PHYSIOLOGICAL RESPONSE OF PATIENTS WITH ACUTE MYOCARDIAL INFARCTION TO A BEDBATH

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We hereby recommend that the Thesis prepared under our supervision by Betty Jean Oliver entitled "Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath"

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CHAPTER I

INTRODUCTION

Coronary heart disease is the leading cause of death in the United States. In America alone 4,000 people suffer acute myocardial infarctions, and 1,500 of these die each day from the disease. From the moment a patient with an acute myocardial infarction enters the coronary care unit, attention is directed toward the ultimate goal of helping the patient return to a full, vital, and productive life. The patient's life style, however, must remain within the limits of the heart's ability to adjust to increases in activity and stress in the presence of an infarcted area.

One of the many functions of the nurse in the coronary care unit is the administration of a bedbath to the patient with an acute myocardial infarction. In addition to the cleansing purpose of the bedbath, it is very refreshing to a patient when he is feeling restless and uncomfortable. Many physiological and psychological stressors that are known to affect the cardiac function are encountered during a bath. Some possible stressors are environmental temperature, movement and massage of the

body, anxiety, and social-clinical interaction. They affect the nervous, endocrine, and vascular systems that regulate the function of the heart. This study has been designed to determine the patient's physiological response to the bedbath in terms of alterations in the heart rate and electrocardiogram.

Statement of the Problem

The problem of this study was to determine alterations that occur in selected physiological phenomena in a patient with an acute myocardial infarction during the bedbath procedure.

Purposes

The purposes of this study were to:

1. Determine the alterations that occur in the heart rate of a patient with an acute myocardial infarction during the bedbath procedure

2. Determine the alterations that occur in the ST segment of the electrocardiogram of a patient with an acute myocardial infarction during the bedbath procedure

3. Determine specific arrhythmias that occur in the patient with an acute myocardial infarction during the bedbath procedure

4. Determine the time intervals and duration of the alterations that occur in the heart rate, ST segment, and rhythm of a patient with an acute myocardial infarction during the bedbath procedure

Background and Significance

Acute myocardial infarction is a life-threatening condition. Because the myocardium must function continuously, blockage of blood to the heart muscle and the development of subsequent necrotic areas within the myocardium represents a catastrophic blow to the body which may be fatal to the individual (Luckmann and Sorensen 1974). The very essence of nursing care rests upon the nurse's ability to perceive and understand the behavioral clues that indicate the patient's ability to deal with the problems brought about by threats to optimum health (Bryne and Thompson 1972).

Myocardial infarction is a condition which occurs when the myocardial tissue is destroyed in the regions of the heart that are deprived of blood supply after complete or limited closure of the coronary artery or one of its branches, either by a thrombus or through obstruction of the vessel lumen by atherosclerosis (Brunner and Suddarty 1975). Periods of anoxia or severe hypoxia as short as five minutes can cause irreversible damage to the enzyme

system concerned with aerobic metabolism of the heart. Periods of fifteen to twenty minutes of ischemia produce mitrochondrial swelling; after thirty to forty minutes of ischemia, marked distortion of muscular tissue occurs (Hurst 1974).

The basic functions of the cardiovascular system are to transport oxygen and other nutrients to the cells of the body, to remove metabolic waste products from the cells, and to carry substances such as hormones from one part of the body to another (Hurst 1974). When the heart is no longer able to fulfill its function adequately, changes occur that affect the stability of the individual.

After an individual has a myocardial infarction, it is essential that the workload of the heart be decreased to allow for oxygen supply and better nutrition to the heart muscle and body tissue (Luckmann and Sorensen 1974). This is accomplished by eliminating insofar as possible, any demands on the patient to adapt to the environment. Decrease in activity reduces the tissue demands for oxygen by lowering the blood pressure, thereby diminishing the arterial resistance against which the heart must pump. A decreased heart rate allows for a prolonged recovery period of the cardiac muscle which results in a more efficient cardiac contraction (Luckmann and Sorensen 1974).

The goal of nursing care for a patient with an acute myocardial infarction is directed toward decreasing the work load of the heart. Methods to evaluate the patient's tolerance of the various aspects of care need to be utilized. One component is the administration of a bedbath to the patient with an acute myocardial infarction while in the coronary care unit. Depending on the situation and the temperature of the water used for the bath, the patient may feel stimulated and ambitious following the bath or relaxed to the point that sleep follows soon after. The feeling of cleanliness and relaxation that accompanies a bath is satisfying to the patient.

A bath also produces physiological changes in the individual. Massaging the skin affects the peripheral nerve endings and the peripheral circulation. The bath is also of value to the musculoskeletal system as a form of exercise (Fuerst, Wolff, and Weitzel 1974). The physiological phenomena that occur with this activity however, may be an added stress to which the body needs to adapt.

The cardiac output remains proportional to the overall metabolism of the body. That is, the greater the degree of activity of the muscles and other organs, the greater the cardiac output. The performance of the heart is extraordinarily complex; extrinsic influences such as

neural and hormonal factors, the blood volume and venous return, and the instantaneous impedance of the peripheral vasculature affect the demands placed on the heart (Hurst 1974).

Physiological parameters are available to help the nurse assess the patient's tolerance of stressful situations. The patient's cardiovascular responses can be evaluated by assessing heart rate, heart rhythm, and blood pressure. In the normal person, one cardiovascular response to exercise is cardiac acceleration. However, the patient with an ischemic or infarcted heart may develop bradyarrhythmias, tachyarrhythmias, or ectopic beats (Lavin 1973). Talano and Ronan (1974) state the increase in cardiac work produced by tachycardia places undesired stress on an already ischemic myocardium.

The electrocardiographic monitor affords optimal documentation of the patient's cardiovascular response to a particular level of activity. Criteria have been established in many coronary care units to determine the level of activity the patient can tolerate (Haskell 1974 and Hurst 1974).

A decrease in the level of physical activity for the patient is indicated by the appearance of chest pain or dyspnea, an increase in the heart rate of over 120 beats

per minute, increased ST segment displacement on the electrocardiogram or monitor, the occurrence of significant arrhythmias, or a fall in systolic blood pressure greater than 20 millimeters of mercury (Hurst 1974).

The effect a bedbath has on a patient with an acute myocardial infarction may be evaluated by determining the alterations that occur in selected physiological phenomena. The parameters selected for this study are the heart rate and the ST segment of the electrocardiogram and the occurrence of arrhythmias.

Hypotheses

The hypotheses for this study are:

1. There will be no alteration in the heart rate in a patient with an acute myocardial infarction during the bedbath procedure

2. There will be no alteration in the ST segment of the electrocardiogram in the patient with an acute myocardial infarction during the bedbath procedure

3. There will be no occurrence of arrhythmias in a patient with an acute myocardial infarction during the bedbath procedure

Definition of Terms

For the purposes of this study the following operational definitions were used.

Acute myocardial infarction--a life-threatening condition characterized by the formation of localized necrotic areas within the myocardium.

Arrhythmia--any irregularity of rhythm of the heart's beating.

Bedbath--a method of bathing a patient by a nurse that does not require active movement by the patient.

Bedbath procedure--includes a 15 minute rest period before and after the administration of a bedbath.

Coronary care unit--a highly equipped, fully staffed area of the hospital that is reserved specifically for care of patient- with acute cardiac conditions.

Electrocardiogram--a graphic tracing of the electrical activity of the heart.

Heart rate--the number of cardiac cycles per minute.

Nurse--an individual licensed to practice as a registered nurse in the state of Nebraska.

ST segment--a portion of the electrocardiogram representing the time following depolarization of the ventricles and preceding repolarization of the ventricles.

Limitations

The limitations of this study over which no control was exercised were:

1. The extent of the myocardial infarction
2. The patient's biological, psychological, and social resources
3. Unknown stresses the patient may be experiencing
4. Medications the patient is receiving that may have an effect on the variables of this study
5. The interruptions that occur during the bedbath by unexpected events in the coronary care unit
6. The occurrence of chest pain or extension of the myocardial infarction.

Delimitations

The delimitations for this study were:

1. The subjects must have a diagnosis of acute myocardial infarction
2. The subjects must be without congestive heart failure, cardiogenic shock, extension of the myocardial infarction and acute episodes of arrhythmias
3. The subjects must be in the coronary care unit
4. The subjects must be over eighteen years of age

Assumptions

The assumptions for this study were:

1. Stress in man produces physiological responses that can be measured

2. Increases in mental and/or physical activity causes an increase in the workload of the heart

Summary

The patient with an acute myocardial infarction is subjected to many stresses while in the coronary care unit. All aspects of care which produce stress need to be evaluated in light of the patient's response to these activities.

The topics pathophysiology, management, nursing research related to patient management, and physiological means to evaluate patients with an acute myocardial infarction were reviewed in the literature and are summarized in Chapter II. Chapter III contains the method utilized to obtain and treat the data collected when a patient with an acute myocardial infarction was given a bedbath. Chapter IV contains the analysis of the data obtained before, during, and after the administration of a bedbath. Complete-block design and chi-square were used to analyze the heart rate, ST segment, occurrence of arrhythmias, temperature, and blood pressure. The summary,

conclusions, implications, and recommendations which were derived from this study appear in Chapter V.

CHAPTER II

REVIEW OF LITERATURE

A review of the literature revealed a large body of new knowledge in the field of cardiovascular pathophysiology of acute myocardial infarction as well as the diagnosis, complications, and management of patient care. In order to facilitate the planning, administration, and evaluation of care, the physiological alterations that occur in man when adapting to both internal and external stressors were explored. The various components of the bedbath were related to the appropriate stressor.

Pathophysiology of Acute Myocardial Infarction

Heart disease has become the leading cause of death in North America, Australia, Europe, and New Zealand (Luckmann and Sorensen 1974). It has been estimated that about 1 million people in the United States experience acute myocardial infarction each year. More than 500,000 persons die annually from this disease (Yu 1971). Approximately one-third of these deaths occur in males under sixty-five years of age (Joyce 1972). This disease often affects the "breadwinner" of the family during the most

productive years of the individual's career and creates psychological trauma and anxiety for the family. Since heart disease affects so many people, a national, as well as world-wide, effort is under way to prevent or retard the rapid progress of this disease, and improve the management and rehabilitation of the people affected.

The basic functions of the cardiovascular system are to transport oxygen and other nutrients to the cells of the body, to remove metabolic waste products from the cells and to carry substances such as hormones from one part of the body to another. Acute myocardial infarction alters the heart's ability to maintain these functions, thereby causing a disruption in homeostasis (Hurst 1974).

The primary cause of myocardial infarction is coronary atherosclerosis, a pathological condition of the coronary arteries. Coronary atherosclerosis is characterized by abnormal lipid and fibrous tissue accumulation in the vessel wall with resulting disruption of the vessel architecture and function and variable reduction of blood flow to the myocardium (Hurst 1974). If this blood flow is reduced enough to cause insufficient flow of oxygen and nutrients to the myocardium, the tissue will die. Acute myocardial infarction refers to the process by which the tissue is destroyed because of an insufficient supply of

blood, and subsequently oxygen and nutrients, to the myocardium (Joyce 1972).

The healing process of the myocardial infarction is well established within twenty-four hours after the onset of symptoms. Leukocytic infiltration occurs during this first phase of healing. By the end of the third day the second stage of healing has begun. This second stage is characterized by removal of necrotic fiber and it continues through the fourth week after the infarction has occurred. With the beginning of the third week following the infarction, the stage of scar formation becomes apparent (Hurst 1974).

Epidemiological studies and observations by cardiologists have established that the incidence of coronary atherosclerosis is increased in individuals with certain risk factors (Hurst 1974). Some of these risk factors are nonmodifiable and some are modifiable. The risk factors that cannot be modified are age, sex, and family history. The development of atherosclerosis is dependent on time, therefore, it is increased in advancing age. Men are more prone to clinical manifestations of coronary atherosclerosis than are women of childbearing age because of the protective effects of estrogen.

Individuals with either parents or siblings affected by the disease prior to age fifty have a greater risk of developing the disease (Hurst 1974).

Risk factors that can be modified are elevated serum lipids, hypertension, cigarette smoking, carbohydrate intolerance, obesity, sedentary living, and psychosocial tension (Brunner and Suddarth 1975). Although there is a strong correlation between these risk factors and the development of coronary atherosclerosis, the exact cause is not known (Hurst 1975).

Rosenman (1972) believes that the personality of an individual, not the risk factors, plays a major role in accelerating coronary heart disease. This was first suspected approximately fifteen years ago when the rising coronary rate in the middle-aged American was believed to stem from emotional interplay associated with the new stresses imposed by industralized civilization, in conjunction with high-fat diet, diminished physical activity, and relatively high serum lipids. This suspicion was strengthened by noting that groups free of such socio-economic stresses were free of coronary heart disease no matter what the dietary, smoking, or exercise habits were. This personality type has been termed Behavior Pattern Type A.

Type A personality is characterized by such personality traits as aggressiveness, ambition, drive, competitiveness, and a profound sense of time urgency. The individual's speech is usually forceful, rapid, often explosively uneven, emphatic, and accompanied by sudden gestures such as fist-clenching and taut facial grimaces. Locomotion and mannerisms are rapid, reflecting the person's drive, competitiveness, chronic restlessness, impatience, and particularly the habitual sense of time urgency (Rosenman 1972).

Diagnostic Measures

There are several tests available to confirm the diagnosis of an acute myocardial infarction and to evaluate the patient's progress. The electrocardiogram is of great value since it will detect changes in the electrophysiological process within the heart which is very sensitive to alterations in perfusion of the myocardium. Ischemia of the myocardium is seen by inverted T waves. Injury to the myocardium, which is a stage beyond ischemia but is still reversible, is seen with ST segment elevation. Necrosis of the myocardium, which is irreversible, is noted by the presence of an abnormal Q wave (Marriott 1972). The age of the infarction can be determined by evaluating the sequence of occurrence of all these findings.

Location of the myocardial infarction can be determined by the occurrence of abnormal Q waves in specific leads. An anterior infarction produces Q waves in lead V_1, V_2, V_3, or V_4; lateral infarction produces Q waves in leads I and AVL; and the inferior infarction produces Q waves in leads II, III, and AVF. A posterior infarction produces the exact opposite pattern of the anterior infarction (Dubin 1974).

The laboratory tests that are of value in confirming an acute myocardial infarction are nonspecific indices of tissue necrosis and inflammation and the serum enzyme changes. The nonspecific reaction to myocardial injury is associated with leukocytosis, which often reaches levels of 12,000 to 15,000 cells per cubic millimeter. The magnitude of the leukocytosis yields some information about the size of the infarction. It appears within a few hours after the onset of pain and persists for three to seven days. The second nonspecific change is the elevation of the erythrocyte sedimentation rate, which peaks during the first week and remains elevated for several weeks (Conn, Clohecy, and Conn 1974).

Enzymes in the tissues are released into the blood when the myocardium is infarcted. The rate of enzyme elevation aids in determining what tissue has been damaged.

The three most important enzyme studies used to confirm the diagnosis of an acute myocardial infarction are creatine phosphokinase, serum glutamic oxaloacetic transaminase and lactic dehydrogenase (Meltzer et al. 1976). Serum glutamic oxaloacetic transaminase becomes elevated within six to twelve hours, peaks on the second day and returns to normal within approximately four days. The creatine phosphokinase is short-lived and may be missed. This enzyme becomes elevated in the serum within four to six hours, reaches its peak serum concentration in about twenty-four hours and returns to normal by forty-eight to seventy-two hours. One of the more sensitive indicators of acute myocardial infarction is serum alpha lactic dehydrogenase. This enzyme reaches its peak late in myocardial injury, on about the fourth or fifth day, and stays elevated up to two weeks (Conn, Clohecy, and Conn 1974). These enzyme studies help determine the size of the infarction since the larger the infarction, the greater the enzyme response.

Additional diagnostic measures that are available are echocardiograms, phonocardiograms, vectorcardiograms, arteriograms, and radioisotope studies (Hurst 1974 and Wintrobe et al. 1974). Echocardiograms are a method of acquiring echosignals from the structures of the heart for study of the valves and ventricular wall motion and

thickness. The phonocardiograms are recordings of audible vibrations enamating from the heart and great vessels. Vectorcardiograms produce a pathway of instantaneous vectors from the electrical activity of the heart usually during one cardiac cycle. Coronary arteriograms allow visualization of the coronary arteries by injection of contrast medium directly into each coronary artery. Radioisotope studies, such as scintiangiocardiography, are done by the addition of an isotope to the blood and utilization of a scanner which makes possible the study of the pericardiac space, cardiac chambers, great vessels, and myocardium and detection of pericardial effusion.

Complications of Acute Myocardial Infarction

Prevention of life-threatening complications or the early detection of a complication alters the possibility of death of the patient with an acute myocardial infarction. Arrhythmias occur in more than 90 percent of patients with myocardial infarction (Grace 1975). The greatest impact on the mortality rate in myocardial infarction is through the detection and control of ventricular arrhythmias. Because ventricular arrhythmias are greatest in the first four hours after the infarction, there is great urgency for bringing the patient into an environment where complications

can be prevented or if present, treated (Meltzer and Cunning 1972).

Ectopic rhythms arise in or near the borders of intensely ischemic and damaged myocardial tissue. Damaged myocardium may also interfere with the conduction system of the heart causing dissociation of the atria and ventricles (Luckmann and Sorensen 1974). Second-degree or third-degree heart block occurs in 8 to 10 percent of the patients. Heart block occurs more commonly in inferior wall myocardial infarction because the atrioventricular nodal artery arises from the right coronary artery. When heart block is a complication of anterior wall infarction, the prognosis is poor since the heart block is usually due to extensive septal damage (Romhilt and Fowler 1973).

Now that arrhythmias are being more effectively treated, shock or pump failure is the most important fatal complication of myocardial infarction. Cardiogenic shock occurs in about 20 percent of the patients with an acute myocardial infarction and accounts for at least 50 percent of the deaths (Wintrobe et al. 1974). Shock develops when cellular oxygenation is impaired because there is an inadequate flow of blood. The profound decrease in effective blood flow is the result of the inability of the left ventricle to pump blood throughout the body and perfuse the

brain and the vital organs; they, therefore, become inadequate to sustain life (Stude 1974).

The earliest indication of impending shock is a change in the patient's mental condition or general behavior. The patient is often restless, agitated, and confused, which indicates decreased cerebral oxygenation. With the progression of shock, the patient becomes listless, apathetic, extremely weak, and may eventually slip into a coma. Initially the skin is pale and cold due to vasoconstriction and decreased cardiac output. As the condition progresses pallor and cyanosis develop. The systolic pressure fails initially causing a decrease in pulse pressure, as well as oliguria (Stude 1974).

Congestive heart failure, which is failure of the cardiac muscle to pump sufficient blood to meet the body's metabolic needs, occurs in 60 percent of the patients with myocardial infarction (Meltzer et al. 1976). Structural damage to the myocardium or metabolic changes within the muscle cell lead to decrease in contractility of the heart and consequently a decrease in cardiac output. Signs of congestive heart failure include sinus tachycardia, tachypnea, orthopnea, elevation of the jugular venous pressure, pulmonary rales, a third heart sound, enlargement of the liver, and peripheral edema (Romhilt and Fowler 1973).

Complications from thromboembolism are seen clinically in approximately 10 percent of the patients with myocardial infarction; however, thrombotic lesions have been found in up to 45 percent of the patients, suggesting that thromboembolism is often unrecognized clinically (Wintrobe et al. 1974). The left ventricle is the most common location for mural thrombus, and the rarity of mural thrombi in the right ventricle is consistent with the belief that most pulmonary emboli develop in the deep veins of the lower extremities and travel through the right ventricle to become lodged in the pulmonary tree.

Mitral regurgitation occurs in more than half the patients during the first five days after the onset of a myocardial infarction. This occurs as a result of left ventricle papillary muscle dysfunction because of the decrease in blood supply to the muscle. Only a small percentage of the patients show any hemodynamic changes of importance (Meltzer and Dunning 1972).

Pericardial friction rub, which is usually the result of inflammation of the myocardium, is seen in 10 to 15 percent of the acute myocardial infarctions. The rub is characteristically of a leathery quality and occurs from two to three hours to several days after the occurrence of the myocardial infarction (Romhilt and Fowler 1973).

Myocardial rupture is a dramatic complication of myocardial infarction and accounts for approximately 10 percent of the hospital deaths. Approximately one-third of the ventricular ruptures occur within the first day and two-thirds occur by the end of the second week of hospitalization. Clinically there is a sudden disappearance of the pulse, blood pressure, and consciousness while the electrocardiogram continues to show sinus rhythm. The myocardium continues to contract but the forward blood flow is not maintained. Blood is pumped into the pericardium and cardiac tamponade ensues (Meltzer and Dunning 1972).

Management of Patients with Acute Myocardial Infarction

An understanding of the concepts related to the management of patients with myocardial infarction is necessary for both the physician and the nurse. These concepts allow for planning, implementing, and evaluating individualized patient care and provide the physician and nurse with mutual guidelines to help the patient obtain his optimum level of wellness (Dunn 1973).

Two main objectives of management are to prevent death due to arrhythmias and to minimize the mass of infarcted tissue (Wintrobe et al. 1974 and Hurst 1974). Additional objectives of care are prevention of complications, physiological and functional rehabilitation,

halting the progression of atherosclerosis, and education of the patient and family (Brunner and Suddarth 1975).

The development of coronary care units has resulted in improved care of the patients with myocardial infarctions and a reduction in mortality rates. Because of the specialized care in the coronary care units, the anticipated mortality of patients admitted to the hospital with an acute myocardial infarction has dropped from 30 to 35 percent in 1962 to 20 percent in 1972.

The coronary care unit is a specially designed nursing unit, the most important feature of which is a staff of highly trained personnel with authority to take immediate action in emergency situations. The unit is equipped with a system to permit continual monitoring of the heart of each patient in addition to emergency equipment including drugs, defibrillators, pacemakers, and respirators. The personnel in the unit recognize arrhythmias, adjust and/or administer antiarrhythmic drugs, perform cardiac resuscitation, and apply electroshock when necessary (Meltzer and Dunning 1972). Although emphasis in the coronary care unit is on detection and treatment of arrhythmias and on hemodynamic measurements and corrections, careful attention to the physical examination alone may alert the personnel to early changes in the condition of the patient with an acute myocardial infarction (Romhilt and Fowler 1973).

