

# THE ELECTROCARDIOGRAM OF THE STOKES-ADAMS ATTACK

BY

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The combination of a slow pulse with syncopal, epileptiform, or pseudo-apoplectic attacks was briefly mentioned by Morgagni in 1761; then it was more fully described by an Edinburgh physician, Spens (1793), and by Adams (1827) and Burnett (1827), equally well in the same year. Mayo (1838), reviewing examples of slowness of the pulse and quoting Spens, included one such case; and Holberton (1841) added another. Stokes (1846) wrote the important paper that drew general attention to the subject: because of this, and because Huchard (1899) originally proposed the term Stokes-Adams disease, we strongly favour its retention in this order and excluding the use of the word "syndrome", which would imply that all similar cases whatever the underlying pathology were to be included.

Shortly after Stokes's publication, the discovery was made of the slowing effect on the heart of vagal stimulation, and this governed the views on bradycardia until the end of the last century. Since 1900, with development of our knowledge of heart block, the newer conception placed bradycardia in general as due to an intrinsic myocardial lesion and seldom to a vagal effect. But from this division into neurogenic and myocardial causes another difficulty arises when we consider the definition of Stokes-Adams disease to-day. As our knowledge of the disease is infinitely greater, there is need to define it afresh. Shall it include all cases of cardiac syncope whether these result from vagal action or from ventricular asystole of myocardial origin? We think not, believing that heart block of some grade should be present at some time if the term Stokes-Adams attack is to retain a distinctive meaning. Our reasons are that the great majority of the clinical cases in question are due to myocardial disease with block and that their course, prognosis, and treatment are similar; whereas the reported cases of neurogenic origin (usually without block) are rare and of varied ætiology and prognosis, and may reasonably be classified as cardiac syncope.

*Definition.*—Stokes-Adams disease is a name applicable to patients with heart block who suffer from recurrent attacks of loss of consciousness due to ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these.

During a Stokes-Adams attack from ventricular standstill the auricle continues to beat, whereas in other cardiac syncopes as a rule there is total cardiac standstill.

The term cardiac syncope may be reserved for attacks in patients without heart block due to total cardiac standstill from neurogenic or myocardial causes.

*Cardiac syncope of neurogenic origin.*—An ordinary faint in healthy people is most often of vaso-vagal origin with a fall of blood pressure combined with bradycardia (Cotton & Lewis, 1918), and we have recorded this event in a healthy man of 40, who happened to faint in the cardiographic chair (Fig. 1). In healthy people, too, vagal, carotid sinus, or ocular pressure (Fig. 2) may

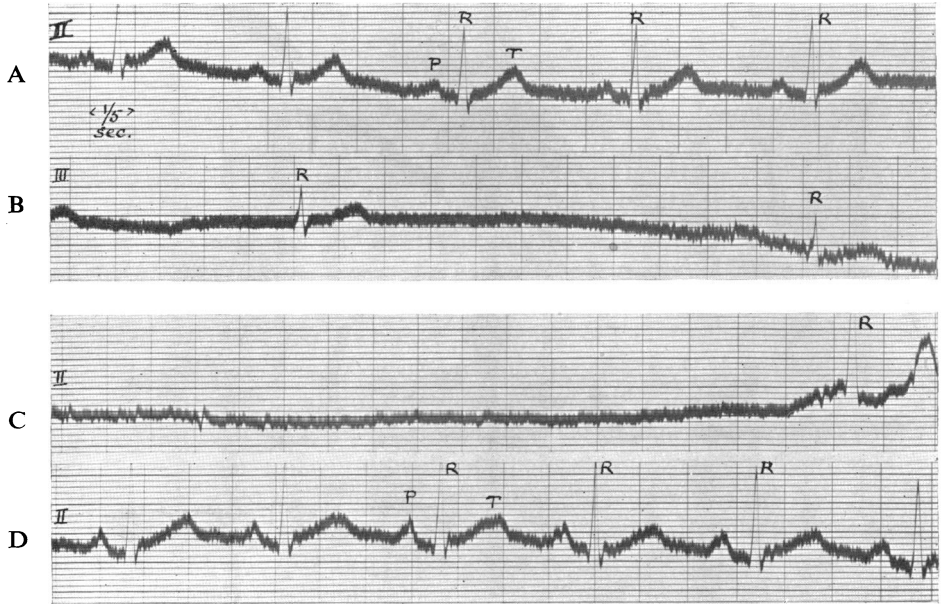


FIG. 1.—Syncope in a healthy man. (A) Before; (B) During faintness; (C) Syncope; (D) Recovery.

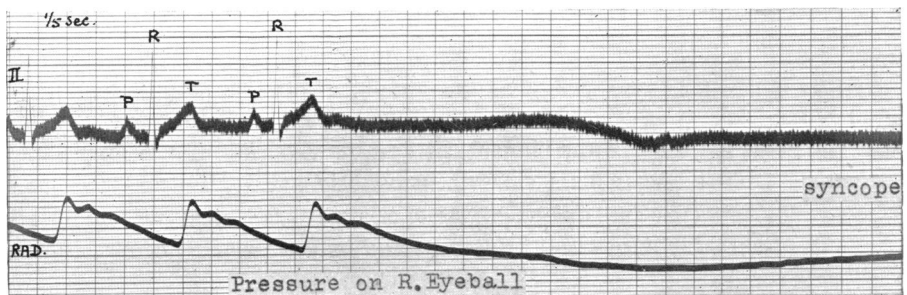


FIG. 2.—Induced syncope in a healthy boy. Pressure on right eyeball.

produce bradycardia and even ventricular standstill with syncope (Lewis, 1925). Transient A-V block may thus be induced (Weiss & Baker, 1933). There are rare but interesting cases of syncope from disease of or near the carotid sinus, or affecting the vagus in its course (Cassidy, 1928; Cassidy & Page, 1928; Gluch, 1932; Weiss & Ferris, 1934; Levy, 1939). A few of

them have been reported as Stokes-Adams attacks, but such cases are not included under our definition, though they are on the borderline, certainly when associated with transient and neurogenic A-V block (Weiss & Ferris, 1934).

*Cardiac syncope of myocardial origin.*—Paroxysmal ventricular tachycardia (Allan, 1926; Lukl, 1937; Grædel & Kisch, 1939), the same as a complication of nodal bradycardia (Dressler, 1929) or from severe myocardial damage during a fatal infection (Schwartz & Jezer, 1934), may produce loss of consciousness and approximate to a Stokes-Adams attack. The episodic nature of the attacks, and the absence of heart block distinguish them from it.

In auricular flutter, syncope may be determined by the assumption of a 1:1 rhythm at the high auricular rate. Fæssler (1939) has described the case of an infant in which nodal bradycardia without standstill produced syncopal attacks.

#### INCIDENCE OF AND VARIETIES OF STOKES-ADAMS ATTACKS

Only a proportion of patients with heart block get Stokes-Adams attacks. Of Cowan and Ritchie's (1935) 78 cases of complete heart block, one third gave a history of attacks. In Graybiel and White's (1936) series of 72 cases, there were 44. Figures for their incidence in partial or variable block are scarce, but Downie (1929) found Stokes-Adams attacks distinctly more common in partial block than in complete block. It seems likely that a block changing from partial to complete, and a rapidly developing block are periods most susceptible to Stokes-Adams attack, but it is a mistake to assume that with established complete heart block a patient becomes immune from attacks.

It is widely believed that ventricular standstill is the only common disturbance of mechanism which, supervening in heart block, determines the loss of consciousness; and this is implied in many text-book descriptions. But other disturbances of the cardiac mechanism may be responsible for the cerebral attack, and it was with the object of deciding the relative frequency and importance of the mechanisms of the actual Stokes-Adams attack that this investigation was made.

We have restricted our inquiry to cases in which an electrocardiogram was recorded during the unconsciousness of a Stokes-Adams attack. Of these we have 8 at our disposal, and we have collected and studied 56 reported cases. As a consequence we have been led to adopt the following classification :—

##### *Group I.—Ventricular standstill alone.*

28 reported cases; and our Cases 1, 2, 3, 4, 5. (Table I).

##### *Group II.—Ventricular tachycardia followed by ventricular standstill.*

(a) Low ventricular tachycardia (low V.T. in this paper means a rate below 160) followed by ventricular standstill; 3 reported cases and our Case 6. (Table IIa.)

(b) High ventricular tachycardia (high V.T. means in this paper a rate between 200–500), usually also with ventricular fibrillation, followed by ventricular standstill; 13 reported cases and our Case 7. (Table IIb.)

