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# Family Practice Grand Rounds

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## The Cardiac Cripple

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**DR. DAVID SCHUTT** (*Chief Resident in Family Practice*): Today, Family Practice Grand Rounds involves the case of Mr. T., who is a 47-year-old married white male with severe atherosclerotic coronary artery disease and secondary congestive heart failure. Mr. T. was recently admitted to the Malcolm Grow USAF Medical Center with a left pleural effusion and increasing dyspnea of three months duration. Dr. Royal (Mr. T.'s family physician) will present the past medical history and I will discuss the patient's recent hospitalization. Dr. Selle (clinical pharmacist) will discuss the clinical, laboratory, and mathematical techniques for managing digoxin therapy. Major Steele (clinical social work consultant) will discuss the effect that endstage heart disease has had on the patient and his family. He will suggest methods for dealing with this problem.

**DR. LOUIS ROYAL** (*Director, family practice*

*residency and Mr. T.'s family physician*): Mr. T.'s past medical history includes the onset of angina pectoris in his late 30s, a myocardial infarction at age 41 (in 1972), and a second myocardial infarction four months later. Because Mr. T. subsequently developed multifocal premature ventricular contractions, he was started on procainamide. Two years later, he suffered a right cerebrovascular accident with left hemiparesis. Mr. T.'s problems following his stroke were persistent angina pectoris, exercise induced dyspnea, limited use of his left arm, an adverse reaction to procainamide, and low self-esteem with significant depression.

In February 1977, the patient presented to the Family Practice Clinic with concern over impotence which he related to the ingestion of clofibrate. He noted that temporary cessation of this drug seemed to improve his symptoms. Several months of monitoring with assessment of cholesterol and triglyceride levels while off clofibrate showed some improvement of his impotence, while cholesterol and triglycerides remained in an acceptable range on diet alone. However, the patient became very concerned over the potential dangers of "elevated fats," and wished to continue the clofibrate and accept his impotence. Organic factors as a cause of impotence were nega-

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tive, and serial observations of Mr. T. made it much more likely that his impotence had a psychologic basis.

In December 1977, Mr. T. was hospitalized with symptoms of marked fatigue, without an associated increase in dyspnea. Laboratory studies showed leukopenia, thrombocytopenia, elevated erythrocyte sedimentation rate, a positive antinuclear antibody (ANA), a positive lupus erythematosus prep, positive VDRL, and positive Coombs test. The patient was admitted for evaluation of suspected procainamide induced lupus. At the time of admission, Mr. T. had been on procainamide for four years. Of interest, an ANA had been negative one month prior to this admission. Mr. T.'s platelet count reached a nadir of 10,000/ $\mu$ mm on the third day of admission. He was immediately started on prednisone therapy and responded dramatically to it, with an increase in platelet count and white blood cell count.

In February 1978, Mr. T. had a severe bout of chest pain and was admitted to the hospital. A subendocardial myocardial infarction was documented. In April 1978, he was again admitted, this time in heart failure.

Mr. T.'s family history shows that he has a 13-year-old daughter with congenital rubella syndrome and deafness. He has two healthy sons living away from the home and his wife is in good health. Though the patient's daughter was deaf with significant attendant problems in education and care, it was clear in many interviews with the family that the patient himself occupied the primary "sick role" in the family. In interviews with his wife, the acceptance of the patient's illnesses was taken as a part of life, though frequent trips to the hospital interfered with her job status, sometimes with feelings that "it would be easier to have him in the hospital."

This brings us to his present admission for a left pleural effusion. His medications at the time of admission included clofibrate, digoxin, furosemide, potassium supplementation, and nitroglycerin.

DR. SCHUTT: On admission, Mr. T. was a thin, slightly depressed middle-aged male, appearing slightly older than his stated age, friendly and cooperative. Vital signs showed a pulse of 88 beats per minute, blood pressure 110/60 mmHg, temperature 98.2 F, weight 62 kg; HEENT, no xanthelasma, no jugular venous distention; Lungs,

clear; Heart, regular rhythm, II/VI systolic murmur, S<sub>3</sub>; Abdomen, no hepatosplenomegaly, no hepatojugular reflux; Genitals, testes normal size; Pulses, all palpable with bruits over both femorals; Extremities, no edema, xanthomas on areas of Achilles tendons, and several xanthomas present on forearms; left arm with some muscular atrophy; Skin, no rashes; and Central Nervous System, weakness and poor fine control left upper extremity and left leg. Laboratory: All admission laboratory work was normal except for a chest x-ray which demonstrated a left pleural effusion, which was confirmed on a decubitus film.