Promotion of rest is an important part of the care for a patient with an acute myocardial infarction. The nurse must provide an environment for both physical and emotional rest since therapeutic rest will provide the most favorable circumstances for the healing of the myocardium. In man most of the necrotic tissue resulting from the infarction is not replaced by connective tissue for about two weeks and complete healing requires four to eight weeks. The myocardium should not be subjected to major increases in heart rate or blood pressure for at least several weeks since the myocardium is at a critical part of the healing process and is weakest at this time (Haskell 1974).

It has been shown that allowing patients with acute myocardial infarction out of bed early during their convalesences does not increase the immediate mortality rate or incidence of ventricular aneurysm, myocardial rupture, congestive heart failure, or recurrent infarction (Duke 1973). Benefits attained from early activity include relief of anxiety, prevention of cardiac decompensation and avoidance of thromboembolism. Hospitalization beyond two weeks does not benefit patients with uncomplicated myocardial infarction and the reduction of hospitalization to less than four weeks is not accompanied by increased mortality.

Similar information was obtained from a study which involved 104 patients under seventy years of age who were hospitalized for acute myocardial infarction and who had no complications during the first two days of hospitalization. The patients were divided into two groups. In the early mobilized group, patients were treated by a physical therapist with a progressive program beginning on day two or three after the infarction. The control group was placed on the traditional strict bedrest for three or more weeks. Follow-up ranged from six to twelve months. There were no statistically significant differences between the two groups with regard to mortality, rate of reinfarction, arrhythmias, heart failure, angina pectoris, ventricular aneurysm, or results of exercise testing (Bloch et al. 1974)

Exercises for the patient with acute myocardial infarction should be rhythmic rather than isometric. Rhythmic exercises involve muscle contraction accompanied by a change in muscle length, such as active and passive flexion of the limbs. Isometric exercise involves muscular contraction with both ends of the muscle fixed and no movement in the involved joint, such as holding objects. Rhythmic exercise is preferred in the prevention of venous stasis, thrombosis, and embolization. These include passive

leg exercise, active flexion and extension of the feet, and turning in bed (Favin 1973).

With improper movement, exercise, and the occurrence of the valsalva maneuver, sufficient myocardial anoxia can occur to produce ventricular fibrillation (Conrad 1971). The valsalva maneuver occurs when the patient causes forced expiration against a closed glottis; this causes a need for the heart to adapt to varying volumes of venous return. During such a maneuver the intrathoracic pressure increases as much as 40 to 100 millimeters of mercury above the atmospheric pressure and interferes with blood entering the large veins. This results in decreased cardiac output of up to 72 percent (Gross 1973). Patients should be instructed to exhale when moving about in bed instead of holding their breath. The cardiac patient should be warned against straining when reaching for an object, supporting himself on the elbows and rising to a sitting position since these actions can produce a valsalva maneuver.

Indications of an inappropriate increase in exertion during care in the coronary care unit may be observed by the nurse if the patient has the appearance of chest pain or dyspnea, an increase in heart rate to over 120 beats per minuts, increased ST segment displacement on the electrocardiogram or monitor, the occurrence of significant

arrhythmias, or a fall in systolic blood pressure greater than 20 millimeters of mercury (Hurst 1974). Haskell (1974) utilizes the following criteria to evaluate the activity of a patient with an acute myocardial infarction--unexpected fatigue, weakness or dyspnea, development of or increase in angina, significant rhythm or conduction disturbances, increased ischemic type ST segment displacement, increase in heart rate by more than twenty beats per minute over the resting rate or a decrease by more than ten beats, and/or a persistent decrease or substantial increase in systolic blood pressure.

If any of these responses occur during activity, bedrest should be resumed until the patient's status is re-evaluated and a revised activity plan developed. Careful evaluation of the patient is indicated while gradually increasing physical activity to the optimum level for that patient.

As progression in activity increases in the recovery phase of a patient with an acute myocardial infarction, stress testing has been found helpful in the detection of possible tendencies toward the development of arrhythmias. Mortality rates of patients with myocardial infarction during the first year after discharge from the hospital is approximately 8 percent; many die suddenly, probably due to

ventricular arrhythmias. Mortality in patients with coronary heart disease appears to be greater in patients with ventricular premature beats than in those without. A two year follow-up of men who had suffered myocardial infarction disclosed a mortality rate twice as high for patients with frequent ventricular premature beats in a single electrocardiogram than for those with rare or no ventricular premature beats (Ericsson et al. 1972). Stress testing was done on patients three weeks after the myocardial infarction to detect possible arrhythmias and assist with prescribing an activity schedule for the patient at the time of dismissal from the hospital. The criteria utilized to stop the test included angina pectoris, frequent premature ventricular contractions, and a heart rate exceeding 140 beats per minute (Ericsson et al. 1972).

DeBusk (1975) states that it is safe to stress test patients at low levels of physical activity three weeks after an infarction to assist in the establishment of guidelines for optimal physical activity in the early post-infarction period. Criteria utilized to stop the testing include the appearance of symptoms of left ventricular failure, significant ventricular arrhythmias, or a heart rate of 130 beats per minute.

Management of care for a patient with acute myocardial infarction included concepts related to decreasing complications, and promoting rest, ambulation, and exercise. These concepts must be utilized when planning, implementing, and evaluating all aspects of patient care. Several aspects of care have been evaluated and revised as a result of research. Many other aspects including the administration of a bedbath to a patient with an acute myocardial infarction need to be researched.

Research in Nursing

Improvements in nursing practice can be achieved through creative study and initiation and evaluation of subsequent change. Research in nursing provides new facts, new knowledge, answers to problems, improvement, and development of new techniques (Treece and Treece 1973). Recent literature reveals studies that have been conducted to improve patient care. Those related to cardiac patients are now reviewed.

The response of patients with acute myocardial infarction to visits from the doctor, nurse, minister, and family were studied by Conn (1973). Visits from the physician caused the greatest increase in heart rate. This was closely followed by the visit from a minister. Visits from family members and friends produced the least amount of

change. From this study it would appear that the rigid visiting regulations imposed upon patients hospitalized in special care units are unnecessary. Judgment regarding such matters should be based on an assessment of each patient's needs (Conn 1973).

The effects of touch as nonverbal communication on seriously-ill patients were studied on sixty seriously-ill patients between the ages of twenty and sixty-four. The results of the study demonstrated that the nurse can establish a rapport with a seriously-ill patient within a short period of time through the use of physical contact (McCorkle 1974).

An exit questionnaire to measure satisfaction with selected aspects of coronary care was given to patients following discharge from the coronary care unit. Although the overall satisfaction was found to be high, the findings suggested the need for more attention to the nontechnical aspects of care, especially reduction of emotional trauma and improved interpersonal relations (Geertsen, Ford, and Castle 1976)

For years myocardial infarction patients have not been permitted to drink ice water or any extremely cold liquids because it was assumed that doing so might precipitate further myocardial ischemia. However, research

indicates that ingestion of ice water does not alter cardiac status in patients with uncomplicated myocardial infarction when the patient is free of other stressors. Fulfilling the myocardial infarction patient's request for ice water provides not only a physiological comfort measure but also a favorable psychological effect by deleting another "do not" from the list of restrictions (Houser 1976).

Gruber (1974) found no consensus in the literature as to whether or not it was dangerous to the patient to take rectal temperature when the patient had suffered an acute myocardial infarction. A study was then conducted by Gruber which showed that no significant cardiac rate changes occurred when rectal temperature was taken on patients with acute myocardial infarction.

Occupied bedmaking was studied to determine the least taxing method of bed making for a cardiac patient on bed rest. The results indicated that oxygen consumption was greater during the "cardiac bed" or top-to-bottom procedure than during the "regular bed" or side-to-side procedure (Flores and Zohman 1970).

The experience of myocardial infarction, admission to a coronary care unit, and transfer from the coronary care unit to less intensive settings are considered psychologically stressful. The degree of stress evidence by individual

patients varies widely and is generally dependent upon the patient's ability to deny the unpleasant reality of his critical condition. Research reveals that a high level of anxiety and discomfort is characterized by increased urinary sodium retention and potassium output (Gentry, Musante, and Haney 1973).

Significant heart rate and rhythm changes occur in coronary care patients during a variety of social-clinical interactions. Results of continuous day-long observations and concurrent monitoring of a coronary care patient disclosed twice as many ectopic beats occurred during routine nursing interactions when compared to resting periods. This study illustrated the need to devote attention to the effect of various social interactions, and the emotional context of these interactions. It may be possible to isolate the type of patients or kinds of social interactions which generally elicit heart reactions such as ectopic beats (Thomas, Lynch, and Mills 1975).

Mitchell (1973) discusses several procedures that may be utilized to decrease a patient's elevated temperature, but recommends controlled nursing studies to determine which method is most successful to decrease the body temperature. A great deal of nursing research still needs to be done. The National Commission for Study cited research as a means

to "determine the relative effectiveness of various forms of nursing intervention and the impact of particular innovation in nursing practice" (Treece and Treece 1973, p. 105).

No information in regard to the effects of a bedbath on a patient with an acute myocardial infarction was found when reviewing literature. However, principles to be utilized when administering a bedbath and the bedbath procedure appear in many fundamental nursing textbooks (Fuerst, Wolff, and Weitzel 1974, DuGas 1972, and Gragg and Reese 1974).

The bedbath is a valuable component of care both for the patient and the nurse. In addition to cleansing and promoting comfort for the patient, the bedbath stimulates blood circulation and provides some exercise for the patient. When administering a bedbath, the nurse has an opportunity to teach the patient desirable hygiene measures and incorporate other health teaching. The nurse also has the opportunity to observe the condition of the patient's skin, nails, and hair, and observe for the presence of edema, assess the quality of respirations, and identify any difficulty or pain the patient may have. This time provides an excellent opportunity for the nurse to establish rapport with the patient and assess the patient's mental state (DuGas 1972).

Nursing research is a means of improving current patient care and implementing and evaluating new concepts in regard to patient care. Before attempting to alter the administration of a bedbath to a patient with an acute myocardial infarction, information regarding the effect of this nursing action needs to be obtained. It is necessary to review the literature related to the physiological alterations to stress that occur in situations similar to that encountered with a bedbath. In addition, means to evaluate the patient's response to the bedbath need to be reviewed.

Physiological Alterations to Stress

Man is constantly exposed to stress both internal and external. In order to maintain homeostasis, man must adapt to these stresses constantly and effectively. During the administration of a bedbath some of these stressors may be more than the patient with an acute myocardial infarction is able to adapt (Mitchell 1973). During the bedbath the environmental temperature to which a patient is exposed is altered frequently. Also, the room and water temperature may vary, clothing and coverings are removed and reapplied, the skin is rubbed, the body and joints are moved about, and the patient is in constant contact with a strange person in unfamiliar surroundings. Since one of the main goals of patient management is to decrease the workload of the heart,

the factors associated with the administration of a bedbath that may affect the workload of the heart rate are explored.

The demands made on the heart and the performance of the heart is very complex. When a person is at rest, the heart must pump only 4 to 6 liters of blood each minute. However, during severe exercise the heart may be required to increase the cardiac output as much as five times the resting amount.

The cardiac output is ultimately determined by two variables, the amount of blood ejected with each heart beat and the number of beats per minute (Frohlich 1972). Normally 90 to 95 percent of the cardiac output regulation is affected by peripheral circulation and only 5 to 10 percent by the heart itself. However, when the heart becomes diseased and unable to provide adequate pumping capacity, it is the heart, not the peripheral circulation, that becomes the limiting factor in cardiac output regulation (Sodeman and Sodeman 1974). This decrease in cardiac output is observed in patients with acute myocardial infarction because the effectiveness of the contraction of the myocardial is impaired (Oliver 1975). Normally, alterations in cardiac output are influenced by the nervous system, hormonal factors, blood volume, and venous return to the heart (Hurst 1974).

Nerves affect carciac pumping by changing the heart rate and strength of the contraction. The central nervous system is in a continuous state of activity as it exerts control over multiple organ systems (Frolich 1972). The parasympathetic stimulation decreases heart rate and sympathetic stimulation increases the heart rate and force of contraction. In general, the more times the heart beats per minute, the more blood it can pump up to a physiological end point. After it reaches a critical level, the heart strength itself decreases, presumably because of overstimulation of metabolic substance in the cardiac muscle. Also, the period of diastole is not adequate to allow time for blood to flow from the atria to the ventricles (Guyton 1976). The increase in cardiac work produced by tachycardia in a patient with acute myocardial infarction places an undesirable stress on an already ischemic myocardium (Talano and Ronan 1974). In addition, stimulation of the sympathetic nervous system results in vasoconstriction and decreased blood flow to the tissue of the extremities, thereby increasing the resistance against which the heart must pump (Luckmann and Sorensen 1974).

Hormones affect the function of the heart. The catecholemines, epinephrine, and norepinephrine, are secreted by the adrenal medulla. Both affect the heart in a

manner similar to the sympathetic division of the autonomic nervous system in that they produce an increase in the heart rate and force of contraction (Shepro, Belamarich, and Levy 1974), and cause an increase in the tone of the arterial wall with an increase in peripheral vascular resistance, thereby imposing a greater workload on the damaged heart (Adams 1973). The release of catecholemines occurs very soon after coronary occlusion resulting in hypoxia and stimulation of the sympathetic nerves (Oliver 1975).

Thyroxine is synthesized by the thyroid gland and increases the metabolism of all cells. When metabolic rates are increased during periods of increased metabolism and stress, the need for nutrients and waste removal is also increased. The heart responds by an increase in cardiac output. Thyroxine also increases the heart's excitability, already a major problem and concern in a patient with an acute myocardial infarction (Shepro, Belamarich, and Levy 1974).

The body attempts to maintain normal blood volume by the retention of fluids primarily by the kidney and by the movement of water from other extracellular fluids into the blood (Nordmark and Rohweder 1975). The volume of blood entering the heart must be neither too small nor too great. Too small a blood volume entering the heart will

cause a decrease in cardiac output and circulatory collapse and too great a volume of blood entering the heart will overwork it, resulting in a circulatory overload. Alterations in volume may be caused by shock, hemorrhage, dehydration, defects in clotting mechanism, and altered circulation (Luckmann and Sorensen 1974).

The venous return of blood to the heart is controlled by the peripheral circulation. Peripheral circulation changes in accord with the needs of the tissues. The causes of vasodilatation at the local level is still unknown, but blood flow increases to the area of hypoxia and/or increased metabolism very rapidly, from seconds to minutes (Sodeman and Sodeman 1974). The amount of blood allowed to flow through the small vessels from tissue into the veins and hence into the heart, determines the cardiac output. Frank-Starling's law of the heart states that the greater the heart is filled during diastole, the greater will be the cardiac output (Guyton 1976).

The nervous system, hormonal factors, blood volume, and venous return to the heart are the main factors that regulate the function of the heart. These factors can be influenced by many physiological and psychological stressors. Some of these stressors are similar to the ones that the patient comes in contact with during the bedbath.

Stressors that can affect the workload of the heart, and possibly produce myocardial ischemia in a damaged heart, are thermal, circulatory, muscular, and emotional conditions (Houser 1976).

Heat production by exercise, shivering, disease, and metabolism and heat loss by sweating, skin circulation, radiation, evaporation, convection, and conduction are balanced in health to maintain the narrow range of normal. Multiple factors affect temperature maintenance and regulation. The total environment, sex, age, time of day, emotional status, activity, diet, and drugs are all reputed to affect temperature balance.

Heat production is increased up to five times during shivering and up to 600 percent with chills (Mitchell 1973). Heat production is also increased by stimulation of the sympathetic nervous system, thyroxin and adrenocortical secretion, exercise, excitement, and anxiety (Nordmark and Rohweder 1975). Jarvis (1976) points out that increases in temperature may create hypoxia simply because the oxygen requirement of tissue metabolism increased 7 percent for every degree of Fahrenheit rise in temperature. This causes an increase in heart rate thereby increasing the cardiac output in order to meet the cellular need for oxygen. A decrease in temperature brings about a decrease in heart rate (Guyton 1976).

Four physical processes by which the body dissipates heat are radiation, conduction, convection, and evaporation of water (Mitchell 1973). Radiation is the transfer of heat from the surface of one object to that of another without physical contact between the two. Conduction is the transfer of thermal heat from one molecule to another, or from one atom to another. Convection is a process whereby transfer of heat from the body occurs as air next to the body is heated, moves away, and is replaced by cool air. Evaporation is the conversion of water into a vapor which is then carried off the body into the environmental air. Evaporation of water is almost continuously occurring as insensible perspiration and through the respiratory passageway (Guyton 1976).

The amount of heat loss from the surface by radiation and conduction varies with the amount of body insulation, the amount of skin area exposed, the external environmental temperature, and the amount of blood flow in peripheral capillaries. The amount of heat loss from evaporation depends upon production of perspiration, amount of skin exposed, amount of blood flow in peripheral capillaries, humidity of the surrounding atmosphere, and air currents. The amount of heat loss from convection depends on the

amount of surface area exposed and the air current (Nordmark and Rohweder 1975).

Heat conduction to the skin by the blood is controlled by the degree of vasoconstriction of the arterioles that supply blood to the venous plexus of the skin and this vasoconstriction is controlled almost entirely by the sympathetic nervous system. The usual suit of clothes decreases the rate of heat loss from the body to about one-half of that from a nude body. The effectiveness of clothing in preventing heat loss is almost completely lost when it becomes wet because there is no entrapped air to act as an insulator (Guyton 1976). Houser (1976) points out cold stimuli can increase cellular oxygen demands and myocardial work.

The patient's circulation can be greatly altered during a bedbath by massage. Friction produced by rubbing the skin with a wash cloth and towel stimulates peripheral circulation and increases nutrition to the cells (Mitchell 1973, Brunner and Suddarth 1975). The increase in peripheral circulation occurs as a result of the nerve endings being stimulated by massaging the skin. Long smooth strokes on the arms and legs directed from the distal to the proximal increase the rate of venous flow to the heart, thereby increasing the cardiac output (DuGas 1972).

Movement during the bath process provides muscle exercise and allows many of the joints to be put through their range of motion. This movement also increases circulation and improves respiratory function by stimulating deep breathing. During exercise, muscle metabolism increases and there is an inadequate amount of oxygen available. The deficiency brings about vasodilatation of the muscle vessels, allowing the blood to pass through the tissue with little resistance and return rapidly to the heart. The net result of the added flow to the heart is increased cardiac output. In addition, physical exertion brings the sympathetic portion of the autonomic nervous system into play, which stimulates the heart (Shepro, Belamarich, and Levy 1974).

Even before exercise is begun, the thought of exercise stimulates the autonomic nervous system. Tightening or contraction of muscles around the blood vessels increases the mean systemic pressure from 7 to 20 millimeters of mercury (Guyton 1976). Frohlich (1972) refers to this as an exercising muscle squeezing blood out of the vessels to increase venous return to the heart. Cardiac output usually remains proportional to the overall metabolism of the body. Increase in cardiac output is proportional to increase in exercise (Guyton 1976).

Anxiety is a normal reaction to stress. It is highly personalized and is usually precepitated by a threat to the person's biological integrity or self-esteem (Mitchell 1973). There are many ways in which the performance of the various aspects of patient care can be a source of psychological stress and unnecessary anxiety. Some of these sources are: change in care; change in personnel; remarks in conversation (Beland and Passos 1975); body integrity; exposure; embarrassment; discomfort from pain, cold, and fatigue; restriction from movement; and inconsistent unpredictable behavior of authority figures on whom the patient's welfare depends (Brunner and Suddarth 1975). Mitchell (1973) points out that stress increases when the patient is forced to become a dependent recipient of care, rather than an independent or partner in his own care, or when the patient is unfamiliar with hospital experience.

Psychosocial interaction can have major effects on cardiac function. Interactions between a nurse or doctor and a patient, such as a nurse taking a patient's pulse, can cause alterations in the heart rate, rhythm and frequency of ectopic beats (Thomas, Lynch, and Mills 1975). Houser (1974) reported a correlation between anxiety level at the time of transfer from the coronary care unit to

general care and the incidence of physiological complications. The higher the anxiety the more likely the complications. Physiological reaction to anxiety is primarily a reaction of the autonomic nervous system. This includes increased heart rate and respiration, shift in blood pressure and temperature, relaxation of the smooth muscles of the bladder and bowel, cold clammy skin, and increase in perspiration (Brunner and Suddarth 1975).

Thermal conditions, circulation, physical activity, and emotional conditions all have a dynamic effect on the cardiac output and demands made on the heart. Means to evaluate the patient's tolerance to these factors after experiencing an acute myocardial infarction need to be utilized by the nurse when planning, implementing, and evaluating patient care.

Evaluation of Patient's Response to Stress

Patients with acute myocardial infarction or extensive coronary disease may maintain adequate myocardial perfusion in the resting state and objective evidence of myocardial ischemia may be lacking. However, when myocardial oxygen requirements are increased, the diseased coronary arteries are incapable of increasing myocardial blood flow proportional to the demands (McHenry, Morris, and Jordan

1974). Criteria to evaluate the heart's ability to meet the demands placed on it by stress have been set up in many coronary care units and centers doing stress or exercise testing (Wintrobe et al. 1974 and Hurst 1974).

Stress or exercise testing utilizes the sensitivity of the electrocardiogram to demonstrate anoxia of the myocardium. When myocardium oxygen requirements are increased and the diseased coronary arteries are incapable of increased myocardial blood flow proportional to the demands, the ST segment of the electrocardiogram will be altered (McHenry, Morris, and Jordan 1974). Changes in the ST segment during and immediately following exercise are the most significant electrocardiographic indicators of myocardial ischemia (Friedewald 1976). In addition, a decrease in systolic blood pressure or a decline in the heart rate during exercise is highly suggestive of myocardial failure and/or severe myocardial ischemia (McHenry, Morris, and Jordan 1974).

Talano and Ronan (1974) believe the heart rate is one of the determinants of myocardial oxygen consumption. Hurst (1974) and Haskell (1974) utilize heart rate, ST segment displacement on the electrocardiogram, the occurrence of significant arrhythmias, change in systolic blood pressure, and the occurrence of pain to evaluate the patient's

tolerance of activity. McHenry, Morris, and Jordan (1974) state that increases in heart rate and systolic blood pressure are two of the major determinants of increased myocardial oxygen requirement.

The electrocardiogram is one of the many parameters utilized to evaluate the cardiac patients because of its sensitivity to subtle as well as significant changes in the myocardium. Changes in heart rate and ST segment as well as the occurrence of arrhythmias can be observed with an electrocardiogram tracing (Marriott 1972). There are many factors that will alter the electrocardiogram and these need to be kept in mind when utilizing this tool. Some of the major factors were explored by Dubin (1974) and Marriott (1972).

