor (1972)) were present in all patients with cardiac tamponade and in most patients with pericardial effusion. Electrical alternans, however, was present only in the four patients with tamponade. The alternans was total (P–QRS–T) in the first three cases (Figs 2, 3, and 4) and ventricular (QRS) in the fourth. In the first case it was seen only in leads I, II, and aVF, in the second it was present in all leads, while in the third and fourth cases it was better seen in lead $V_1$. The alternans was not very constant in the fourth case. The alternating complexes varied in height from complex to complex even in the same lead (Fig. 2), the difference being greater in the right ($V_1$, $V_2$) and left ($V_6$) chest leads. The electrical alternans disappeared in cases 1, 2, and 3 after aspiration of 30, 65, and 670 ml of pericardial fluid respectively (Figs 3 and 4), and after treatment with frusemide in the fourth patient, but the tachycardia persisted in case 3 (Fig. 3). During reaccumulation of the pericardial effusion in cases 2 and 3 several types of alternans were observed.

The heart and alternans rate and the various electrocardiographic types of alternans before pericardial aspiration are shown in Table III. A heart rate above 100 per minute was present in two patients on three occasions, while in the rest of the electrocardiograms the heart rate was below 100 per minute; total alternans was present with both fast and slow heart rates. The ratio between heart rate and alternans rate varied; it was always 2 : 1 when exact (1 : 1) electrical alternans was

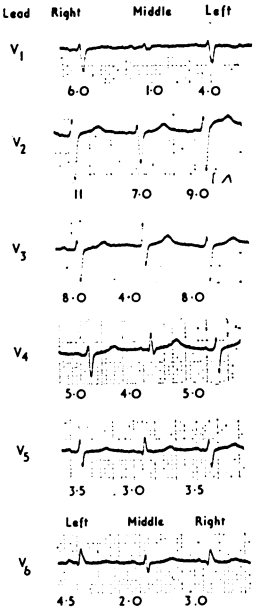


FIG. 2. *Case 2. Total electrical alternans (best seen in lead $V_6$) before pericardiocentesis; the alternans is 1 : 1 (exact alternans); the numbers represent the largest part of the QRS in millimetres, positive or negative. See text for details.*

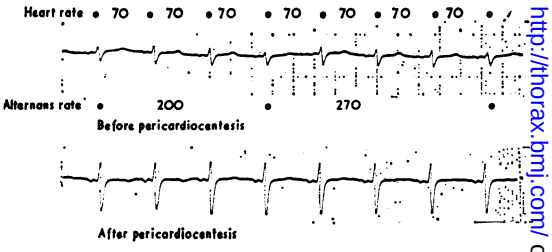


FIG. 3. *Case 3. Heart rate and alternans rate ratio is nearly 3 : 1 and 4 : 1, but the type of total alternans is 2 : 1 and 3 : 1 respectively. The heart rate is 111 per minute; after pericardiocentesis of 670 ml of fluid the heart rate remains the same but the alternans has disappeared.*

present (Figs 2 and 4), but 3 : 1 and 4 : 1 when 2 : 1 and 3 : 1 respectively alternans was present (Figs 3 and 5; Table IV). In addition a varying type of alternans was seen in case 2 soon before that patient's death; no constant relationship could be seen between the normal and alternating complexes, and the alternating complexes differed greatly from each other (Fig. 6). Conduction defects were not observed in the tamponade group.

## TABLE III

HEART AND ALTERNANS RATE BEFORE AND AFTER PERICARDIOCENTESIS; VARIOUS TYPES OF ELECTRICAL ALTERNANS ARE SHOWN

| Case | Before Pericardiocentesis | | | | | | After Pericardiocentesis |
|---|---|---|---|---|---|---|---|
| | | | | | ECG Types of Electrical Alternans | | |
| | Heart Rate[1] (HR) | Alternans Rate (AR) | Ratio HR/AR | Approx Ratio HR/AR | Ratio (normal/ alternating QRS) | Alternating Part | Heart Rate |
| 1 | 94 | 55 | 1·7:1 | 2:1 | 1:1 | Total | 71 |
| 2 | 94 | 48 | 1·9:1 | 2:1 | 1:1 | Total | 83 |
| 2 | 88 | 21 | 4·3:1 | 4:1 | 3:1 | Atrial | — |
| 2 | 83 | 27 | 3·0:1 | 3:1 | 2:1 | Total | — |
| 3 | 115 | 41 | 2·8:1 | 3:1 | 2:1 | Ventricular | 112 |
| 3 | 115 | 28 | 4·1:1 | 4:1 | 3:1 | Total | 112 |
| 4[2] | 136 | 75 | 1·8:1 | 2:1 | 1:1 | Ventricular | 83 |
| 2 | 83 | Varying | | Varying | Varying | Total | — |

[1]Marked sinus arrhythmia was present in all electrocardiograms.
[2]No pericardiocentesis, patient treated with frusemide.