The patient was maintained on all admission medications, placed at bedrest, and put on a salt and fat restricted diet. His weight remained stable between 61.8 and 62.7 kg. A serum digoxin level was drawn eight hours after his last oral dose of 0.25 mg. A value of 0.7 ng/ml was reported, slightly below our accepted therapeutic level.

On his third hospital day, Mr. T. experienced an acute exacerbation of dyspnea with the appearance of basilar rales bilaterally. His dyspnea improved subjectively with the attending physician's arrival on the ward. His furosemide and digoxin were then increased empirically. Mr. T. expressed displeasure at the physician's slow response to his page. According to Mr. T., the physician apparently did not realize how ill he was! He complained repeatedly that his medications were not delivered on time and that his symptoms would worsen if he did not receive them *exactly* on time.

Serial chest radiographs showed resolution of the effusion over the next seven days. His weight decreased to 60.4 kg and his dyspnea improved.

Toward the end of his hospitalization he developed diarrhea. Mr. T. was sure this was due to his furosemide and he felt the drug should be stopped.

About four days after increasing Mr. T.'s daily digoxin (from 0.25 mg/day to 0.25/0.375 mg alternating days), a repeat serum digoxin level was drawn. A value of 0.8 ng/ml (normal range 0.9-1.5 ng/ml) was obtained. Suspecting an error, another level was immediately drawn. This second value was zero ng/ml. A creatinine clearance was performed and showed a clearance of 65 ml/minute, which was unchanged from one year previously. At this time, Mr. T.'s diarrhea was not improving, and his serum digoxin levels were completely inappropriate for the amount of drug Mr. T. was receiving. A consultation was requested of the

clinical pharmacist for evaluation of digoxin therapy. His findings will be discussed later.

Because Mr. T. expressed considerable anxiety over the type and timing of his medication and also appeared to have subjective symptoms out of proportion to his objective findings, we also asked our clinical social work consultant to interview Mr. T. His findings and advice will be discussed later.

DR. DONALD MARSHALL (*Staff family physician*): Since the serum digoxin levels did not seem to correlate with Mr. T.'s dosage, was any thought given to impaired absorption?

DR. SCHUTT: At the time of discharge Mr. T. was changed to the liquid form of digoxin which is completely absorbed.

DR. ROBERT SELLE (*Clinical pharmacist*): I would like to address briefly the subject of digoxin dosing. As you are well aware, digoxin is primarily metabolized via the kidneys, approximately 60 to 90 percent of the administered dose being excreted unchanged in the urine.<sup>1</sup> Thus, one must adjust dosage in patients with compromised renal function. It is also well known that there are bioavailability problems with digoxin.<sup>2</sup> For this reason, it is best to use only a product with demonstrated bioavailability.

Digoxin, like most drugs, is subject to variable pharmacokinetics. Problems in bioavailability have already been mentioned. Concomitant administration of anticholinergic drugs may increase the bioavailability of digoxin tablets and subsequently result in higher serum levels.<sup>3</sup> Digoxin distributes poorly in fat, thus dosages should be calculated on lean body mass.<sup>4</sup> Also, increased biotransformation may occur in patients with hyperthyroid disease; the opposite being true for hypothyroid patients.<sup>5</sup> These problems are compounded by the fact that there is a high percentage of patients who fail to take medications as they are prescribed. Doses are missed for various reasons, and some patients still believe if one dose is good then two are better. Patients must be continually questioned concerning compliance to their drug regimen.

A couple of days prior to admission a serum digoxin level was drawn for Mr. T. The results returned during his admission and were subtherapeutic (0.7 ng/ml). About this time, the patient's symptoms of congestive heart failure worsened and the dosage of digoxin was increased empirically to 0.25 mg alternating with 0.375 mg

daily. Another digoxin level was ordered six days later in order to assess his steady state with this dosage. During this time, Mr. T. was doing very well clinically. At the time of discharge, the digoxin level came back again subtherapeutic (0.8 ng/ml)—normal for this laboratory is 0.9-1.5 ng/ml. Since the patient was in the hospital, we have to assume that he was compliant. We also tested our brand of digoxin (Lanoxin) for potency. The results showed them to be of the correct strength. The only other possibility would be a flaw in the assay method. There was no way for us to expeditiously determine this.

DR. ROBERT KITCHEN (*Third year family practice resident*): Are you suggesting that serum digoxin levels are of little help in the management of patients on this medication?