ABBREVIATIONS USED IN TABLES

- |        |                            |          |                           |
|--------|----------------------------|----------|---------------------------|
| A.     | auricle                    | H.F.     | heart failure             |
| A.F.   | auricular fibrillation     | L.B.B.B. | left bundle branch block  |
| A.P.   | angina pectoris            | N.R.     | normal rhythm             |
| B.B.B. | bundle branch block        | R.B.B.B. | right bundle branch block |
| B.P.   | blood pressure             | Sl.      | slight                    |
| C.H.B. | complete heart block       | St. Ad.  | Stokes-Adams attack       |
| C.T.   | coronary thrombosis        | V.       | ventricle                 |
| EC.    | electrocardiogram          | V.F.     | ventricular fibrillation  |
| Ex.s.  | extrasystole (ventricular) | V.St.    | ventricular standstill    |
| H.B.   | heart block                | V.T.     | ventricular tachycardia   |

TABLE I  
GROUP I.—STOKES-ADAMS ATTACKS DUE TO VENTRICULAR STANDSTILL ALONE

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Wilson and Robinson (1918)	48	F.	St. Ad. one day	(a) C.H.B.; V. 66, A. 110 (b) 4: 1 and 2: 1 H.B. (c) N.R. (110) P-R 0.32 sec.	C.H.B.; V. 90, A. 110	V. St., A. to 120	C.H.B. V. 90	(V.St. 7-11 sec.)	Improved
Hay (1921)	—	—	—	—	N.R. 50 to 64, P-R 0.26, B.B.B.	V.St., A. 66-88	—	(V.St. 5.5 sec.)	—
Wiltshire (1923)	64	M.	Slow reg. pulse	C.H.B.	—	V.St., A. rapid then slow; occas. flutter	—	1-1.5 min.	—
Wenckebach and Winterberg (1927) Case 1.	—	—	—	(a) N.R., B.B.B. partial (b) partial H.B. (2-5: 1)	Normal P-R, then A. rapid; dropped beats	V.St., A. reaches 150	V.T. (140 in one attack)	(V.St. 18-23 sec.)	—

Wenckebach and Winterberg (1927) Case 2.	16	F.	—	(a) P-R+ (b) Partial B.B.B.	H.B.,	C.H.B.; V.39, A.110; occas. 2: 1 H.B.	V.St.	—	(V.St. 11 sec.)	—
Wenckebach and Winterberg (1927) Case 3.	—	M.	St.Ad. weeks: produced on emotion, never on exertion	Variable H.B.	H.B.	2: 1 then 3: 1	V.St., A to 120	—	(V.St. 7-8 sec.)	Died during attack
Stecher (1928)	65	M.	St.Ad. one week	C.H.B.; V. 20-30, A. 60-70	—	—	V.St., A. 58-76, ectopic P.	3: 1 H.B. P-R 0-36 sec.	10-20 sec. (V. St. 10 sec.)	Attacks ceased
Heimann (1929) Case 1.	60	M.	—	(a) C.H.B. (b) 2: 1, 3: 1 H.B. (c) P-R+; B.B.B.	—	—	V.St., A. 80 irreg.	—	34 sec.	—
Heimann (1929) Case 2.	—	—	—	—	—	—	V.St., occas. with A.F.	—	8 sec.	—
Yater and Willis (1929)	74	M.	St.Ad. 3 months. B.P. 240/105	(a) 3: 1, 2: 1 H.B. (V. 38; A. 75). (b) N.R. 75	C.H.B.; B.B.B. V. 55; irreg., A. 120	—	V.St., A. 130, then 50, then V.St., no P.	Basic compl. C.H.B.; V.10, A. 100	(V. St. 16-35 sec.)	Died during attack. Necropsy
Cheer and Tang (1932)	66	M.	St.Ad. 10 months	N.R. 85	C.H.B.	—	V.St., A. 120	N.R.; P-R 0-18-0-20 sec.	(V.St., 3-6 sec.)	Attacks ceased
Condorelli (1932)	72	M.	—	(a) P-R+ (b) C.H.B. Partial B.B.B.	C.H.B. 20	—	V.St.	V.T.	18 sec.	—
Wood (1932)	48	M.	St.Ad. 9 months	C.H.B.; V. 20-30, B.B.B.	—	—	V.St. A. 75-86	—	2-3 min. (V. St., 7-5 sec.)	Attacks ceased
Géraudel and Others (1933)	78	F.	B.P. 210/70; uraemia	(a) 2: 1 H.B.; V. 30, A. 60; partial B.B.B. (b) 1: 1 H.B.; V. 60	H.B.; V. 63, A. 71	—	V.St., P. quickens	H.B. as preceding	15-40 sec.	Death during attack. Necropsy

TABLE I—continued.

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Pardee (1933)	—	—	—	—	C.H.B.; V. 54, A. 96, R.B.B.B. B. then normal QRS	V.St.	—	(V.St. 15 sec.)	—
Sachs and Traynor (1934)	43	M.	St.Ad. months. Normal heart	N.R. 80	—	V.St. A. 110	C.H.B.; V. irreg., A. 90; R.B.B.B. then N.R.	1 min. (V.St. 5 sec.)	Attacks ceased
Gilchrist (1934)	61	F.	St.Ad. one year	(a) 2:1 H.B. P-R 0.18 sec. (b) C.H.B. V. 28, A. 70	2:1 H.B. P-R 0.19 sec.	V.St.	3:1 H.B.	(V.St. 10 sec.)	Attacks ceased
Lauer (1934)	68	M.	St.Ad. preceded by angina	(a) P-R 0.26-0.32 sec. Partial B.B.B. (b) 2:1, 3:1 H.B.; C.T. (post.)	Sinus tachycardia, P-R 0.36, dropped beats	V.St. A. 92-142	2:1, 3:1 H.B.	(V.St. 11 sec.)	Attacks ceased
Cowan and Ritchie (1935)	49	F.	—	—	—	V.St. (A. 120)	—	(V.St. 11 sec.)	—
Clerc and Levy (1936). Case 1	67	M.	St.Ad. months. H.F.	C.H.B. V. 38; A. 95	—	V.St. occas. P; then V.T.	V.T. 100-48 —27	(V.St. 30 sec.)	Died after 4 years (cerebral thromb.)
Schwartz (1936,b)	53	M.	St.Ad. one day, with C.T.	(a) C.H.B. (b) N.R.	—	V.St. A. 110	C.H.B.; then N.R.	(V.St. 9.5 sec.)	Attacks ceased Necropsy
Hermann and others (1937)	65	M.	St.Ad. 14 years. P. 80, reg.	(a) N.R. 75; partial B.B.B. (b) C.H.B. only day before death.	N.R. A. 100; dropped beats	V.St. A. 110-50 Ectopic P.	Nodal (A-V) rhythm, then N.R. also V.T.	(V.St. 45-150 sec.)	Died in observed attack Necropsy

Norris and Landis (1938). Case I	—	—	—	—	V.St., variable P.	—	(V.St. 4 sec.)	—
Cossio (1939)	—	—	—	C.H.B.; V. 34, A. 105	V.St., A. to 120	—	(V.St. 8.5 sec.)	—
Laplace (1939)	29	M.	—	C.H.B.; V. 50, A. 135	V.St., P. slow then absent	—	—	Died in observed attack
Sigler (1939, A)	57	M.	A.P.	N.R.; R.B.B.B.	V.St., P. 100-116	2: 1 H.B., also N.R.	30-60 sec.	Attacks ceased
Scherf and Boyd (1940)	—	—	H.B. after C.T.	C.H.B.	V.St.	C.H.B.	(V.St. 90 sec.)	—
Teran (1941)	51	M.	St.Ad. 4 months. B.P. 190/100;	(a) C.H.B.; V. 15 A. 100 (b) 2: 1 H.B. R.B.B.B. (c) N.R. 66	V.St., A. 60-95. Ectopic P.	N.R. 66; P-R 0-22; R.B.B.B.	(V.St. 8-10 sec.)	—

The above Table I comprises 28 reported cases in Group I, to which our Cases 1, 2, 3, 4, 5 also belong.

TABLE II  
 GROUP II.—VENTRICULAR TACHYCARDIA FOLLOWED BY VENTRICULAR STANDSTILL  
 (a) Low ventricular tachycardia (below 160) followed by ventricular standstill.

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Gager and Pardee (1925)	59	M.	St.Ad. 2 weeks	(a) C.H.B.; V. 27-15; A. 92 (b) 2:1 H.B. (c) N.R. 60	V.T. (V. 60; A. 100) then V.St.	V.St. A. 100	—	0.5-1 min. (V.St. 6-20 sec.)	Died after 6 months during attack
Herapath (1926)	63	M.	B.P. 210	(a) C.H.B.; V. 35; Ex.s. (b) C.H.B.; QRS. of the ex.s. type (c) 2:1 H.B.; L.B.B.B.	V.T. QRS of ex.s. type, rate 62	V.St. A. rate +	—	(V.T. 40 sec. V.St. 12-15 sec.)	No further attacks
Coelho (1932)	—	—	—	—	V.T.	V.St.	—	—	—
(b) High ventricular tachycardia (200-500), usually also with ventricular fibrillation, followed by ventricular standstill.									
Kerr and Bender (1922)	68	M.	H.F. 4 years	C.H.B.; A.F.; R.B.B.B. V. 30-36	V.T. 180-220	V.F. then V.St.	—	1-2 min.	Recovery from attack.
Hesslin (1925)	31	F.	Slow pulse	C.H.B.; V. 32-34; Ex.s.	—	V.F. then V.St. P. modified during V.St.	V.T. 132-134; of each group QRS has different shape	—	—
Levine and Matton (1926)	52	F.	—	(a) C.H.B.; R.B.B.B. (b) 3 months later, inversion of T <sub>2</sub> and T <sub>3</sub>	Variable Ex.s.	V.T. (? V.F.) 240; V.St. A. 45	V.T. 140; after 7 min. 70	V.F. 200 sec. V.St. 80 sec.	Attack ceased.
Freundlich (1932)	69	F.	B.P. 250/70; St.Ad. one day, preceded by angina	C.H.B.; V. 48; Ex.s.	Multiple, variable Ex.s. V.T. at increasing rate	Irreg. V.T. (? V.F.) 280; V.St.	T. inverted in all leads; coronary R-T	5 min. (V.F. 80 sec.; V.St. 7.5 sec.)	Attacks ceased



