calcium may stop P.A.T.B. This can be achieved experimentally by sodium ethylenediamine tetra-acetic acid, which is a chelating agent with the property of binding calcium *in vivo* (Popovici *et al.*, 1950). Treatment of digitalis toxicity by this means has been employed by Gubner and Kallmann (1957). In our experience, however, stopping the digitalis preparation and diuretics, supplementing potassium, and preventing further potassium loss have always proved successful.

Summary

Although digitalis has been in use for over a century and a half and its toxic effects on the ventricles are well documented, its importance in the production of atrial arrhythmias has only recently been stressed. Of these, the commonest atrial disorder is P.A.T.B.

The potential seriousness of this arrhythmia when induced by digitalis is emphasized. Conditions which render the production of the arrhythmia likely, even with doses of digitalis that are small and have been unchanged for long periods, are discussed. The most important of these is a low myocardial potassium, the commonest cause of which is the use of diuretics.

We believe that P.A.T.B. is not a fixed specific entity, and that it merges into atrial flutter, from which at times it may be indistinguishable electrocardiographically or by any other means. Infinitely more important than trying to distinguish atrial flutter from P.A.T.B. is to determine whether digitalis is responsible for the arrhythmia. The problem of distinguishing P.A.T.B. due to digitalis from that arising without digitalis is discussed, and a regime is outlined by means of which it may be possible to differentiate them without submitting the patient to the risks of the acetyl-strophanthidin test.

The differential diagnosis is described in detail. We find the prognosis to be less serious than that usually stated; this is dependent upon the early recognition of the role of digitalis in precipitating the arrhythmia and stopping the drug immediately. Often no other treatment is necessary.

It has been claimed that serious myocardial disease with congestive heart failure is almost invariably present, but this was not so in our series; neither could we confirm the importance of fresh cardiac infarction as a precipitating factor. Details are given of a patient in whom no heart disease was present and yet in whom P.A.T.B. was induced by digitalis in the presence of a lowered serum potassium.

Although depression of the S-T segment was found in all patients, and ventricular premature beats in one-half of them, we wish to emphasize the uncommonness of other symptoms or signs of digitalis toxicity during the arrhythmia. Their absence cannot be taken as evidence that digitalis is not responsible for the P.A.T.B. Estimation of the serum potassium may also be misleading, as it does not necessarily reflect the myocardial potassium, and the administration of potassium, therefore, should not be withheld because the serum estimation is normal.

Addendum.—Since this paper was submitted for publication we have had two further cases, thus emphasizing the increasing frequency of this arrhythmia. (1) A man aged 85 was admitted in congestive heart failure of ischaemic origin and in sinus rhythm. Digoxin, 0.25 mg. b.d., with mersalyl, 2 ml. I.M. twice a week, was given and sodium restriction maintained. One week later P.A.T.B. developed. Digoxin and mersalyl were discontinued and potassium chloride, 8 g. daily, was given by mouth. Four days later sinus rhythm was restored. (2) A woman aged 70 was admitted in gross

congestive heart failure with P.A.T.B. She had received digitalis folia, $\frac{1}{2}$ gr. (32 mg.) t.d.s., for 18 months, with mercaptopurine, 2 ml. twice weekly, and her sodium intake had been restricted. On her admission the serum potassium was 3.4 mEq/l. and the blood urea 40 mg./100 ml. Digitalis was reduced to $\frac{1}{2}$ gr. (32 mg.) b.d., and she was given potassium chloride, 1 g. t.d.s. by mouth. Atrial fibrillation developed and has remained.

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ATRIAL TACHYCARDIA WITH ATRIOVENTRICULAR BLOCK DUE TO DIGITALIS POISONING

BY

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Digitalis can cause most types of cardiac arrhythmia. Ectopic beats (commonly ventricular), ventricular tachycardia, ventricular fibrillation, and heart-block are well-recognized manifestations of digitalis intoxication. It is not yet widely appreciated that atrial arrhythmias of various kinds may be caused by digitalis, and that one variety, atrial tachycardia with atrioventricular (A.V.) block, is almost always due to digitalis poisoning. This arrhythmia, which was first recognized as a complication of digitalis treatment by Sir James Mackenzie (1910), has received scant attention in this country, although several papers have been published about it in the United States during recent years (Lown and Levine, 1954, 1958; Goldberg, Bristow, Parker, and Ritzmann, 1958; Lown, Wyatt, and Levine, 1960).