DR. SELLE: The clinician would be wise to use serum blood levels only as an adjunctive tool in the management of the patient. Let the clinical assessment of the patient be the determinant factor. This is especially true for digoxin, for which there is considerable evidence that concentrations in plasma correlate with its inotropic effect, ie, the inotropic effects of digoxin increase linearly with increasing dose and there is no apparent threshold for such an effect.<sup>5</sup> Thus, many patients with congestive heart failure may respond adequately with subtherapeutic serum levels of digoxin. In such patients, it would not be necessary to increase the dosage just to "satisfy" the therapeutic range.

As with any laboratory test, there are pitfalls in the determination of serum digoxin. Values vary from one laboratory to another, since different laboratories may utilize different methods.<sup>4</sup> However, in spite of these problems, the serum levels of digoxin correlate better with pharmacologic effects and toxicity than does the dose of digoxin.<sup>6</sup> Thus, serum levels can be very useful in explaining unanticipated responses, in serving as a guide to modifying dosage, especially in patients with severely compromised renal function, and in monitoring patient compliance. There is evidence that digoxin toxicity can be reduced through serial monitoring of serum levels of the drug.<sup>7</sup>

There is a formula which some clinical pharmacists use to estimate serum digoxin levels.\* It is

\*Suggested reading on pharmacokinetics: Wagner JG (ed): *Fundamentals of Clinical Pharmacokinetics*. Hamilton, Ill, Drug Intelligence Publications, 1975

Table 1. Relationship Between Clearance and K*	
Creatinine Clearance	K
110—120 ml/min	0.408
90—100 ml/min	0.348
70— 80 ml/min	0.324
50— 60 ml/min	0.303
30— 40 ml/min	0.252
10— 20 ml/min	0.210

\*Rough correlations mathematically determined by co-author (R.S.) based upon work published by Dettli L et al<sup>8</sup>

simple to use, and yields results which are fairly accurate. The formula is as follows:

$$C_{ss} = \frac{F \times D}{V_d \times K \times T}$$

C<sub>ss</sub> = Steady state of the drug (desired plasma level)

F = Bioavailability of the drug (fraction of oral dose absorbed)

D = Maintenance dose

V<sub>d</sub> = Apparent volume of distribution

T = tau = Dosing interval

K = Elimination rate constant

For digoxin, C<sub>ss</sub> is generally 0.9 to 1.5 ng/ml, F is 0.6 (Lanoxin tablets), V<sub>d</sub> is 7 to 10 liters/kg, K varies with creatinine clearance, and tau is usually 1 day but may be greater in certain patients with severely compromised renal function. Table 1 presents various K values as related to creatinine clearance. Note that as the clearance decreases, K also decreases. With this information, the clinician can accurately determine the maintenance dose of digoxin.

Example: Mr. T. is a 46-year-old white male with congestive heart failure. His creatinine clearance is 60 ml/min. Mr. T. weighs 60 kg (lean body mass). What would be the maintenance dose of digoxin in this patient?

Rearrangement of above formula to solve for D:

$$D = \frac{C_{ss} \times V_d \times K \times T}{F}$$

$$D = \frac{1 \mu\text{g/liter} \times (8 \text{ liter/kg} \times 60 \text{ kg}) \times 0.303 \times 1 \text{ day}}{0.6}$$

$$D = 240 \mu\text{g}$$

Thus 0.25 mg tablet per day should yield a steady state serum level of digoxin (achieved after five half-lives) of approximately 1 μg/liter or 1 ng/ml.

DR. SCHUTT: We would now like to introduce Major Steele, our clinical social work consultant, for further insights into the social and emotional dynamics of Mr. T.

MAJOR JOHN STEELE, JR (*Clinical social work consultant*): A basic treatment philosophy for patients, as they move in the direction of either an improved or stabilized physical condition, is to work for a return to their home and community living. Such was seemingly the medical treatment intent of the medical team as they planned for Mr. T.'s discharge from the hospital. But as is so often the case, "the best laid plans of mice and men" go awry, especially when the patient is resistant to such plans and makes the remark upon leaving the

hospital, that he would "be back." When considering such behavior one should take into consideration several points that might underlie the patient's behavior. They are as follows:

1. In the case of Mr. T., as we view the possibility of his becoming a chronic cardiac cripple, we see him undergoing much role change involving his personal role concept, his personal role expectations, and his present and eventual role performance within the hospital and home environment. The patient has a background history of having been a highly successful and success oriented military noncommissioned officer. Upon retirement from the service he began working as an appliance salesman for a nationally known firm, and compiled an excellent and enviable work record. He was viewed by family, peers, and himself as a hard working and highly productive individual. His own high self-concept as father, husband, and citizen was very much a factor in his total pattern of self-esteem and self-performance. He expected much of others but he gave much of himself to and for others. He was an individual who gave his best at all times. To do otherwise was unthinkable.