Lian and Deparis (1934)	52	F.	St.Ad. 2 years; B.P. 220/90	C.H.B.; V. 35, A. 78; partial B.B.B.	—	V.F. 300; V.T. 200; then basic complexes. Once V.St. A. to 120	C.H.B.	1-1.5 min. (V.St. 12 sec.)	Died during attack
Clerc and Lévy (1936) Case 2	75	F.	St.Ad. 9 years	(a) C.H.B.; V. 32; (b) N.R., P-R Sl. +, R.B.B.B	—	V.F. (280) then V.St.	V.T. 200 for 7 min., then C.H.B.	6 min. (V.St., 60 sec.)	Alive after 10 months; few attacks
Jezer and Others (1936) Case 1	68	M.	St.Ad. 5 months; H.F.	C.H.B.; 30	V.T. 60-120	V.St., A. up to 165, then V.T.	—	Hours: (V.St. 90-180 sec.)	Died after few hours of V.St. and V.T.
Jezer and Others (1936) Case 2	39	M.	St.Ad. 18 months; B.P. 175/85	C.H.B.; A. 70, V. 25.	V.T. 120 then C.H.B. 25, then V.St.	V.St.	V.T. 75 to 120, then V.St. again	(V.St. 30-60 sec.)	Died in observed attack
Spühler (1936)	51	F.	St.Ad. 4 months; A.P. 16 months	(a) C.H.B.; B.B.B.; (b) 2:1 H.B.; B.B.B.	C.H.B., B.B.B., multiform Ex.s.; A. 125	V.T. (270). V.St., A. 40, irreg.	V.T. (130); then C.H.B.; V55, A. 170; with normal QRS	2-8 min. (V.T. 115 sec.; V.St. 8 sec.)	—
Froment and Gomin (1938, A)	60	M.	St.Ad. 10 years; H.F.	C.H.B.; V. 13, A. 35; L.B.B.B.	V.T. 60.	V.St. P. later disappears. Occasional V.T. 300	V.T. 40-48-28, then V.St.; 4 hours alternate V.St. and V.T.	(V.St. 30 sec.)	Died in V.F.
Gertz and Others (1938)	59	F.	St.Ad. years	(a) N.R. (b) 2:1 H.B.	C.H.B. Ex.s.	V.T. 250 and V.F.; V.St.	C.H.B.	(V.F. 83 sec. V.St. 50 sec.)	Died in observed attack
Norris and Landis (1938). Case 2	—	—	—	C.H.B.	—	V.T. 160, V.St. (A. 100); V.F. 150-200	—	20 min.	Died after two days
Soulié (1938)	68	F.	St.Ad. 2 years, following C.T.	2:1 H.B.; L.B.B.B.	2:1 H.B.; C.H.B.	V.St. absent P. V.T. for 600-800 sec. then again V.St.	Multiform Ex.s. C.H.B. with aur. flutter	(V.St. 70-130 sec.)	Died in observed attack

The above Table II, comprises (a) 3 reported cases in Group II (a), to which our own Case 6 also belongs; and (b) 13 reported cases in Group II (b), to which our own Case 7 also belongs.

TABLE III  
 GROUP III.—HIGH VENTRICULAR TACHYCARDIA, OR VENTRICULAR FIBRILLATION, OR BOTH. NO VENTRICULAR STANDSTILL

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Gaillard (1923)	62	M.	St.Ad.; chronic nephr., B.P. +	C.H.B. with A.F. (V. 40)	—	V.T. (200)	V.T. at low rate	—	Died after 1 month
Gallavardin and Bernard (1924)	55	F.	St.Ad. 4 years	C.H.B.; V. 35-42, A. 85	—	V.F. (336)	C.H.B.	2-5 min.	—
Heimann (1929) Case 3	46	F.	H.F., B.P. +	Partial H.B.	—	V.F. (? V.T.) 200	—	10 sec.	Died during attack
Gallavardin and Froment (1931)	39	M.	Subacute bact. endocarditis; St.Ad. 5 days before death	C.H.B. (40)	N.R. then 3:1, 2:1 H.B., partial B.B.B., then V.T.	V.T.	—	—	Died after 5 days
Schwartz and Jezer (1932)	65	F.	St.A. 5 months; H.F.	C.H.B.; V. 28, A. 66; QRS. normal or B.B.B.	Multiple Ex.s.	V.T. and V.F., rate 250-500	Low V.T. to 90, then basic rhythm	10-180 sec.	—
Bizzozero (1934)	60	F.	Diabetes; St.Ad. 1 month. H.F.	C.H.B.; V. 40, A. 110; partial B.B.B.	—	V.F. (and V.T.) 230	—	—	—
Schwartz and Hauswirth (1934)	56	F.	St.Ad. 6 months; B.P. 230/120	(a) Sinus tachyc., normal P-R, R.B.B.B. (b) 3:2 H.B. (c) C.H.B.	Multiple ex.s.	V.T. and V.F. 250-300	—	Several min.	Died after 1 year with syncope
Kahall (1935)	68	M.	St.Ad. 3 days; B.P. 200/100; P. 34-40	—	C.H.B. multiple ex.s.	V.T.; V.F.; or V.T. only	C.H.B., slow ventr. rate	20-120 sec.	Died after 6 weeks in attack.

Sigler (1938)	58	M.	St.Ad. months; A.P.	Normal P-R, 3:2 H.B.	As between attacks; runs of Ex.s.	V.T. 220-250; V.F. 370-195	C.H.B.; after 300 sec. V.T. 130	1-2 min.	Attacks ceased
Turrey and Leammann (1929)	—	—	—	C.H.B.	Multiform Ex.s. in runs	V.T. (240), V.F., V.T. with occas. V.St. and absent P.	V.T., C.H.B., Ex.s.	(V.F. 42 sec.)	—
Sigler (1939, B)	66	F.	Diabetes, C.T.; 6 months later St.Ad.	C.H.B.	—	V.T. (250), V.F. (370), V.T.	—	5 hours inter- mittently	—
Fishberg (1940)	—	—	—	C.H.B.	C.H.B., multi- form. Ex.s.	V.T. 200-300, then V.F.	—	(V.F. 19 sec.)	—

The above Table III comprises 12 reported cases in Group III, to which our own Case 8 also belongs.

*Group III.—High ventricular tachycardia, or ventricular fibrillation, or both, without ventricular standstill.*

12 reported cases and our Case 8. (Table III).

*Group IV.—Extreme bradycardia with complete heart block.*

No table is given for this group because it is small and less important. Many early reports tell of extreme slowness of the pulse as the sufficient cause of the faint, yet electrocardiograms of such cases are hard to find. That of Gilchrist (1934) is an example, yet that was the result of stoppage of ephedrine therapy. The case of Dubbs (1938) is a clear example though it is surprising that the ventricular rate was not lower than 20.

#### AUTHORS' CASE REPORTS

*Case 1, male, aged 54.*

*History.*—Three years ago and six months ago he lost consciousness. Recently the attacks became frequent and he was admitted to hospital, where he died four days later during an attack. There was no history of rheumatic fever, chorea, or diphtheria.

*Examination.*—The pulse was regular and 30 a minute, but next day it fell to 16, persisting at this rate between the attacks until he died. There was no clinical evidence of cardiac enlargement and there were no murmurs. Signs of heart failure, including distension of the liver and œdema of the ankles, appeared on the third day in hospital. The blood pressure (B.P.) was 120/65. The Wassermann reaction (W.R.) was negative.

*Course.*—He was seen in several Stokes-Adams attacks which occurred about every hour during the last two days of life. A momentary feeling of faintness preceded unconsciousness, then the respiratory movements became exaggerated and profuse sweating accompanied a great pallor. The pupils dilated; the upper limbs twitched. With the onset of unconsciousness the pulse stopped and the ventricles ceased to beat (auscultation)—for 25 seconds in one attack. When the pulse returned, it was regular at 16 and consciousness was quickly regained. Occasionally the heart sounds were absent for periods over 10 seconds without complete loss of consciousness. Vomiting commonly followed the attacks, but there was no incontinence of urine.

On one occasion, adrenalin (3 minims of 1 in 1000 solution intravenously) quickened the pulse to 72 within one minute, but two minutes later it was 32, and 30 minutes later, 16. On another occasion, two successive injections (5 minims subcutaneously) had no effect on the pulse rate.

#### *Electrocardiographic features*

(a) *Between* the attacks there was complete heart block (C.H.B.) with a regular ventricular rate of 33 to 25 and an auricular rate of 100. The ventricular complexes were of right bundle branch block (B.B.B.) type (QRS, 0.16 sec.) with deep inversion of T<sub>2</sub> and T<sub>3</sub>.