Alterations in the coronary perfusion of the myocardium will produce characteristic changes in the myocardium. Ischemia or decreased blood supply to the myocardium is characterized by inverted T waves. As the blood supply decreases and injury to the myocardium progresses, the ST segment will become altered. ST segment elevation is seen with acute infarction and a positive master's test or ventricular strain. A myocardial infarction is synonymous with the presence of a Q wave, and the location of the infarction can be determined by noting

the lead in which the Q wave is present (Dubin 1974 and Marriott 1972).

Other forms of pathology produce changes in the electrocardiogram. Emphysema produces a low voltage in all leads and often right axis deviation. Pulmonary infarction may produce a large S wave in lead I, and a Q wave in lead III, depression in lead II and T wave inversion in leads V_1 through V_4. Right bundle branch block is common. Pericarditis produces an elevated ST segment, usually flat or concave, and the entire T wave may be elevated off the baseline (Dubin 1974).

Electrolyte disturbances effect the electrocardiogram. A high serum potassium will produce flat p waves, a wide QRS and peaked T wave. A low potassium will produce a flat T wave and a prominant U wave. Hypercalcemis shortens the QT interval while hypocalcemia prolongs the QT interval.

Administration of drugs may also product alterations of the electrocardiogram. Digitalis produces a gradual downward sloping of the ST segment and may lead to AV blocks of many varieties. Digitalis toxicity may produce ectopic ventricular arrhythmias. Quinidine causes notching on the wide p wave, widening of the QRS complex and ST depression (Marriott 1972).

The blood pressure is essentially a reflection of the cardiac output and vascular resistance (Mitchell 1973) and varies not only with the moment but with the condition (Jarvis 1976). The baroreceptors, chemoreceptors, and central nervous system ischemic response play a major role in regulation of the arterial pressure and prevent the pressure from rising extremely high or falling extremely low from minute to minute. The baroreceptors are sensitive to arterial pressure, the chemoreceptors are sensitive to deficiency in oxygen, and the central nervous system ischemic response becomes active if the arterial pressure falls below 50 millimeters of mercury (Guyton 1976). Factors affecting the blood pressure include heart rate, stroke volume, autonomic system reaction, metabolic rate, physical activity, body position, nervous excitement, and condition of the vascular bed (Mitchell 1973). The blood pressure increases with physical exercise, change in position, and emotional states (Nordmark and Rohweder 1975).

Many parameters are available to evaluate the heart's ability to adapt to stress. Utilization of the electrocardiogram tracing while the patient with an acute myocardial infarction is in the coronary care unit, allows for observation of the heart rate, ST segment changes, and the occurrence of arrhythmias. These strips can be obtained

before, during, and after the administration of the bedbath without disturbing the patient. Obtaining a blood pressure reading disturbs the patient and interrupts the bedbath procedure, but if taken before and after the administration of a bedbath, adds more data to the patient assessment.

Summary

Chapter II contains a review of literature related to myocardial infarction, its pathophysiology, diagnosis, complications, and management. Recent research in nursing affecting the management of cardiac patients was presented. Physiological alterations that occur due to physical and psychological stress similar to that which occurs with the administration of a bedbath were also explored. Factors that regulate and change these physiological alterations were discussed, as well as means to evaluate these alterations when a bedbath is given to a patient with an acute myocardial infarction.

CHAPTER III

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

A descriptive study was conducted to determine the physiological response of patients following an acute myocardial infarction to a bedbath. A standardized bedbath was administered to each patient in the study. Data to determine the physiological response of the patient were obtained from an electrocardiogram tracing taken before, during, and after the bedbath.

Setting

This study was conducted at Mary Lanning Memorial Hospital in Hastings, Nebraska, a community in mid-Nebraska with a population of approximately 25,000 people. Mary Lanning Memorial Hospital, with a bed capacity of 177, admitted and treated a total of 8,524 patients in 1975. In 1975, seventy-nine patients with myocardial infarction as a primary diagnosis were treated in the coronary care unit.

The coronary care unit and the intensive care unit are combined into a single unit. The combined bed capacity is eleven. Three of the beds are in a ward located at the nurse's station, two are private rooms, and the remaining

six beds are in semi-private rooms which are utilized primarily for cardiovascular patients. The six beds, located in semi-private rooms, are equipped with monitors that provide an electrocardiogram which meets the American Heart Association's specifications for twelve-lead electrocardiogram diagnosis.

Population

The people eligible for this study were any individuals over the age of eighteen who were admitted to the coronary care unit with the diagnosis of acute myocardial infarction. No individual was included in the study if severe complications or arrhythmias were demonstrated.

The convenience method was used to select a sample that would represent patients with acute myocardial infarction. In this method, the subjects are selected because they happen to be available for participation in the study at a certain time (Abdellah and Levine 1965). This study was conducted from the middle of December 1975 to the end of May 1976. A total of twenty-eight patients were studied.

Methodology

A pilot study was conducted in the summer of 1975 to determine the time intervals to be utilized in the collection of data. Five patients were selected by the convenience method as they were admitted to the coronary care unit in a hospital located in a metropolitan city in Texas. The subjects had a diagnosis of acute myocardial infarction without severe complications of congestive heart failure, cardiogenic shock, or significant arrhythmias. The monitoring lead was utilized to obtain all electrocardiogram tracings. An electrocardiogram tracing was obtained for baseline data after the patient had rested for a minimum of fifteen minutes. The bedbath was administered by a nurse. An electrocardiogram tracing was obtained every minute during the bedbath and for fiteen minutes after the bedbath had been completed. The heart rate and ST segment were evaluated for alteration that indicated stress on the heart. The greatest alterations that occurred in the heart rate and ST segment were within the first five minutes of the bedbath, and the first ten minutes after the completion of the bedbath. The following recommendations were made as a result of this study:

1. Electrocardiogram tracings be taken
    a. after a fifteen-minute rest period

b. every minute for five minutes then every five minutes until the bath is completed

c. every minute for five minutes then every five minutes for two times after the bedbath is completed

2. The patient not be moving about in bed or changing positions when obtaining the electrocardiogram tracings

3. The bedbath procedure be standardized for water temperature, room temperature, duration, patient covering, and order of bathing

From the pilot study a tool was developed to collect pertinent information for this study. This information included patient's name, code number, hospital number, bed number, doctor, date of admission, date of onset of symptoms, birthdate, age, sex, marital status, diagnosis, drugs being taken that may affect the electrocardiogram, room temperature, time intervals for running the electrocardiogram, patient's temperature, blood pressure, monitor lead, interruptions that occurred from unexpected events, and unusual behavior of the patient (see Appendix A).

Permission to conduct this study was obtained from the Human Research Review Committee of Texas Woman's University (see Appendix B), the administrator of the

hospital where the study was conducted (see Appendix C), and the patient's attending physician (see Appendix D). This study was conducted with the knowledge and verbal permission of each patient.

An assistant was selected to administer the bedbaths. This assistant was a registered nurse who graduated from an accredited school of nursing in 1962 and obtained a Bachelor of Arts degree in 1964. The assistant had three years of experience as a staff nurse and over three years of experience on the faculty of a school of nursing.

A second pilot study consisting of one student nurse and three patients was done in the fall of 1976 to clarify the bath and data collection procedure. The subsequent procedure was then utilized for each patient studied. From this pilot study it was determined that the temperature of the bath water could be maintained at 110° and 120° Fahrenheit by using a large, preheated metal bath basin. The length of time required for the bath averaged twenty-two minutes.

A brief description of the study was discussed with the patient. This discussion included information about the qualifications and job description of the researcher and assistant. The reason given for the study was evaluation of the bedbath as it is usually administered to patients in the coronary care unit. The same procedure would be utilized,

only an additional rest period would be required before and after the administration of the bedbath. During the rest and bath time electrocardiogram tracings would be made at the nurse's station which would provide information about the patient's heart rate. As a result of studies similar to this one, alterations in patient care are made to provide improved care and comfort for the patient. The patient was told that permission to conduct this study had been given by his physician. All the patients eligible to be included in this study consented to participate.

The room temperature was maintained between 68° and 74° Fahrenheit. All equipement needed for the bedbath was assembled and placed at the bedside. The head of the bed was elevated 15 to 35 degrees. The bath was scheduled to be given one-half to two hours after breakfast. The bedpan was offered to each patient approximately thirty minutes before the bath was scheduled. The monitoring electrodes were checked for placement and conduction and the electrocardiogram standard was checked. Baseline blood pressure and oral temperature were obtained on each patient after a minimum rest period of fifteen minutes. The patient was then allowed to rest without interruption for fifteen minutes before the bedbath was started. After this fifteen-minute rest period, a baseline electrocardiogram tracing was run at the nurse's station.

The bath basin was filled with tap water of 120° Fahrenheit. The assistant then entered the room and started the bedbath. The bathblanket was placed over the patient and the top linens and patient's gown were removed. The wash cloth was utilized in a mitten technique, and firm but gentle strokes were used. Small sections of the body at a time were washed, rinsed, and dried to prevent chilling. The sections included first the head, neck, and ears, each arm done separately, chest, abdomen including the area of the thighs near the groin, and each leg and foot were done separately. The water was then changed and the patient was rolled to the side so the back of the neck, shoulder, back, buttocks, and posterior upper thigh could be completed. The back and buttocks were gently rubbed with lotion. The patient was rolled on to his back, and a gown and dry bathblanket were placed on the patient. Conversation was kept to a minimum. Equipment was left at the bedside and the patient allowed to rest for fifteen minutes. The genital area was not done on any of the patients to maintain consistency with the bath procedure that is conducted in the institution. Policy for the coronary care unit of the hospital utilized for this study allowed for an orderly to do the genital area of a male patient.

The administration of the bedbath was monitored via closed circuit television at the nurse's station. Electrocardiogram tracings were run every minute for the first five minutes, then every five minutes for three times. When the bath was completed, the electrocardiogram tracings were run every minute for five times, then every five minutes for two times. The monitor was observed during the entire procedure to detect any arrhythmias. At the completion of the final rest period the patient's blood pressure and oral temperature were taken. Interpretation of the electrocardiogram tracings were made by a cardiologist.

Procedure for Treatment of Data

The procedure for treatment of data was the application of randomized complete-block design and chi-square. Randomized complete-block design is applied whenever a set of variants is classified according to two criteria of classification, as with time and people (Alder and Roessler 1968). The chi-square test of significance, tells how significant the difference is between the frequencies observed and the frequencies expected (Treece and Treece 1973).

Summary

A study to determine the physiological alterations that occur when a patient with an acute myocardial infarction is given a bedbath was conducted in a community hospital in mid-Nebraska. The population was selected by the convenience method. A standardized bath was administered by one nurse and data were collected from electrocardiogram tracings. The electrocardiogram tracings were interpreted by a cardiologist and the resulting data compiled for statistical analysis.

CHAPTER IV

ANALYSIS OF DATA

A study to determine the physiological alterations that occur when a patient with an acute myocardial infarction is given a bedbath was conducted from the middle of December 1975 to the end of May 1976. A sample population of twenty-eight patients was selected by the convenience method. Data were collected before, during, and after the administration of a standardized bedbath. Chapter IV contains the data collected, a statistical analysis of the data, and a summary of the findings. Tables are used to facilitate presentation of the data.

Data

Information pertaining to the age, sex, marital status, location of the infarction, the number of days since the onset of symptoms, duration of the bedbath, and room temperature were obtained for each subject. The sample population ranged from forty to eighty-nine years of age with the mean age of 69.7 years. There were seventeen (61 percent) male and eleven (39 percent) female subjects. In the sample population, twenty-one (71 percent) were married,

six (21 percent) were widowed, one (3 percent) was single, and one (3 percent) was divorced.

Time for the administration of the bath ranged from twenty-one to twenty-four minutes with the average time being 22.5 minutes. Room temperature ranged from 74° to 79° Fahrenheit with the mean being 74.6° Fahrenheit. The bedbath was administered to the patient one to ten days after the initial onset of symptoms, the mean being three days after the onset of symptoms (see table 1).

Heart Rate

The first hypothesis states there will be no alteration in the heart rate in a patient with an acute myocardial infarction during the bedbath procedure. The heart rate of the subjects was determined from the electrocardiogram tracings obtained at selected time periods before, during, and after the administration of the bedbath (see Appendices E and I). The heart rate was analyzed by randomized complete-block design.

The first analysis looked at sixteen time periods to detect subtle changes that occurred at any one time before, during, or after the administration of the bedbath. The analysis of variance for the sixteen time periods for the twenty-eight patients obtained an F ratio of 1.414, giving a

TABLE 1

DEMOGRAPHIC DATA

Patient Number	Sex	Age	Marital Status	Location of Infarction	Days after Infarction	Room Temperature Fahrenheit
1	Male	40	Married	Inferior wall	1	74
2	Male	56	Married	Inferior wall	5	74
3	Female	69	Married	Anterior and lateral wall	2	74
4	Female	67	Widowed	Anterior and inferior wall	2	76
5	Female	85	Widowed	Anterior and lateral wall	4	74
6	Female	74	Widowed	Inferior wall	1	74
7	Male	85	Married	Inferior wall	1	74
8	Male	68	Married	Anterior and lateral wall	2	75
9	Female	68	Married	Lateral wall	1	72
10	Male	75	Married	Inferior wall	1	74
11	Female	79	Married	Subendocardial	1	76
12	Female	51	Divorced	Anterior wall	2	74
13	Male	84	Widowed	Anterior wall	3	75
14	Female	73	Widowed	Inferior wall	2	75

Table 1 (Continued)

Patient Number	Sex	Age	Marital Status	Location of Infarction	Days after Infarction	Room Temperature Fahrenheit
15	Male	55	Married	Anterior wall	2	77
16	Male	75	Married	Inferior	5	74
17	Female	72	Widowed	Inferior wall	5	75
18	Male	83	Widowed	Inferior and anterior wall	10	75
19	Male	64	Married	Inferior wall	1	74
20	Male	89	Married	Anterior wall	2	74
21	Male	76	Widowed	Lateral wall	2	74
22	Male	77	Married	Posterior	3	74
23	Male	50	Married	Anterior-septal	6	79
24	Female	68	Married	Anterior wall	7	74
25	Male	57	Married	Inferior wall	3	74
26	Male	51	Married	Inferior wall	3	76
27	Male	79	Married	Anterior, lateral, and inferior wall	4	74
28	Female	83	Single	Inferior wall	5	74

p value of approximately 0.25 which is not significant at the 0.05 level.

The second analysis looked at the mean heart rate of five time periods. Each time period represented the occurrence of specific activity. These time periods were after a fifteen-minute rest period, first five minutes of the bedbath, last fifteen minutes of the bedbath, first five minutes of rest after the bedbath, and the last ten minutes of rest after the bedbath. The second heart rate determinations made it possible to analyze any event that occurred with the overall activity. The analysis of variance for the five time periods for the twenty-eight patients obtained an F ratio of 2.19, giving a p value of approximately 0.20 which is not significant at the 0.05 level.

Eight interruptions occurred during the period under study. These interruptions included a nurse checking the patient's condition, visitors walking into the room, and doctors checking the patient's condition. It is interesting to note that there was an increase in the heart rate during two of the four visits by the doctors. No other alterations occurred during the interruptions (see table 2).

The analysis of data obtained from the electrocardiogram tracing does not support rejection of the null

hypothesis at the 0.05 level of significance. The hypothesis stating there will be no alteration in the heart rate in a patient with an acute myocardial infarction during the bedbath procedure is accepted as stated.

ST Segment

The second hypothesis states there will be no alteration in the ST segment of the electrocardiogram in the patient with an acute myocardial infarction during the bedbath procedure. The alteration of the ST segment from the baseline electrocardiogram tracing occurred in one of 420 tracings (see appendix I). This alteration occurred in patient number 14 ten minutes after the completion of the bedbath. The chi-square test for qualitative variable is usually not done in studies where the frequency in any one cell is less than ten (Abdellah and Levine 1965). Therefore, it can be assumed that a significant change did not occur.

The analysis of data obtained from the electrocardiogram tracings does not support rejection of the null hypothesis. The hypothesis stating there will be no alteration in the ST segment of the electrocardiogram in the patient with an acute myocardial infarction during the bedbath procedure is accepted as stated. Table 2 shows the interruptions during the bedbath procedure.

TABLE 2

INTERRUPTIONS DURING THE BEDBATH PROCEDURE

Patient Identification	Interruption by Whom	Time of Interruption
6	Nurse	5 minutes after completion of the bedbath
7	Minister	14 minutes after completion of the bedbath
13	Visitor	7 minutes after completion of the bedbath
14	Nurse	1 minute after completion of the bedbath
22	Doctor	2 minutes after starting the bedbath
25	Doctor	16 minutes after starting the bedbath
26	doctor	2 minutes after starting the bedbath

Arrhythmias

The third hypothesis states there will be no occurrence of arrhythmias in a patient with an acute myocardial infarction during the bedbath procedure. Arrhythmias did occur during the administration of the bedbath. During or immediately following the administration of the bedbath, six (21 percent) of the patients experienced a minor arrhythmia. The arrhythmias that occurred were sinus arrhythmia, premature atrial contractions,

premature nodal contractions, and trigeminy of premature nodal contractions. Refer to appendices F and I for total response and table 3 for variations in the rhythm. Of the total population, three (11 percent) had arrhythmias during the administration of the bedbath, two (7 percent) had arrhythmias immediately following the bedbath, and one (3 percent) had an arrhythmia during and immediately following the bedbath. No major or death-producing arrhythmias were noted.

TABLE 3

OCCURRENCE OF ARRHYTHMIAS

Patient Identification	Type of Arrhythmia	Length of Arrhythmia
1	Sinus arrhythmia	Three 1/2-minute periods
7	Premature atrial contractions	3 minutes
8	Premature nodal contractions	2 minutes
17	Premature atrial contractions	3 minutes
27	Premature atrial contractions	1 minute
28	Premature nodal contractions	1-1/2 minutes

Analysis of data obtained from the electrocardiogram tracing supports the rejection of the null hypothesis stating there will be no occurrence of arrhythmias in a patient with an acute myocardial infarction during the bedbath procedure. Minor arrhythmias were experienced by 21 percent of the patients. The data supports the acceptance of the alternate hypothesis which states there will be occurrence of minor arrhythmias in a patient with an acute myocardial infarction when given a bedbath.

Temperature

The temperature of each patient was taken fifteen minutes before the administration of the bedbath and fifteen minutes after the bedbath had been completed. Refer to appendix G for the total response and table 4 for the statistical data of the temperature.

TABLE 4

TEMPERATURE BEFORE AND AFTER THE BEDBATH

Time Period	Mean	Standard Deviation	Standard Error
Before the bedbath	98.21	0.851	0.161
After the bedbath	98.28	1.052	0.199

The mean elevation of oral temperature after the bedbath for fifteen (54 percent) of the twenty-eight patients was 0.52° Fahrenheit. The mean decrease or oral temperature for twelve (43 percent) of the twenty-eight patients was 0.46° Fahrenheit. No change in oral temperature occurred on one (3 percent) of the twenty-eight patients. The temperature alteration from the base reading ranged from -1.1° to +1.2° Fahrenheit. Analysis of the data yielded a mean difference of -0.0679 and a T statistic of -0.63, giving a p value of -.534.

Evaluation of temperature changes was made from this data. When comparing the temperature before and after the bedbath, the p value of 0.534 obtained was not significant at the 0.05 level of significance. Therefore, it can be stated that an alteration in temperature did not occur in patients with an acute myocardial infarction when given a bedbath.

Blood Pressure

The blood pressure of each patient was taken after a minimum rest period of fifteen minutes before the bedbath procedure was started (the bedbath procedure included a fifteen-minute rest period before the administration of the bedbath) and fifteen minutes after the bedbath. Refer

to appendix H for total response and table 5 for statistical data of the blood pressure.

TABLE 5

SYSTOLIC BLOOD PRESSURE BEFORE AND AFTER THE BEDBATH

Time Period	Mean	Standard Deviation	Standard Error
Before the bedbath	118	19.452	3.676
After the bedbath	114.68	17.355	3.280

The mean elevation in the systolic blood pressure following the bedbath for eleven (39 percent) of the patients was 8.5 millimeters of mercury. The mean decrease in blood pressure after the bedbath for seventeen (61 percent) of the patients was 11.4 millimeters of mercury. The change in systolic blood pressure ranged from +20 to -40 millimeters of mercury. A blood pressure change greater than 20 millimeters of mercury did not occur in twenty-five (89 percent) of the patients. Blood pressure elevation of greater than 20 millimeters of mercury occurred in one (3 percent) of the patients and decreased greater than 20 millimeters of mercury in two (7 percent) of the patients (one 42 and the other 40 millimeters of mercury). Analysis of the data yielded a mean difference of 3.321 and a T statistic of 1.28, giving a p value of 0.21.

Evaluation of the blood pressure changes was made from this data. When comparing the systolic blood pressure in this sample before and after the bedbath, the p value of 0.21 obtained was not significant at the 0.05 level of significance. Therefore, it can be stated that an alteration in the systolic blood pressure did not occur in patients with an acute myocardial infarction when given a bedbath.

Summary

A study was done to determine the physiological alterations that occur when a patient with an acute myocardial infarction is given a bedbath. Data were collected from electrocardiogram tracings obtained before, during, and after the administration of a bedbath. Analysis of the data does not support rejection of two null hypotheses. The hypotheses stating there will be no alteration in the heart rate and ST segment of the electrocardiogram in the patient with an acute myocardial infarction are accepted as stated. Data supports rejection of the hypothesis stating there will be no occurrence of arrhythmias in a patient with an acute myocardial infarction when given a bedbath. The data supports the acceptance of the alternate hypothesis which states there will be occurrence of minor arrhythmias in a patient with an acute myocardial infarction when given a bedbath.

The temperature and systolic blood pressure were analyzed for alterations before and after the administration of the bedbath. Significant alteration did not occur in either the temperature or blood pressure in the patient with a myocardial infarction when giving a bedbath.

CHAPTER V

SUMMARY, CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS

Summary

This study was conducted to determine the alterations that occur in selected physiological phenomena when a patient with an acute myocardial infarction is given a bedbath. The parameters selected for this study were the heart rate, ST segment of the electrocardiogram, and the occurrence of arrhythmias.

A review of the literature was done to determine the concepts related to myocardial infarction, its development, clinical course, management, and evaluation. Factors that affect the function and healing process of the heart following an acute myocardial infarction were also reviewed.