The diagnosis of atrial tachycardia with atrioventricular block can be made only from the electrocardiogram. Clinically, its presence may be suspected

in a patient receiving digitalis who develops an increase in ventricular rate, usually regular, or whose initially high ventricular rate does not fall in the expected fashion. The atrial arrhythmia which it most resembles is atrial flutter, and the response to carotid sinus pressure is similar. The principal electrocardiographic criteria for the diagnosis were described by Lown and Levine (1954). These are an atrial tachycardia of

not more than 250 per minute, a variable degree of A.V. block, and small upright P waves, particularly in lead I and lead II (Table I). Fig. 1 shows a typical example of this arrhythmia.

This paper reports 22 incidents which occurred in 19 patients of atrial tachycardia with A.V. block.

Results

Of the 19 patients with atrial tachycardia and A.V. block, seven had ischaemic, four had hypertensive, four had rheumatic, three had pulmonary, and one had congenital heart disease. Thirteen of these patients were over 50 years of age. Sixteen had congestive cardiac failure. Other details are given in Table II. Cases 1, 5, and 13 each had two separate episodes of atrial tachycardia.

Relationship to Digitalis.—Only one of these 19 patients had not received digitalis; he was recovering from a recent myocardial infarct. Before digitalis was started, 13 had sinus rhythm and two had atrial fibrillation;

of the three remaining patients, two were thought to have atrial fibrillation, and one sinus rhythm, but electrocardiograms were not recorded. The smallest quantity of digitalis taken before the atrial tachycardia was recognized was 2.25 mg. of "digoxin" orally over three days. Two other patients had received less than 5 mg. of digoxin over the preceding four days. The rest had had digitalis in conventional maintenance doses for periods of up to 22 months. In two patients intravenous digoxin had been given shortly before the arrhythmia in addition to maintenance doses by mouth.

Relationship to Diuretics.—Before the onset of the atrial tachycardia, seven patients had received mersalyl, 10

TABLE I.—E.C.G. Features of Atrial Flutter and of Atrial Tachycardia with A.V. Block

	Atrial Flutter	Atrial Tachycardia with A.V. Block
Atrial rate	250-350	120-250, usually 150-200
A.V. block	Usually 2:1, 3:1, 4:1, or variable	All grades to A.V. dissociation
P wave	"Saw-tooth" in leads II and III	Upright and small in lead II
Isoelectric interval between P waves	Absent in lead II	Present in all leads
P-P interval .. .	Unvarying	May be variable
Carotid sinus stimulation	Increase of A.V. block	Increase of A.V. block
Response to potassium	Does not restore sinus rhythm	Restores sinus rhythm if digitalis-induced

FIG. 1.—A typical example of digitalis-induced paroxysmal atrial tachycardia with 2:1 A.V. block. The response of this arrhythmia to carotid sinus pressure is similar to that of atrial flutter, an example of which is shown for contrast.

FIG. 2.—Another example of digitalis-induced atrial tachycardia with variable A.V. block. After carotid sinus pressure, 29 atrial contractions were recorded before A.V. conduction was re-established. The oral administration of potassium chloride rapidly restored normal sinus rhythm.