(b) *During* the attack (Fig. 3) ventricular standstill was recorded for 3.8 seconds with an increased auricular rate of 150.



FIG. 3.—Case 1. Stokes-Adams attack. Ventricular standstill. Complete heart block.

*Case 2, male, aged 76.*

*History.*—Some heart affection had been recognized for years. Fainting attacks, some with convulsions, occurred for the first time on the day before admission to hospital.

*Examination.*—The pulse was regular, owing partly to extrasystoles; the rate varied between 30 and 72. The heart sounds were normal, and on X-ray examination the heart was of normal size and the aorta was unfolded. There was no evidence of heart failure, nor of any other than cardiovascular disease. B.P., 180/80.

*Course.*—In hospital the attacks were at first frequent, sometimes 3–4 a day and each lasting about 30 seconds. He lost consciousness suddenly and his breathing became stertorous; no pulse could be felt and no heart sounds could be heard. He had oral treatment with ephedrine and thyroid, and injection treatment with adrenalin. Gradually he improved and there were no attacks during the last weeks in hospital. He became quite well, and when last seen, four years later, he was still free from attacks.

#### *Electrocardiographic features*

(a) *Between* the attacks there was at first partial or complete heart block with a ventricular rate of 50–75, apart from multiform extrasystoles. The basic complexes were of supraventricular type (QRS, 0.07 sec.). Fig. 4 was taken at this period. Three days after admission, sinus rhythm returned with a P–R interval of 0.3 sec. The successive records taken in hospital and a fortnight after his discharge were similar. Five months after his discharge, partial heart block was recorded with an irregular ventricular rate of 50–63 and a regular auricular rate of 80. Sinus rhythm with prolonged P–R interval returned one month later, and the last record taken two and a half years later was similar, with only a widened ventricular complex (QRS, 0.12 sec.) of partial bundle branch block appearance.

(b) *During* the attack (Fig. 4) there was ventricular standstill recorded during 4 seconds, during which the auricular rate increased from 66 to 86. At the end of the standstill there was partial heart block.

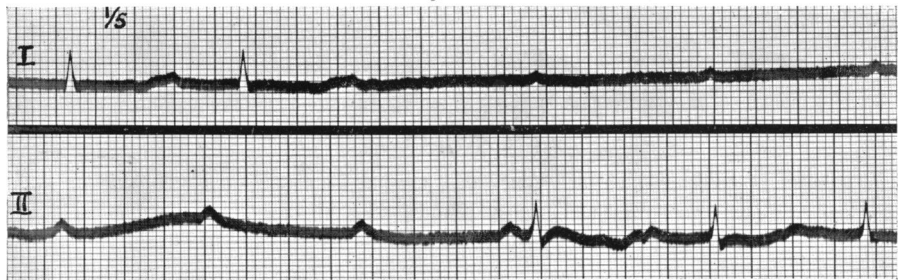


FIG. 4.—Case 2. Stokes-Adams attack. Ventricular standstill. Partial heart block.

*Case 3, male, aged 68.*

*History.*—For years had been treated for high blood pressure. For one year, recurrent attacks of anginal pain. A few days before examination had attacks of giddiness, sometimes followed by loss of consciousness. The day before examination the syncopal attacks became very frequent.

*Examination.*—The blood pressure was raised; there were no signs of failure. While being examined, he fainted repeatedly. The colour of the face alternated between pallor and flushing, the breathing became irregular and sometimes ceased for seconds. The slow, regular pulse of about 50 stopped suddenly for some seconds, the heart sounds were inaudible and the patient fell back unconscious. Even when the pulse returned, he was mentally clouded the whole day. He became worse, and died a few days later.

*Electrocardiographic features.*

(a) *Between* the attacks there was complete heart block with a ventricular rate of 57, and an auricular rate of 80, both regular. The basic ventricular complexes were supra-ventricular (QRS, 0·08 sec.) with an inverted T<sub>1</sub>.

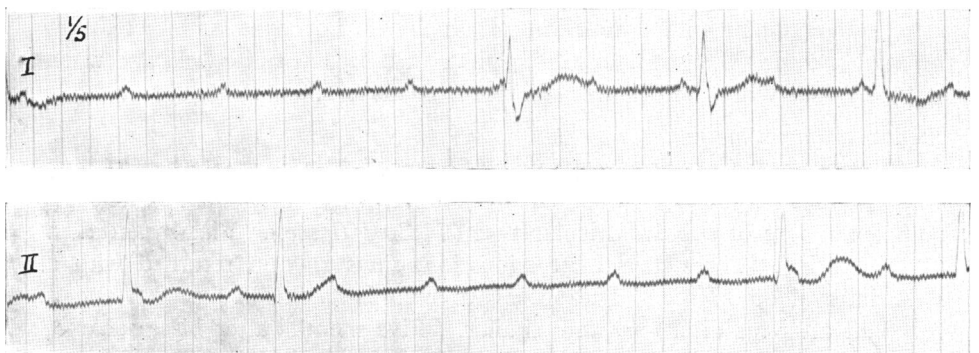


FIG. 5.—Case 3. Stokes-Adams attack. Ventricular standstill. Complete heart block.

(b) *During* the attacks (Fig. 5) there was ventricular standstill recorded during 4 and 3·6 seconds respectively. The first ended with an ectopic ventricular complex followed after 1·4 seconds by a similar one. The second attack ended with two basic ventricular complexes at a rate of 46 a minute, but from the third complex onwards the rate was again 57. At the end of the first and longer standstill the auricular rate slightly increased to 92, and the original rate

of 80 was regained only 5 seconds after the appearance of the basic ventricular complex. The second and shorter ventricular standstill left the original auricular rate unaltered.

*Case 4, male, aged 43.*

*History.* The first attack of unconsciousness happened 14 months ago. No attacks for 4 months, then they became frequent. He was admitted to hospital for observation as epilepsy was suspected. There was no history of acute rheumatism, chorea, or diphtheria.

*Examination.*—The pulse was regular, 70 a minute. The heart was not enlarged and there were no murmurs; other systems showed no abnormal signs. B.P., 140/80; W.R., negative. During a stay of 38 days in hospital no fewer than 590 attacks were noted. In many he remained fully conscious and complained of a burning sensation over the stomach which spread upwards to the chest, to the neck and face, and sometimes along the arms to the fingers and down the legs to the toes. During such paroxysms he expected to faint, but only turned pale and breathed rapidly. In some of them the pulse stopped and the ventricles ceased beating (auscultation) for periods of 5 seconds or more, jugular pulsation (auricular contraction) continuing. This ventricular standstill was often recorded by electrocardiograph. In almost as many attacks consciousness was lost, and then ventricular standstill lasted 10 to 20 seconds, the patient becoming unconscious towards the middle of the period, the breathing stertorous, and the face muscles twitching.

*Course.*—The attacks gradually became less frequent, and a month after discharge from hospital they ceased. Later he reported none for three years, and was able to do light work.

*Electrocardiographic features.* In all, 15 electrocardiograms were taken and they showed that the block was paroxysmal.

(a) *Between* the attacks there was most often sinus rhythm at a rate of 85, with a normal P-R interval and normal ventricular complexes (Fig. 6A). On

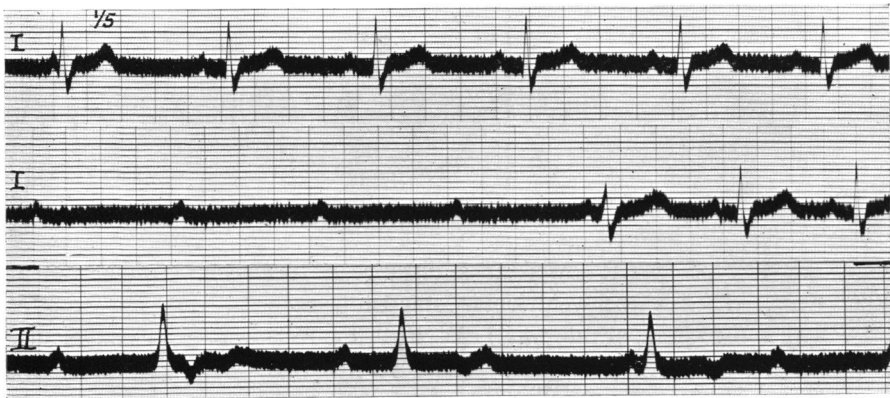


FIG. 6.—Case 4. Stokes-Adams attack. Paroxysmal heart block. (A) Sinus rhythm, normal P-R. (B) Ventricular standstill ending with sinus rhythm. (C) Complete heart block. (B) was taken immediately after (A); (C) on another day.

other occasions complete heart block was recorded with a ventricular rate of 55 and an auricular rate of 90 (Fig. 6C).

(b) *During* the attack there was ventricular standstill recorded during 3 seconds. It began abruptly during sinus rhythm with a delayed auricular beat not followed by the ventricular complex (not shown), and ended with an ectopic beat 0·10 second after P, when the sinus rhythm continued at the usual rate of 85 (Fig. 6B). During the standstill the auricular rate increased from 66 to 75.

