ANIL AGGRAWAL'S
Internet Journal of
Forensic Medicine and Toxicology
The world's first online journal devoted entirely to forensic medicine and toxicology · Published 1 January and 1 July

VOL. 10, NO. 2
Downloaded from www.anilaggrawal.com · ISSN 0972-8074 (Online) · https://www.anilaggrawal.com/ij/vol_010_no_002/reviews/tb/page004.html

Book Reviews: Technical Books Section

...THE VERY FIRST BOOK ON FORENSIC ASPECTS OF CARDIOVASCULAR DISORDERS ...The very first book on Forensic aspects of cardiovascular medicine...Should be useful not only to forensic and medicolegal professionals but also to cardiologists and cardiovascular surgeons...

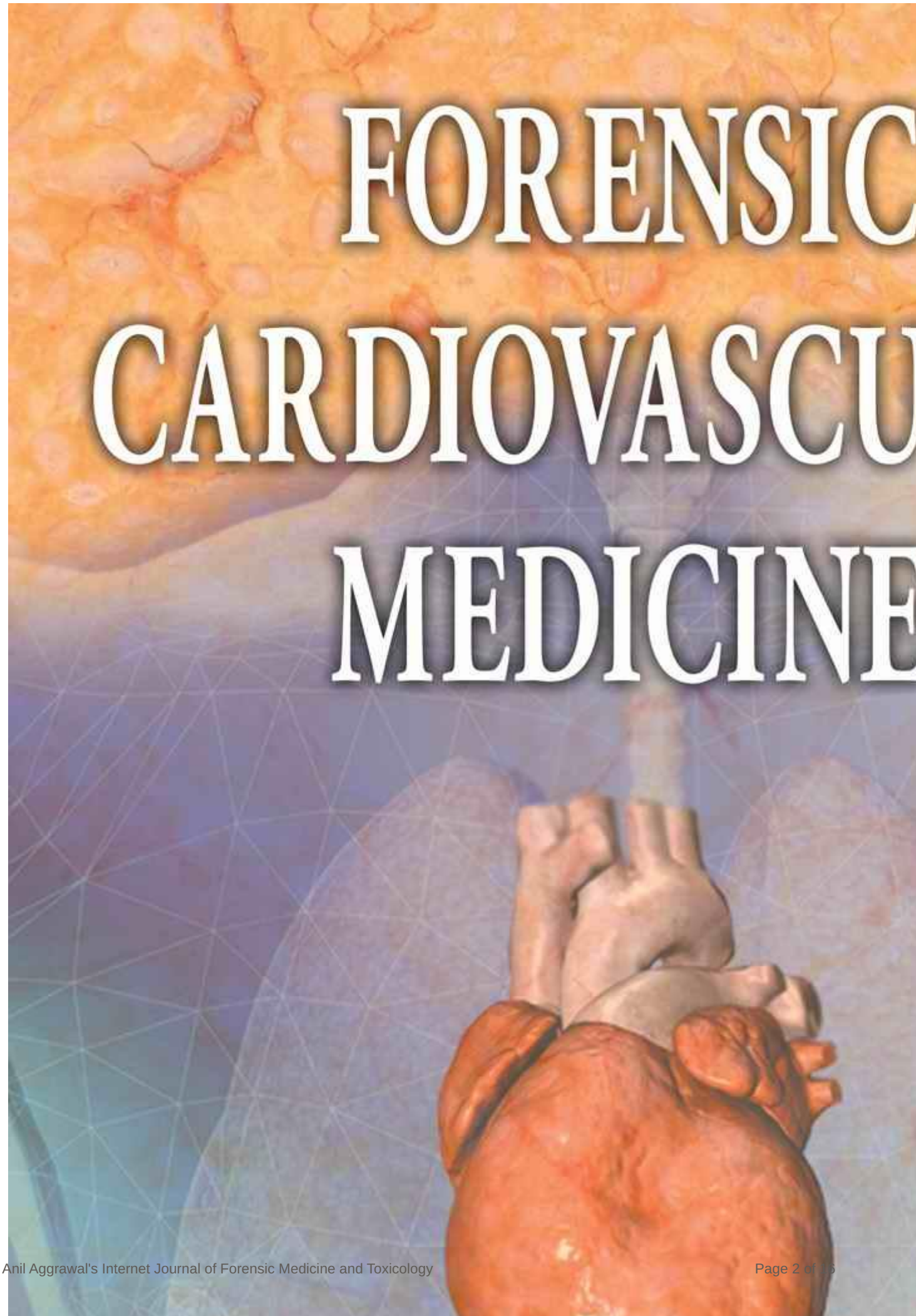
Forensic Cardiovascular Medicine, 1st Edition, by Basil RuDusky, Hard bound, 9.3" x 6.2" x 0.8". CRC Press LLC, 2000 Corporate Blvd., N.W., Boca Raton, Florida 33431, , . Publication Date April 15, 2009. 210 pages, ISBN-10: 1420094319; ISBN-13: 978-1420094312 (alk. paper). Price: $99.95