A descriptive study was conducted from the middle of December 1975 to May 1976 in a coronary care unit of a 177-bed hospital located in a midwestern state. Subjects were selected by the convenience method as they were admitted to the coronary care unit with a diagnosis of acute myocardial infarction. Subjects with congestive

heart failure, cardiogenic shock, extension of the myocardial infarction, or acute episodes of arrhythmias were not included.

Data were collected on twenty-eight subjects. An electrocardiogram tracing was run on each patient at selected time periods before, during, and after the administration of a standardized bedbath. The heart rate was analyzed by complete-block design. The heart rate did not alter significantly at the 0.05 level of significance. The ST segment of the electrocardiogram did not alter frequently enough for statistical analysis. Minor arrhythmias did occur in 21 percent of the patients during and immediately following the bedbath. The temperature and blood pressure were taken before and after administration of the bedbath. Alterations did not occur in either the temperature or blood pressure at the 0.05 level of significance.

Conclusions

Based on the findings of this study, the following conclusions are offered.

1. The heart rate, temperature, systolic blood pressure, and ST segment of electrocardiogram were not significantly affected in the twenty-eight patients with

acute myocardial infarction when given a standardized bedbath

2. No life-threatening arrhythmias were observed on the electrocardiogram during or immediately following the administration of a standardized bedbath

3. Minor arrhythmias did occur in 21 percent of the patients with acute myocardial infarction during and immediately following the administration of a standardized bedbath

4. A standardized bedbath given with controlled water and environmental temperature, order and manner of administration, and allotted rest periods is not harmful to the patient with an acute myocardial infarction as demonstrated by the heart rate and ST segment of the electrocardiogram

5. Nursing care administered to the patient with an acute myocardial infarction in the coronary care unit can be evaluated as to the physiological effect the care has on the patient by using the electrocardiogram

Implications

The physiological response of patients with acute myocardial infarction to a bedbath was studied. These findings have implications for the coronary care nursing staff who are responsible for the nursing care of the

patient with an acute myocardial infarction. A bedbath administered in a therapeutic manner is not harmful to the patient with an acute myocardial infarction and can provide the patient with physical and psychological benefits. All aspects of the patient's care should be evaluated in light of information obtained from careful assessment of the patient. Nursing care should be planned and revised according to the patient's tolerance. Actions that produce the least stress and the most satisfaction should be utilized.

These findings have implications for nursing educators who plan and implement a course of study to prepare an individual to become a Registered Nurse. The student should be taught the scientific principles necessary for the administration of a therapeutic bedbath. The student should be taught the parameters needed to evaluate the patient's response to care. Development and revision of a curriculum must be done in the light of research, new concepts, and revision of present concepts that are made in nursing.

These findings have implications for nursing research which is the foundation of the nursing profession. A scientific approach has been utilized to determine the effect of the standardized bedbath on a patient with an

acute myocardial infarction. Alterations from this standardized bedbath should be researched to determine the best nursing action to be taken that will provide the patient with safe, effective, and satisfying nursing care.

Recommendations

Based on the findings of this study, the following recommendations have been made.

1. That a similar study be conducted utilizing patients with an acute myocardial infarction with specific complications such as congeative heart failure, cardiogenic shock, and major arrhythmias

2. That a similar study be conducted using variations in the bedbath procedure such as change in room temperature, temperature of the bath water, patient covering, order of bathing, positioning of patient, and patient participation

3. That a similar study be conducted using different levels of personnel to administer a standardized bedbath such as aides, orderlies, licensed practical nurses, registered nurses without advanced education in the care of cardiac patients, and registered nurses with advanced preparation in the care of cardiac patients

4. That a similar study be conducted to determine alterations that occur during other aspects of nursing care

such as patients being fed; patients feeding themselves; patients ambulating; patients watching television, reading, playing cards, or similar activities; male patients standing to void; catheterization of a patient; waking patients for nursing care and back rubs

5. That a similar study be conducted using a larger population where the placement of patients within the coronary care unit is different

6. That a similar study be conducted comparing and contrasting such factors as age, sex, location of the infarction, and the age of the infarction to alterations in heart rate, ST segment, and the occurrence of arrhythmias

7. That a study be conducted to determine the psychological effects that the bedbath has on the patient with an acute myocardial infarction

Developing the art of nursing is a life-time process (Rogers 1966). Research is one of many components that is involved in the development and growth of the art of nursing. Review of the literature revealed some of the physical and psychological benefits that occur in the patient when given a bedbath. From the results of this study it can be stated that a bedbath given in a therapeutic

manner to a patient with acute myocardial infarction is not harmful to that patient.

Nursing is developing a sound body of scientific knowledge that is the requisite of a profession. A challenge is directed to every nurse to utilize all opportunities available to add to this growing body of knowledge.

APPENDIX A

INFORMATION ON PATIENTS WITH AN ACUTE

MYOCARDIAL INFARCTION

Code number_____ Date_____

Patient's name_____ Date of admission_____

Hospital number_____ Date of onset of s/s

CCU bed number_____ _____

Sex_____ M S D W Monitor lead_____

Doctor_____ Time started_____

Diagnosis_____ Time finished_____

Medications: Total time_____

Times for EKG tracings: Baseline:

Baseline_____ Blood pressure_____

q min. X 5 _____ Temperature_____

_____

_____ After the bath and rest

_____ Blood pressure_____

_____

q 5 min. X 3 _____ Temperature_____

_____

_____

Bath finished Interruptions:

q min. X 5 _____

_____

_____

_____

_____ Unusual behavior:

q 5 min. X 2_____

_____

APPENDIX B

TEXAS WOMAN'S UNIVERSITY
DALLAS, TEXAS 75235

83

August 7, 1975

COLLEGE OF NURSING

Betty Barratt
Texas Woman's University
1810 Inwood Rd.
Dallas, Texas 75235

Dear Ms. Barratt,

The Human Research Review Committee has reviewed and approved your protocol, "Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath."

Sincerely,

Lois Hough

Lois Hough, Chairman
Human Research Review Committee

OFFICE OF THE ASSOCIATE DEAN
TEXAS WOMAN'S UNIVERSITY
DALLAS CENTER
1810 INWOOD ROAD
DALLAS, TEXAS 75235

OFFICE OF THE DEAN
TEXAS WOMAN'S UNIVERSITY
BOX 23026, TWU STATION
DENTON, TEXAS 76204

OFFICE OF THE ASSOCIATE DEAN
TEXAS WOMAN'S UNIVERSITY
1130 M. D. ANDERSON BLVD.
TEXAS MEDICAL CENTER
HOUSTON, TEXAS 77025

APPENDIX C

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING
DENTON, TEXAS

DALLAS CENTER
1810 Inwood Road
Dallas, Texas 75235

HOUSTON CENTER
1130 M.D. Anderson Blvd.
Houston, Texas 77025

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE MARY LANNING MEMORIAL HOSPITAL

GRANTS TO BETTY JEAN BARRATT, B.S.N.

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem:

The problem of this study will be to determine alterations that occur in selected physiological phenomenon when a patient with an acute myocardial infarction is given a bedbath.

The conditions mutually agreed upon are as follows:

1. The agency (may) (may not) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (may not) be identified in the final report.
3. The agency (wants) (does not want) a conference with the student when the report is completed.
4. The agency is (willing) (unwilling) to allow the completed report to be circulated through interlibrary loan.
5. Other: _____

Date December 17, 1975

Signature of Agency Personnel Administrat

Signature of student

Linda B. Brown
Signature of Faculty Advisor

*Fill out and sign three copies to be distributed as follows: Original -- Student; first copy -- agency; second copy -- T.W.U. College of Nursing.

APPENDIX D

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date 12/12/75

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date 12-15-75

Signature of Physician _____

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date January 13, 1976

Signature of Physician _____

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date January 15, 1976

Signature of Physician [signature]

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date 2-10-76

PHYSICIAN'S CONSENT FORM

I grant to Betty Jean Oliver, B.S.N., a student enrolled in the Medical-Surgical Nursing Program leading to a Master's Degree at Texas Woman's University, permission to utilize my patients who are in the coronary care unit to study the Physiological Response of Patients with Acute Myocardial Infarction to a Bedbath.

Date 5-12-76

APPENDIX E

HEART RATE OF SUBJECTS BEFORE, DURING, AND AFTER

THE ADMINISTRATION OF A BEDBATH

Patient Number	Base-line	Number of Minutes after Starting the Bedbath								Number of Minutes after Starting the Bedbath						
		1	2	3	4	5	10	15	20	1	2	3	4	5	10	15
1	68	68	68	71	88	68	83	68	63	83	63	63	63	63	63	68
2	83	83	83	83	83	79	83	83	75	79	79	83	83	79	79	83
3	115	125	115	115	115	115	115	115	125	115	115	115	115	107	115	115
4	83	83	83	83	83	83	83	83	83	79	79	79	79	79	79	79
5	83	88	88	88	88	83	88	79	88	83	83	83	88	88	88	88
6	94	83	94	88	94	94	94	107	107	107	100	100	100	100	107	100
7	75	75	79	75	75	75	75	75	71	71	68	68	68	71	71	71
8	100	100	94	94	94	94	94	94	94	94	94	94	94	94	94	94
9	71	75	75	71	75	75	75	75	75	71	71	75	71	75	71	75
10	56	56	54	56	58	56	54	58	54	54	56	52	56	54	54	54
11	68	68	71	71	71	71	68	71	71	71	75	71	68	71	65	68
12	68	65	60	68	65	63	63	68	63	63	65	63	65	65	65	68
13	83	83	83	83	83	83	83	79	79	79	83	83	83	79	79	88
14	71	65	79	71	71	71	68	68	65	63	63	63	63	63	58	58

HEART RATE OF SUBJECTS BEFORE, DURING, AND AFTER

THE ADMINISTRATION OF A BEDBATH (CONTINUED)

Patient Number	Base-line	Number of Minutes after Starting the Bedbath								Number of Minutes after Starting the Bedbath						
		1	2	3	4	5	10	15	20	1	2	3	4	5	10	15
15	79	94	79	83	83	83	79	75	79	79	83	83	79	79	79	79
16	88	88	83	83	88	83	83	83	83	79	79	79	79	79	83	83
17	71	75	75	71	75	75	75	75	79	75	75	75	75	75	75	79
18	79	75	71	75	75	71	65	83	71	71	71	71	68	71	79	65
19	56	65	63	71	63	68	65	60	68	56	54	52	54	52	52	54
20	71	71	71	68	68	71	71	71	68	68	71	68	68	68	68	71
21	94	88	94	94	94	94	94	94	100	115	115	115	115	115	100	107
22	100	100	100	100	100	100	100	100	94	94	94	94	88	94	94	88
23	94	94	94	94	94	94	94	94	94	94	94	94	94	94	94	94
24	100	107	107	100	100	100	100	107	100	100	100	100	107	107	100	100
25	83	83	83	83	83	83	83	88	83	88	83	83	83	79	79	83
26	83	79	100	83	88	83	88	88	83	94	71	83	83	94	79	83
27	68	71	68	68	71	71	71	71	71	68	65	68	71	71	68	65
28	71	75	71	71	71	71	75	71	75	68	71	71	68	68	68	71

APPENDIX F

TIME OF OCCURRENCE OF ARRHYTHMIAS

Patient Number	Base-line	Minutes After Starting the Bedbath								Minutes After Completing the Bedbath						
		1	2	3	4	5	10	15	20	1	2	3	4	5	10	20
1					X		X			X						
2																
3																
4																
5																
6																
7				X			X									
8										X	X					
9																
10																
11																
12																
13																
14																
15																
16																
17										X	X	X				
18																

TIME OF OCCURRENCE OF ARRHYTHMIAS (CONTINUED)

Patient Number	Base-line	Minutes After Starting the Bedbath								Minutes After Completing the Bedbath						
		1	2	3	4	5	10	15	20	1	2	3	4	5	10	20
19																
20																
21																
22																
23																
24																
25																
26																
27					X											
28				X		X										

APPENDIX G

TEMPERATURE BEFORE AND AFTER THE BEDBATH

Patient Number	Before	After	Difference	Patient Number	Before	After	Difference
1	97.4	97.5	+0.1	15	98.5	98.8	+0.3
2	97.7	97.5	-0.2	16	98	99.2	+1.2
3	97.8	97.6	-0.2	17	96.9	96.4	-0.5
4	97.5	97.7	+0.2	18	97.2	97.6	+0.4
5	100.5	100.8	+0.3	19	97.7	97	-0.7
6	98.5	97.9	-0.6	20	98.5	98.9	+0.4
7	98.9	98.5	-0.4	21	99.9	100.9	+1.0
8	97.6	98.6	+1.0	22	99	99.2	+0.2
9	97.4	97	-0.4	23	98.6	98.3	-0.3
10	98.8	97.7	-1.1	24	97.5	97.6	+0.1
11	98.4	98.8	+0.4	25	99	98.9	-0.1
12	98	98.4	+0.4	26	97	97	0
13	98.6	98.4	-0.2	27	98.6	99.5	+0.9
14	98.9	98.1	-0.8	28	97.5	98	+0.5

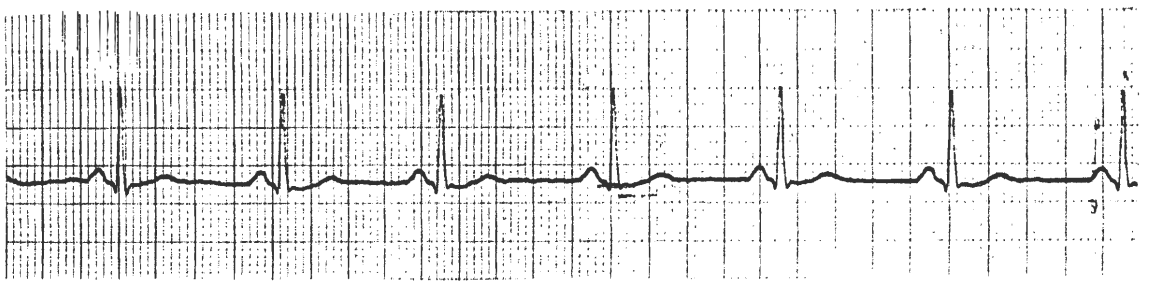
APPENDIX H

SYSTOLIC BLOOD PRESSURE BEFORE AND AFTER THE BEDBATH

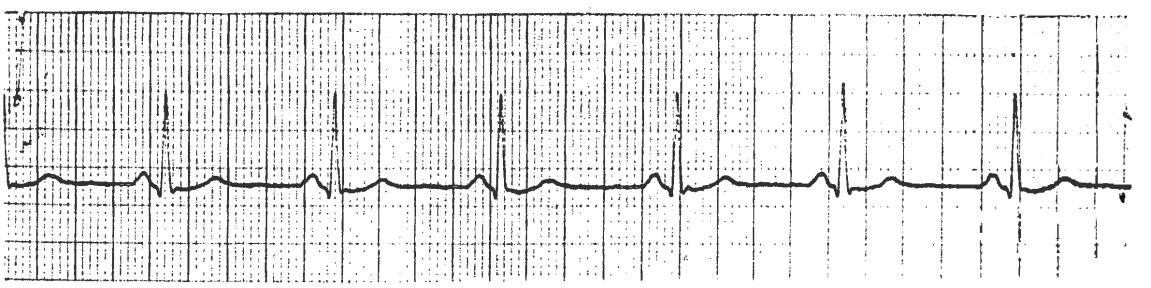
Patient Number	Before	After	Difference	Patient Number	Before	After	Difference
1	140	152	+12	15	110	112	+ 2
2	160	152	- 8	16	104	102	- 2
3	160	150	-10	17	92	90	- 2
4	96	102	+ 6	18	98	102	+ 4
5	140	130	-10	19	120	110	-10
6	110	106	- 4	20	110	102	- 8
7	142	138	- 4	21	106	100	- 6
8	112	118	+ 6	22	102	122	+20
9	94	98	+ 4	23	110	106	- 4
10	130	120	-10	24	116	132	+16
11	148	106	-42	25	112	98	-14
12	98	104	6	26	120	118	- 2
13	110	117	+ 7	27	110	100	-10
14	114	124	+10	28	140	100	-40

APPENDIX I

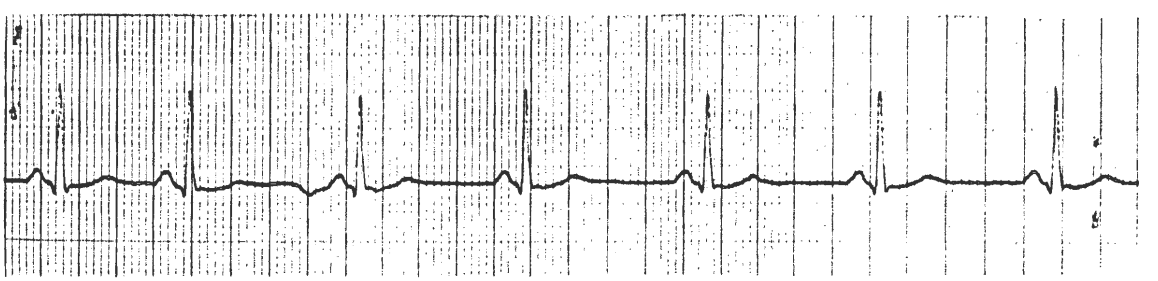
Patient 1 Monitor lead II Medication



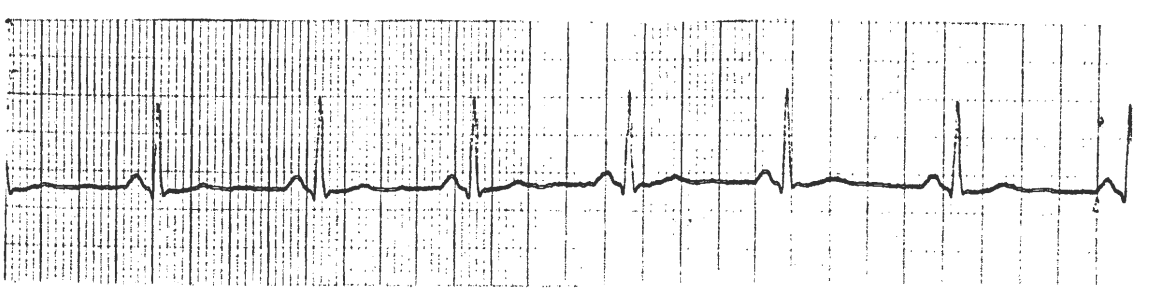
Baseline



1 minute

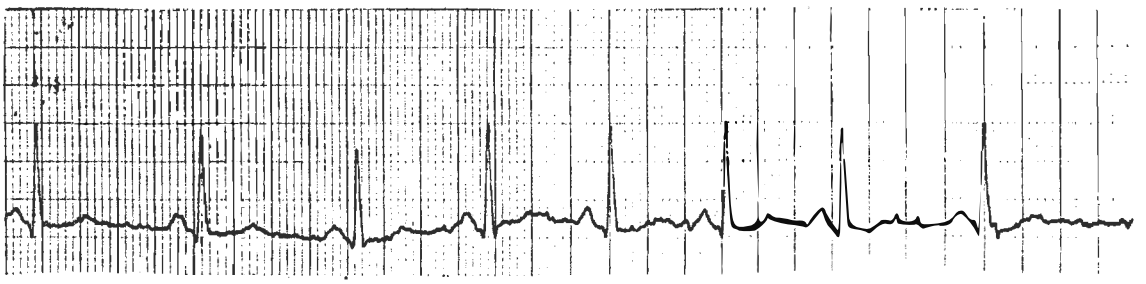


2 minutes

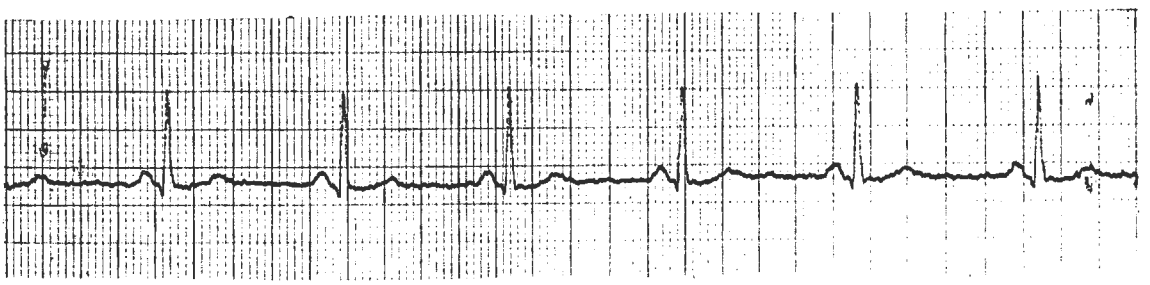


3 minutes

Patient 1



4 minutes



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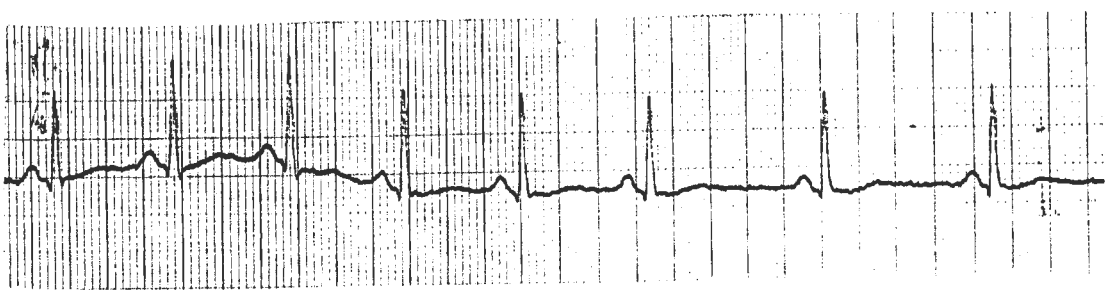

