

JOURNAL
OF THE
ALLENTOWN HOSPITAL

ALLENTOWN, PENNSYLVANIA

VOL. IV

FEBRUARY, 1955

NUMBER 3

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JOURNAL OF THE ALLENTOWN HOSPITAL

Published Quarterly

BY THE STAFF AND TRUSTEES
OF
THE ALLENTOWN HOSPITAL
ALLENTOWN, PENNSYLVANIA

FEBRUARY

1955

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THE TOXIC EFFECTS OF DIGITALIS

RAY F. BEERS, JR., M.D.

With the introduction of low salt diets and effective mercurial diuretics as well as purified digitalis glycosides, it was thought that the incidence of digitalis intoxication would decrease considerably. However, this has not been the case and intoxication is becoming an increasing problem. This is partially explained by the fact that patients are living longer with badly diseased hearts. The marked electrolyte shifts secondary to low salt diets and profound mercurial diuresis, as well as standardized purified glycoside schedules of the type originally promulgated by Gold,¹ have had a distinct effect. Digitalis therapy is seldom the pure white of therapeutic effect, or the black of advanced intoxication, rather it is usually shades of grey, for any digitalis preparation requires 60 per cent of the toxic dose to produce a recognizable therapeutic effect. In this respect Digoxin, Digitoxin and anatosidec-(Cedilanid) offer no advantages over the whole leaf²⁻⁴ and have the distinct disadvantage of not producing local irritating effects on the gastric mucosa. This allows more toxic doses to be administered than is usually possible with whole leaf. Gitalin, an amorphous water soluble mixture of digitalis glycosides that can be extracted from *Digitalis purpurea*, may be an exception since Batterman and his co-workers report that but one third of the toxic dose is required to produce a therapeutic effect with this preparation.⁴ However, this remains to be confirmed by other critical observers.

Every form of arrhythmia and conduction disturbance has been attributed to digitalis overdosage,⁵⁻⁹ but while disturbances in the ventricular rhythm and the conduction of the cardiac impulse are well recognized consequences of toxicity, abnormal auricular mechanisms have usually been regarded as rare and non-specific.¹⁰ Recently, clinical experience seemed to indicate that this view was untrue and with this in mind all the disorders of the heart beat due to disorders of impulse formation above the level of the ventricles, with the exceptions of sinus tachycardia, sinus bradycardia, sinus arrhythmia, and premature supraventricular contractions, that occurred in the Allentown Hospital during the period of July 1, 1953 to June 30, 1954 were reviewed with respect to the etiology of the arrhythmia.

170 arrhythmias were identified in 160 patients and the following incidence was found:

	<i>No. of Cases</i>	<i>% of all Cases</i>
Paroxysmal auricular tachycardia		
without block	13	7.6%
Paroxysmal auricular tachycardia		
with block	11	6.4%
Auricular fibrillation	127	74.5%
Auricular flutter	4	2.3%
All A-V nodal rhythms	11	6.4%
Coronary sinus rhythm	2	1.2%
Wandering pacemaker	5	2.9%
All other A-V nodal rhythms	4	2.3%
Sinus arrest	0	0%
Auricular standstill	4	2.3%

PAROXYSMAL AURICULAR TACHYCARDIA WITHOUT BLOCK

No statistics are available on the incidence of paroxysmal auricular tachycardia without block caused by digitalis, but many authors believe that it frequently precedes auricular fibrillation induced by this medication. ⁶ No instance of digitalis induced auricular tachycardia without block was encountered in this series. In two instances the arrhythmia appeared in fully digitalized patients, but it was not associated with toxicity and seemed to have little effect on the rhythm other than the fact that both ventricular responses were relatively slow (150/min.). Both cases were terminated easily with neostigmine potentiated carotid sinus pressure. The causative factors for this arrhythmia are listed below. Where there was no evidence of heart disease or specific predisposition such as thyrotoxicosis, the etiology is listed as idiopathic.

- Idiopathic - 6
- Arteriosclerotic heart disease plus pneumonia - 2
- Pulmonary embolus - 1
- Myocardial infarction - 1
- Inactive rheumatic heart disease with valvular deformity - 2
- Luetic aneurysm of the ascending aorta - 1
- Digitalis intoxication - 0

The age range was from three weeks to 67 years and the majority of cases showed no heart disease. The two instances of arteriosclerotic

heart disease cannot be considered to be due primarily to heart disease for neither had ever experienced any rapid or irregular heart action prior to the onset of the infection, although both had been in mild congestive failure.

PAROXYSMAL AURICULAR TACHYCARDIA WITH BLOCK

Of the 11 cases of this type of arrhythmia encountered, all but one were due to digitalis intoxication. The one exception appeared as a transient rhythm in the early stages of an acute myocardial infarction. In none of the cases could excessive mercurial diuresis or gross derangements of electrolytes be implicated although several were receiving small doses of mercaptomerin in accordance with their weight gain. The importance of a relative deficit of potassium ion in the cardiac muscle cell has been stressed by Levine and he has produced the arrhythmia in humans both by maintaining constant digitalization and employing methods to deplete the body potassium and by keeping potassium constant and employing increasing doses of digitalis.¹⁰ He notes that there is no constant relation between the serum potassium and increasing sensitivity to digitalis except that a definitely low figure makes higher doses of digitalis exceedingly hazardous.