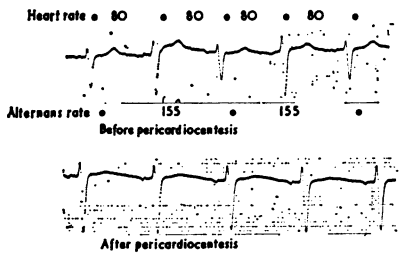


FIG. 4. *Case 2, lead $V_2$. Total electrical alternans 1 : 1 type (exact alternans). Heart and alternans rate is nearly 2 : 1. Sinus arrhythmia is present. After pericardiocentesis of 65 ml of fluid the alternans has disappeared.*

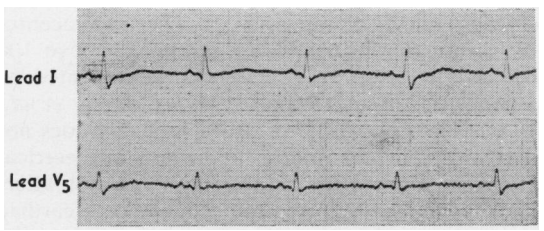


FIG. 5. *Case 2. Recurrence of cardiac tamponade. Lead I, a 2 : 1 electrical alternans is present; lead $V_5$, 3 : 1 alternans is present.*

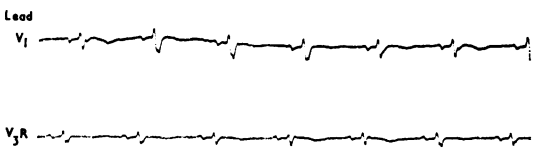


FIG. 6. *Case 2. Varying electrical alternans.*

The duration of the QRS did not vary greatly between the normal and alternating complexes.

## TABLE IV

DURATION OF QRS (SECONDS) IN PATIENTS WITH TAMPONADE BEFORE AND AFTER PERICARDIOCENTESIS

| Case | Before Pericardiocentesis | | | After Pericardiocentesis |
|---|---|---|---|---|
| | Lead | Normal QRS | Alternating QRS | Normal QRS |
| 1 | II | 0·10 | 0·08 | 0·06 |
| 2 | I | 0·08 | 0·10 | — |
| 2 | $V_1$ | 0·08 | 0·10 | 0·12 |
| 2 | $V_5$ | 0·06 | 0·08 | — |
| 3 | $V_1$ | 0·08 | 0·08 | 0·08 |
| 4 | $V_1$ | 0·06 | 0·06 | 0·08 |

## DISCUSSION

INCIDENCE AND AETIOLOGY OF ELECTRICAL ALTERNANS The incidence of electrical alternans has been estimated as varying between 1 in 1,212 and 1 in 10,000 tracings (Hamburger *et al.*, 1936; Kalter and Schwartz, 1948). To date about 80 cases, including the present four, have been reported in the literature (Brody and Rossman, 1937; McGregor and Baskind, 1955; Colvin, 1958; Usher and Popp, 1972). In two-thirds of the reported cases the alternans was due to cardiac tamponade caused by malignant haemopericardium or by massive pericardial effusion. The clinical, radiological, and other laboratory findings of the present study support the view that total electrical alternans is diagnostic of pericardial tamponade or massive effusion. The abnormality disappeared after aspiration of varying amounts of pericardial fluid, and this coincided with clinical improvements and reduction of the heart size on the chest radiograph. To my knowledge, electrical alternans due to either cardiac tamponade after acute myocardial infarction or large pericardial effusion (hydropericardium) complicating gluten-induced enteropathy has not previously

been described. Other causes are listed in Table V.