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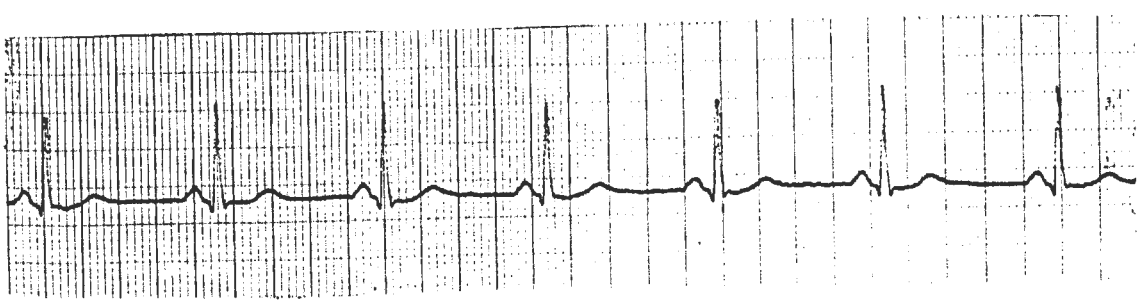
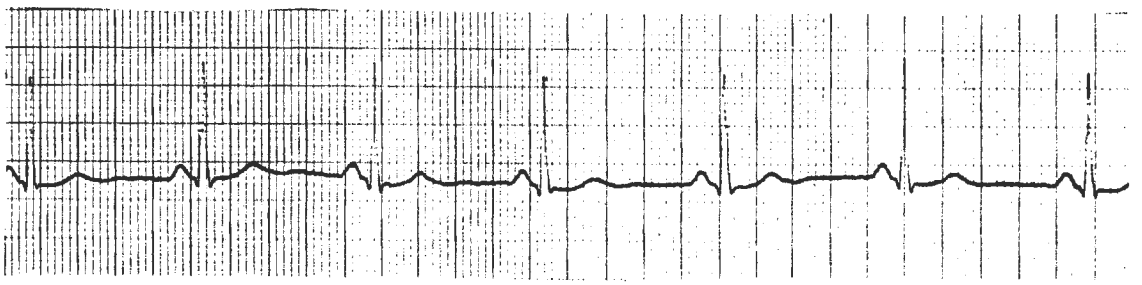


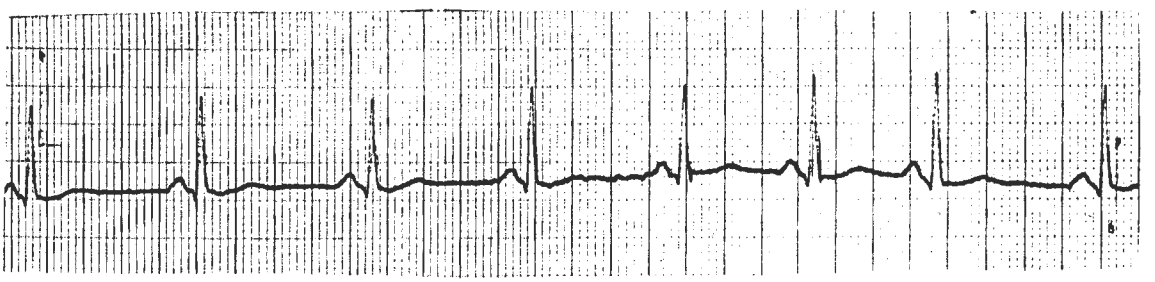
15 minutes

Patient 1



20 minutes

Bath Completed



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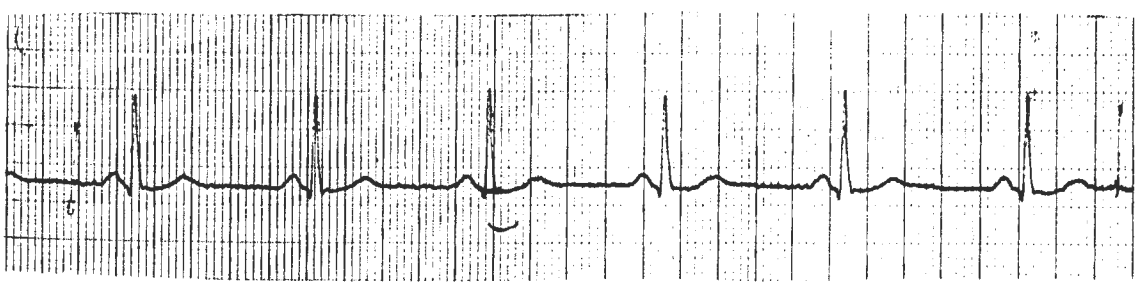
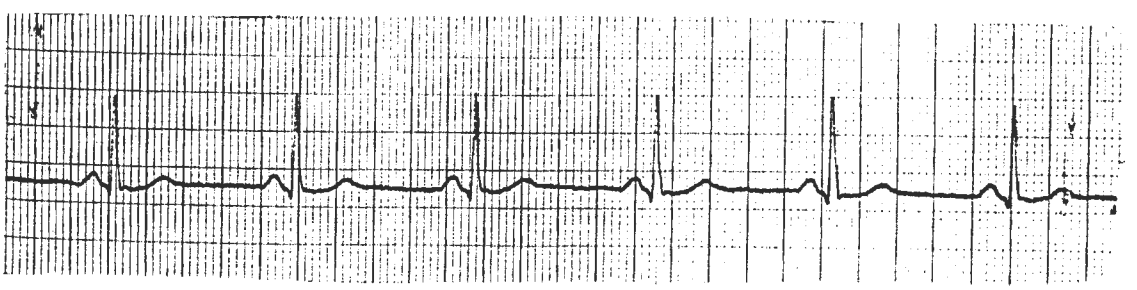
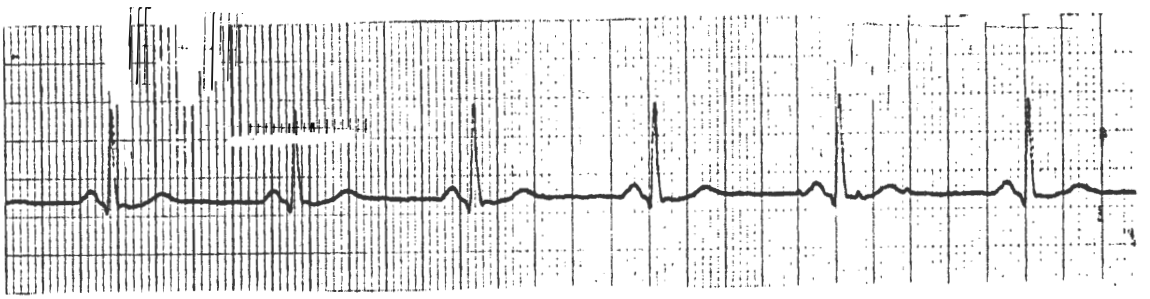