Paroxysmal auricular tachycardia with block is a hybrid arrhythmia that shows some of the characteristics of both auricular tachycardia without block and auricular flutter. The auricular rate varies from 150 to 250/min., being usually in the range of 150-to 190/min. In Levine's series only thirty per cent showed a 2:1 ventricular response, while the remainder showed a variable degree of A-V block, with the Wenckebach type of conduction being common.¹⁰

The natural development of auricular tachycardia with block has been observed in three humans poisoned with acetylstrophanthidin given I.V.¹⁰ This glycoside is more rapid and transient than ouabain, showing its earliest effects in 1.5 minutes and its peak effect in 12 minutes. The duration of toxicity is only 30 minutes making continuous electrocardiographic tracings during the development and recession of toxic rhythms possible. The natural development of the arrhythmia was seen by Levine to be as follows.¹⁰

At first the auricular rate increased and the shape and magnitude of the P-wave changed, but not its direction; representing the gradual usurpation of the S.A. node by a more excitable auricular focus. There followed a gradual continuing acceleration of the auricular rate with a

1:1 ventricular response. The P-waves then gradually migrated towards the preceding T-wave as the degree of A-V block increased. When a critical rate of 150-250/min. was reached a 2:1 ventricular response occurred and the arrhythmia was fully established. Reestablishment of normal sinus rhythm was the exact mirror image of its development, with a gradual slowing of the auricular rate and a migration of the P-waves toward the succeeding QRS-complexes as the degree of A-V block diminished. A sharp change in the size and contour of the P-wave signalled the return to sinus rhythm.

The vital statistics of the cases encountered at the Allentown Hospital during the period covered by this review are listed below:

<i>Patient</i>	<i>Age</i>	<i>Type of Heart Disease</i>	<i>Excessive Diuresis</i>	<i>Excessive Digitalis</i>	<i>Systemic Signs of Dig.Tox.</i>
A.K.	76	Arteriosclerotic	No-but potassium loss due to vomiting	yes	Vomiting & delirium
J.H.	90	Arteriosclerotic	No diuretics	yes	Anorexia
E.S.	76	Arteriosclerotic	No	yes	None
P.B.	84	Arteriosclerotic	No diuretics	yes	Anorexia
G.W.	67	Acute myocardial infarction	no	no	none
G.D.	80	Arteriosclerotic	no	yes	Vomiting & delirium
P.F.	64	Hypertensive-arteriosclerotic	no	yes	Vomiting, blurred & dbl. vision
S.A.	47	Rheumatic-mitral stenosis	no	yes	Vomiting & vertigo
T.W.	68	Arteriosclerotic	no	yes	Vomiting & vertigo
E.G.	69	Arteriosclerotic	no	yes	Nausea & mental confusion
K.V.	61	Arteriosclerotic	no	yes	Nausea & vomiting

OUTCOME

A.K.	Expired	G.W.	Recovered	T.W.	Recovered
J.H.	Expired	G.D.	Expired	E.G.	Expired
E.S.	Expired	P.F.	Recovered	K.V.	Expired
P.B.	Expired	S.A.	Recovered		

Six of the eleven cases died soon after the onset of the arrhythmia, giving rise to a mortality rate of 54.5% which compares with Levine's reported mortality of 60% in 23 cases studied at the Peter Bent Brigham Hospital.¹⁰ Decherd, Hermann and Schwab report a 55% mortality,¹¹ and Askey reported a 100% mortality in five cases.¹²

Auricular tachycardia with block can resemble practically every other auricular mechanism when only single tracings are available of a phase in its natural development and without serial tracings or special leads it may be impossible for even the experienced cardiologist to identify the true nature of the arrhythmia.

1. **Sinus Tachycardia.** Before the onset of significant A-V block, when the auricular rate is relatively slow and previous tracings are not available to compare the size and contour of the P-waves, the arrhythmia may be indistinguishable from sinus tachycardia.

2. **Paroxysmal auricular tachycardia without block.** When the auricular rate exceeds 150/min. and the migrating P waves have fused with the preceding T waves and the ventricles are in a 1:1 response, the arrhythmia is apt to be called supraventricular tachycardia.

3. **Nodal Rhythm.** When the ventricular rate is slow because of a high degree of A-V block and the P waves are very small and are not in evidence in the standard or unipolar leads, the arrhythmia is apt to be confused with nodal rhythm. V leads to the right of the precordium or exploration of the precordium with C-R leads may resolve this conflict at times.

4. **Auricular Fibrillation.** When the P waves are diminutive and the degree of A-V block is constantly changing, the arrhythmia is apt to be called auricular fibrillation. Despite special leads and carotid sinus pressure, it may be impossible to distinguish auricular tachycardia with block from other auricular mechanisms and for this reason Levine has recently advocated a digitalis tolerance test employing acetlstrophanthidin.¹³ This type of arrhythmia is illustrated by five type cases briefly summarized below.