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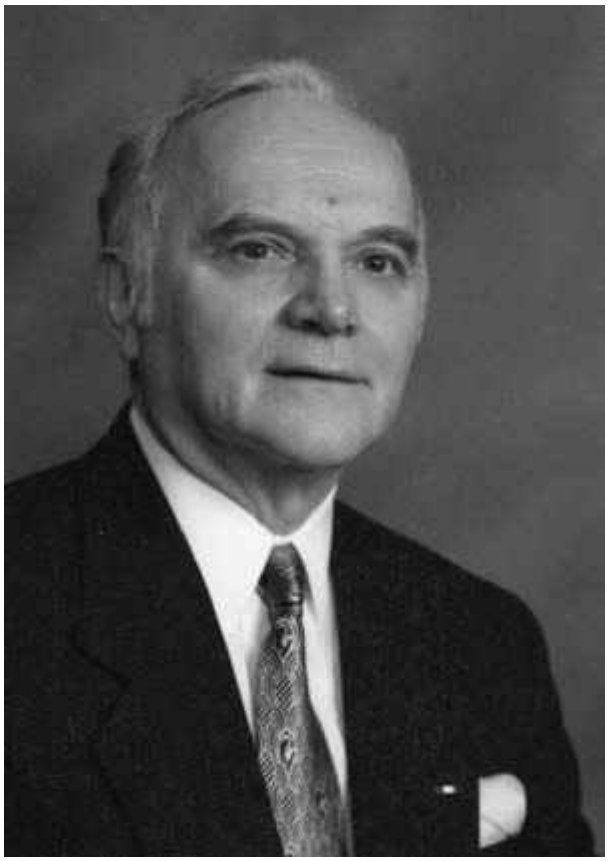
Read an interview with the author Dr. RuDusky by clicking here.

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FORENSIC
CARDIOVASCU
MEDICINE

The book under review is a unique resource for medical and medico legal professionals. It covers clinical aspects of the investigation of cardiac disease in a forensic context. Dr. Basil RuDusky's has had a vast experience in dealing with medico legal cases dealing with cardiovascular trauma, and in this book, he draws upon his experiences to churn out a masterpiece. The book emphasizes some of the most frequently encountered cardiovascular medical problems facing the medical examiner and forensic medical specialist. An introductory overview covers death certificates, autopsies, and the role of the medical examiner. Enough emphasis is given on how to deal with litigation concerning claims of posttraumatic cardiac injury which have been steadily increasing. The increase in litigation is often due to a presumption of injury or cardiac problem actually unrelated to an injury or, in fact, without substantial evidence for aggravation of the claimants' prior cardiac status. The medical-legal implications of blunt chest trauma are horrendous in terms of cost and time, and all too often are plagued by erroneous or fallacious argumentation and reasoning. As the book informs us, it is especially important in cases of suspected or proven myocardial contusion that the forensic medical specialist obtains as much previous medical history, information, and records regarding the presence of previous heart problems as possible.

Basil RuDusky, author of Forensic Cardiovascular Medicine (Please Click to enlarge)



In the chapter entitled "**Coronary Arterial Afflictions**", among other things, RuDusky touches and elaborates upon the current controversy whether there is a role of *Helicobacter* and *Chlamydia* organisms in the onset and progress of coronary artery disease and myocardial Infarction. Mention is also made of the curious condition of "**Myocardial bridging**". Muscle overlying the intramyocardial segment of an epicardial coronary artery (first mentioned by Reyman in 1737) is termed a myocardial bridge, and the artery coursing within the myocardium is called a **tunneled artery**. Myocardial bridging is said to occur in 5 to 80% of patients at necropsy and in 0.5 to 12% of

patients undergoing cardiac catheterization. This condition can have significant medico legal implications, which are discussed in the book.

View this book on Amazon

If we encounter a case of sudden death in a female during peripartum period of pregnancy, we know what all we have to look for. However we are informed that in such cases (peripartum period of pregnancy) dissection of a coronary artery (with or without association with coronary artery aneurysms), and we must not overlook this fact. Peripartum coronary arterial dissection usually occurs during the first 2 weeks postpartum. It is believed to be due to an enigmatic vasculitis and most often affects the left anterior descending coronary artery. Multiple dissections have been known to occur but are rare.

Click here to read an exclusive interview with Dr. Basil RuDusky.

Perhaps the most illustrative part of the book is section VII, which gives as many as 25 illustrative case reports, some of which have been excerpted below for the benefit of the reader. These case studies illustrate the topics discussed earlier in the book, including cardiac trauma, vascular abnormalities, specific cardiopathic disorders, and toxic, physical, technical, epidemiological, and social influences.

This book is the very first which deals with forensic and medico legal aspects of cardiovascular medicine. In my opinion, it is an absolute must for all medical and medico legal professionals and medical examiners.

Something about the book. The book is divided into 7 sections and 18 chapters. There are three appendices. The detailed list of contents is as follows:

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Excerpts from the book:

This book is the very first which deals with forensic and medicolegal aspects of cardiovascular medicine. It would thus be of immense use not only to forensic and medicolegal professionals, but also to cardiologists and cardiovascular surgeons. Toxicologists, especially forensic toxicologists would find section **Section V (Toxic and Physical Influences)** very useful. A range of cardiotoxic drugs and chemicals is discussed here along with their medicolegal implications. Here is a sampling of some of them (pages 97-8)

Carbon Monoxide

The primary physiologic effect of carbon monoxide (CO) on the body is its high affinity for hemoglobin, which produces tissue hypoxia. The end result is determined by the concentration and time of exposure, an individual's hemoglobin content, and the presence of coronary heart disease or previous heart muscle damage. Acute poisoning can cause ST-T abnormalities on the ECG and supraventricular or ventricular dysrrhythmia. Patients who have coronary artery disease are more likely to sustain angina pectoris, and near-fatal exposure has been reported as causing myocardial necrosis and cardiomyopathy in persons without coronary occlusion. Cardiac effects may appear acutely or may be delayed for several days. Chronic toxicity secondary to high-concentration exposure on a daily basis may also occur. Carbon monoxide's relationship to the production of coronary atherosclerosis remains debatable, perhaps less so in those concomitantly exposed to cigarette smoke and individuals exposed on a daily basis in specific occupations. The exposure of firefighters to carbon monoxide poses less risk, as this is generally not on a daily, continual basis or

in high concentrations because of protective external breathing devices. Guidotti, after an extensive review of occupational mortality among firefighters concluded the following:

1. There is no evidence of an increased risk of death from heart disease.

2. Sudden death, myocardial infarction, or fatal arrhythmia occurring on or soon after near maximal stress on the job are likely to be workrelated.