*Case 5, male, aged 70.*

*History.*—No complaints until two and a half years before, when he suddenly fell down unconscious in the street. Occasional short faints followed at varying intervals, and for one week as frequently as every day. The day before admission, the attack was more severe; he became extremely pale, and unconscious, and seemed about to die. For months he had noticed dyspnoea and palpitation.

*Examination.*—He was pale, and every few minutes fainted. The pulse was irregular, about 24 a minute; there was slight enlargement of the left ventricle, and moderate uniform widening of the aortic shadow on radioscopy. The heart sounds were normal; there was no evidence of failure. B.P., 190/75; W.R., negative. The urine contained a trace of albumin.

*Course.*—Adrenalin (5 minims subcutaneously) did not increase the ventricular rate and did not prevent the recurrence of attacks. During the following 5 months he fainted about once a week, except that on one particular day he had twelve faints. The longest period of freedom was ten days.

*Electrocardiographic features.*—Polygrams showed complete heart block with frequent pauses of the radial pulse, the longest one recorded being of 8 seconds duration.

(a) *Between* attacks, the electrocardiogram showed complete heart block with a regular auricle at 86, and a regular ventricle at 33. The ventricular complexes were of small voltage and of left bundle branch block type (Fig. 7).

(b) *During* the attack (Fig. 7), the basic ventricular complex of left bundle

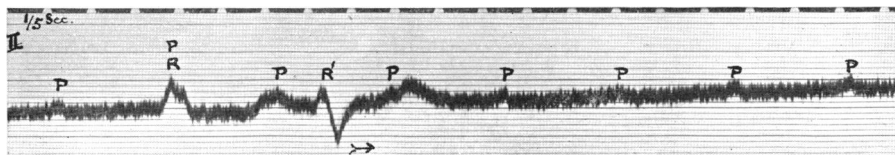


FIG. 7.—Case 5. Stokes-Adams attack. Ventricular standstill. Complete heart block.

branch block type was followed at a distance of 0·7 sec. by a ventricular extrasystole. Then came ventricular standstill, recorded for 2·6 seconds during which the auricular rate was 110. With the reappearance of ventricular action the auricular rate slightly decreased to 100. Ectopic ventricular complexes recurred at irregular intervals.

At the end of another attack (not shown), ventricular extrasystoles were followed after 2 seconds by the returning basic complex. The ventricular rhythm



later increased from 35 to 46 and then changed into a regular ventricular tachycardia at a rate of 60 with widened ventricular complexes of another shape.

*Case 6, male, aged 40.*

*History.*—Nine years dyspnoea, more severe for two years, appearing even on slight effort and compelling him to relinquish heavy work. Seven years, momentary faints (loss of consciousness) about once a month, ascribed to heavy meals or to effort. Ten months ago he had the first severe syncope; its duration was unknown, but there was twitching of face and hands, with incontinence of fæces during the attack, and vomiting after it. Since then he has had up to 100 similar attacks a day, though sometimes he was free for as long as 9 days.

*Examination.*—The pulse was regular, 28 a minute, and the rate could not be increased by exercise. The apex-beat was forcible but not displaced, and the cardiac dullness was normal. The heart sounds were distant and a faint systolic murmur was heard at the mitral area. The liver was palpable, but there were no other signs suggesting failure. B.P., 125/60; W.R., negative.

*Course.*—While in hospital few days passed without attacks, which were of two different kinds. Some were accompanied by disappearance of the pulse, loss of consciousness, clonic contractions, and incontinence; and they lasted 1–2 minutes. The others, during which he did not lose consciousness, started with a feeling of epigastric oppression, followed by “thumps of the heart”, then by a feeling of pins and needles in the neck which extended over the head. Both types of attack might often recur indifferently over a period of several hours. Adrenalin (5 minims subcutaneously) and barium chloride (0.5 grain, t.i.d.) did not influence the length of the attacks, nor prevent their recurrence. Attacks proved more frequent during a trial course of digitalis (tinct. digitalis, 10 minims, t.i.d.) for seven days. During five months of observation little change was noted and no signs of failure developed. He died in hospital after a succession of attacks in which he complained of epigastric tightness and flatulence. Necropsy was refused.

*Electrocardiographic features*

(a) *Between* the attacks there was always complete heart block with a regular or irregular ventricle at a rate of 20–30. The auricle was fairly regular at 66–86. The basic ventricular complexes were of right bundle branch block type (Fig. 8A). Ectopic ventricular complexes were frequent and varied in shape; they appeared both as extrasystoles following the basic complex, and as regular sequences of a slow ventricular “tachycardia” at 55–75.

(b) *During* the attacks several standstills of the ventricle were recorded, the longest lasting 6.8 sec. in which the auricular rate increased from 75 to 100 (Fig. 8B). They were always preceded by a low ventricular tachycardia. Periods with extrasystoles sometimes preceded the ventricular tachycardia.

*Case 7, female, aged 62.*

*History.*—Hypertension was discovered two years before when the patient was in hospital for pulmonary infarction following phlebitis. There was no history of rheumatic fever, chorea, or diphtheria. During the six weeks preceding her present admission she had two Stokes-Adams attacks.

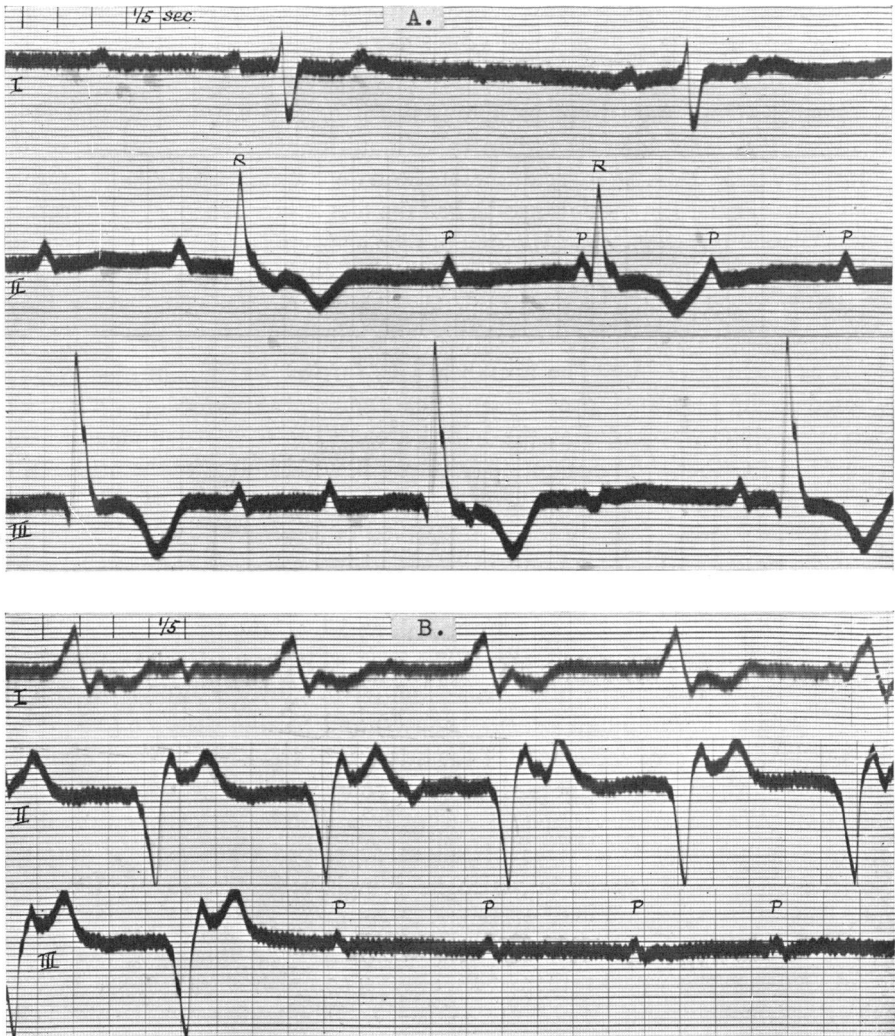


FIG. 8.—Case 6. Stokes-Adams attack. (A) Complete heart block, right bundle branch block. (B) Low ventricular tachycardia preceding ventricular standstill.

*Examination.*—She looked well and was rather stout. There were no abnormal signs except in the cardiovascular system. As a rule the pulse was regular and infrequent, 38. On radioscopy, the left ventricle was seen to be moderately enlarged. A systolic murmur was heard in both mitral and aortic areas. There was no evidence of heart failure. B.P., 205/110; W.R., negative. Many Stokes-Adams attacks were observed in hospital. On some days there were as many as five, but there might be no attacks for a week or longer.

*Clinical features of the attacks.*—Often one followed soon after a meal. The patient would complain of feeling suddenly ill and might call for a bed-pan.