(a) **Conversion of sinus rhythm to auricular tachycardia with block.** P.F. A 64 year old white laborer with benign essential hypertension was admitted to the Allentown Hospital on July 6, 1953 because of his first episode of congestive failure. He was placed on a low salt diet and digitalized with 18 grains of whole leaf. Thereafter he was maintained on 1.5 grains daily of whole leaf. He responded well on this regimen and was discharged in normal sinus rhythm (see fig. 1)

on July 17, 1953, with thirty tablets containing 1.5 grains of leaf digitalis each. He was instructed to take one tablet daily, but he misunderstood and took all thirty tablets over the next six days (45 grains). He appeared in clinic on July 22, 1953 complaining of severe anorexia and nausea and repeated vomiting. In addition he had double and blurred vision. An EKG taken on this day showed auricular tachycardia with block (see fig. 2). All medication was stopped and one week later on July 29, 1953 tracings showed what seemed to be auricular fibrillation (see figs. 3 & 3a). On August 5, 1953, two weeks after digitalis was stopped he had returned to normal sinus rhythm. Subsequently he has been well maintained on leaf digitalis, grains 3/4 daily.

(b) **Conversion of acute auricular fibrillation to auricular tachycardia with block.** T.W., a 68-year-old diabetic white male, was admitted to the surgical service of the Allentown Hospital on November 2, 1953 for the treatment of a cellulitis of the right forearm. He was in moderate congestive failure and was fibrillating rapidly on admission. He was digitalized with 1.2 mgm. of digitoxin and placed on a daily ration of .1 mgm. daily. On this regimen his failure cleared quickly and he returned to sinus rhythm in 48 hours. He did well until November 12, 1953 when his ventricular rate suddenly increased to over 200. A tracing was taken at that time and with the aid of neostigmine potentiated carotid sinus pressure, coarse auricular fibrillation was demonstrated (see fig. 4). Digitoxin was stopped and he was placed on one mgm. of digoxin daily. By November 15, 1953 tracings showed typical auricular tachycardia with block (see fig. 5). All digitalis was stopped, and potassium chloride was given by mouth. The arrhythmia persisted in serial tracings over the next five days and on November 21, 1953 he returned to sinus rhythm (see fig. 6). Subsequently he underwent several operations including skin grafting without untoward event and he was discharged in good condition.

(c) **Conversion of chronic auricular fibrillation into auricular tachycardia with block.** The presence of many years of auricular fibrillation does not protect the patient against the development of auricular tachycardia with block as is illustrated by S. A., a 49 year old white diabetic female who had undergone mitral commissurotomy for advanced mitral stenosis. She was admitted to the Allentown Hospital because of severe anorexia and nausea with repeated vomiting and vertigo. Although she had experienced a few episodes of sinus rhythm, her dominant rhythm for several years had been auricular fibrillation. She had been maintained for many months on .2 mgm of digitoxin daily. Her admission EKG on April 6, 1954 showed paroxysmal auricular tachycardia with block (see fig. 7). Digitalis was stopped

and serial tracings over the next 15 days showed persistence of the arrhythmia (see fig. 8). On April 25, 1954 (19 days after digitalis was stopped) she returned to auricular fibrillation. Leaf digitalis grains, one daily, was begun when the ventricular response to the fibrillation became more rapid and she has since been maintained on this dosage without ill effect.

(d) **Use of special leads to demonstrate auricular tachycardia with block.** G.D., an 80-year-old white male with arteriosclerotic heart disease, who had been maintained on a daily ration of .2 mgm of digitoxin daily for a considerable period of time was admitted to the Allentown Hospital on October 16, 1953 because of anorexia and mental confusion amounting at times to delirium. His initial tracing showed a regular ventricular rhythm (see fig. 9). No P waves were discernible in the routine standard and unipolar leads, but when the precordium was explored the true nature of the arrhythmia became apparent when a unipolar precordial lead was taken midway between the right nipple and the right parasternal line in the fourth interspace. This showed paroxysmal auricular tachycardia with block of varying degree (see fig. 9). The significance of the arrhythmia was not realized and digitalis therapy was continued because of signs of congestive failure. The patient died eight days later.

(e) **Auricular tachycardia with block confused with auricular fibrillation.** E.G., a 69-year-old white male with arteriosclerotic heart disease was admitted to the Allentown Hospital on October 16, 1953 because of rapidly progressing dyspnea and orthopnea coupled with anorexia, nausea and mental confusion. He had been maintained for many months on leaf digitalis grains 1.5 daily. He failed to improve significantly and on October 19, 1953 the dosage of leaf was increased to grains 4.5 daily. The next day his rhythm, which had been regular, had become rapid and irregular and an EKG showed what was thought to be auricular fibrillation, but which in reality was auricular tachycardia with block (see fig. 10). The digitalis was continued in the same dosage for the next three days and then dropped to grains 1.5 daily again. Since the ventricular rate had slowed, it was thought that a therapeutic effect had been achieved, but a repeat tracing again showed auricular tachycardia with block and a Wenckebach type of A-V condition (see fig. 11). This was correctly interpreted, but digitalis was not stopped and the patient died the next day.

The difficulties encountered in definitely indentifying auricular tachycardia with block have been noted above and it is safe to estimate that for each case identified at least one was probably missed. If all

are considered, including those missed, it can be seen that about two cases a month of this type of arrhythmia will be encountered in a 500 bed general hospital.

AURICULAR FIBRILLATION

This arrhythmia, which predominates all the abnormal auricular mechanisms encountered, was most commonly caused by arteriosclerotic heart disease, which accounted for 82 cases or 64.5%. Inactive rheumatic heart disease with valvular deformity was next in frequency, being the etiologic agent in 26 cases or 20.4%. Acute myocardial infarction gave rise to auricular fibrillation in 7 cases or 5.5% as did hypertensive heart disease. Thyrotoxicosis and pulmonary embolus accounted for one case each. Digitalis was considered as possibly the causative agent in four cases or 3%. Because of the controversy surrounding the causation of auricular fibrillation by digitalis intoxication these four cases will be briefly summarized.

