Intensive Cardiac Care and Digitalis Glycosides

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Toxicity Caused by Foxglove — “I found him incessantly vomiting, his vision indistinct, his pulse forty in a minute.” William Withering, 1785.

There are unique problems attendant to the administration of digitalis glycosides in intensive cardiac care units. What are the diagnostic signs of overdosage and under what circumstances can the coronary care unit nurse assist in achieving early diagnosis? What is the most effective treatment for digitalis toxicity in patients who require surveillance in intensive care units?

Although investigators differ in their choice of glycoside and the method of administration, there is agreement that digitalis compounds are of value in coronary care units for managing certain supraventricular arrhythmias. Digitalis glycosides are also a major aspect of therapeutic regimens for the treatment of pump failure, particularly when pump failure is manifested by congestive heart failure rather than cardiogenic shock. The term “coronary care” suggests primarily management of acute myocardial infarction, but in most centers such care includes treatment of life-threatening arrhythmias not necessarily associated with obstructive coronary vessel disease. The etiologic role of digitalis is more quickly identified if ventricular arrhythmias appear or if there is marked bradycardia secondary to sinus node inhibition or advanced heart block. However, we have not sufficiently emphasized that digitalis in excess not only depresses A-V transmission, but also enhances impulse formation in atrioventricular junctional tissue. Under these circumstances, A-V dissociation with nonparoxysmal A-V nodal tachycardia may appear. An irregular ventricular response to atrial fibrillation may become regular when A-V dissociation occurs. Another source for confusion exists in the small group of patients with atrial fibrillation in whom digitalis excess is characterized by an increased rather than a decreased ventricular response to fibrillatory impulses. These aspects of digitalis toxicity require personnel in intensive care units to be aware of the possibilities signified by acceleration or regularization of the pulse rate.

A variety of excellent myocardial depressant drugs are available for treatment of ventricular arrhythmias. These agents include lidocaine, quinidine, procaine amide, propranolol and potassium, if there has been depletion of body potassium stores. However, depressant drugs are contraindicated if the ventricular arrhythmia occurs in the presence of heart block or profound sinus bradycardia. This combination of phenomena is not infrequently found with digitalis intoxication since digitalis in excess may precipitate advanced heart block (particularly in patients with rheumatic heart disease) or it may further inhibit conduction in a chronically diseased atrioventricular junction. Recent studies of ventricular dysrhythmias assign a fundamental etiologic role to underlying bradycardia. Episodes of rapid or chaotic ventricular activity may be secondary to the inadequate cardiac output of a markedly slow ventricular rate. Cardiac pacemakers and more specifically the demand pacemaker can provide major assistance in the management of these clinical situations. There are theoretic dangers to the use of electric pacemakers in the presence of digitalis overdosage. Fortunately, however, many clinicians in this country and overseas have demonstrated that endocardial pacing may be used safely in subjects with digitalis toxicity. Such pacing can maintain an adequate cardiac output until advanced myocardial manifestations of drug excess disappear. On rare occasions even demand pacing can precipitate ectopic ventricular rhythms; in these instances, propranolol or a comparable agent should be used in addition to pacing. Temporary endocardial demand pacing may be valuable not only in the treatment of digitalis intoxication, but also in anticipation of untoward effects, as for example when digitalis or depressant drugs are administered to patients with sinus bradycardia or heart block.

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What about the “unique advantages” ascribed to diphenylhydantoin (Dilantin)? Preliminary observations suggest that diphenylhydantoin may improve conduction through A-V junctional tissue with simultaneous suppression of digitalis-induced ventricular arrhythmias. Unfortunately, in the dosage range necessary for suppression of these arrhythmias, diphenylhydantoin often causes significant impairment of A-V conduction. This is further evidence that the pharmacologic management of digitalis-induced conduction abnormalities frequently entails unacceptable risks unless a temporary demand endocardial pacemaker is functioning or immediately available.

Digitalis toxicity is one of the most frequent forms of untoward drug responses. Fewer episodes of intoxication would occur if more clinicians appreciated the linear relationship between the dose of digitalis and its inotropic effect. The total digitalizing dose need not be given to achieve a clinically satisfactory initial response. In patients with congestive failure secondary to acute myocardial infarction, digitalis should be given in smaller than average dosages. Certainly, from the diagnostic viewpoint, evaluation of arrhythmias is enormously complicated by the coexistence of acute myocardial infarction and digitalis toxicity since the same rhythm and conduction abnormalities may occur in both states.

The problem of toxicity is aggravated by misconceptions perpetuated in standard reference sources. A 1969 syllabus prepared for medical students notes that approximately 50 percent of digoxin is absorbed from the gastrointestinal tract. This instruction conflicts with recent repeated observations using radioisotope techniques which indicate that between 80-90 percent of orally administered digoxin is readily absorbed. Many faculty members teach that the initial loading dose of digoxin is 2-3 mg. Jelliffe recently emphasized that in most instances a loading dose of 0.75-1.5 mg of digoxin in three divided doses approximately five hours apart is entirely adequate. Indeed, administration of the proper maintanance dose would result in adequate total body digoxin stores in approximately eight days even if no loading dose were given, if renal function is normal. It may be prolonged to the range of 18 days for a patient with no renal function. Possibly these dosage recommendations will be challenged, but surely we must commend the sound therapeutic principles enunciated by Jelliffe. Total glycoside losses from the body are proportional to the total amount of drug present and a reasonable estimate of daily losses can be readily achieved in patients with normal renal function, as well as in those with reduced renal function. No longer need initial loading doses be given intuitively since the likely range of reasonable loading doses can be determined for most patients. A rational approach to the administration of digitalis is possibly the most important single measure in the prevention of digitalis intoxication in coronary care units, hospital wards, and in office practice.

Reference

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THE SERENE BEAUTY OF WEDGWOOD THROUGH INDUSTRIAL HYGIENE

Free silica may be the only constituent in crushing flint, or it may occur in the dust mixed with other substances, either naturally, like the quartz in granite, or artificially like the ground flint in earthenware pottery. In 1720 John Ashbury introduced finely powdered calcined flint into the body of his chinaware so as to produce on firing a remarkably white and hard product. In 1865 in her Life of Josiah Wedgwood, Eliza Meteyard states that at first the flint when reduced to powder in large iron mortars was extremely injurious as the dust was inhaled by the workmen and produced lung diseases of various kinds. She relates in detail how a painter employed in the potteries was struck by overhearing the story of the fatal effects of flint. It occurred to him that the crushing could be performed by millstones under water, and the adoption of this method led to a considerable reduction in the amount of dust. Hunter, D.: The Diseases of Occupations, (Third ed.), Little, Brown & Co, Boston, 1962

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