TABLE II.—Details of 19 Patients with Atrial Tachycardia and Atrioventricular Block

Case No.	Preceding Rhythm	Treatment Before Tachycardia	Atrial Rate	A.V. Conduction	Subsequent Rhythm	Remarks
1	Sinus	Digitalis leaf 386 gr. (25 g.) in 27 weeks. Mersalyl 2 ml. twice weekly for 27 weeks	152	2 : 1	Sinus	Digitalis withheld when nauseated. Discharged taking digitalis. Died at home 1 month later
			136	2 : 1 and 1 : 1	"	
2	? A. fib.	Digitalis leaf 36 gr. (2.4 g.) in 3 days. Pot. iodide 1 g. thrice daily before and during A.T.	164 148	2 : 1 A.V. dissociation. Vent. rate 78	Atrial paralysis. Vent. rate 41 with periods of asystole	Died
3	Sinus. P-R 0.26 sec.	Digoxin 13 mg. over 7 days. 1 ml. mersalyl 4 days before	158	4 : 1 with vent. es. 1 : 1 with L.B.B.B.	No reversal observed	Died suddenly
4	Complete A.V. block or Wenckebach	None	213 213	A.V. dissociation. Vent. rate 71. 3 : 1	A. fib., later sinus	Recovering from acute myocardial infarction. Recovered from arrhythmia
5	A. fib.	Digoxin 8 mg. over 16 days. Mersalyl 4 ml.	160	2 : 1 and 1 : 1	Sinus	C.S.P. when in sinus rhythm produced A.V. block. Died in uraemia 4 months after second episode
	" "	Digoxin 5 mg. over 14 days	200	A.V. dissociation. Vent. rate 120	"	
6	Sinus	Digoxin 30 mg. over 9 weeks. Chlorothiazide 3 g. in 1 day. Mecamylamine	167-300 (one record)	A.V. dissociation. Vent. rate 68	No reversal observed	Digoxin continued after A.T. developed. No potassium given. Died 4 days after A.T. developed
7	"	Digitalis leaf 19.5 gr. (1.3 g.) over 13 days. Digoxin 0.5 mg. I.V. on 15th day, A.T. on 16th day. Mersalyl ? amount. Chlorothiazide 16 g. over 3 weeks. Pot. citrate 4 g. (one day)	136	1 : 1 with long P-R interval and Wenckebach periods	Sinus	Digoxin stopped and pot. citrate continued after A.T. developed. Glucose and insulin also given. Recovered
8	? A. fib.	Digoxin ? 17.5 mg. over 5 weeks	200	2 : 1 with coupled vent. es.	Sinus 2 hours after KCl	Recovered. Remained sensitive to C.S.P. 12 weeks after last digoxin given (A.V. block with ventricular escape; A.V. dissociation)
9	Sinus	Digoxin in unknown maintenance dose for 1 year. Chlorothiazide in unknown amount for 1 year	115	1 : 1 P-R 0.22 sec.	Sinus	Digoxin 0.5 mg. I.V. and 0.25 mg. t.i.d. orally after A.T. developed. Pot. chloride 8 g. orally over 2 days. Died on day sinus rhythm returned
			213	A.V. dissociation. Vent. rate 120		
			167	1 : 1		
10	"	Digoxin 8.25 mg. over 15 days. Mersalyl 14 ml. over 14 days	230	2 : 1 multiple vent. es.	"	Mersalyl-resistant oedema prior to A.T. No further episode despite continued digoxin, chlorothiazide, and prednisolone
			136	A.V. dissociation, multiple vent. es.		