3. "Heart attacks" occurring away from work cannot be presumed to be work-related.

4. Job-related sedentary lifestyle and personal risk factors (smoking, lipidemia, hypertension, diabetes, family history, etc.) are more reflective of an adverse health effect being involved in the causation of heart disease in these individuals, rather than any work-related involvement.

5. Some evidence from clinical studies suggests a risk of sudden cardiac decompensation and a risk of a heart attack with sudden maximal exertion and after exposure to CO, and suggests that this did not translate into an excess risk of fatal heart attack at a later date.

6. If a firefighter did have a heart attack during or within a day after a fire, it would be reasonable to call it a work-related event.

Glueck et al., concluded that firefighters who later developed coronary heart disease (CHD) and those without CHD did not differ by history of smoke inhalation. They noted that cigarette smoking, hypertension, hyperlipidemia, and family history were more important risk factors than exposure to CO. Recent studies have indicated that up to 37% of patients following moderate to severe exposure to CO (poisoning) may sustain myocardial toxicity (injury) based on the elevation of cardiac enzymes and abnormal electrocardiograms. It was also concluded that short- and long-term mortality increased in this patient population. An investigative report by Kalay et al. studied 20 patients diagnosed as having CO poisoning and found that the extent of myocardial toxicity and dysfunction was dose- and time-related. The degree of left ventricular dysfunction (ejection fraction) correlated with the level of carboxyhemoglobin on blood sampling. All patients had normal coronary angiograms. It was concluded that most of the myocardial dysfunction dissipates within 24 hours.

Particulate Air Pollution

The harmful effects of air pollution are finally becoming a recognized and highly important factor in the development of disease and in the aggravation of preexisting disease states. These include cancer, lung and heart disease, and other conditions that receive less attention. Particulate air pollution is a mixture of suspended solid and liquid particles of various sizes and compositions. These include various kinds of soot, carbon monoxide, sulfur dioxide, nitrogen dioxide, ozone, many types of volatile organic compounds and gasses, pollens, dusts, spores, and a host of harmful legal and illegal substances produced by various industries.

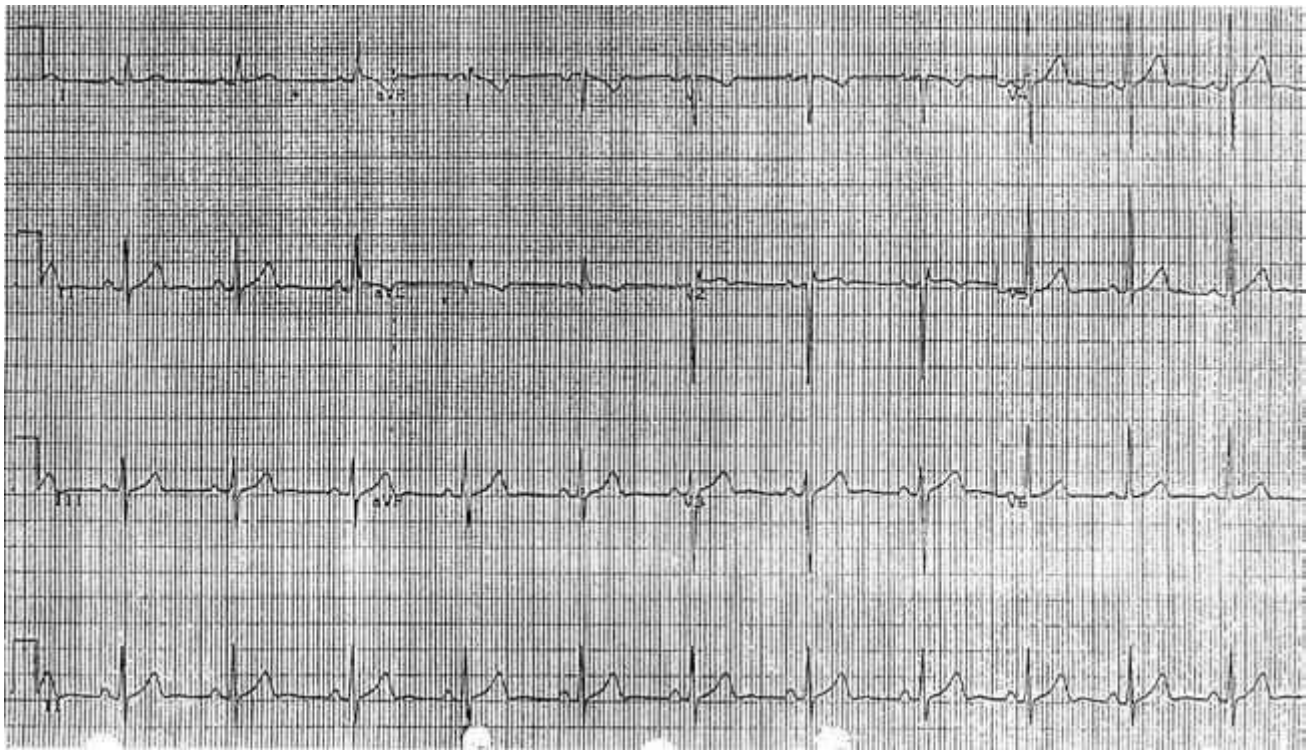
The production and progression of atherosclerosis resulting from air pollution is now proven fact, experimentally and epidemiologically. The subsequent effects of plaque production and rupture, coagulopathic and thrombotic effects, arterial vasoconstriction, systemic inflammatory response, and various neuro-humeral responses have been implicated in the production of angina pectoris, heart failure, and cardiac arrhythmia. These adverse effects can lead to myocardial infarction, a proclivity for enhanced artificial pacemaker activity, and sudden death.