1. P.F., a 64-year-old white laborer, already summarized as Case No. 1 under paroxysmal auricular tachycardia with block. If figures 3 and 3a are referred to, it can be seen that the mechanism certainly seems to be auricular fibrillation and if so, its causation by digitalis is clearly established. However, it must be admitted that the precordium was not explored with special leads in search of distinctive P waves.

2. E.G. a 69-year-old white male, already summarized as Case No. 5 under paroxysmal auricular tachycardia with block. If figs. 10 and 10a are referred to it will be seen that this tracing closely resembles auricular fibrillation and it was so considered both clinically and electrocardiographically. However close inspection of the malformed P waves in leads V-1 and AVF, leads one to the conclusion that this is probably a developmental phase of paroxysmal auricular tachycardia with block.

3. K.S., a 73-year-old diabetic white female, with no evidence of clinically significant heart disease, developed bronchiolitis two weeks prior to her admission to the Allentown Hospital on February 13, 1954 and was digitalized with grains 21 of leaf digitalis in the belief that she was in cardiac failure. She was on a daily ration of grains 1.5 daily when admitted to the hospital because of increasing rales in her chest and repeated vomiting. Her admission EKG showed typical auricular fibrillation with a strong digitalis effect on RS-T-segments and T waves. Digitalis was stopped and antibiotic therapy instituted. On this regimen she made an uneventful recovery and returned to normal sinus rhythm seven days later. Her previously abnormal EKG was within normal limits on discharge.

4. W.M., a 67-year-old white female with arteriosclerotic heart disease was first admitted to the Allentown Hospital on February 22, 1954 because of repeated vomiting. She had been maintained on .1 mgm. of digitoxin daily for at least four months prior to admission because of moderate dyspnea and orthopnea with mild ankle swelling. Her EKG on admission showed typical auricular fibrillation. Digitalis was stopped and she reverted to sinus rhythm three days later. Her vomiting abated, her appetite returned and she was discharged in good condition. Shortly after arriving home, she was again placed on .1 mgm of digitoxin daily. Three weeks later on 29 March, 1954 she was again admitted to the Allentown Hospital this time for psychiatric evaluation because of her mental confusion. She was again vomiting repeatedly. The EKG showed she was in auricular fibrillation. Digitalis was again stopped and no other therapy added. By April 6, 1954 she again returned to sinus rhythm and was eating well without vomiting. Her mental status had cleared markedly and she was discharged in good condition.

In all these cases of auricular fibrillation digitalis was probably a significant factor in the development of the arrhythmia. However, the arrhythmia in Case No. 2 is not auricular fibrillation, though it was thought to be. Case No. 1 seems to be typical auricular fibrillation but it might represent a waning phase of auricular tachycardia with block, just as the arrhythmia in Case No. 2 definitely represents a waxing phase. Case No. 3 did not have clinically evident heart disease, but the development of auricular fibrillation can be caused by infection alone in elderly individuals, however it took seven days for her to return to sinus rhythm which time span is compatible with the excretion of digitalis. Case No. 4 definitely had heart disease and fibrillation might have occurred whether or not digitalis was used, but the development of two distinct episodes of fibrillation associated with severe systemic signs of toxicity and its regression to sinus rhythm on both occasions when digitalis was stopped make it highly probable that digitalis poisoning on an already diseased myocardium created the arrhythmia.

An incidence of 3% of cases in which digitalis toxicity is a contributing factor is admittedly small, but it serves to further remind us of the fact that not all rapid, irregular rhythms of supraventricular origin demand increasing doses of digitalis to slow the ventricular rate.

AURICULAR FLUTTER

Four cases of auricular flutter were encountered. In two cases the etiologic agent was arteriosclerotic heart disease; one case followed an acute myocardial infarction and one case was produced by thyrotoxic

heart disease. No cases due to digitalis intoxication were identified although several cases of paroxysmal auricular tachycardia with block were tentatively considered to be flutter until the true nature of the arrhythmia was established.

A-V NODAL RHYTHMS

A-V nodal rhythms were encountered in 11 cases and of these, two were of the coronary sinus type. Both cases of coronary sinus rhythm were encountered in the early stages of acute myocardial infarction. The pacemaker wandered to the A-V node in five cases. Of these, one case was associated with arteriosclerotic heart disease and one case was encountered during the acute stage of myocardial infarction. The remaining three cases were all due to digitalis. Of the cases due to digitalis, two were associated with systemic signs of toxicity while the appearance of the rhythm in the third patient showed little influence on his continuing improvement. One of the cases associated with systemic signs of toxicity is briefly summarized below.

J.E., a 74-year-old white female with arteriosclerotic heart disease was admitted to the Allentown Hospital because of increasing congestive failure. She had been on a daily ration of digitalis for many months, but the amount could not be determined. Digitoxin, 1.4 mgm. was administered within 48 hours of admission whereupon the patient became very ill and began to vomit repeatedly. An EKG take at this time showed that the pacemaker wandered to the A-V node and strong digitalis type RS-T segment deviations and T wave, inversions were noted. Digitalis was stopped and within seven days all vomiting had ceased and the patient was eating well. At this time, EKG showed normal sinus rhythm with a moderate digitalis effect on the ST-segments and T waves. She was subsequently discharged in good condition.