11	"	Digoxin 2.25 mg. over 3 days. Hydroflumethiazide 600 mg. over 2 weeks	125	Wenckebach periods	Sinus with long P-R interval. Later sinus tachycardia with normal P-R interval	Digitalis stopped and pot. citrate given when A.T. developed. Death 12 days later. S.B.E. and pyrexia possibly contributed to tachycardia
			115	1 : 1 P-R 0.28 sec. (3 days later and no digoxin)		
12	A. fib.	Digitalis maintenance 4 years 3.5 mg. digitalin orally. Mersalyl 8 ml. Chlorothiazide 16 g. Pot. citrate 84 g. within preceding 20 days. 2.75 mg. digoxin I.V. within 7 days	150	A.V. dissociation with R.B.B.B.	No reversal observed	Died in attack of A.T. with complete A.V. block. Given calcium gluconate for tetany before the onset of complete A.V. block
			160	Complete A.V. block		
13	Sinus	Digoxin 59.75 mg. over 83 days. Chlorothiazide 18 g. over 36 days. Pot. citrate 210 g. over 35 days. Low-sodium diet	142	2 : 1 or 1 : 1	Sinus	Died 2 days after second attack of A.T. Each attack ended after 4 g. of KCl given by mouth. Between 1st and 2nd attacks digoxin had been restarted and a big diuresis had occurred
			130	1 : 1 P-R 0.24 sec.	"	
14	Sinus with supravent. es.	Digoxin in unknown maintenance doses for several weeks. Digoxin 0.75 mg. daily for at least 1 month. Chlorothiazide 22 g. over 39 days	150	A.V. dissociation.	"	A.T. lasted 11 days. Reversion occurred despite continuance of digoxin and chlorothiazide
			214	2 : 1		
			200	A.V. dissociation. Vent. rate 60		
15	Sinus	Digoxin 21.5 mg. over 7 weeks. Chlorothiazide 64 g. over 8 weeks	152	2 : 1 P-R 0.28 sec.	Complete A.V. block	Poorly controlled diabetes, with vomiting, before admission. Died
16	"	Digoxin 30 mg. orally in 40 days. Chlorothiazide 84 g. over 14 weeks. Mersalyl 96 ml. over 24 weeks. Pot. chloride 414 g. over 42 weeks. Thyroid extract 22.5 g. over 1 year	176	2 : 1 P-R 0.24 sec.	Not observed	Died 3 weeks after arrhythmia discovered. Digoxin had been continued (3.75 mg. week) with mersalyl and KCl. Duration of A.T. unknown
17	Unknown	Digoxin 4 mg. orally over 4 days. Mersalyl 2 ml. I.M. 2 days before	125	1 : 1 P-R 0.24 sec.	No reversal	Died 4 days after A.T. first noted still taking digitalis. Before A.T. had frequent vomiting with dehydration. ? Nutritional deficiency. Diabetic ketosis
			150	1 : 1 P-R 0.28 "		
			125	1 : 1 P-R 0.32 "		
18	Sinus	Digoxin 3.5 mg. orally in 4 days	166	2 : 1	Sinus	Digitalis was stopped and KCl 1 g. 2-hourly begun when A.T. first noted. Died 5 days later in sinus rhythm
19	"	Digoxin 255 mg. in 22 months. Chlorothiazide 3 g./week for 10 months	230 230	2 : 1 2 : 1	"	Digitalis stopped 7 days after A.T. first noted and KCl 3 g. day started. Next day S.R. was restored. Recovered