The effects on the pulmonary system include acute and chronic bronchitis, pneumonitis, bronchial asthma, hypersensitivity syndrome, and lung cancer. Allergic rhinitis and dermatitis are also frequent accompaniments of particulate air pollution. Unbridled diesel fumes are one of the most hazardous producers of various disease states in today's world. This has been reviewed in more detail in the book ***Your Car Can Be Hazardous To Your Health***.

So much for the toxicology portion. I would imagine that one of the most useful segments of this book is **Section VII**, where the author gives many "**Illustrative Case Reports**". This is an important section in that these case reports serve to consolidate information given in earlier parts of this book. All cases are followed by their forensic implications. It is for this reason that we at the editorial office decided to give some excerpts from this section of the book too. Here are some illustrative case reports from this section.

Case 2: Myocardial Contusion, Cardiac Rupture, and Sudden Death

Figure 18.2 ECG post-accident revealing small q and negative T in AVL, negative T in V1, and biphasically negative T in V2, with high takeoff of the ST segment. (This figure appears on page 119 of this book. Please Click to enlarge)



A 17-year-old male was taken to the emergency room following a motor vehicle accident during which his vehicle hit a tree. He admitted to not wearing a seat belt. He complained of frontal head pain, knee and ankle pain, and central chest pain on inspiration. Physical examination revealed sternal ecchymosis and was otherwise negative. Laboratory tests revealed a hemoglobin of 12.0 g/dl and a white blood count of 13,200 K/µl. The chemistry profile, urinalysis, and chest x-ray were negative. Cardiac enzymes were not performed. A single preadmission ECG revealed normal sinus rhythm, negative T-wave in lead AVL and V-l, high takeoff of the ST segments in leads V1 and V2, and a biphasic T-wave in lead V2 (Figure 18.2). A repeat ECG was not done. The patient was admitted to orthopedic service for suturing of his knee and forehead lacerations and discharged the

following morning. Two days later, the visiting nurse noted bilateral basilar rales. The patient complained of weakness, nausea, and light-headedness. Twenty-four days following hospital discharge he collapsed and could not be resuscitated.

Figure 18.3 Autopsy revealing resected pericardium and ruptured pseudoaneurysm of the left ventricle. (This figure appears on page 120 of this book. Please Click to enlarge)



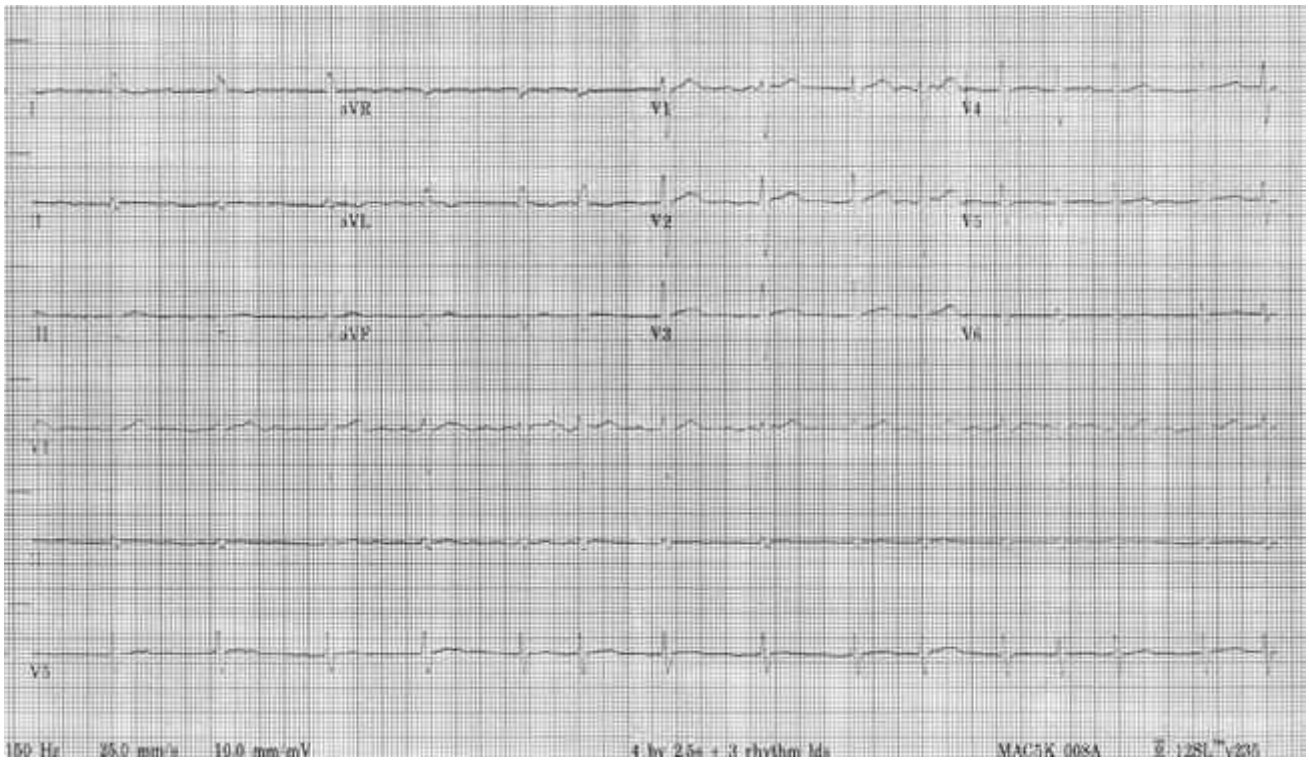
The autopsy revealed 600 cc of blood in the pericardial sac due to a ruptured pseudoaneurysm of the anterior left ventricle, obviously causing pericardial tamponade (Figure 18.3).