All other nodal rhythms were encountered in four cases, in two of which arteriosclerotic was present. One 53-year-old man had no evidence of clinically significant heart disease. One case was due to digitalis and was associated with systemic signs of toxicity. She rapidly improved when digitalis was stopped and potassium salts were given by mouth. Concomitantly with her improvement, she returned to normal sinus rhythm.

Thus it can be seen that a type of nodal rhythm appeared as one manifestation of digitalis toxicity in better than 27% of such rhythms encountered. While the appearance of a nodal rhythm during digitalis therapy is not of itself an indication of overdosage, still it is a hallmark of digitalis effect and the patient should be carefully observed for

systemic signs of toxicity. Certainly the appearance of the arrhythmia plus systemic signs of toxicity, merits at least a reduction in digitalis dosage.

SINUS ARREST

No cases of omitted beats due to sinus arrest were encountered during the period reviewed. However, it has been well documented that the arrhythmia is often initiated by the vagotonic action of digitalis.¹⁴

AURICULAR STANDSTILL

While this type of arrhythmia is secondary to either sinus arrest or sino-auricular block, it is characterized by an idioventricular rhythm during the period when no auricular activity occurs. Four such cases were encountered during the period reviewed and in all, advanced digitalis toxicity was the etiologic factor. One case summary will serve to illustrate the arrhythmia.

K.V., a 61-year-old white female with chronic auricular fibrillation (see fig. 12) was admitted to the Allentown Hospital on November 11, 1953 because of a second cerebral embolus within a six month period. It was decided that conversion to sinus rhythm should be attempted and after two weeks of adequate anticoagulant (dicoumerol) therapy, she was digitalized with grains 18 of leaf digitalis and placed on a daily ration of grains 1.5. Subsequently increasing doses of quinidine sulfate were tried and on December 11, 1953, after 54 grains of quinidine (grains 9 q 2 h for 6 doses), the EKG showed auricular tachycardia with block. (See figs. 13 and 13a). However, since this type of arrhythmia was known to occur during the conversion of auricular fibrillation to sinus rhythm and was considered of no clinical significance,¹⁰ it was assumed that the next higher dosage schedule of quinidine would probably accomplish the conversion. Quinidine sulfate grains 12 q 2 h was subsequently tried on two occasions, but the patient began to vomit repeatedly after one or two doses had been given. This was thought to be due to quinidine despite the fact that she had taken 54 grains previously without gastro-intestinal upset. Quinidine therapy was abandoned, but when the vomiting continued for 72 hours after the cessation of therapy and an EKG revealed auricular standstill (see fig. 14) it was realized that the patient's daily ration of digitalis had produced toxicity. Digitalis was discontinued and potassium salts were given by mouth. The patient's appetite and sense of well being slowly improved although she still had anorexia on December 21, 1953 when the standstill suddenly converted to sinus rhythm. (see fig. 15) Eight days later she returned to auricular fibrillation. She was

discharged in good condition on a daily ration of leaf digitalis gr. 3/4 daily.

From the foregoing, it can be seen that digitalis toxicity is not an uncommon cause of abnormal auricular mechanisms since it was encountered in 21 instances or 12.3% of the cases reviewed and furthermore overdosage of this drug was clearly established in 17 cases or 39.5% of all abnormal auricular mechanisms other than auricular fibrillation. If the rhythm is true auricular fibrillation with a rapid ventricular response, then the chances of overdosage are unlikely, but it must be remembered that occasionally digitalis toxicity will be a contributing factor in this type of arrhythmia. Furthermore, auricular tachycardia with block is at times extremely difficult to differentiate from fibrillation and its presence is almost always an indication of advanced digitalis toxicity. A rapid, irregular rhythm of supraventricular origin per se does not, therefore necessarily call for increasing doses of digitalis. The arrhythmia must be identified with certainty and even more so is this true of the patient who has been previously well maintained and who is now not doing well. Both paroxysmal auricular tachycardia with block and auricular standstill are absolute indications for the discontinuance of digitalis therapy, so that the regularization of ventricular response in a digitalized patient with auricular fibrillation should be viewed with suspicion even when the ventricular response is rapid, as digitalis toxicity is a good possibility in these cases. Nodal rhythms and wandering pacemaker are not per se indications of toxicity, but they can be caused by overdosage.

Paroxysmal auricular tachycardia with block is the most frequently encountered disturbance of auricular activity due to intoxication with digitalis accounting for 11 cases or 52.3% of the toxic supraventricular rhythms. Its ability to mimic mechanisms requiring digitalis for control make it especially important and it must be considered and carefully ruled out in a digitalized patient with an abnormal auricular mechanism, before digitalis therapy can be safely continued or increased. Paroxysmal auricular tachycardia with block is an indication of advanced digitalis toxicity and is associated with an extremely high mortality.

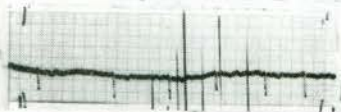
SUMMARY

Digitalis intoxication affects the auricles as well as the ventricles and 12.3% of all abnormal auricular mechanisms, exclusive of sinus tachycardia and bradycardia and premature supraventricular contractions, encountered at the Allentown Hospital during the period July 1, 1953 to June 30, 1954 were shown to be due to this factor.