es. = extrasystoles. supravent. = supraventricular. vent. = ventricular. A.T. = atrial tachycardia. R. or L.B.B.B. = Right or left bundle branch block. C.S.P. = carotid sinus pressure. S.B.E. = subacute bacterial endocarditis.

chlorothiazide or hydroflumethiazide, and three mersalyl and chlorothiazide. Five of the 10 patients who were given chlorothiazide or hydroflumethiazide also had regular doses of potassium.

Electrolyte Disturbances.—Serum electrolyte concentrations were estimated in eight patients when the arrhythmia was recognized. In three there was a definite reduction of sodium, potassium, or chloride concentrations.

Outcome.—The immediate outcome is shown in Table III. Of eight patients who continued to take digitalis, six died without reverting to sinus rhythm. When

TABLE III.—Outcome in 18 Patients with Digitalis-induced Atrial Tachycardia with A.V. Block

	Reverted to Sinus Rhythm	Died Without Observed Return to Sinus Rhythm
Digitalis stopped. K not given	2	1
" " given	7	—
Digitalis continued. K not given	1	5
" " given	1	1

digitalis was stopped, nine out of ten patients regained sinus rhythm; three of these ten did not receive potassium, and one of the three died before regaining sinus rhythm.

Effect of Carotid Sinus Stimulation.—Compression or massage of one or both carotid sinus areas was carried out in eight patients. In six the degree of A.V. block was increased: an extreme example, with 29 uncondacted beats during carotid sinus stimulation, is shown in Fig. 2. In one patient there was a change in the form of the P wave without alteration in the degree of block. There was a slight decrease in the atrial rate in some cases, but in none was the arrhythmia abolished. In the remaining patient carotid sinus stimulation was ineffective. After sinus rhythm had been restored and digitalis stopped, carotid sinus stimulation continued to produce atrioventricular block in several of these patients (Fig. 2). In one patient this effect could still be produced four and a half months after withdrawal of digitalis.

Discussion

Atrial tachycardia with A.V. block appears to have been caused by digitalis in 21 of the 22 incidents reported here. Of 112 episodes of this arrhythmia reported by Lown (1957), 83 were caused by digitalis and in only 13 could the drug be exonerated. Thus atrial tachycardia with A.V. block is usually a complication of digitalis administration. Its incidence is difficult to assess. Thirteen of our cases were recognized during the last 18 months, usually during routine reporting of electrocardiograms. We have been unable to arrive at an estimate of the number of patients at risk during this period.

The recognition of tachycardia in a patient receiving digitalis is often regarded as an indication for increasing the dose of digitalis. If atrial tachycardia with A.V. block is the cause of a high ventricular rate, the giving of more

digitalis may be lethal. Digitalis poisoning is likely to have been the immediate cause of death in eight patients (Cases 1, 2, 3, 6, 12, 15, 16, and 17), but digitalis cannot be incriminated with certainty in their deaths, since these eight patients had either cardiac failure or recent myocardial infarction at the time their arrhythmia began. Atrial tachycardia with A.V. block is particularly likely to develop in patients who already have serious heart disease.

Diagnosis

The main criteria for the diagnosis of atrial tachycardia with A.V. block have been stated. When the atrial rate is less than 150 a minute there is usually 1:1 A.V. conduction. Rates greater than 150 a minute are usually associated with 2:1 or higher grades of block. The degree of block may vary from a long P-R interval to A.V. dissociation. Pressure on the carotid sinus nearly always increases the degree of A.V. block, and may temporarily stop A.V. conduction, with little or no slowing of the atrial tachycardia. This response to carotid sinus stimulation is like that seen in atrial flutter and unlike that usually seen in paroxysmal atrial tachycardia. It is of great value in confirming the diagnosis. In several of the cases here described P waves were obscured in the QRST complex. When this happens in the presence of 2:1 A.V. block, atrial tachycardia can easily be overlooked (Fig. 3).

Atrial tachycardia with A.V. block may be confused with atrial flutter, for the two possess many features in common and it may be impossible to distinguish them with certainty. Their chief features are summarized in Table I. The characteristic "saw-tooth" baseline of atrial flutter is most pronounced in leads II and III and may not be seen in other leads; thus leads II and III are the leads upon which the distinction is most reliably made. In atrial tachycardia, however, P waves are often best recognized in the right chest leads. It is impossible to tell digitalis-induced atrial tachycardia with A.V. block from sinus tachycardia due to

FIG. 3.—A further example of digitalis-induced atrial tachycardia with 2:1 and 1:1 A.V. block. The P waves are superimposed on the T waves.

some other cause associated with a long P-R interval induced by digitalis, unless there has been a clear-cut alteration in the shape of the P wave.

Potassium Depletion

Like other digitalis-induced arrhythmias, atrial tachycardia with A.V. block is especially prone to occur in the presence of potassium depletion. Cardiac failure is associated with cellular potassium depletion, and potassium salts given by mouth (even if absorbed) do not always prevent great losses of this ion during treatment with diuretics. The incidence of atrial tachycardia with A.V. block may be increasing since the introduction of chlorothiazide. In 10 of the 14 cases which have been observed since 1958, chlorothiazide or hydroflumethiazide had been given before the development of atrial tachycardia with block. The danger of these drugs seems to lie partly in the ease with which they can be given compared with injections of mercurial diuretics and partly because of the considerable potassium loss which they produce. Determinations of serum electrolyte concentrations were not made in all the cases under review. Of eight cases in which they were made, five showed no abnormality of sodium, potassium, or chloride concentration. Severe cellular potassium depletion may be present with normal serum concentrations of these ions. Most of the patients in this series probably had potassium deficits of some degree resulting from cardiac failure, diuretics, sodium restriction, vomiting with dehydration, poorly controlled diabetes, or various combinations of these.