Forensic Implications

This case represented several causes of neglect: failure to consider a slightly decreased hemoglobin and increased white blood cell count, failure to consider suspicious ECG abnormalities, and not obtaining a repeat ECG prior to discharge. There was failure to properly evaluate, recognize, and observe a patient who had a high probability of having a myocardial contusion. Preadmission emergency room care was inappropriate and inadequate, as were admission and postdischarge care. The case was a guaranteed verdict for the plaintiff and was settled out of court for a very large monetary sum.

Case 15: Toxic Myocarditis from a "Herbal Energy Capsule"

Figure 18.5 Admission ECG. (This figure appears on page 142 of this book. Please Click to enlarge)



A 56-year-old male professional presented himself for a cardiology consult at the request of his family physician because of a "rapid heart", new onset. The clinical evaluation revealed an irregularly irregular rhythm at a rate of 146 beats/min, characteristic of atrial fibrillation. Other than obesity and hypertension (B.P. 156/104 mm Hg), the examination was unremarkable. The CBC, chemistry profile, T4 and TSH, and lipid profile were unremarkable, as were the cardiac enzymes. The echocardiogram revealed global hypokinesia with an ejection fraction of 48%. An adenosine stress test with a thallium and technetium scan revealed similar abnormalities. He was hospitalized and placed on warfarin and fractionated heparin, digoxin, quinidine, and atenolol. The heart rate decreased to approximately 86/min and converted to normal sinus rhythm in less than 72 hours. The ECG preconversion (Figure 18.5) revealed atrial fibrillation with a controlled ventricular response, low voltage, poor R-wave progression across the precordium intraventricular conduction defect, left axis deviation, and generalized repolarization abnormalities. The postconversion ECG abnormalities were similar except for normal sinus rhythm. Subsequent follow-up revealed the presence of quinidine-induced thrombocytopenia (platelet count of 86,000/μL). The quinidine was discontinued.

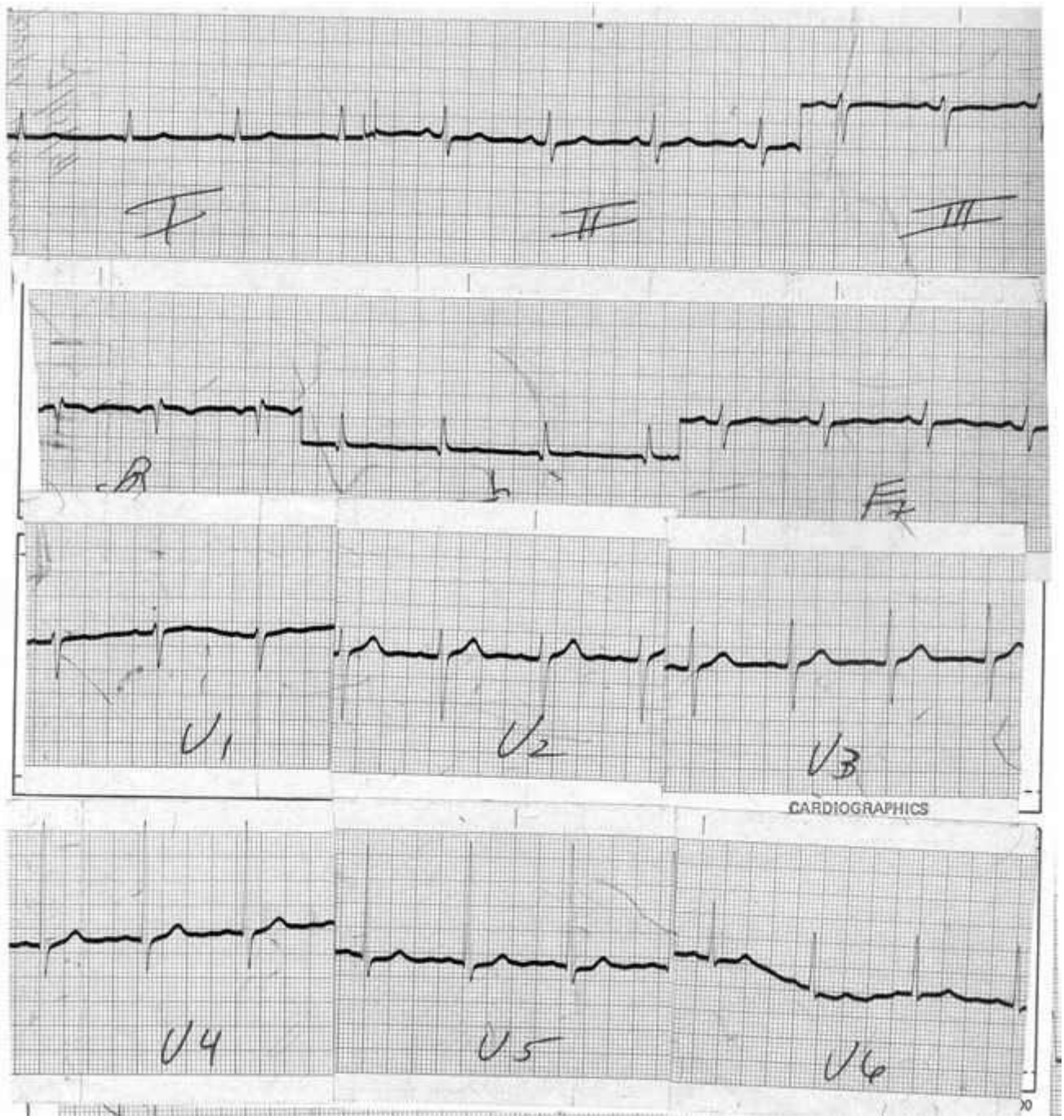
Four weeks later the atrial fibrillation recurred, and disopyramide 100 mg four times daily was prescribed. Pharmacologic conversion to normal sinus rhythm was noted five days later. The platelet count subsequently normalized. The patient chose not to continue warfarin, and was prescribed 81 mg of enteric-coated aspirin once daily and aggrenox twice daily (timereleased dipyridamole 200 mg with 25 mg aspirin, Boehringer Ingelheim Pharma). During the initial consultation the patient noted that he was not taking any medications, however, of his own volition, and on the advice of a friend was consuming an energy supplement. He was advised to discontinue the energy supplement and bring the bottle on his next visit. The capsules contained a multiherbal preparation, which included 24 mg of ephedra.