1. Lead II
Normal sinus rhythm



2. Lead V-2 Par. Auricular
tachycardia with block



3. Lead III
Auricular fibrillation



3a. Lead V-1
Auricular fibrillation

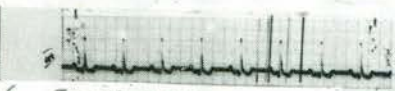
Small
Carotid pressure
hypoxia



4. Lead III Impure Flutter
with carotid sinus pressure



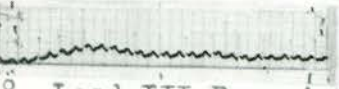
5. Lead III Par. Auricular
Tachycardia with block



6. Lead III
Normal sinus rhythm

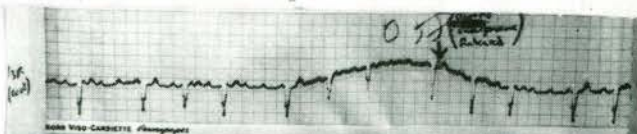


7. Lead III Par. Auricular
Tachycardia with block



8. Lead III Par. Auricular
Tachycardia with block

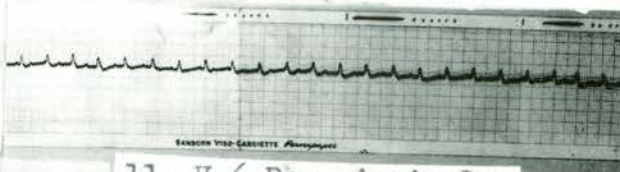
9. Lead aVL Nodal Rhythm
simulated by PAT with block



9a. V-3R-PAT with block clearly
shown with special lead



10. aVF Auricular Fibrillation
PAT with block-probable



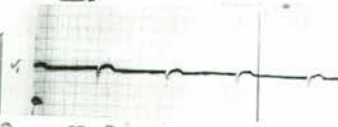
11. V-6 Par. Auricular
Tachycardia with block



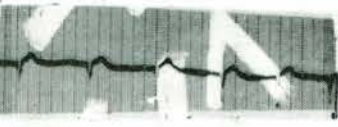
12. V-1 Auricular Fibrillation



13. Lead II Par. Auric.
Tachycardia with block



13a. V-1 Par. Auric.
Tachycardia with block



14. V-1 Auricular Standstill



15. Lead II - Normal Sinus
Rhythm

Eleven cases of paroxysmal auricular tachycardia with block were seen in one year. This is the most common auricular manifestation of digitalis toxicity accounting for 52.3% of the cases reviewed. Its presence indicates advance intoxication and is associated with a high mortality.

Four cases of auricular fibrillation in which digitalis was probably the main causative agent were encountered during the period reviewed.

Four cases of auricular standstill were encountered. All were due to advanced digitalis intoxication.

Eleven cases of A-V nodal rhythms were encountered. The appearance of this rhythm was associated with digitalis toxicity in 3 cases or 27%.

No cases of paroxysmal auricular tachycardia without block, auricular flutter or sinus arrest, due to digitalis were encountered, but such causation is noted in the literature.⁵⁻⁹

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INTRAHEPATIC CHOLANGIO-JEJUNOSTOMY

(A short review with a case presentation)

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INTRODUCTION

This procedure, which is an anastomosis of one of the intrahepatic bile ducts to the jejunum following a partial resection of the left lobe of the liver, was devised and described by W. D. Longmire, Jr., M.D., in 1948. ¹ Since then 23 cases have been reported in the literature, 6 of them having been done for malignant and 17 for benign extra-hepatic bile duct obstructions.

INDICATIONS

Complete obstruction, with destruction of the extra-hepatic ducts, where other more popular or standard procedures are either technically too difficult or would make the operative time too long, thus jeopardizing the life of a patient immediately or subsequently because of the extra burden added to an already markedly damaged liver.

CAUSES OF OBSTRUCTION

Strictures, most of which are due to operative trauma ^{2,3} at the time of cholecystectomy or other upper abdominal surgery, have increased in frequency. The trauma responsible for these strictures may be due either to crushing, excision, incision, ligation especially when transfixion sutures are used, or perforation of the bile ducts. Localized or extensive strictures may thus result, the extensive fibrosis with complete occlusion believed to follow the bathing of the exposed extra-hepatic ducts in a pool of irritating bile in the infra-hepatic region when external drainage is inadequate. Calculi, neoplasms, benign or malignant of the pancreas, ampulla or the ducts, inflammatory conditions especially of the pancreas and congenital anomalies account for most of the other causes.

METHODS OF REPAIR OF STRICTURES

End-to-end anastomosis. This is accomplished by finding and mobilizing both the proximal and distal ends of the ducts which in certain cases might be extremely difficult, anastomosing mucosa-to-mucosa and splinting and decompressing the duct with a T tube either

distal or proximal to the anastomosis. Lahey ³ reports best results with this type of a repair and strongly recommends it because of the preservation of the sphincter of Oddi. The one important factor in this repair is leaving the T tube in long enough so that no contracture of the anastomosis develops. The time recommended by Lahey ³ is at least a year. The other factor which also applies to other types of repair is the excision of all the scar tissue from the ends of the ducts so that normal mucosa may be anastomosed to normal mucosa and thus minimize the formation of scar tissue.

Anastomosis of the proximal duct or ducts to the duodenum or jejunum.