2. Subsequent to his debilitating illness Mr. T. has undergone a complete role reversal experience; one that has lowered his feelings of self-worth and caused his whole outlook on life and himself to change. He no longer views himself as the protector and major provider for his family. His wage earning capacity has decreased, though he stresses that if he should die that he has made satisfactory arrangements for his family that would "keep the wolf from the door." From a position of strength and autonomy, the patient has been reduced to one of dependency and inertia. His self-concept is shattered and he must now face the often devastating emotional task of letting others—namely his wife and sons—take over the management of the home and, in many respects, his personal existence. Intellectually, Mr. T. is capable of letting go of some of these role tasks; but emotionally, he is extremely resistant to such a process and requirement. Inherent in this struggle is the patient's need to still be recognized as a person of worth and dignity. Such needs are often expressed by such patient manipulative action as excessive demands of hospital personnel, consistently inquiring of the physician's competency to treat the patient's particular illness, and a need to

criticize the actions and values of those with whom the patient comes into contact on a daily basis. In essence, the major defense mechanisms used by the patient are an unconscious denial of his probable inability to function as a totally recovered individual and his tendency to project many of his own frustrations and failures onto others—especially those on whom he is most dependent for survival.

3. Personally, Mr. T. is confronted daily with the possibility that he might die. This he can discuss in an intellectual manner; but in reality, he avoids discussing the inevitability of such an occurrence. Rather, he puts the onus of responsibility upon the physician, stressing that with good and complete medical care that he will eventually be as good as new. As noted earlier, the patient avoids contact with people and in effect appears to be isolating himself from family members and friends. This can be seen by the patient's lack of marital relations with his wife, his avoidance of parental responsibilities with his children, and a decrease in social contact with many of his friends. Such avoidance guarantees no failure: to attempt to perform and then fail at the various life tasks set before him, whatever they are, would only serve to weaken his already weak ego and self-concept.

In conclusion, there are several recommendations that could be made that might enable Mr. T. to become more involved in day-to-day living activities:

A. Have both Mr. and Mrs. T. be seen jointly by the family practice social work consultant and his physician for couples therapy. By doing so, the couple would be afforded the opportunity of discussing the effect that the patient's illness possibly has had on their marital relationship. This is particularly important as efforts are made to aid Mr. T. to reach a psychological resolution of his impotence which, as stated earlier, appears to have no organic basis.

B. Structure the patient/physician encounters so that physical and emotional matters are not discussed at the same time. This will enable the family physician to deal with each area of concern separately and neither issue will be diluted. This is particularly true when counseling patients who find dealing with personal feelings difficult.

C. Since Mr. T. has a great desire and need to be of help to someone, perhaps he could be

encouraged to become a volunteer within the hospital and work with patients who could benefit from his knowledge of the military system and also the hospital regimen. It is often true that physical symptoms disappear when one feels needed; especially those of psychological origin. Two sources that might provide such guidance for the patient are the Red Cross Volunteer Program and the occupational therapy department's Work Therapy Program.

D. It is of the utmost importance that all members of the family practice treatment team (the physician, the social work consultant, the clinical pharmacist, and the nurse) communicate with each other regularly regarding their input and recommendations concerning the patient's total care and recovery process. Only by doing so can manipulation by the patient of the family practice treatment team members be avoided and an effective treatment program implemented.

DR. SELLE: Major Steele, do you feel that Mr. T. has the potential for intentionally discontinuing his medications as an outpatient in order to manipulate his way back into the hospital?

MAJOR STEELE: Yes, if Mr. T. does not get satisfaction as an outpatient, I feel he will manipulate the medical establishment in order to receive satisfaction and recognition of his illness by making an effort to be readmitted. Potentially this could occur through discontinuance of his medications.

DR. ROYAL: I concur that Mr. T. may have this potential; however, in my visits with him, I think it is far more likely that he will take medications *exactly* as prescribed. He is in a tenuous state of balance regarding his congestive failure; and he realizes the serious implications of his illnesses. I think his responses are more likely to be a pattern of blaming his physicians when medications and treatment plans fail; he shifts the responsibility for his health to his physicians and further perpetuates his mold of dependency.

DR. CHRIS MARQUART (*Family practice staff physician*): How is Mr. T. doing now as an outpatient? Does he still have diarrhea?

DR. ROYAL: Relatively speaking, he is doing quite well. He has returned to approximately where he was prior to his admission. He moves about in his home and yard with only occasional angina. His diarrhea has not recurred since hospitalization.

DR. SCHUTT: I would like to thank the participants and audience for their contribution to the Grand Rounds.

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