TABLE V
AETIOLOGY OF ELECTRICAL ALTERNANS

| | |
|---|---|
| Cardiac tamponade | Malignant haemopericardium<br>Massive pericardial effusion due to:<br>Tuberculous pericarditis<br>Suppurative pericarditis<br>Idiopathic pericarditis<br>Uraemia<br>Acute myocardial infarction |
| Constrictive pericarditis<br>Congestive cardiac failure | Rheumatic heart disease<br>Ischaemic heart disease<br>Myocarditis<br>?Congestive cardiac failure |
| Hypertension<br>Pneumonectomy<br>Tension pneumothorax[1] | |

[1]Niarchos, A. P., unpublished.

MECHANISM OF ELECTRICAL ALTERNANS The electrocardiographic findings of this study do not support the aberrant conduction theory as the mechanism for electrical alternans (Spodick, 1962) since no conduction defects were observed and the duration of the alternating QRS complexes did not differ greatly from that of the normals (Table IV). On the contrary, the present electrocardiographic findings lend support to the theory which presumes exaggerated anatomical motion of the heart within the pericardial sac enlarged by massive effusion. Indeed, several types of such a cardiac motion have been demonstrated in patients with pericardial effusion and total electrical alternans by cineangiography (Price and Dennis, 1969) and echocardiography (Feigenbaum *et al.*, 1966; Usher and Popp, 1972), and by using a laboratory model (Brody *et al.*, 1973). With the last method both oscillatory and twisting cardiac motions with varying frequency, plane, and amplitude were observed.

The variation in size of the alternating QRS complexes as seen in the present cases can be explained by accepting the view that the heart is oscillating within the distended pericardial sac from left to right and vice versa (Fig. 3). When the heart is close to the right chest leads $V_1$ and $V_2$ a large QRS (QRSr) is recorded; when in the middle the recorded complex (QRSm) is small because the heart is surrounded by fluid; and when the heart is close to the left chest wall the recorded QRS (QRSl) is larger than the QRSm, but smaller as compared with the QRSr because it is recorded from a distance. In lead $V_6$ the opposite sequence of events takes place. In the middle chest leads ($V_3$, $V_4$, and $V_5$) the QRSr is equal to the QRSl because both are recorded from a more or less equal distance from the middle. The variation of the QRS size in the posterior leads only, as seen in case 1, may be explained on the same basis but assuming that the heart is moving along an anteroposterior plane. The observed variations in heart rate/alternans rate ratios 2 : 1, 3 : 1, and 4 : 1 (Table III) can be explained by assuming that the frequency of the cardiac cyclic motion is one-half, one-third, and one-quarter respectively of the heart rate (Figs 3, 4, and 5). Likewise a cardiac motion with varying frequency and plane could account for the varying alternans.

The factors which probably determine the frequency and form of the cardiac motion and hence the appearance of electrical alternans are the heart rate, the pericardial pressure, the rate of accumulation, volume, and viscosity of the pericardial fluid, an aortic root fixed by secondary deposits, the rigidity and configuration of the pericardial sac, and the mobility of the pendulum-like heart within the pericardial sac (Usher and Popp, 1972; Brody *et al.*, 1973). A combination of at least three factors is probably necessary to produce the type of heart motion that is associated with electrical alternans, since the presence of two of them (Tables I and II) did not produce alternans in the effusion group of patients. It has been suggested that electrical alternans is present only when the heart rate is 100 per minute or greater (Littman and Spodick, 1958), but this is not confirmed in the present study as alternans was present with heart rates below 100 per minute (Fig. 5), and it disappeared after pericardiocentesis, although the heart rate remained above 100 per minute (Fig. 3), as has been documented by others (Usher and Popp, 1972; Cokkinos *et al.*, 1974). The pericardial pressure probably does not play an important role in the genesis of electrical alternans since bradyarrhythmias and not alternans have been reported to occur in acute cardiac tamponade (Meurs *et al.*, 1970; Editorial, 1972).

CLASSIFICATION AND PROGNOSIS It is clear from the present study that in addition to the well-known 1 : 1 or exact alternans a 2 : 1 and 3 : 1 alternans exist, and both can be either total or ventricular. Furthermore, a varying type of electrical alternans, characterized by marked variation and completely irregular QRS alternation, has been documented. All types of alternans when due to pericardial effusion should be considered an indication for pericardiocentesis, which may be a life-saving procedure. The prognosis, however, of electrical alternans

associated with malignant haemopericardium is grave because most of the reported cases, and two from this series, died a few days after its onset despite temporary improvement and disappearance of the alternans following pericardiocentesis.

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Requests for reprints to: Dr. A. P. Niarchos, Research Division, Cleveland Clinic, 9500 Euclid Avenue, Cleveland, Ohio 44106, USA.