Walters ⁴ reports best results where the common duct was anastomosed to the duodenum and he has also shown that cholangitis is due to recurrent stricture with biliary obstruction rather than to reflux of duodenal contents into the biliary tree.

Cole ² prefers anastomosis of the proximal duct to a Roux-y arm of jejunum with which he has obtained best results, rather than jejunal loop anastomosis which occasionally is responsible for cholangitis due to regurgitation. He also has stopped utilizing splints for the anastomosis. His end results have been better in cases where no splints were used.

Intrahepatic cholangio-jejunostomy. This method should be added to the surgeon's armamentarium, for it is a very expedient method in a poor risk patient where the dissection at the hilus of the liver would be long and tedious because of adhesions, marked scarring, loss of landmarks and troublesome bleeding.

In cases of long standing obstruction, the liver function may be and usually is markedly decreased because of biliary cirrhosis. Portal hypertension with esophageal varices may be present. But because of the obstruction, different degrees of liver enlargement will occur and with long standing obstruction the liver is usually very large unless the condition is almost terminal in which case the liver starts to shrink in size. The fact that the liver is large and the intrahepatic bile ducts dilated and thickened and the Gibson's capsule thickened the operative procedure which has been adequately described by Longmire (1,^{5,6}) becomes not too unusually difficult.

CASE REPORT WITH A THIRTEEN MONTH FOLLOW-UP

Mrs. E. A., a white female aged 44 was admitted to the Allentown Hospital on March 13, 1953 for the investigation and treatment of recurrent jaundice. On January 24, 1950 a cholecystectomy for chronic calculous cholecystitis was performed on her at a private hospital (unfortunately the record of the operative findings and the procedure carried out could not be obtained). She had a drain (not a T tube) and she claims that she started draining greenish-yellow bile immediately after the operation and kept on draining until March 25th, when the drainage stopped spontaneously. She felt well until July, 1950 when she became jaundiced, her urine became dark brown and her stool acholic. After about three weeks her jaundice cleared up but it recurred again in November, 1950, and this time accompanied by anorexia, pruritis, weakness and tiredness. She went through nine such episodes. With similar symptoms she presented herself to the hospital.

Physical examination revealed a relatively well-nourished female weighing 124 lbs. having bronze colored weather-beaten like skin, and whose apparent age was greater than her chronological age. The temperature was 99.6, pulse 80 and respirations 18. The sclera were markedly icteric. The head, neck, chest and heart were normal. The abdominal examination revealed a markedly enlarged liver, the anterior edge which was sharp and uneven being a good hand's breadth below the costal margin. The spleen was not palpable. A diagnosis of stricture of the common duct with biliary cirrhosis and mild hepato-cholangitis was made. The laboratory reports done were WBC - 10,600; P-70; L-26; M-2; B-2; coag. time-3 min.; icterus index - 40.2; serum protein-5.2gm; A/G ratio-1.7/1; serum bilirubin- 4 plus (immediate direct); alkaline phosphatase-9.1 units; prothrombin time-16.5 sec. (85% conc) control 15.5 sec; B.U.N. - 9.5 mgm; urine was positive for bile; stool was acholic.

She was placed on a high protein, high vitamin including Vitamin K, low fat diet and was given four pints of blood. On March 18, 1953 she was operated upon under continuous spinal anesthesia. Dense fibrotic adhesions between the stomach, duodenum, hepatic flexure, omentum and the liver bed were encountered upon the entrance of the upper abdomen. These were carefully dissected off and the hepato-duodenal ligament identified. A knob of fibrous tissue measuring approximately 1½ cm. in diameter was found at the mid-lateral portion of the hepato-duodenal ligament. After dissecting this free the distal end of the common duct was found attached to it. Further dissection

was carried out proximally, the portal vein and hepatic artery were dissected free up to the hilus. There was no sign of the proximal common or hepatic ducts except for a narrow, fibrotic, tubular-like atresic structure which disappeared in the hilum of the liver. By this time the operating time had come up to approximately three hours so a decision was made to put drains in the Morrison's pouch and mark the distal duct with the idea of re-operating within several days. Post-operatively the patient did relatively well; on the third post-operative day some bile started draining through the cigarette drains. This drainage increased for the next three days and then decreased so that by March 30, 1953 the drainage had practically stopped again. Her blood picture was good because of the transfusions, so on April 1, 1953 she was re-operated upon. Much to the surgeon's chagrin the adhesions were surprisingly dense about the hilus of the liver. When these were separated, oozing was so marked that it was hopeless to try and proceed with further dissection. A decision was then made to do an intra-hepatic cholangic-jejunostomy. The procedure carried out was slightly different from that described by Longmire. The triangular ligament did not have to be cut because of the marked enlargement of the liver, the liver, literally speaking was busting at the seams. Mattress sutures were placed in the anterior inferior edge and immediately after the first needle puncture white bile just kept pouring out. When the liver was cut into approximately $2\frac{1}{4}$ inches a duct measuring about 1 cm. in diameter was encountered. A wedge shaped piece of liver was removed, the duct mobilized for about 1 cm. Then the jejunum was severed about 12 inches distal to the ligament of Treitz and an end-to-end, mucosa to mucosa anastomosis of the intrahepatic duct to a Roux-y arm of the jejunum was performed using interrupted 4-0 silk sutures. The anastomosis was made around a long arm No. 18 French T tube which was inserted into the jejunum about 3 inches away from the open end with one arm threaded through the open end into the bile duct and the other arm threaded distally into the jejunum. The long arm of the T tube was brought out through a stab wound in the left anterior abdominal wall after purse string sutures were placed in the jejunum around the tube. The end-to-side entero-enterostomy was next completed, the raw liver area was covered with gelfoam, cigarette drains placed into the upper abdomen and the abdominal incision was closed. The patient spiked a temperature of 103° after the operation, due to a blood transfusion reaction but after that made a very uneventful recovery. The temperature ranging from 99° to 101° the first five days, then gradually returned to normal within 10 days or so. The stool became colored within six days. Bile drained well through the T tube. The cigarette drains which drained a slight amount of bile