Forensic Implications

Ephedra is a sympathomimetic stimulating alkaloid, the active constituent being ephedrine. It is known to act as a cardiovascular stimulant causing increased heart rate and hypertension. Other side effects include the production of various cardiac arrhythmias, including ventricular tachycardia, coronary vasospasm, and sudden death. The federal drug administration subsequently legislated that ephedra-containing preparations were to cease being produced and over-the-counter medications containing ephedrine be discontinued. No legal action could be taken against the manufacturers, as the patient was consuming up to nine capsules daily (a total of 216 mg of ephedra), which was markedly in excess of the recommended dose of two capsules daily. After several months his blood pressure became normal and his ECG improved but did not fully recover to normalcy. The patient further noted that he had been taking the preparation for more than two years. He was told by his cardiologist that he was fortunate to be alive and that in all likelihood, he sustained a certain degree of cardiac toxicity that resulted in permanent heart damage (cardiomyopathy). It is important that physicians make an attempt to discover self-prescribed drugs and preparations that patients frequently imbibe, especially when abnormal pathophysiologic states are present.

Case 18: Oncologic Cardiomyopathy

Figure 18.6 Normal ECG pre-chemotherapy. (This figure appears on page 147 of this book. Please Click to enlarge)



A 66-year-old male presented to his physician with "a lump in his left neck." Subsequent diagnostic study and biopsy was positive for non-Hodgkin's lymphoma, disseminated to the chest, abdomen, and bone marrow. Physical examination revealed normal vital signs, a split first heart sound, and abnormally palpable lymph nodes in the left neck, left supraclavicular area, and both inguinal areas. The ECG was normal. Initial MUGA (multigated acquisition) scan revealed normal myocardial contractility and an ejection fraction of 59%. The patient was begun on an aggressive chemotherapy program, with MUGA scans taken every two to four months. Initial medication consisted of a combination of thiotepa, velban, and methotrexate. Approximately 13 months later following varying degrees of recurrence, his program was changed to cytoxan, mitoxantrone, adria, and vincristine.

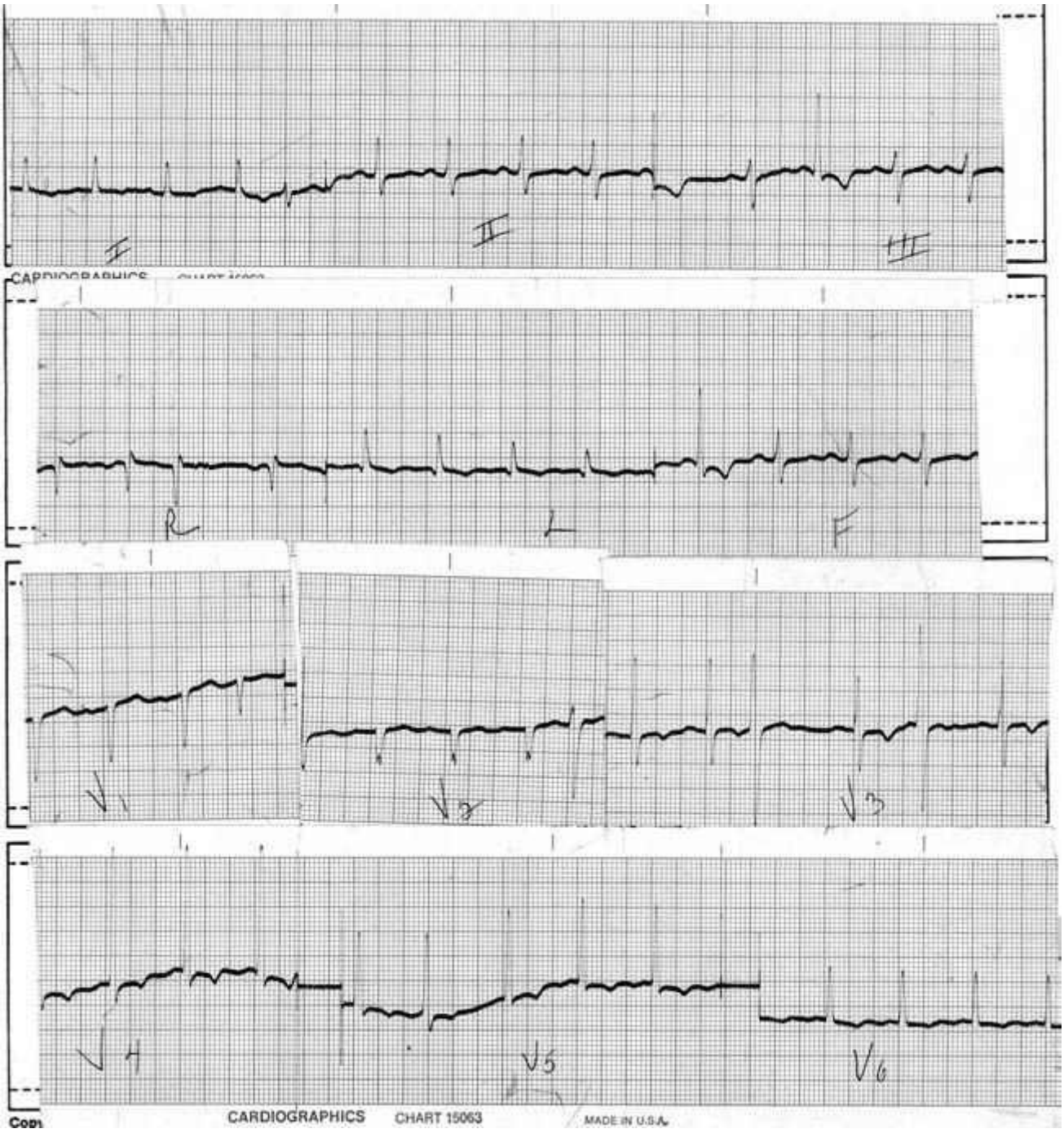
Careful oncologic and cardiologic follow-up resulted in various alterations of treatment. Over the next 11 months the ejection fractions decreased to 51%, 49%, 29%, and a low of 19%, necessitating