Sometimes she would cry out that an attack was coming on. She then became unconscious, the breathing and the pulse stopped, and the heart ceased beating (auscultation). The cyanosed face twitched, and there were spasmodic movements of the arms and legs. The eyes turned up and the pupils dilated widely. The pulse returned after one to two minutes, when it was rapid (about 150 a minute) and usually regular, but later it would fall to about 38 a minute. The breathing, returning soon after the pulse, was stertorous and accompanied by a heaving movement of the chest. Cyanosis gradually disappeared and was usually replaced by slight pallor. After 5 to 15 minutes with returning consciousness she perspired, was exhausted and confused, uttering loud and wild shouts. Incontinence of urine often occurred during an attack.

*Electrocardiographic features*

(a) *Between* the attacks the electrocardiogram changed frequently from complete heart block to 2 : 1 or 3 : 1 heart block when, apart from the pauses, the P-R interval was normal. The ventricular complexes varied from left to right bundle branch block. The ventricular rate was often irregular owing to multiform ventricular extrasystoles. When regular, it varied between 32 and 40 and the auricular rate between 80 and 92.

(b) *During* the attack, Fig. 9 was recorded, and the sequence of events is portrayed in Fig. 10 (Chart). Immediately before the attack, the pulse was regular (38 a minute). While the camera was being loaded she lost consciousness suddenly and the breathing ceased. The pulse stopped, but after 30 seconds it returned and the electrocardiogram (Fig. 9A & B) first showed rapid ventricular tachycardia (270 a minute), and the irregular ventricular deflections of *ventricular fibrillation*. This phase lasted 30 seconds when the pulse again disappeared and Fig. 9C & D showed *cardiac standstill* for one minute, and rudimentary P waves 20.8 and 24 seconds after its onset. A subcutaneous injection of 10 minims of 1 in 1000 solution of adrenalin hydrochloride was given at the beginning of the standstill period. The pulse when it returned was regular and infrequent, and the Fig. 9E showed *complete heart block* with an auricular rate of 95 and a ventricular rate of 42. This phase lasted 12 seconds, and then the pulse rose to 135 a minute at the onset of tachycardia with changing ventricular complexes (Fig. 9F) lasting 5 minutes, the auricular rate increasing from 90 to 150 meanwhile. The breathing returned but became slow with long inspiratory and short expiratory phases. At this stage, although still unconscious and not responding to external stimuli, the patient began to utter wild and loud shouts. Fig. 9G showed complete heart block, the ventricular rate was 37 and the auricular rate was as rapid as 150 a minute. The second paroxysm of ventricular tachycardia shown on Fig. 10 (Chart) is not published as it was similar to Fig. 9F. After two minutes, during which the patient ceased to shout and recovered consciousness, the heart rhythm returned as complete heart block at 38. Altogether the patient had been unconscious for nine and a half minutes. We consider it unlikely that adrenalin had any effect upon the sequence of events just described.

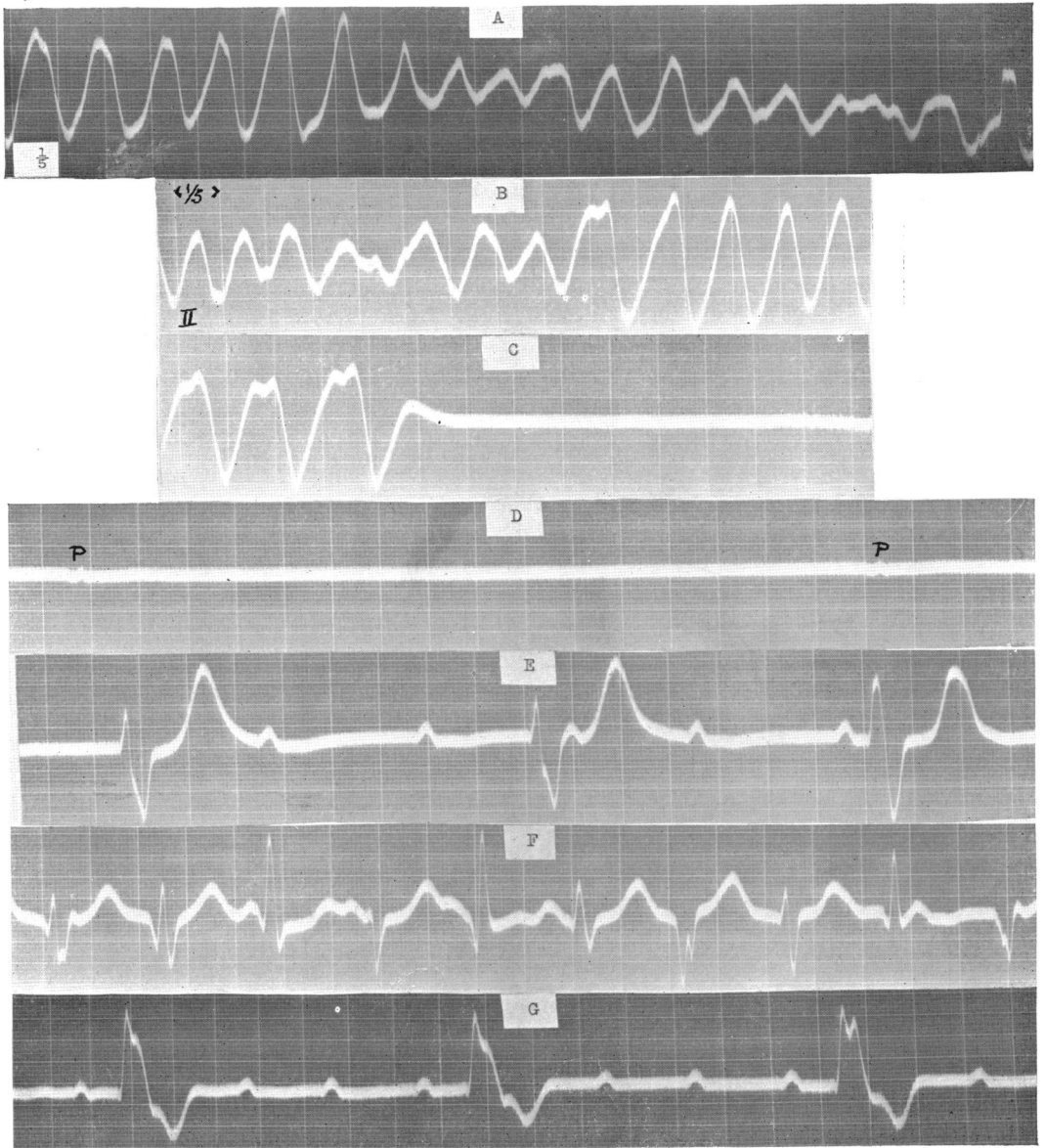


FIG. 9.—Case 7. Stokes-Adams attack. (A) and (B) High ventricular tachycardia and ventricular fibrillation. (C) End of ventricular tachycardia and onset of complete cardiac standstill. (D) Cardiac standstill with two ectopic P waves. (E) Complete heart block, right bundle branch block. (F) Tachycardia with changing ventricular complexes. (G) Complete heart block, left bundle branch block (basic complexes). All records are lead II.

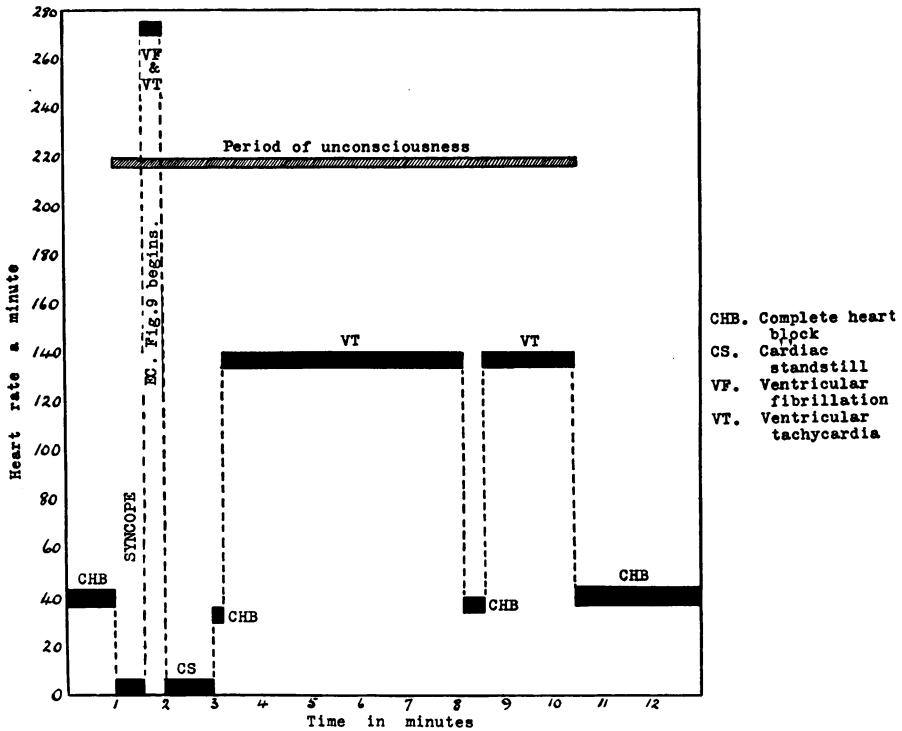


FIG. 10.—Chart of Stokes-Adams attack recorded in Fig. 9 (Case 7).

Case 8, male, aged 65.