2 minutes

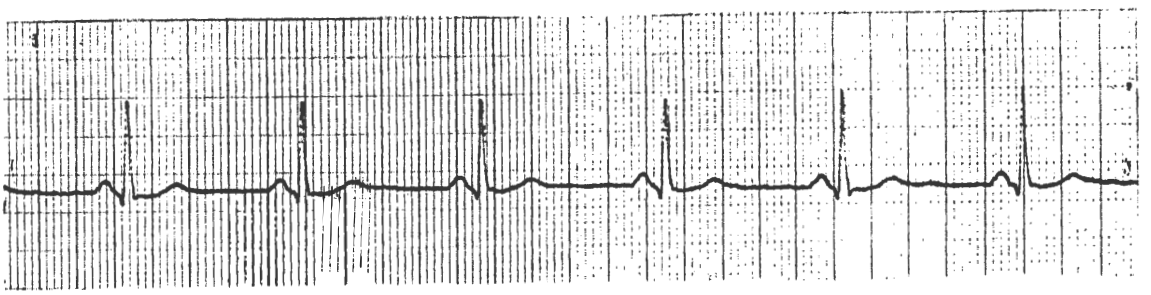


3 minutes

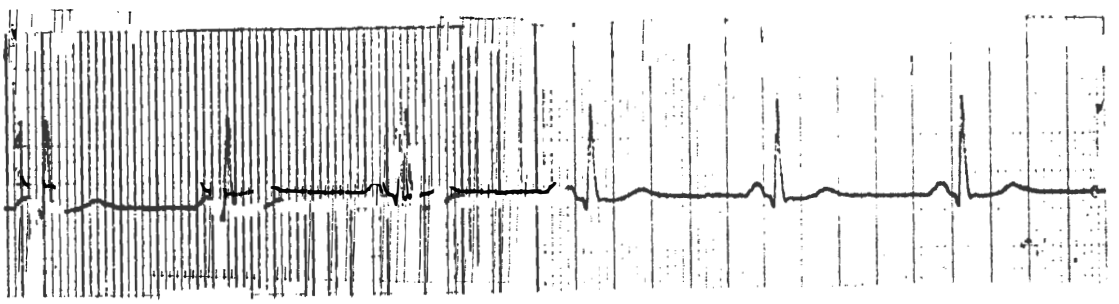
Patient 1



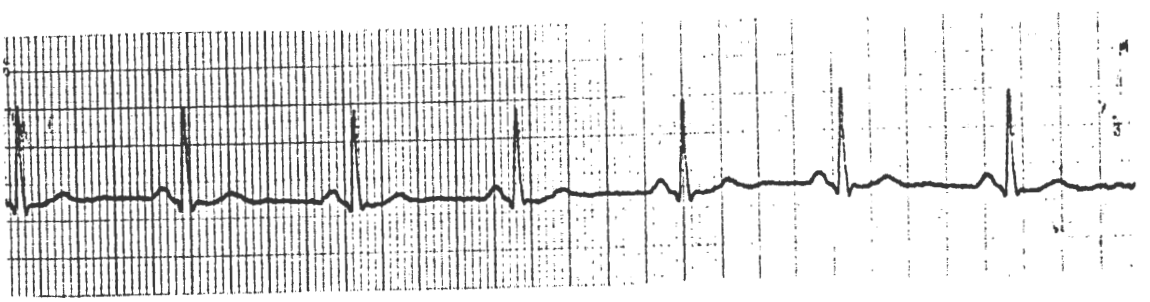
4 minutes



5 minutes

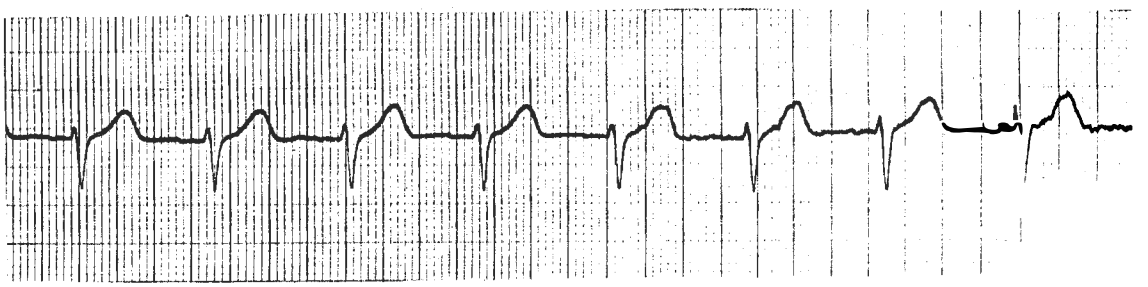


10 minutes

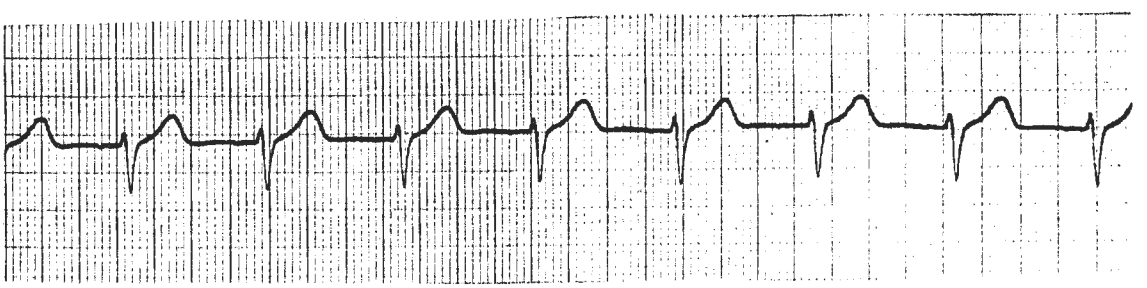


15 minutes

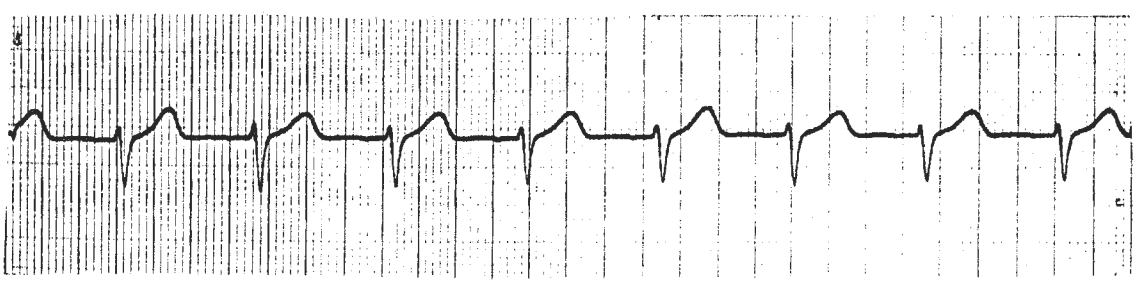
Patient 2 Monitor lead V_1 Medication



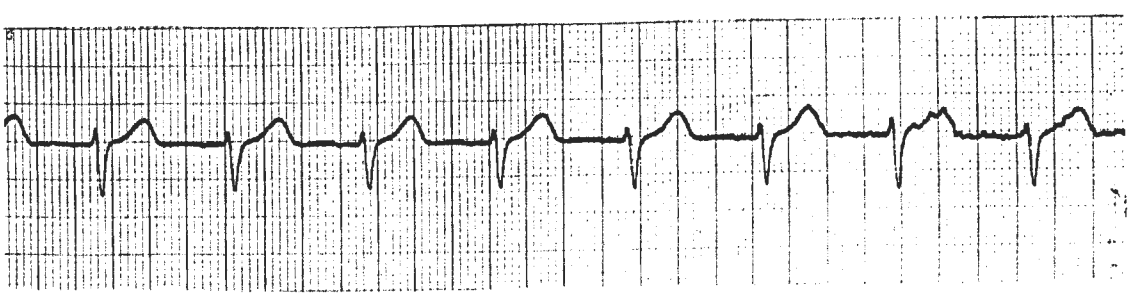
Baseline



1 minute

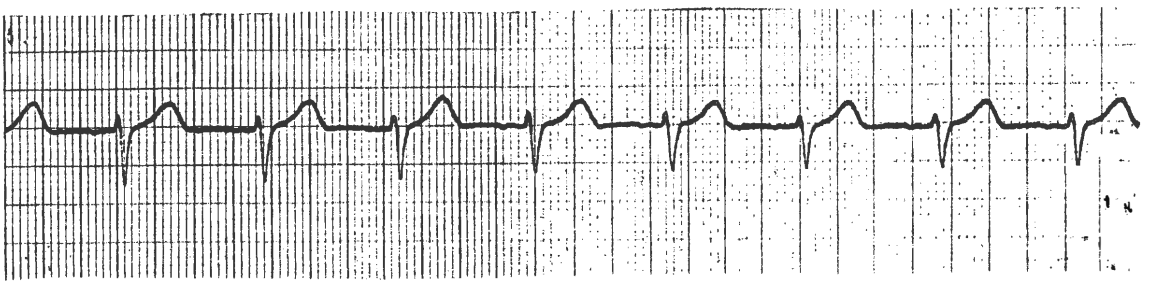


2 minutes

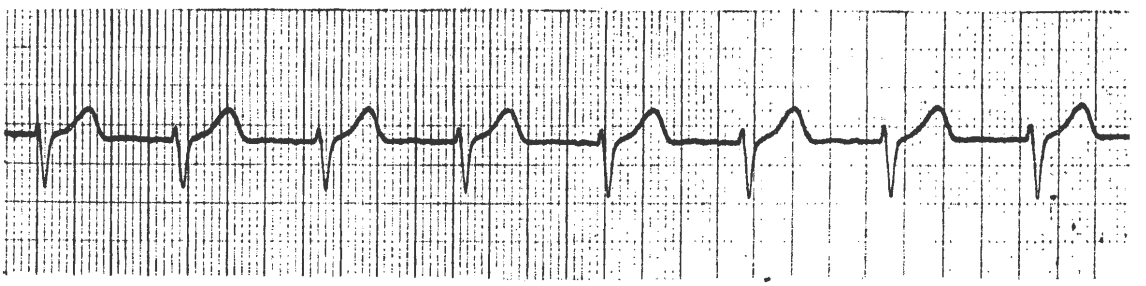


3 minutes

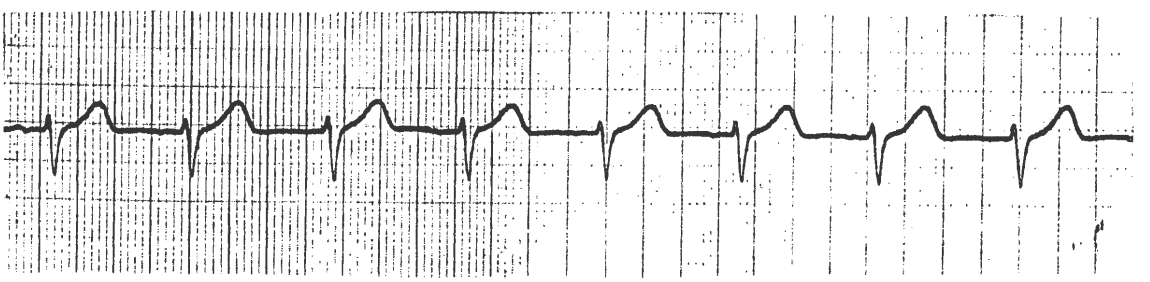
Patient 2



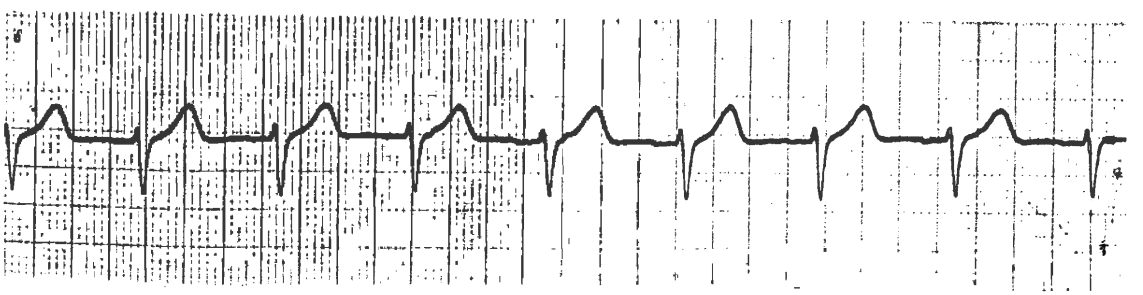
4 minutes



5 minutes

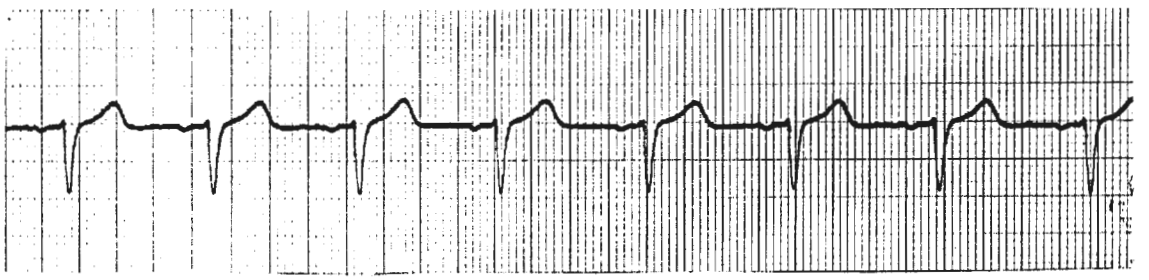


10 minutes



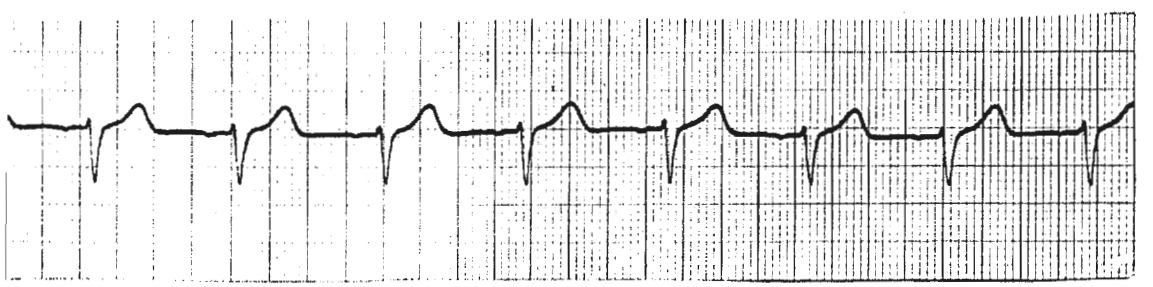
15 minutes

Patient 2

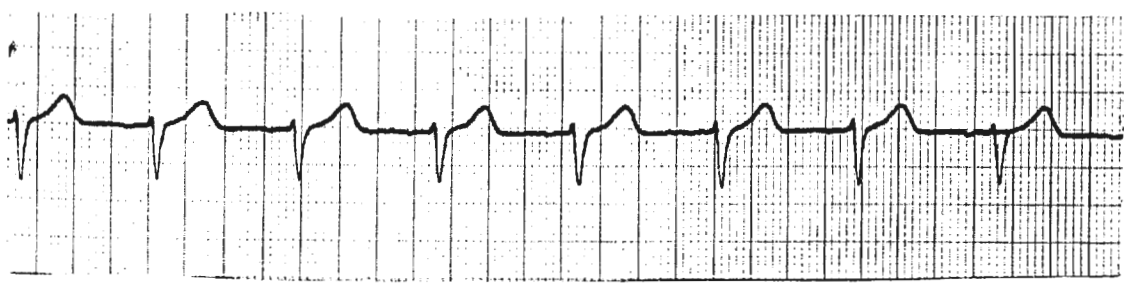


20 minutes

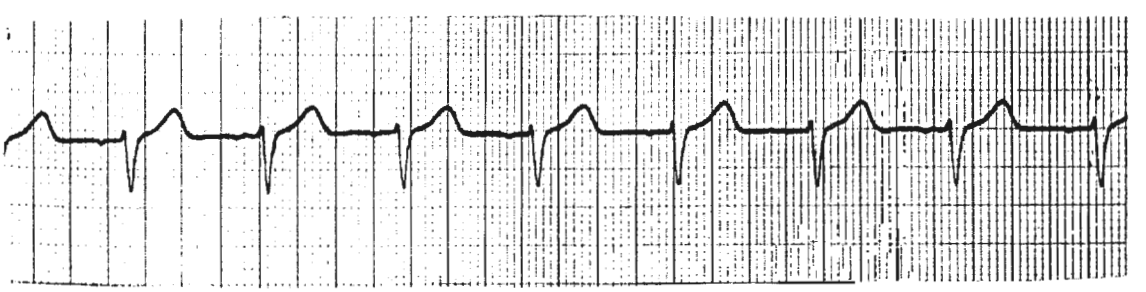
Bath Completed



1 minute

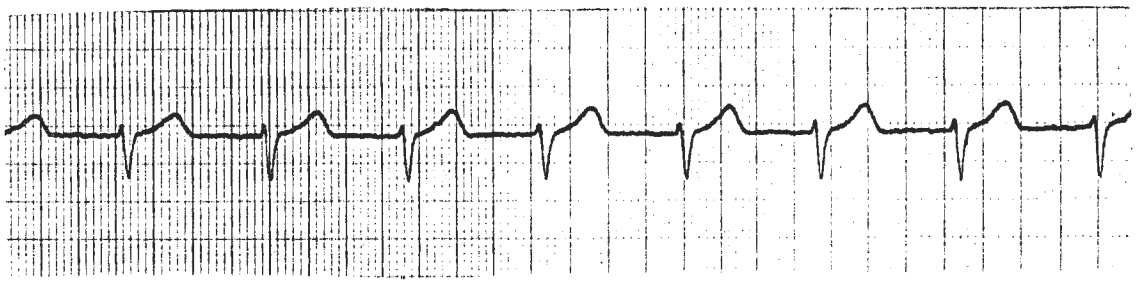


2 minutes

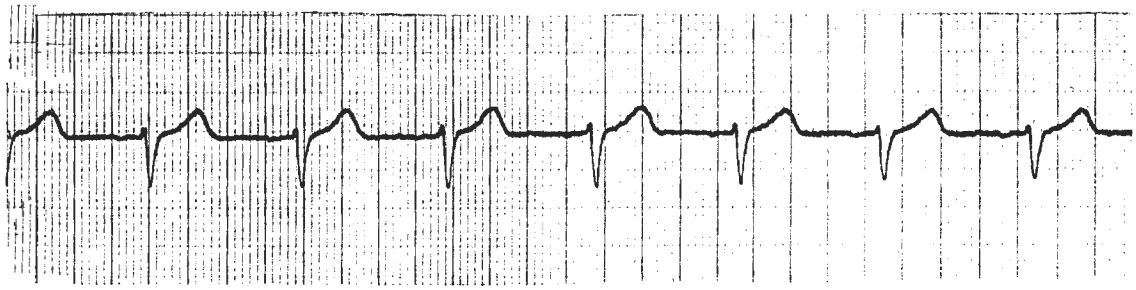


3 minutes

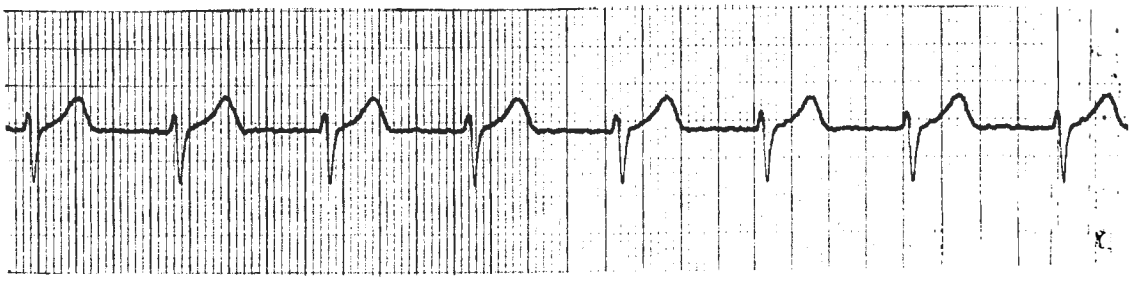
Patient 2_____



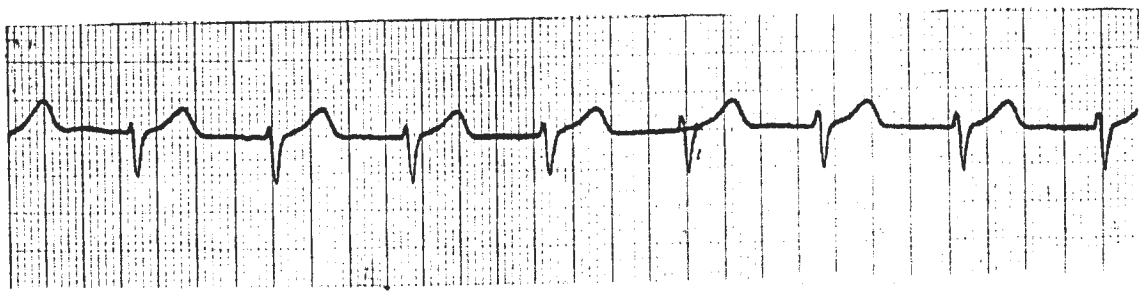
4 minutes



5 minutes

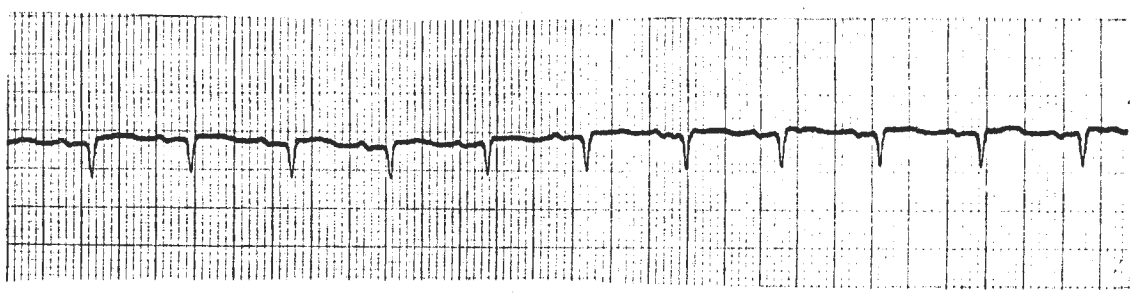


10 minutes

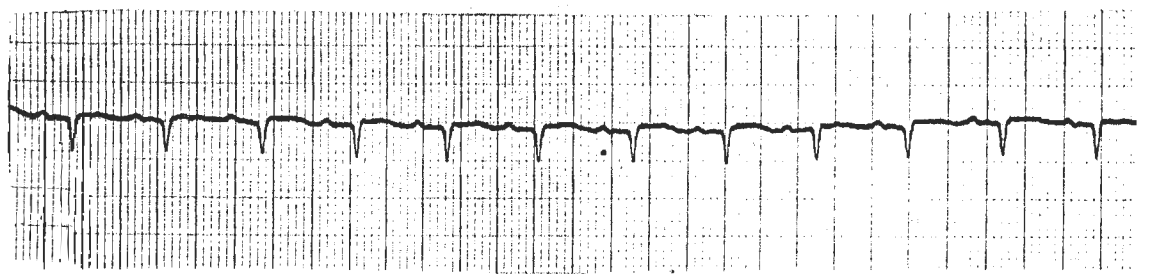


15 minutes

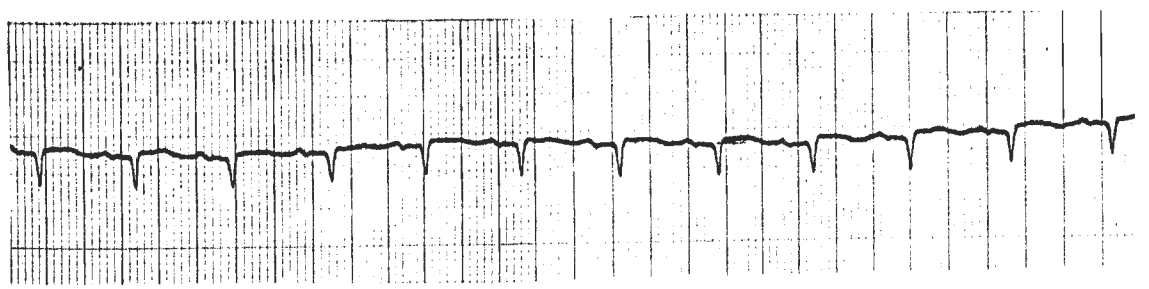
Patient 3 Monitor lead V_1 Medication



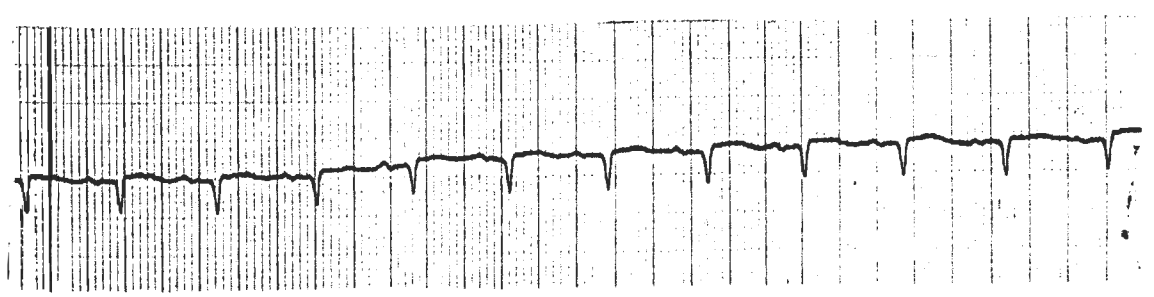
Baseline



1 minute

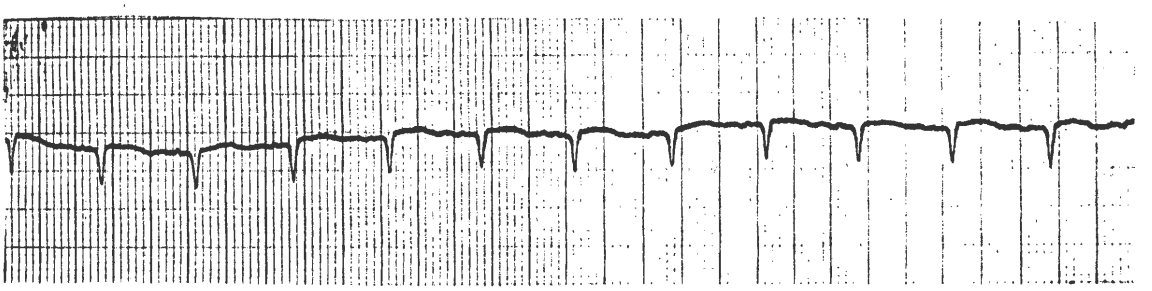


2 minutes

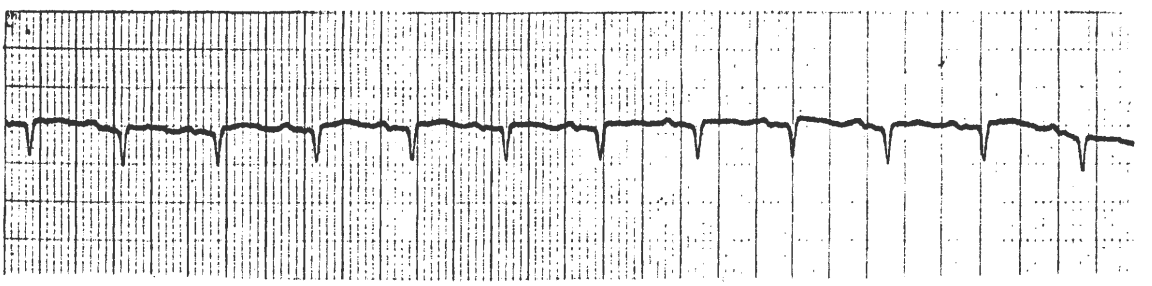


3 minutes

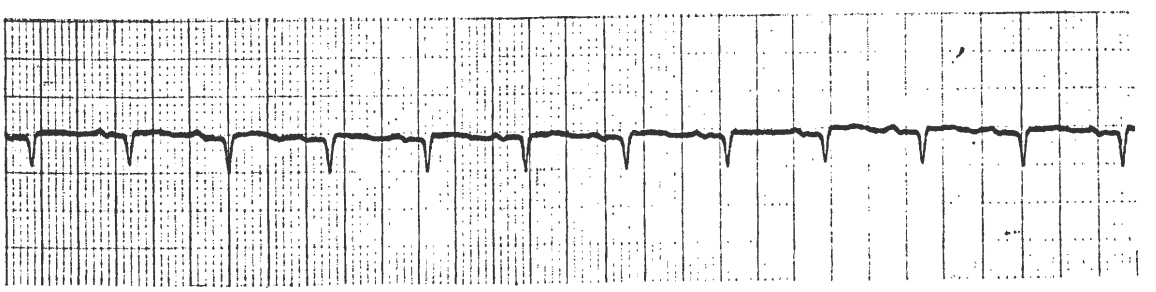
Patient 3



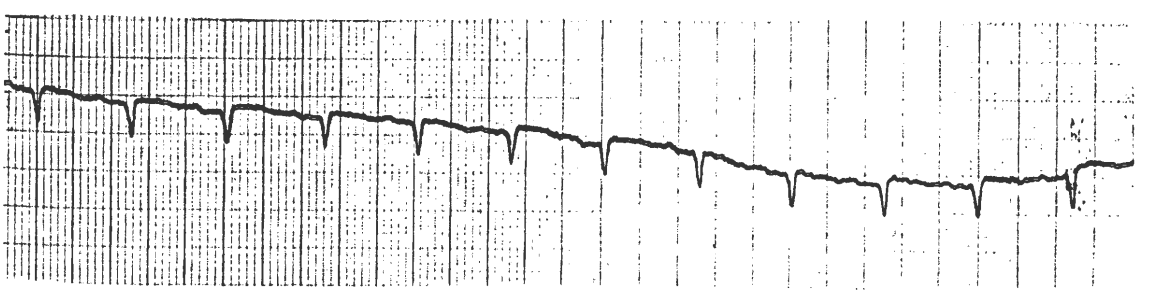
4 minutes



5 minutes

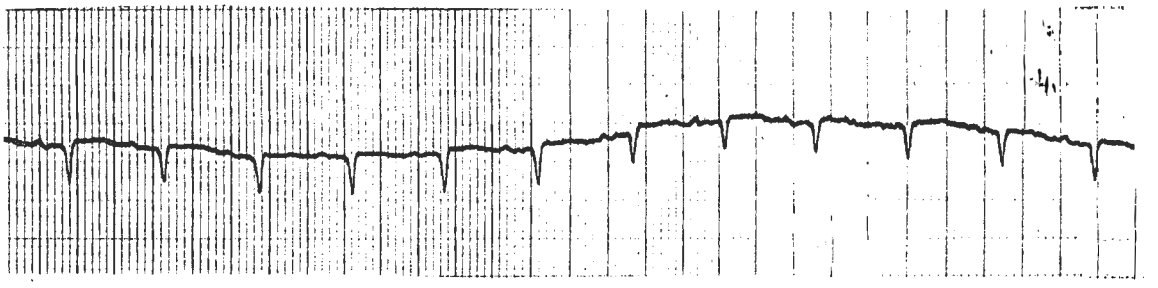