continued changes to the program and improvement in the ejection fraction to 49%. The patient developed symptomatic cardiac decompensation noting dyspnea, leg edema, and orthopnea, all of which improved on digoxin, furosemide, and potassium chloride supplementation.

The MUGA (Multi Gated Acquisition) scan revealed global hypokinesia, decreased left ventricular contractility, and cardiomegaly. The ECG revealed generalized ST-T abnormality with T-waves of the "ischemic-type." Clinical examination revealed a fourth heart sound (S-4) and a grade 1/6 soft, high-pitched, blowing, nonholosystolic murmur at the apex, compatible with hemodynamically insignificant mitral insufficiency. He continued to improve from an oncologic and cardiac standpoint and survived several years thereafter, his demise being due to recurrent lymphoma. The electrocardiograms before and during chemotherapy are noted in Figures 18.6 and 18.7.

Forensic Implications

Figure 18.7 ECG after 11 months chemotherapy. NSR, pre-mature atrial and ventricular contractions, generalized repolarization abnormality, "ischemictype" T-waves and poor R-wave progression in leads V1-V2. (This figure appears on page 148 of this book. Please Click to enlarge)

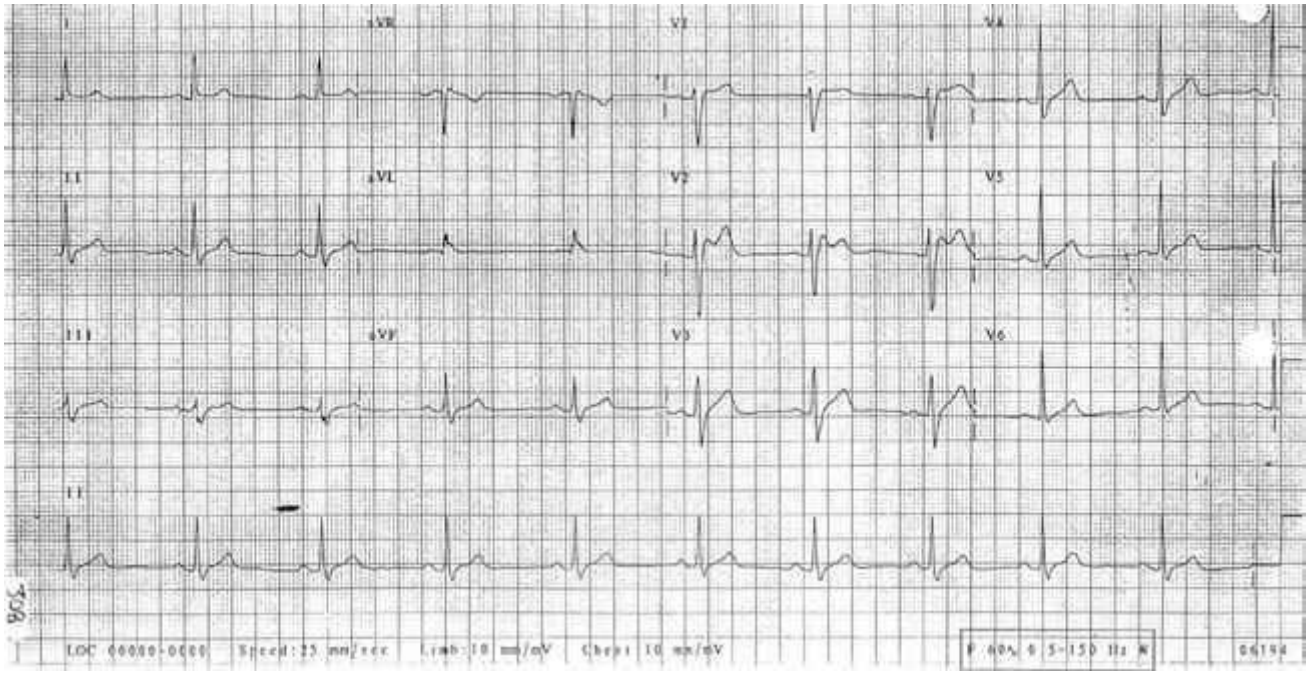


As previously reviewed, almost all chemotherapeutic drugs have cardiotoxic potential, some more than others, with a high degree of patient variability. Patients undergoing chemotherapy must be followed closely by the oncologist and internist or cardiologist. As depicted in this case, under diligent observation and study, probably beyond the norm, cardiac complications secondary to chemotherapy occur and must be managed promptly and appropriately to avoid medical-legal involvement. The chemotherapy was withheld following the echocardiographic assessment that showed the ejection fraction (EF) of 29%. In spite of cessation and alteration of therapy, the EF continued to decline (a not unusual situation) and then began to steadily improve on continued variations in the overall treatment program from an oncologic and cardiac point of view. In these cases, dependent upon the clinical assessments and therapies, improvement is not always forthcoming. Myocardial dysfunction can be permanent and may be accompanied by signs and symptoms of congestive heart failure or cardiac decompensation of the non-congestive type.

Case 20: Brugada Syndrome

A middle-aged male presented with a several-year history of presyncope and syncope that had gone undiagnosed until an episode of ventricular tachycardia and ventricular fibrillation was observed during an emergency room visit. He was otherwise healthy, without evidence for underlying medical or cardiac pathology. Eventually, an astute cardiologist recognized the Brugada pattern on the ECG, with the typical "saddle-back" ST-T change in leads V1 and V2. The ECG also showed a probable left atrial conduction abnormality and a nonspecific intraventricular conduction defect (Figure 18.9). A cardioverter-defibrillator was placed and eliminated the syncopal episodes.

Figure 18.6 Normal ECG pre-chemotherapy. (This figure appears on page 147 of this book. Please Click to enlarge)



The Brugada syndrome is a cardiac oddity believed to be due to a genetic discrepancy involving the myocardial electrolyte action current. It has familial clustering, which requires evaluation and observation for symptoms, with or without associated ECG changes, and must be differentiated from right ventricular dysplasia, which usually features ischemic-type t-wave negativity in the right precordial leads. Both may feature incomplete or complete right bundle branch block ECG patterns. Both the right ventricular bundle block and the "coved" ST elevation has been reported to occasionally be transient in some cases, making the diagnosis quite enigmatic at times.122 Tada et al. have demonstrated the presence of lipomatous infiltration and fibrosis within the myocardium that is not detectable by the usual endomycardial biopsy and other diagnostic modalities.