*History.*—For seven months angina pectoris, and also dyspnœa on exertion. One week dizzy attacks while sitting. On day of admission he had a bout of dizziness, and later fell unconscious; sent to hospital.

*Examination.*—Pulse 40, irregular; no murmurs. B.P. 240/90; W.R., negative. Next day an attack was observed; he felt faint, became pulseless, his face pale, then cyanosed and unconscious, then convulsions for two minutes. Respiration ceased, then returned; he was unconscious from time to time and there were two further convulsions during the next hour. He died one and a half hours after the onset of the attack, during which numerous electrocardiograms were taken.

*Necropsy* showed a moderate degree of coronary sclerosis with calcification, more of the left than of the right coronary artery. No evidence of myocardial infarction could be found, although there was slight patchy fibrosis.

*Electrocardiographic features* (Fig. 11)

(a) *Between the attacks* there was complete heart block, with an irregular basic ventricular rhythm at 25–32. The auricular rhythm was regular, rate 100. Nearly all the basic complexes were followed at an interval of 0.52 sec. by a standard form of extrasystole with the main deflection downward. This was often followed by one or more different extrasystoles, and then the succeeding basic complex was of course delayed; hence the irregularity of the basic ventricular rhythm.

(b) *During* the attack, ventricular tachycardia lasted 6 seconds, and comprised in order : 6 downwards complexes (rate 200 approx.), 2 diphasic complexes, 6 upright complexes (rate 200–210), and 4 downward complexes (rate 200–120) approximating in form to the standard extrasystole. The pause after the tachycardia was no longer than the usual pause after an extrasystole, being about 1·2 seconds. This was confirmed by a short record of the end of another



FIG. 11.—Case 8. Stokes-Adams attack. Paroxysm of high ventricular tachycardia (a continuous record has been divided). Complete heart block.

attack. Following the attack of ventricular tachycardia the electrocardiogram was much the same as that preceding it.

The above analysis applies to Fig. 11, taken while the patient was unconscious, and numerous other records were taken, some on continuous strips; they differed only in the duration of the high ventricular tachycardia and were never followed by ventricular standstill. Ventricular fibrillation appeared only in one record taken shortly before death.

#### DISCUSSION

##### *Ventricular standstill*

The attack was preceded in six of the reported cases by an acceleration of the auricular rate, but in many others and in our own case no sufficient record of the phase preceding the attack was available for reckoning. Hay (1921) called attention to “the gradual quickening of the rate of the auricle previous to

ventricular stoppage", and the matter is chiefly discussed by Wenckebach and Winterberg (1927), who found it in all three of their recorded cases, and by Condorelli (1932). As the auricular rate is so closely under nervous control, one might imagine that an increase of the auricular rate from exertion or emotion would often determine attacks. In Case 3 of Wenckebach and Winterberg, the emotion of an arithmetical exercise was observed to quicken the auricles and to cause ventricular standstill; but any such direct relation between exercise and emotion and the attacks must be rare. With the exception of that case, the rise in auricular rate in recorded cases has been spontaneous, and it originates in the auricle itself, probably in the sinus node. An increased auricular rate is known to worsen conduction to the ventricle, but its influence in the production of the standstill is unknown.

During ventricular standstill of short duration (20 seconds or less) auricular activity continues undisturbed or more often at a somewhat higher rate (Cases 2, 3, 4, 5, and 6). The presence of the P waves during ventricular standstill is an important distinction from a common faint, where with slowing there is a total cardiac standstill (Fig. 1). In the longer ventricular standstill of a Stokes-Adams attack the circulatory stasis affects the auricular activity. While the increase of the auricular rate during short ventricular standstill suggests an asphyxial irritation of the sinus node, the decrease of auricular rate after longer ventricular standstill (20 seconds or more) would suggest a subsequent depression in that node. Ectopic P waves may then appear often irregularly (Hermann, Froment, Gonin, & Mahaim, 1937; Géraudel, Laignel-Lavastine, & Boquien, 1933; Heimann, 1929), or P may disappear (Yater & Willius, 1929); alternatively auricular fibrillation and flutter may supervene (Wiltshire, 1923). Irregular and changing P waves have been observed, however, in short ventricular standstill (Cowan & Ritchie, 1935; Norris & Landis, 1938).

The cardiac standstill following a high ventricular tachycardia and fibrillation (Group II (b), Case 7) is often total. The effect of the high ventricular tachycardia (V.T.) and ventricular fibrillation (V.F.) on the sinus node is comparable with that of ventricular standstill, as the cardiac circulation is practically arrested. The occasional ectopic P waves seen in Fig. 9D (Case 7), is one effect of auricular stasis, and this and other effects of it are reported by Clerc and Lévy, Case 2 (1936), by Soulié (1938), and by Froment and Gonin (1938a), all with ventricular tachycardia and fibrillation (30 seconds or more) preceding the ventricular standstill. Sometimes the auricular rate during ventricular standstill (following high V.T. and V.F.) is accelerated (Lian & Deparis, 1934; Norris & Landis, 1938), and sometimes it is reduced (Gertz, Kaplan, Kaplan, & Weinstein, 1938).

#### *Ventricular Tachycardia*

A low ventricular tachycardia may follow ventricular standstill, but these cases are classed with Group I (Case 5). In our Case 6 (Group II (a)) it preceded ventricular standstill, increasing gradually from 55 to 75. The electrocardiograms between the attacks showed multiform and variable extrasystoles and varying bundle branch block (cf. Cohn & Lewis, 1913; and

Bain, 1941). Such aberrant complexes sometimes replaced the basic ventricular complexes at the low basic rate; at other times they composed a low ventricular tachycardia, with complexes like the extrasystole that followed the basic complex in fixed coupling. The same relation has been described by Herapath (1926) and others. This low ventricular tachycardia may be perceived by the patient as palpitation, as in Case 6, where it was the clear warning of the imminence of unconsciousness due to ventricular standstill.

Whether accompanied or not by ventricular standstill, a close affinity exists between the low and the high types of ventricular tachycardia. They often appear together or follow each other as in our Case 7, and in many of the published cases (see Tables II and III). We divided Group II into (*a*) and (*b*), because low ventricular tachycardia (up to 160) does not produce unconsciousness, but *precedes* a ventricular standstill that does; whereas high ventricular tachycardia with or without ventricular fibrillation does produce unconsciousness. The high ventricular tachycardia differs little from the low in the electrocardiogram, having regard to the rate which gives the undulations a less individual character. Any resemblance to auricular flutter is quite superficial, and there seems no fundamental reason for assuming a corresponding ventricular flutter with a distinctive mechanism as did De Boer (1923), and later Dressler (1929) and Scherf and Boyd (1940).

#### *Ventricular Fibrillation*

Confusion still exists between the high ventricular tachycardia and ventricular fibrillation. Yet, though either may follow the other—and the passage may be a gradual one—the electrocardiographic features are very different. The deflections of ventricular tachycardia are practically regular, and any variation in their shape or size occurs in phases. In any attack the highest rate (200–500) is found at the onset, and towards the end the rate may fall. The rate in ventricular fibrillation may be the same, higher, or even lower, but the deflections are always irregular in time, and constantly varying in shape and size, and so they resemble an artefact rather than a natural electrocardiogram (Fig. 9 A, B).

Schwartz (1936, *a, b*) divided the “transient recurrent ventricular fibrillation” (comprising the high ventricular tachycardia and ventricular fibrillation) into a pre-fibrillatory, fibrillatory, and post-fibrillatory period. The pre-fibrillatory period is one with multiple and variable extrasystoles and slower ventricular tachycardia (the auricles accelerating), and sometimes with a step-like increase of both auricular and ventricular rates with abrupt changes from partial to complete heart block. The fibrillatory period is that “resembling” ventricular tachycardia (i.e. the high ventricular tachycardia) and the phase of varying, irregular complexes (i.e. ventricular fibrillation). The post-fibrillatory period is the ventricular standstill that may last from 1 or 2 seconds (our Group III, here regarded merely as the expected pause after a paroxysm) to 20 or even 80 seconds (our Group II (*b*)), and the subsequent low ventricular tachycardia.

The instability and variability of the widened bundle branch block com-



plexes and of the associated extrasystoles pave the way for the installation of a new rhythm, which supersedes the A-V pacemaker and rules by its higher rate (low or high ventricular tachycardia or ventricular fibrillation). It is supposed that exhaustion of the new centres causes ventricular standstill during which they may recover some activity (post-fibrillatory ventricular tachycardia) until their final suppression by the basic rhythm.