stained sero-sanguinous fluid were removed by the eighth post-operative day. On April 23rd, a cholangiogram was performed by the instillation of a contrast material through the T tube placing the patient in a Trendelenberg position. Good communication between the right and left hepatic radicals was seen present through a remnant of the common hepatic duct. (Fig. I).

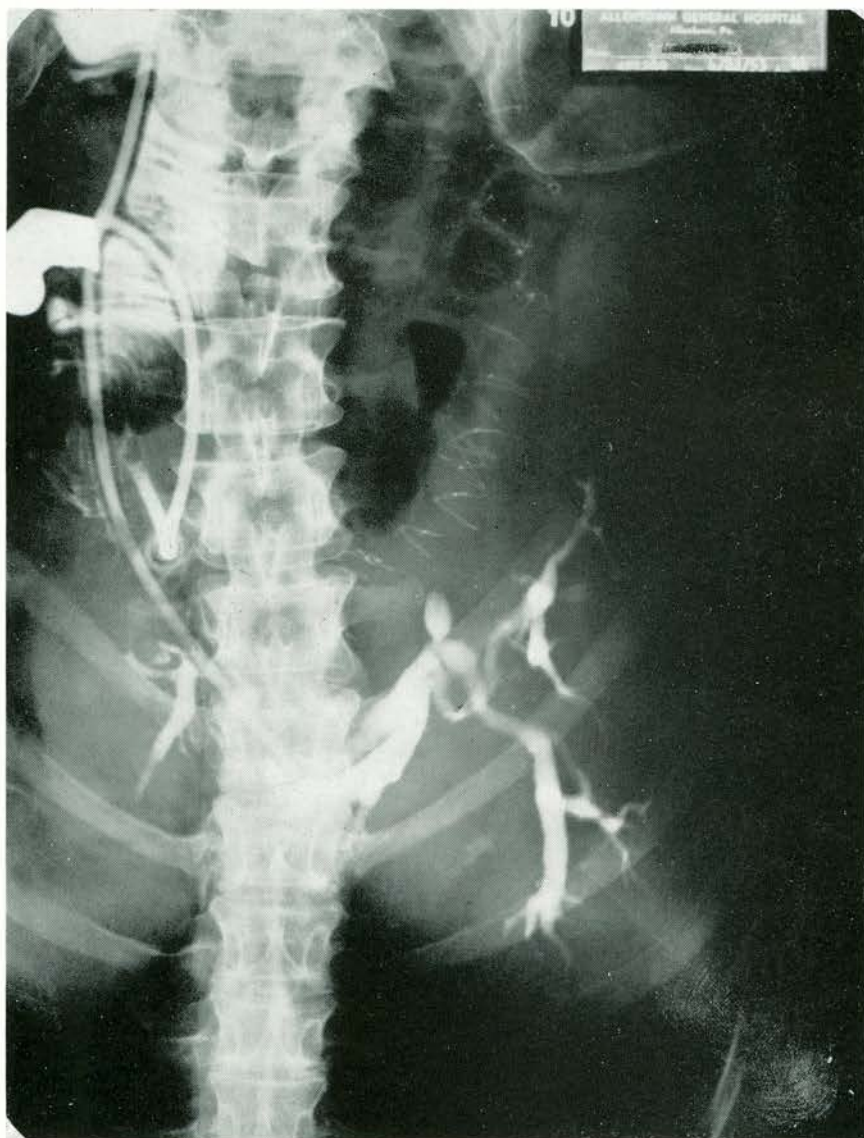


FIGURE I. Post-operative cholangiogram

The patient was discharged on the following day. The pathological report on the liver was chronic hepatitis.

Six months post-operatively the T tube was removed and the opening closed spontaneously within 24 hours. On January 23rd, nine months after discharge, the patient weighed 133 lbs. She generally looked well. The color of the skin appeared normal. She ate everything and had no chills. The liver was no longer palpable. The serum bilirubin was 0.15 mg (direct) and 2.2 mg (indirect); icterus index 10.2; total protein 6.8 gm.; cephalin flocculation - 48 hrs. - 2 plus.

She was again seen in June, 1954 and she feels perfectly well.

Patient was seen in December 1954, she was symptomless and there was no recurrence of jaundice.

SUMMARY

A short review of biliary stricture is presented along with another case report of a successful intrahepatic cholangio-jejunostomy performed upon a patient upon whom a different procedure would have been extremely hazardous if not unsuccessful. The use of a long arm T tube not only gave the anastomotic line a chance to heal without stenosis but also gave the operators an opportunity for a roentgenological follow-up with visualization of the hepatic radicals.

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