10 minutes



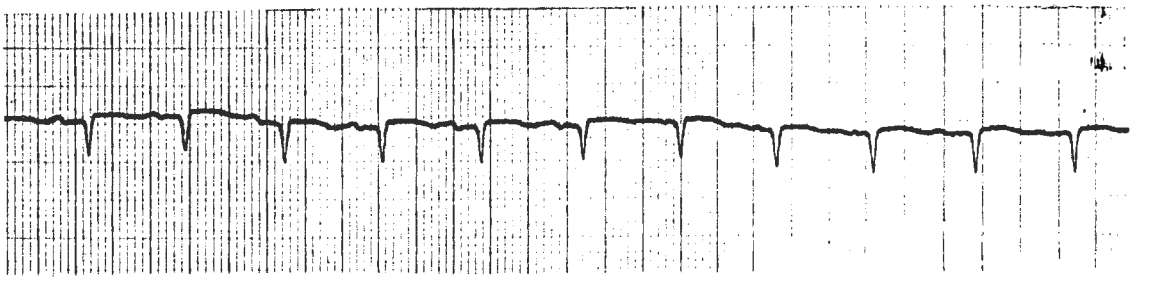
15 minutes

Patient 3

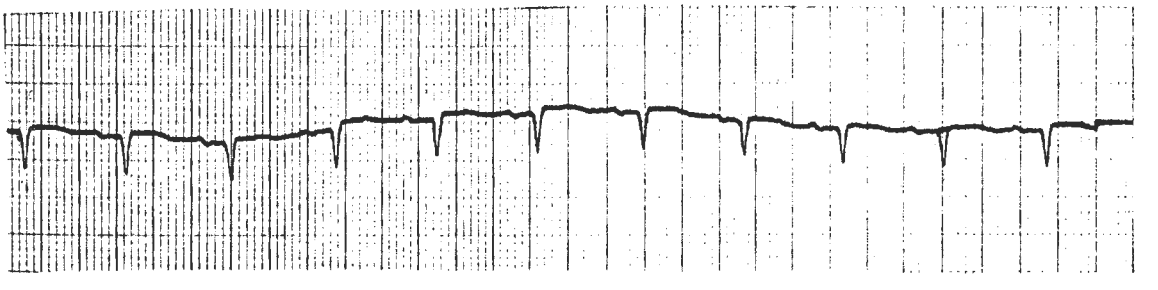


20 minutes

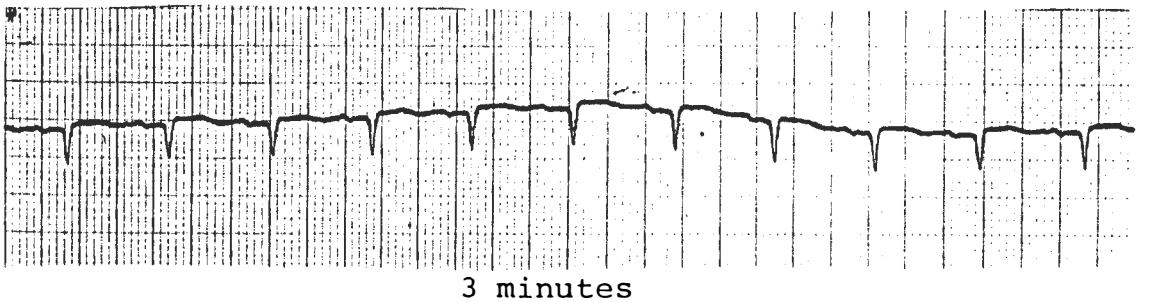
Bath Completed



1 minute

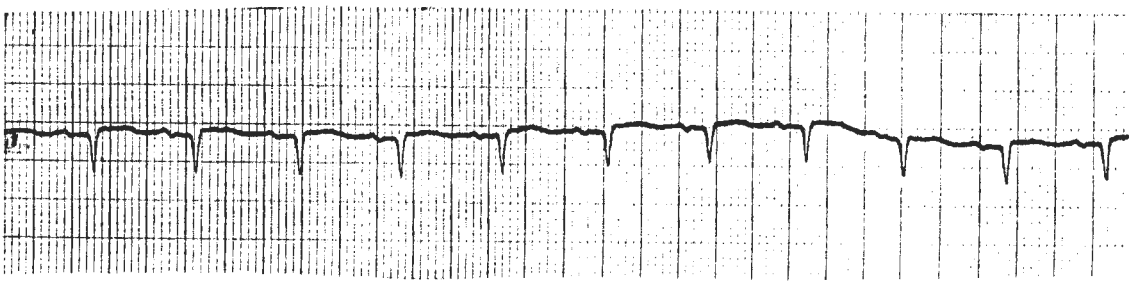


2 minutes

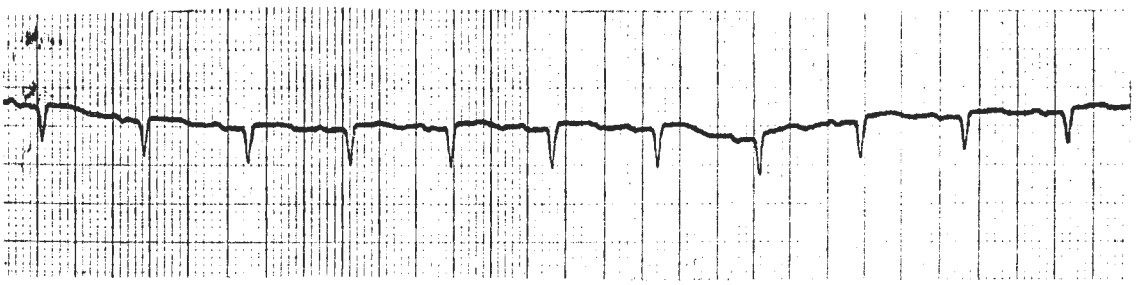


3 minutes

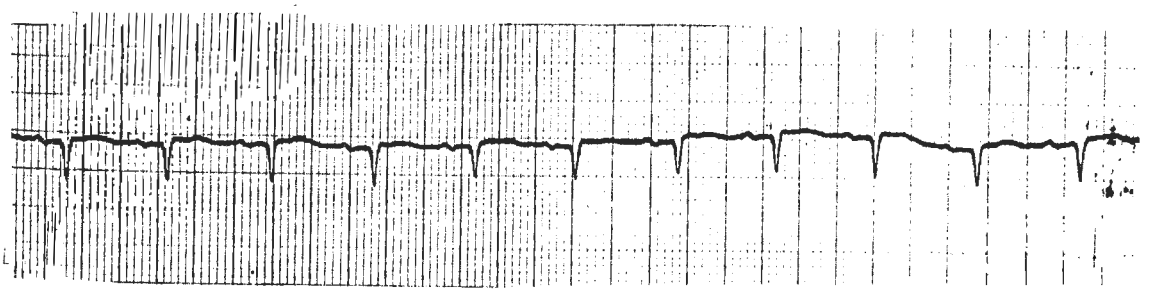
Patient 3



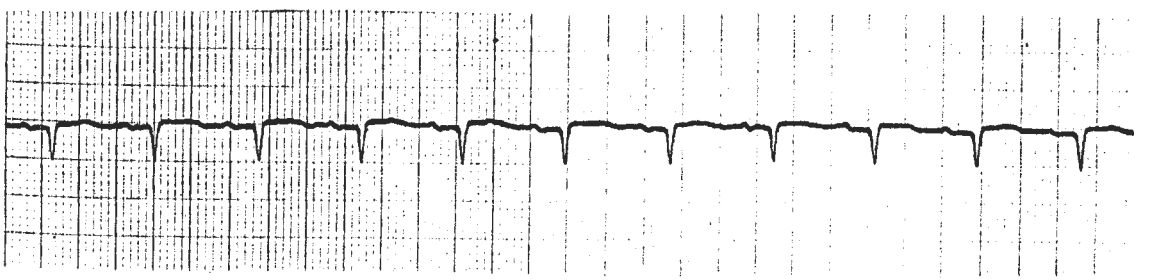
4 minutes



5 minutes

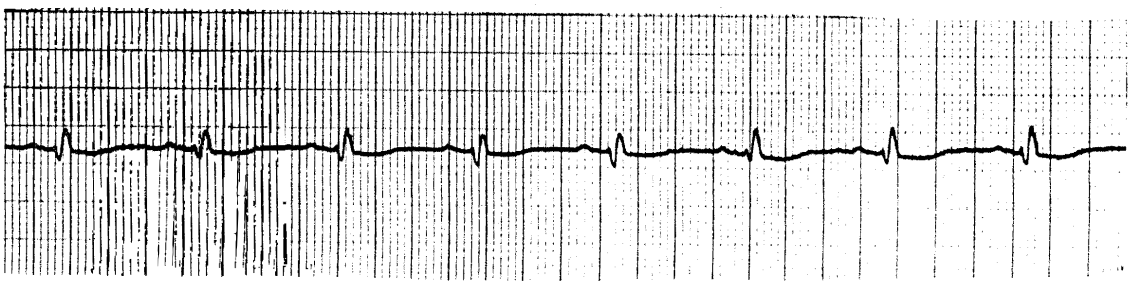


10 minutes

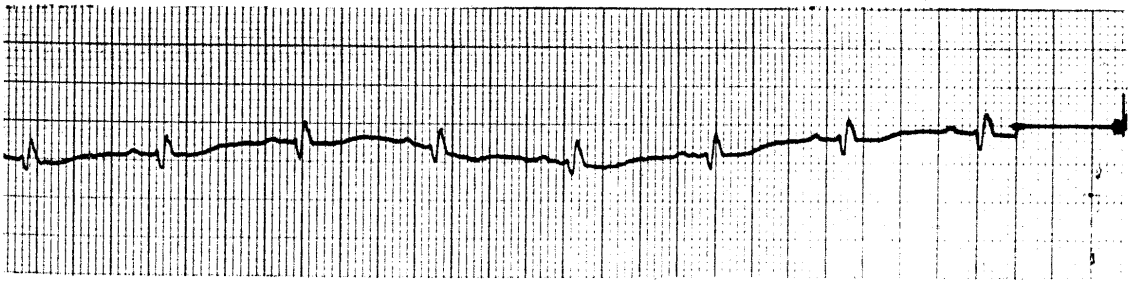


15 minutes

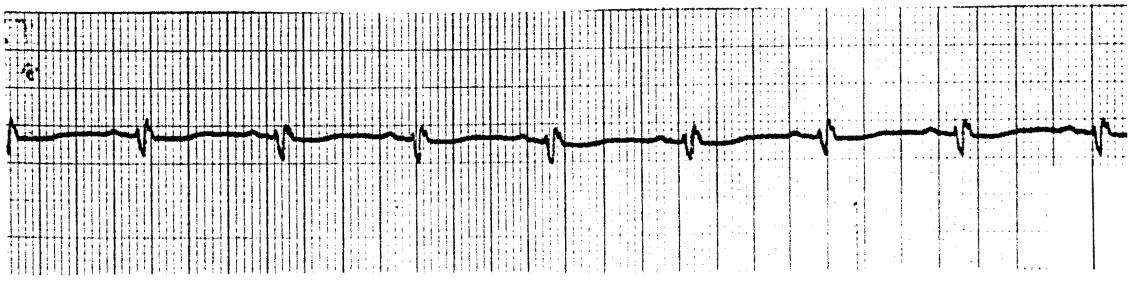
Patient 4 Monitor lead II Medication _____



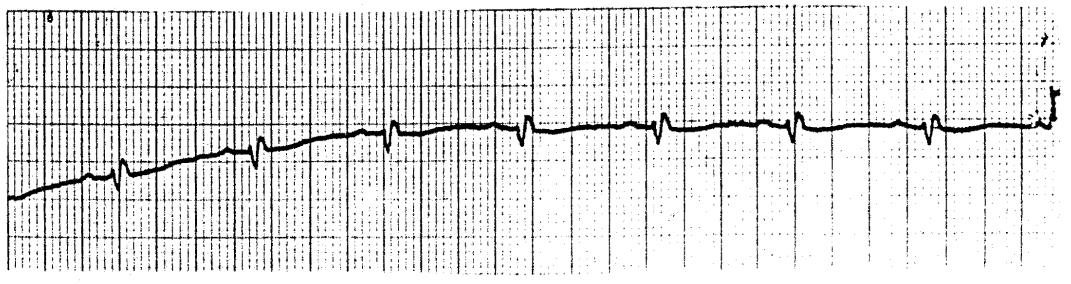
Baseline



1 minute

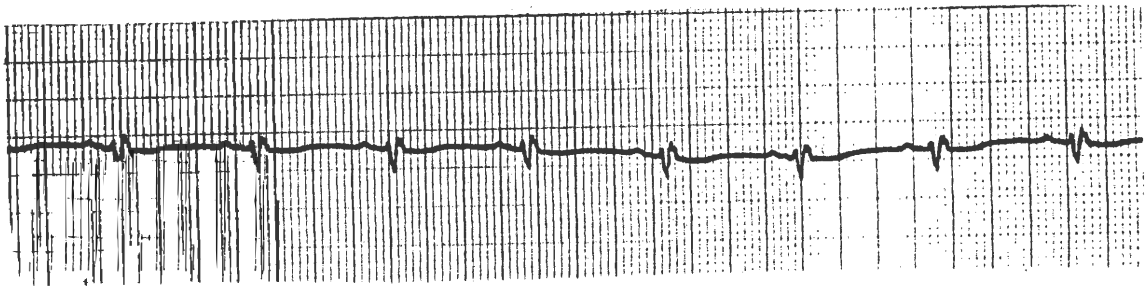


2 minutes

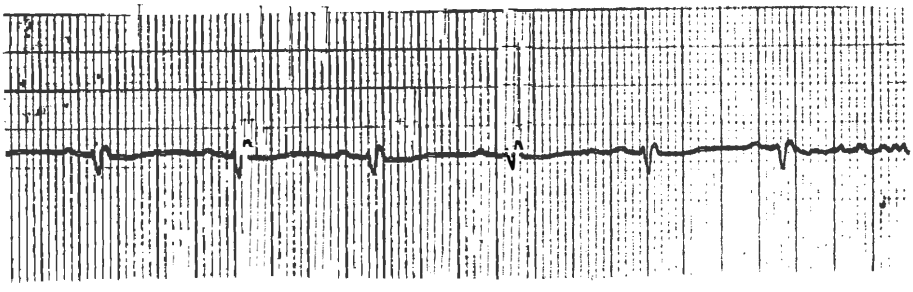


3 minutes

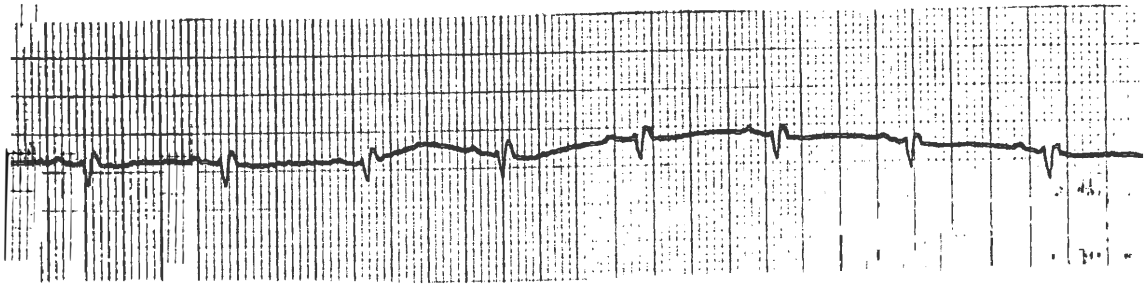
Patient 4



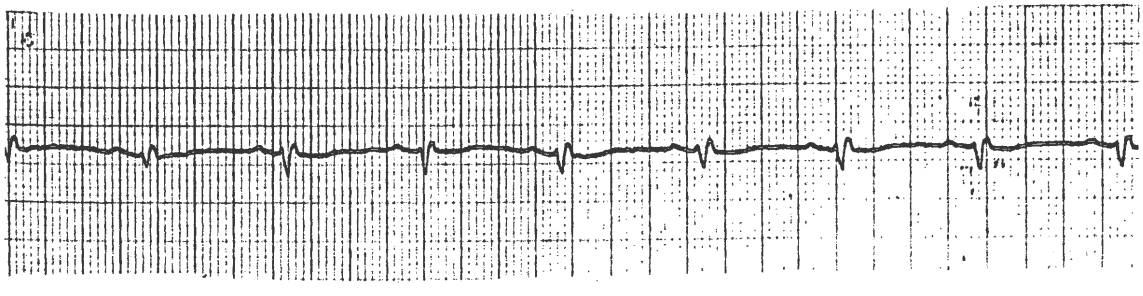
4 minutes



5 minutes

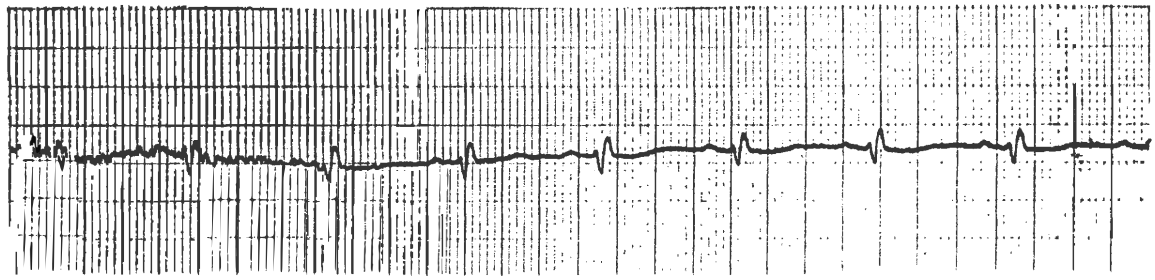


10 minutes



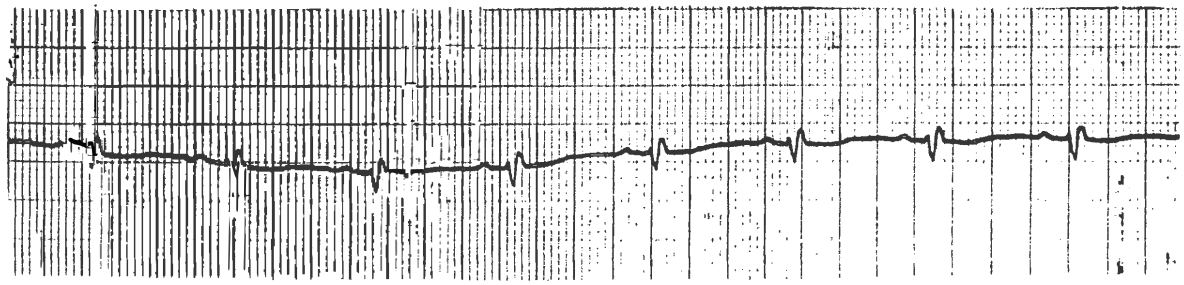
15 minutes

Patient 4

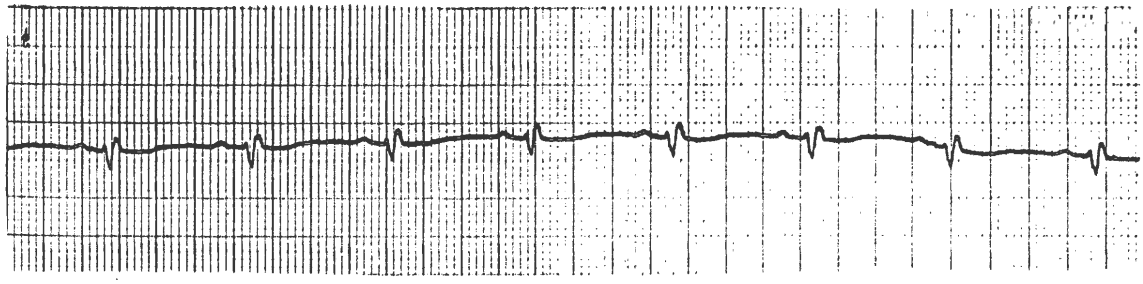


20 minutes

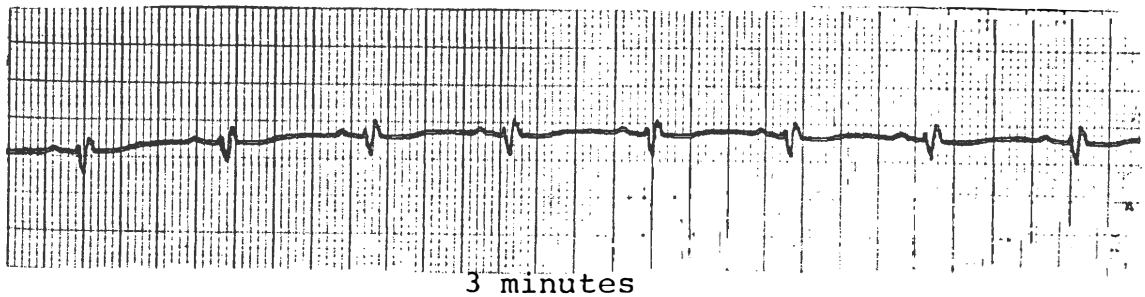
Bath Completed



1 minute

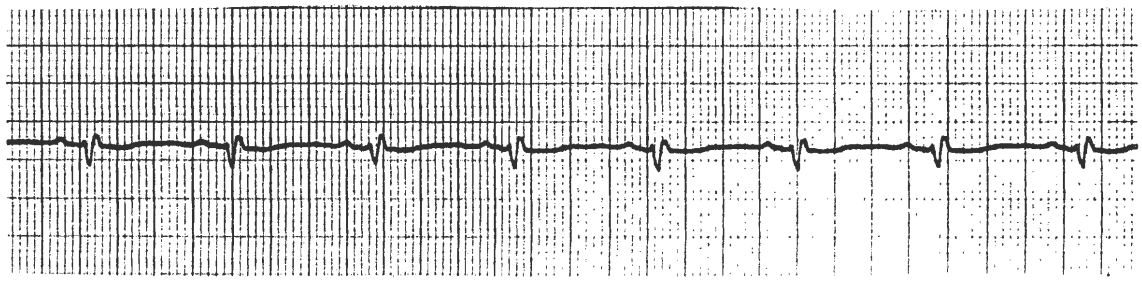


2 minutes

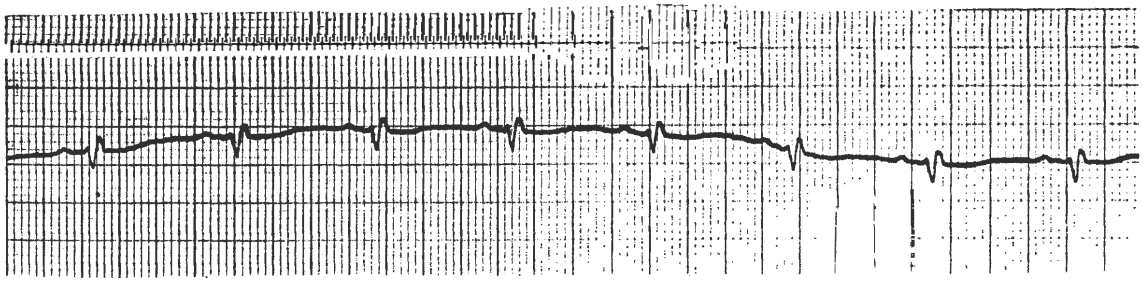


3 minutes

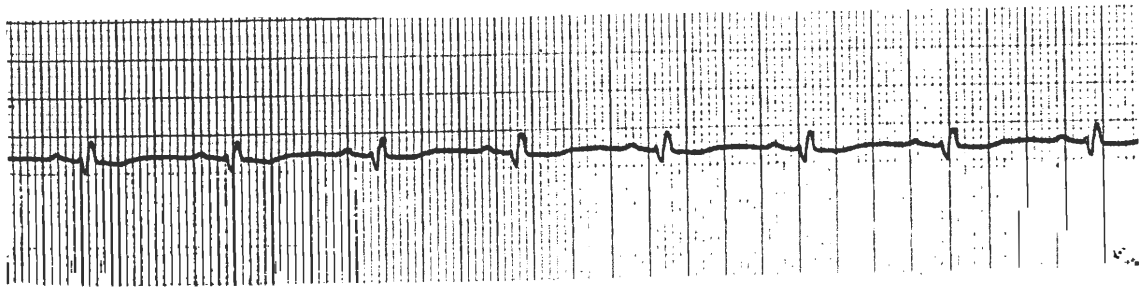
Patient 4



4 minutes



5 minutes



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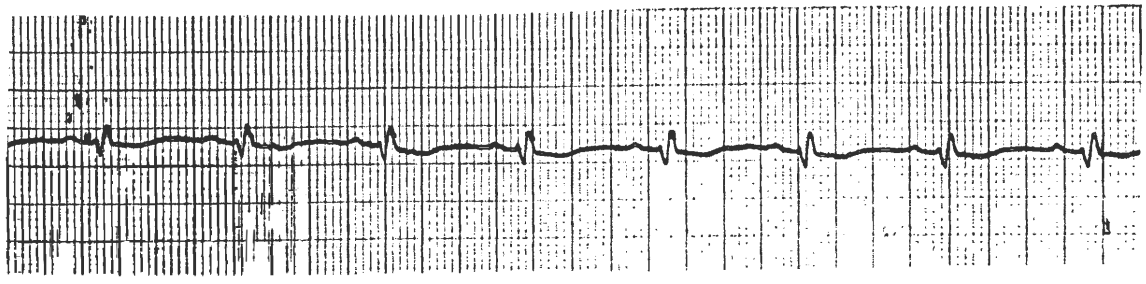
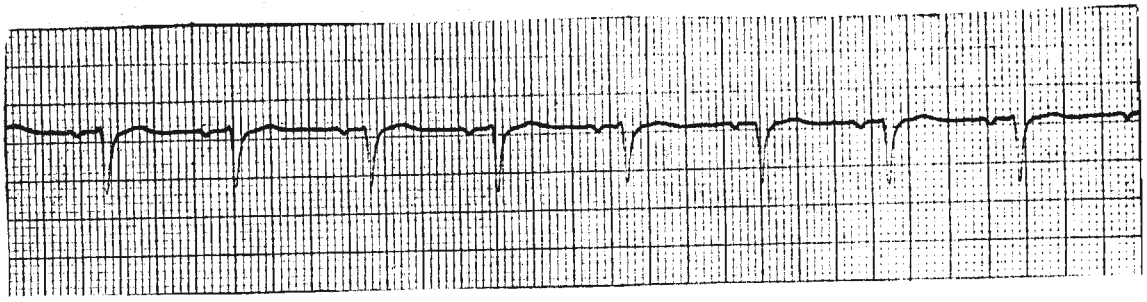
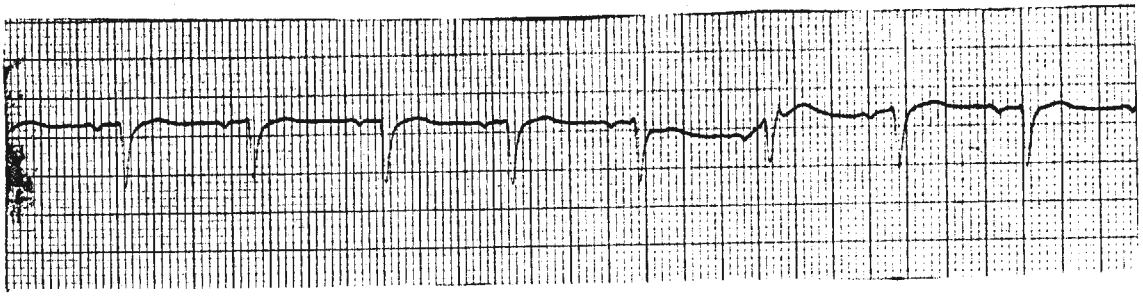


15 minutes

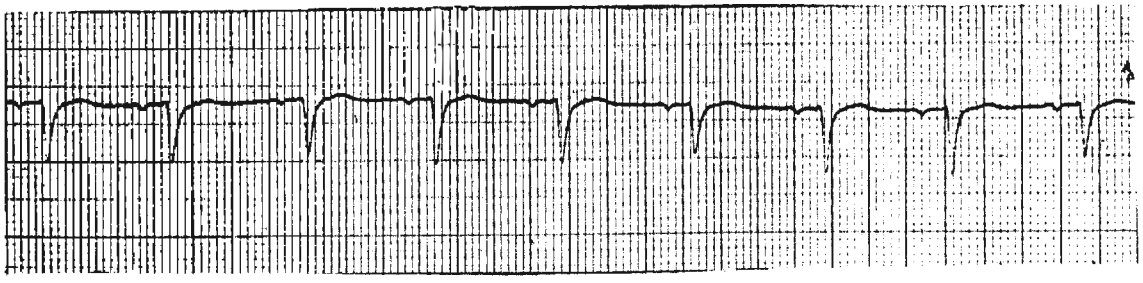
Patient 5 Monitor lead V_1 Medication _____