The Stokes-Adams attack due to the high ventricular tachycardia (and ventricular fibrillation), and that due to ventricular standstill can only be recognized and distinguished by electrocardiogram, because the pulse and the heart sounds are absent in both varieties, and tachycardia may precede each (cf. Groups II and III). Although loss of consciousness of more than three minutes duration points to the high ventricular tachycardia (and fibrillation), our Case 3 with frequently recurring short attacks of ventricular standstill was barely conscious for hours. A late onset of unconsciousness, say that exceeding 20 seconds after the disappearance of the pulse and the heart sounds, is the rule in ventricular tachycardia (and fibrillation) according to Schwartz (1936*a*). But unconsciousness in ventricular standstill may certainly be delayed for over 10 seconds (Cases 1 and 4); and short attacks of ventricular tachycardia (6 seconds in Case 8), if repeated, may induce long periods of unconsciousness. Again, though circulatory arrest is the most important determining factor in the duration of unconsciousness, other factors may contribute, e.g., the rapidity of onset of block and the fall in rate, the frequency of the attacks, a low blood pressure (as after cardiac infarction), and the state of the cerebral arteries. Such being the situation, it is difficult to state the duration of circulatory arrest that produces syncope or convulsions, especially now we know that Stokes-Adams attacks are not due simply to ventricular standstill, and that tachycardia as well as standstill may be in operation. Though admittedly approximate, the onset of unconsciousness in ventricular standstill is given by Mackenzie (1925) as after 10 seconds, and of convulsions as after 15 seconds; by Lewis (1925) as 3-5 and 15-20 seconds; and by Froment and Gonin (1938*b*) as 5-15 and 30-45 seconds respectively.

*The electrocardiogram of the Stokes-Adams attack under special conditions.*

The grade of heart block between the attacks, whether *partial* or *complete*, has no bearing upon the electrocardiographic features of the attack. Our Groups I, II, and III contain an almost equal number of both grades.

Among 10 cases of *paroxysmal heart block*, the attack was due to ventricular standstill alone (Group I) in 8 (Yates and Willius, 1929; Cheer and T'ang, 1932; Sachs and Traynor, 1934; Schwartz, 1936*c*; Hermann, Froment, Gonin, and Mahaim, 1937; Sigler, 1939*a*; Teran, 1941; our Case 4, Fig. 6). In one the ventricular standstill was preceded by the low ventricular tachycardia (Group II (*a*)) (Gager and Pardee, 1925), and in the remaining case it was preceded by the high ventricular tachycardia (Group II (*b*)) (Gertz, Kaplan, Kaplan, and Weinstein, 1938). Thus ventricular standstill seems to be the usual basis of the Stokes-Adams attack in paroxysmal block. Comeau (1937) has discussed paroxysmal heart block as a whole.

No actual electrocardiogram of a Stokes-Adams attack in *congenital heart block* has been found, though Fæssler (1939) has collected 8 such reported cases, and 6 had established complete heart block between the attacks. In his own case there was no block, but nodal rhythm during the unconsciousness.

The incidence of heart block in *coronary thrombosis* is given as 7.6 per cent Kerr (1937), and as 3.2 per cent by Master, Dack, and Jaffe (1938). One of Kerr's 12 cases had Stokes-Adams attacks; and 3 of the 5 cases of Master, Dack, and Jaffe were comatose or semi-comatose, though without distinctive Stokes-Adams attacks. Among 45 cases with Stokes-Adams attacks, 15 were due to posterior coronary thrombosis according to Schwartz (1936*b*). Among the cases we have collected, recent coronary thrombosis was present in 6. In 3 (Laufer, 1934; Schwartz, 1936*b*; Scherf & Boyd, 1940) the Stokes-Adams attack was due to ventricular standstill (Group I), in 2 (Freundlich, 1932; Soulié, 1938) it was due to ventricular tachycardia and fibrillation with standstill (Group II (*b*)), and in 1 (Sigler, 1939*b*) to ventricular tachycardia and fibrillation (Group III). Except in Soulié's case (with variable bundle branch block), the electrocardiogram was of the T<sub>3</sub> type. Although no statistical conclusions can be drawn from 6 cases, it is evident that Stokes-Adams attacks in coronary thrombosis may be due to ventricular standstill, to ventricular tachycardia and fibrillation, or to these combined, and that all are not due to ventricular fibrillation.

### *Prognosis*

The nature of the electrocardiogram during attacks has a great influence upon the prognosis, and this is evident from the 35 cases that were followed up; 15 belong to Group I (ventricular standstill alone) and 20 to Groups II and III (low or high ventricular tachycardia and fibrillation, followed or not by ventricular standstill). Among the 15 of Group I, 5 died during the observed attack and 7 were alive and attack free over a period of from three months to four and a half years; in 3 death was due to other than heart conditions (cerebral thrombosis, peritonitis, and spinal tumour). Among the 20 of Groups II and III, 16 died in an attack (11 during the observed attack and 5 within a year of discharge from hospital), and only 4 were alive at the end of three to ten months. It follows that patients showing ventricular standstill alone have a fair chance of recovery even after severe Stokes-Adams attacks, but that when the attacks include the low or high ventricular tachycardia (and fibrillation) the immediate prospect of recovery is far worse, and even if they recover they seldom survive for more than a year.

### SUMMARY

Stokes-Adams disease is a name applicable to patients with heart block who suffer from recurrent attacks of loss of consciousness due to ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these.

During a Stokes-Adams attack from ventricular standstill the auricle con-

tinues to beat, whereas in cardiac syncope of other types there is as a rule total cardiac standstill.

Cardiac syncope of neurogenic origin (e.g. ordinary fainting, and ventricular standstill from disease affecting the vagus or carotid sinus) and cardiac syncope of myocardial origin without heart block (e.g. in nodal bradycardia and in paroxysmal ventricular tachycardia) are excluded by this definition, though there are borderline cases.

(2) The cardiac mechanism of Stokes-Adams attacks was studied on electrocardiograms recorded during the period of unconsciousness, in 8 of our own cases and in 56 reported cases. These fall into four groups or types and are tabulated according to the electrocardiographic basis of the attack.

*Group I* (28 reported cases and 5 of our own) includes those with *ventricular standstill alone* (Table I).

*Group II* (16 reported cases and 2 of our own) includes both those with (a) *low ventricular tachycardia* and (b) *high ventricular tachycardia and fibrillation, when either is followed by ventricular standstill* (Table II).

*Group III* (12 reported cases and 1 of our own) includes those with the *high ventricular tachycardia and fibrillation* without ventricular standstill (Table III).

*Group IV* includes those rare cases with *extreme bradycardia in heart block* (no Table).

It is evident that ventricular standstill alone is not the only cardiac lapse that determines a Stokes-Adams attack. It is often due to ill action, not to inaction of the ventricle. Ventricular standstill is responsible for about 55 per cent; ventricular tachycardia (with or without ventricular fibrillation), followed by ventricular standstill, for 25 per cent; and ventricular tachycardia without ventricular standstill for 20 per cent.

(3) *Ventricular standstill* is sometimes consecutive to a rise in the auricular rate. As exertion or emotion so seldom determines ventricular standstill, this increase in the auricular rate probably originates locally in the auricle and not from any nervous influence.

During a short ventricular standstill (below 20 seconds), the auricle beats regularly, often at an increasing rate, and the persistence of P waves during the ventricular standstill is a feature distinguishing it from the total standstill of cardiac syncope. During a long ventricular standstill (above 20 seconds), or when ventricular standstill is preceded by high ventricular tachycardia and fibrillation (Group II), the auricle may show slower, irregular, and ectopic P waves, auricular fibrillation and flutter, or it may even stop.

(4) In the group with *ventricular tachycardia*, multiple and variable extrasystoles and varying bundle branch block complexes between the attacks are common, as might be expected. Low ventricular tachycardia (up to 160) does not produce unconsciousness, but it provokes the subsequent ventricular standstill that does produce it (Group II (a)). High ventricular tachycardia and fibrillation (200–500) produce unconsciousness (Group III), and this may be prolonged by the subsequent ventricular standstill (Group II (b)). The electrocardiogram of ventricular tachycardia is composed of regular deflections like bundle branch block, which at high rates merge into simple undulations;

yet the term "ventricular flutter" need not be used, for the resemblance to auricular flutter is superficial.

(5) *Ventricular fibrillation* is distinguished from ventricular tachycardia by its irregularity both in rate and in form, though the rate per minute may be the same, higher, or even lower. High ventricular tachycardia easily passes into fibrillation, which ends with ventricular standstill or with gradual resumption of the basic rhythm through a period of low ventricular tachycardia or of varying extrasystolic complexes.

(6) The essential basis of an attack can only be decided by electrocardiogram. The prolongation of unconsciousness or its late onset in ventricular tachycardia and fibrillation cannot distinguish this group from that of ventricular standstill alone, because other factors may influence unconsciousness, e.g., the suddenness of the development of block or of the fall in rate, a rapid succession of attacks, and the state of the cerebral arteries. For similar reasons it is difficult to state exactly what must be the duration of the circulatory arrest to produce syncope or convulsions.

(7) In established complete heart block or in partial heart block the Stokes-Adams attack may belong to any group, whereas in paroxysmal heart block it is generally in Group I, i.e., ventricular standstill alone. After coronary thrombosis, attacks due to ventricular standstill alone may occur, as well as those due to ventricular tachycardia and fibrillation.

(8) No prognostic significance can be attached to the grade of heart block, partial or complete, obtaining between the attacks, but the electrocardiographic nature of the attack has great significance in prognosis—and doubtless in treatment, though this is not here considered. Patients with ventricular standstill (Group I) have a fair chance of recovery and often survive for many years, whereas those with ventricular tachycardia and fibrillation (Groups II and III) seldom recover and then rarely survive for more than a year.

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