Forensic Implications

If the patient had succumbed to a cardiac arrest secondary to ventricular tachycardia and fibrillation, it would have taken very little plaintiff testimony to recognize the ECG abnormality and the necessity of preventing sudden cardiac death by an Internal Cardioverter Defibrilletor (ICD). A medical-legal catastrophe was therefore averted.

Case 25: Carbon Monoxide Toxicity

A middle-aged husband and wife presented to their respective family physicians with a multitude of complaints. The wife was a two-pack-per-day cigarette smoker and the husband was a nonsmoker. The wife was previously admitted to the hospital for gynecologic surgery during which a carbon monoxide (CO) blood level of 7.25% was noted (laboratory normal being 0 to 1.5%). This CO level would not be unusual for a cigarette smoker. The varying symptoms of both patients, which "waxed and waned" over a period of one year, included lethargy; depression; irritability; memory deficit; anorexia; headaches; irritation of the throat, eyes, and lungs; and insomnia. Shortly thereafter the husband developed neuro-motor dysfunction characteristic of Parkinson's disease. Of additional interest was the fact that two of three dogs in the household died, and all suffered hair loss, one dog having near total hair loss. Plants in the house also suffered from failure to thrive, which was never known to occur in the past. Investigation by several professional services concluded that the entire household-human, animal, and plant life-suffered from chronic CO poisoning caused by improper installation of a new furnace.

Forensic Implications

CO exposure of varying dose and duration is known to affect neurotransmitter metabolism, resulting in central and peripheral neurologic abnormalities. The patients in this case, as well as the plants and animals, suffered from chronic low-grade CO toxicity over a period of one year. Heavy exposure can cause a mixed sensory-motor neuropathy. Movement disorders of the Parkinson's type are known to occur, in addition to hypotonia, dystonia, and disorders of locomotion. Carboxyhemoglobin levels in smokers can vary from 3% to a high of 18% (normal generally being 0.5 to 1%). In addition to the symptoms suffered by the husband and wife as noted in this case report, generalized weakness, visual impairment, and syncope are known to occur. Dependent upon various factors, mood and behavioral disturbances may present themselves with depression that may be long term. Second-hand cigarette smoke contains twice the amount of CO as the directly inhaled smoke of a cigarette smoker. The half-life of CO is approximately 5 hours. CO damages vulnerable areas of the brain including the globus pallidus and substantia nigra. This can result in tremors and a decrease in motor speed, reaction time, and manual dexterity. Delayed neurotoxicity varies in incidence from 2 to 30%.

Short-term exposure to CO can cause angina and myocardial infarction in patients with coronary artery disease. Continued long-term exposure can accelerate the production or progression of arterial atherosclerosis, all complications being magnified in cigarette smokers and those exposed to second-hand smoke. Delayed neurologic and psychiatric symptoms from 3 to 240 days may occur in 10 to 30% of patients. Recovery from these signs and symptoms may take place in 75% of individuals within one year, but some may remain permanently. The plaintiff's case was settled for a substantial but undisclosed monetary award without necessitating a court trial.

The book is full of such cases. It is also a good repository of facts and medicolegal implications related to the issue of Cardiovascular medicine. We are sure our readers would enjoy the book as much as we at the journal office did.

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Editor's recommendation. This book is recommended by the editor.