Prognosis and Treatment

Patients who develop digitalis-induced atrial tachycardia with A.V. block have a poor prognosis. Of the 18 patients in this series, 13 died within four months of the occurrence of the arrhythmia. In five of these, digitalis intoxication was not directly responsible for death. Patients who develop atrial tachycardia with block are already gravely ill, and it is difficult to assess the role of the various factors involved in their rapid deterioration. Although the tachycardia itself may be important in accelerating the downhill course, resistant cardiac failure with electrolyte imbalance and the other toxic effects of digitalis also contribute. The development of this unusual and almost specific arrhythmia may serve as a timely warning of the presence of potassium deficiency and digitalis poisoning. Its prompt treatment may lead to a striking improvement in the patient's general condition and in his response to diuretics.

It is apparent, on theoretical grounds and from practical experience, that both the administration of potassium and the withdrawal of digitalis are essential for the treatment of digitalis-induced atrial tachycardia with A.V. block. The rapid effects of oral potassium are illustrated by Fig. 2, which is representative of other cases, and by the experience of Lown, Wyatt, and Levine (1960), and of Oram, Resnekov, and Davies (1960). Potassium is rapidly absorbed when given by mouth. The dose required to correct the arrhythmia ranges, in our experience, from 2 to 8 g. of potassium chloride. When necessary, 40 mEq of potassium (3 g. of potassium chloride) may be given as an intravenous infusion lasting an hour or two, under continuous electrocardiographic control (Lown *et al.*, 1960). Potassium chloride may have advantages over other potassium salts, since hypochloroemic alkalosis is commonly present in these patients.

If this treatment is undertaken the prognosis for the arrhythmia is good. The subsequent management of congestive heart failure, which is so often present, may, however, present difficulties. A potassium supplement should be given whenever diuretics are administered. Digitalis is not necessarily contraindicated provided potassium is administered at the same time; several of the patients here reported were treated with digitalis after successful management of digitalis-induced atrial tachycardia with block and the arrhythmia did not recur.

Although the prophylactic administration of potassium might prevent the development of atrial tachycardia with A.V. block, the arrhythmia can arise in spite of this precaution. Indeed, in retrospect, we would raise few objections to the treatment received by many of our patients before the development of atrial tachycardia with A.V. block. It is all the more important, therefore, that physicians should be alert to this complication of digitalis therapy.

Summary

Twenty-two episodes of atrial tachycardia with atrio-ventricular block have been observed in 19 patients with various types of advanced heart disease. Eighteen of these patients were receiving digitalis.

Atrial tachycardia with A.V. block is a serious complication of digitalis treatment which is often overlooked or confused with atrial flutter. It is probably increasing in incidence as a result of the widespread use of potassium-depleting diuretics.

Awareness and recognition of digitalis-induced atrial tachycardia with A.V. block is important, since it responds rapidly to withdrawal of digitalis and the administration of potassium.

We are grateful to Professor Sir Derrick Dunlop and Dr. Rae Gilchrist for their advice and encouragement, and to them, and other physicians in the Edinburgh Royal Infirmary, for permission to report cases under their care.

[ADDENDUM.—Six additional episodes of atrial tachycardia with A.V. block have been observed in six patients during the last three months. All had received digitalis before the arrhythmia began, but in one the drug was probably not responsible. Digitalis was continued in four, of whom three died, two without reversion to sinus rhythm.]

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In 1959 there were 30 fatal accidents and 7,000 other reportable accidents in the shipbuilding and ship-repairing industry in Great Britain, and a number of typical occurrences which have been notified to H.M. Inspectors of Factories are included in the latest edition of *Accidents—How they happen and how to prevent them*, which has recently been published (No. 44, H.M.S.O., price 1s. 3d. net).