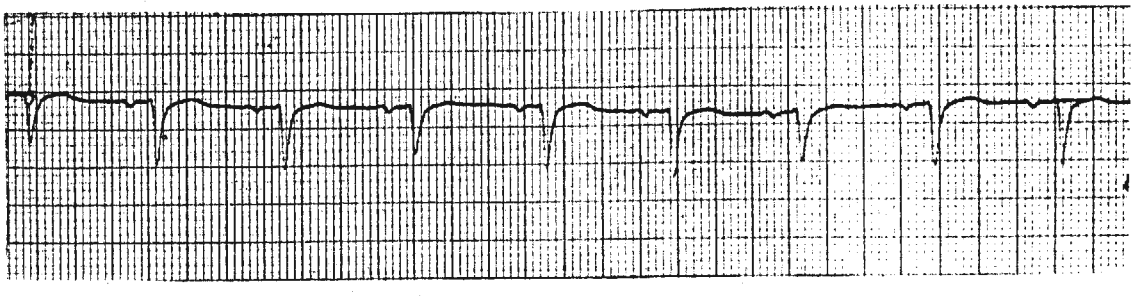
Baseline



1 minute

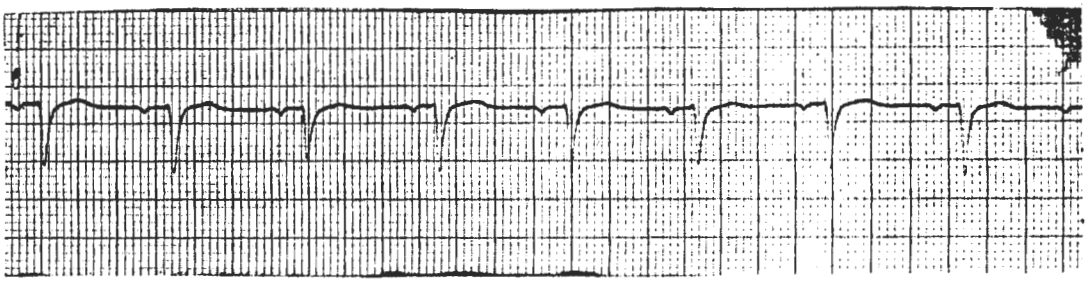


2 minutes

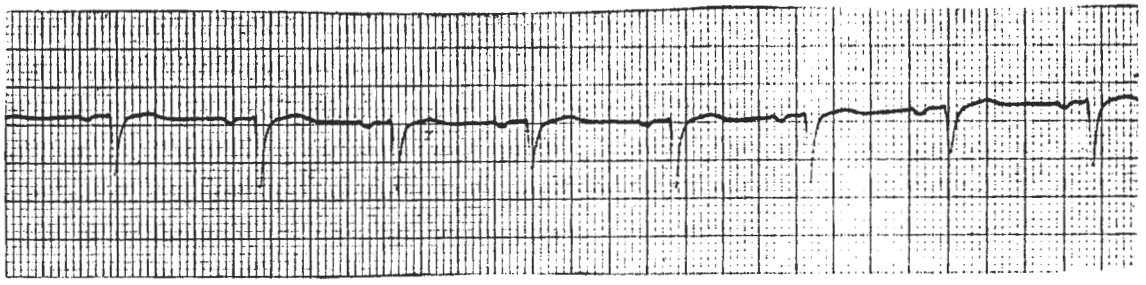


3 minutes

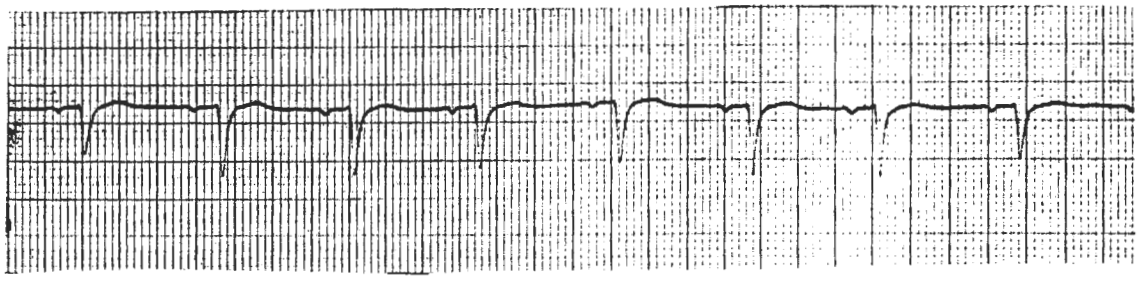
Patient 5



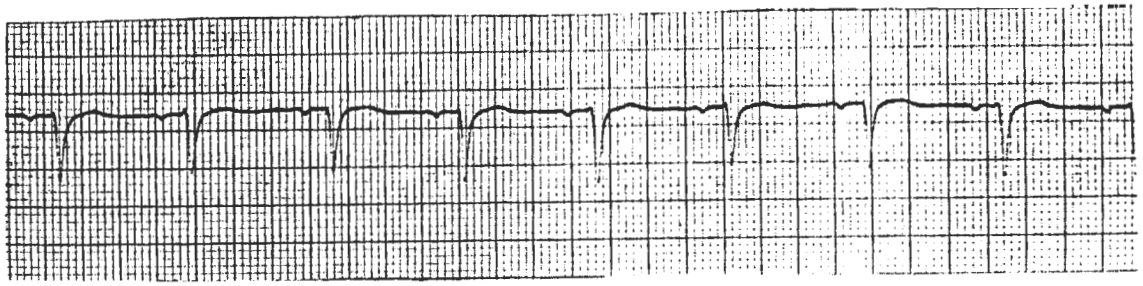
4 minutes



5 minutes

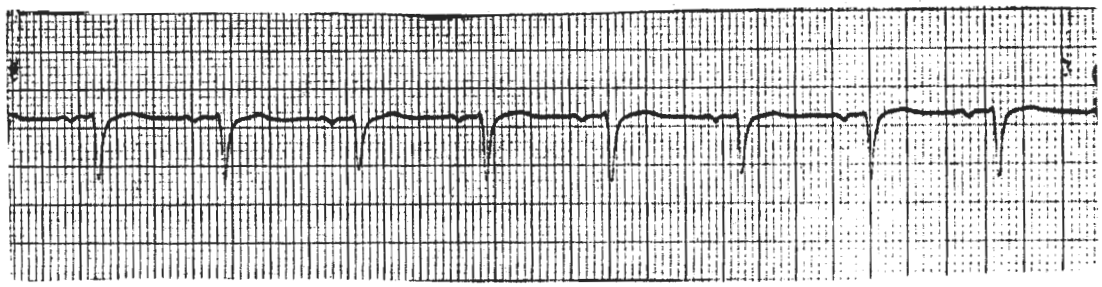


10 minutes



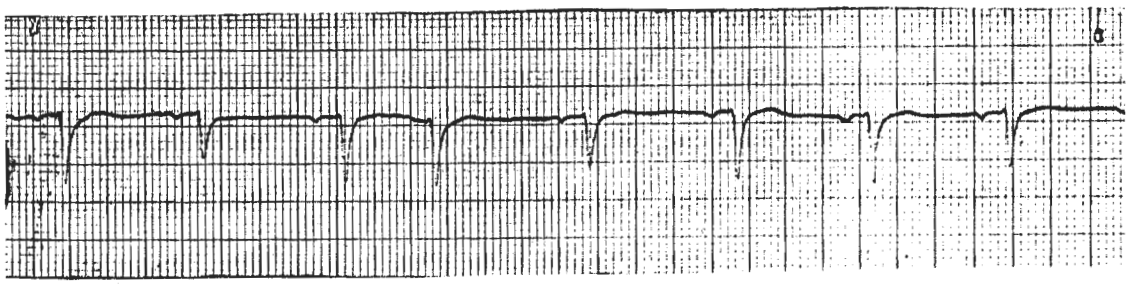
15 minutes

Patient 5

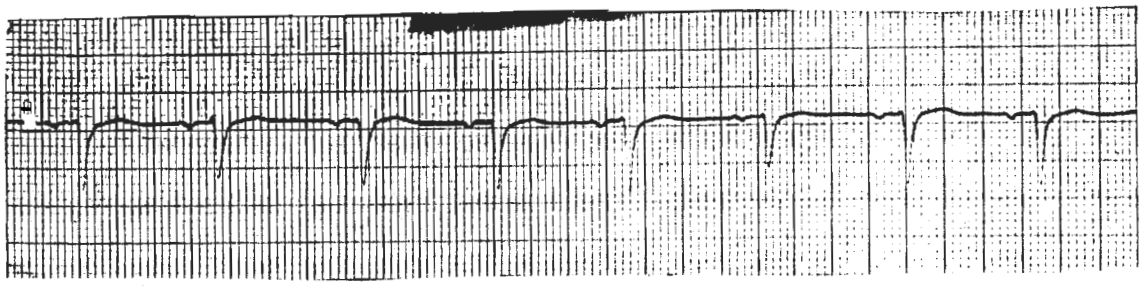


20 minutes

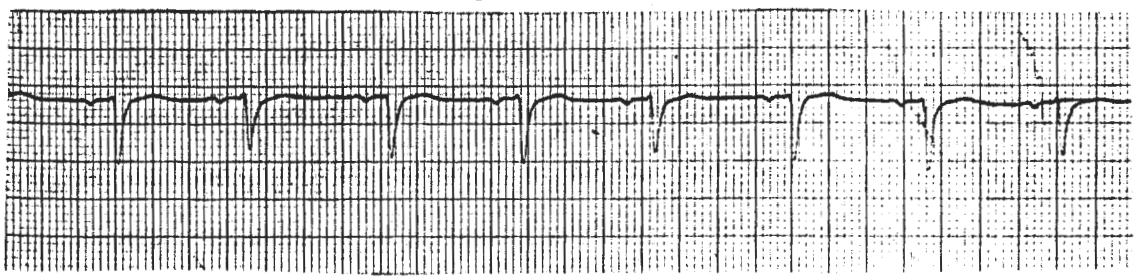
Bath Completed



1 minute

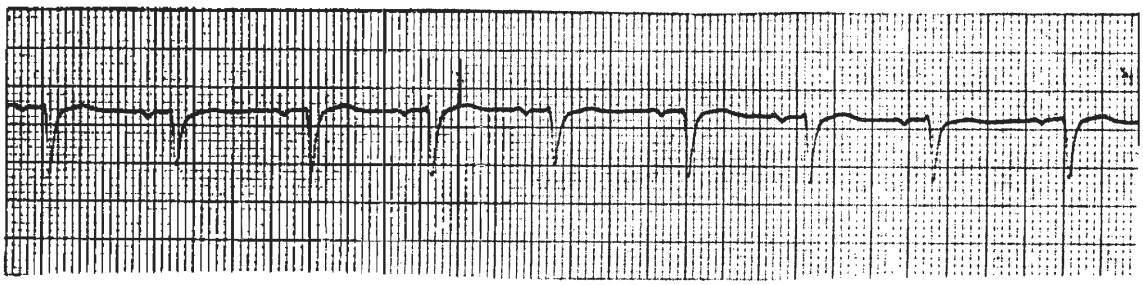


2 minutes

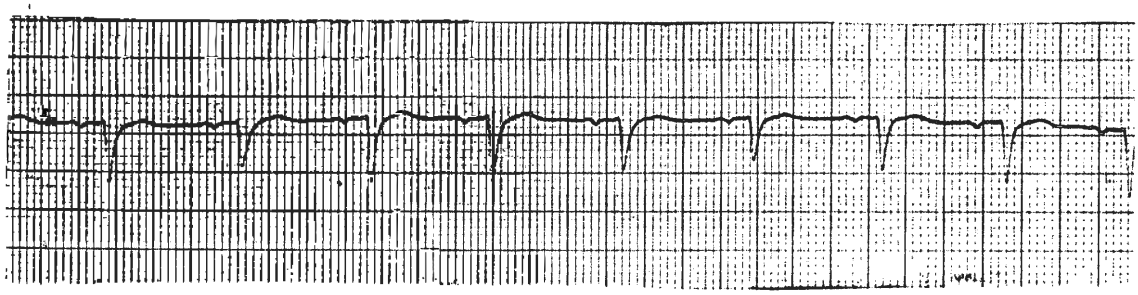


3 minutes

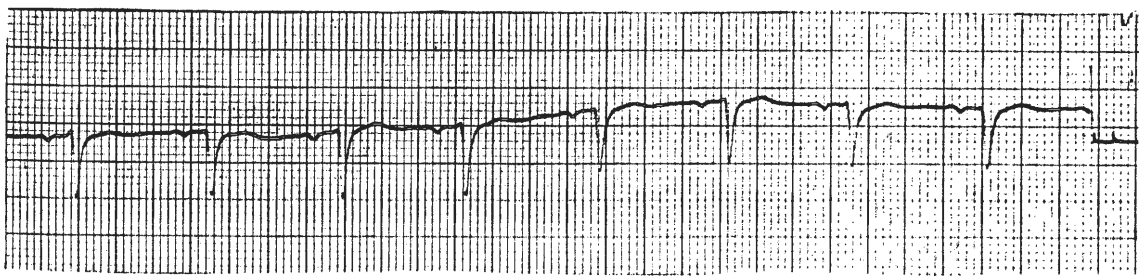
Patient 5



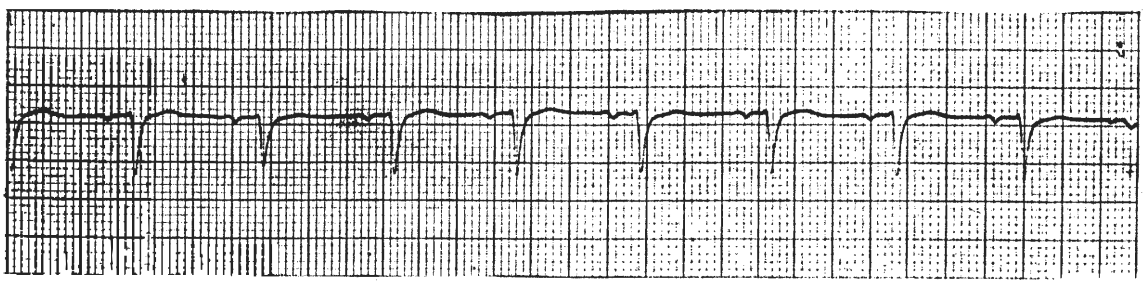
4 minutes



5 minutes

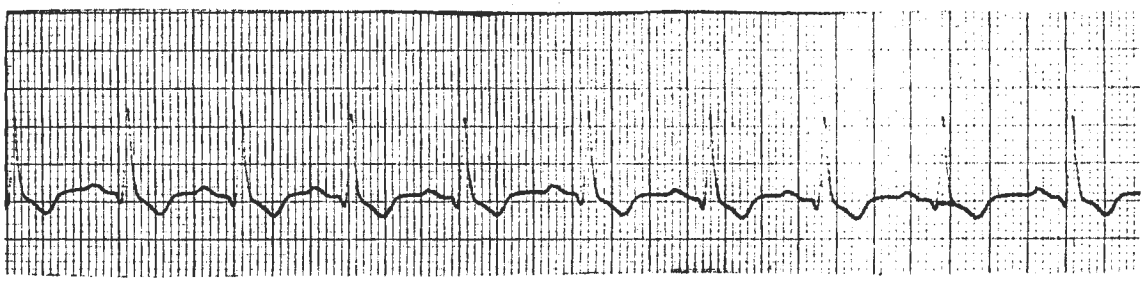


10 minutes

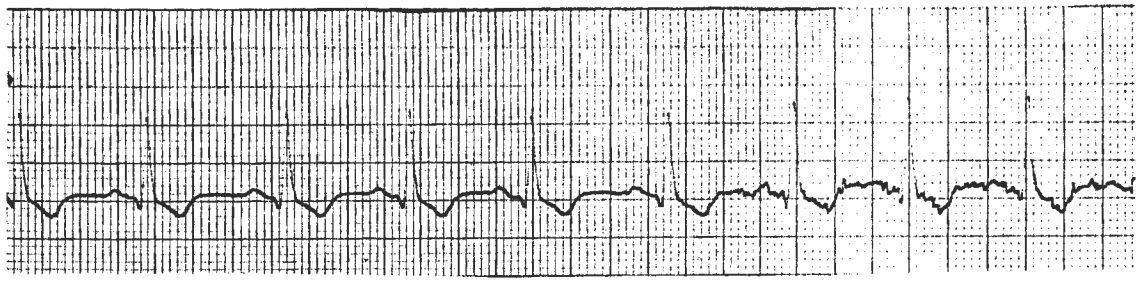


15 minutes

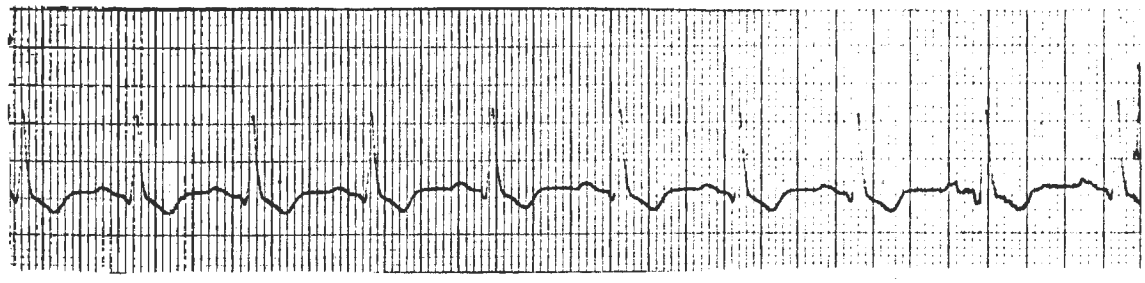
Patient 6 Monitor lead II Medication Lanoxin



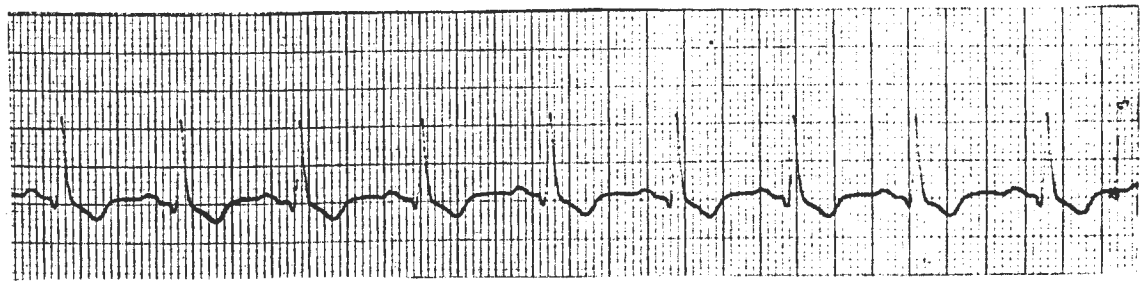
Baseline



1 minute

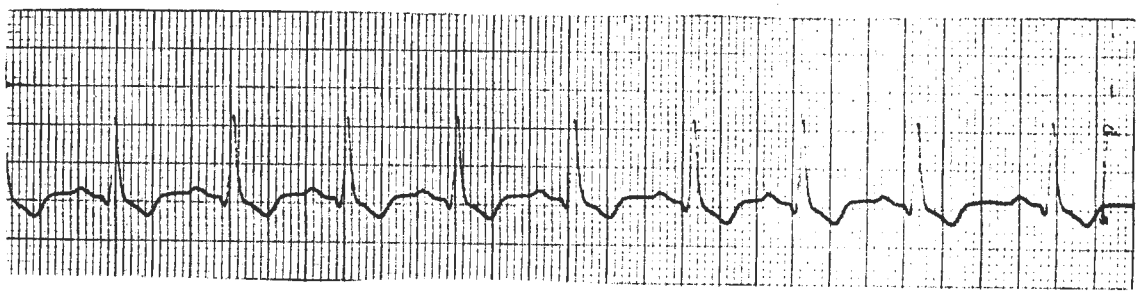


2 minutes

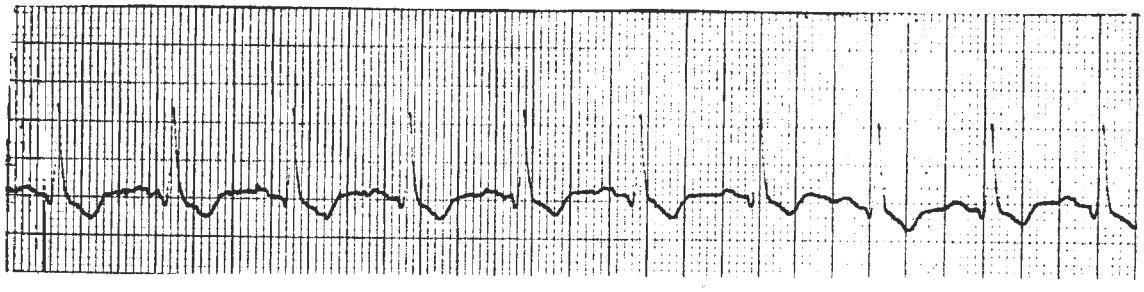


3 minutes

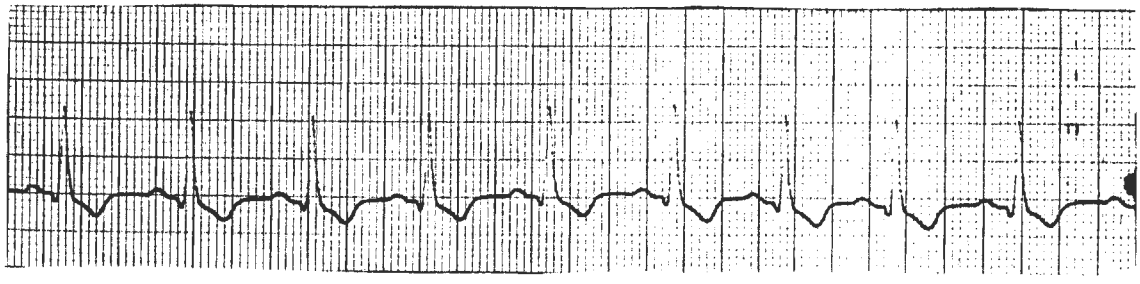
Patient 6



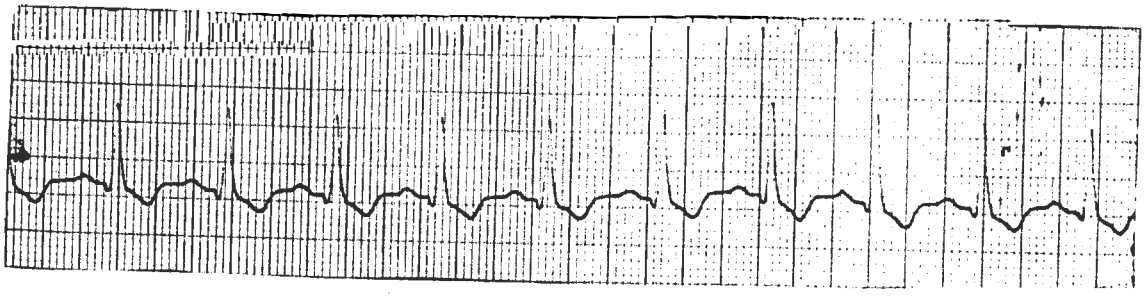
4 minutes



5 minutes

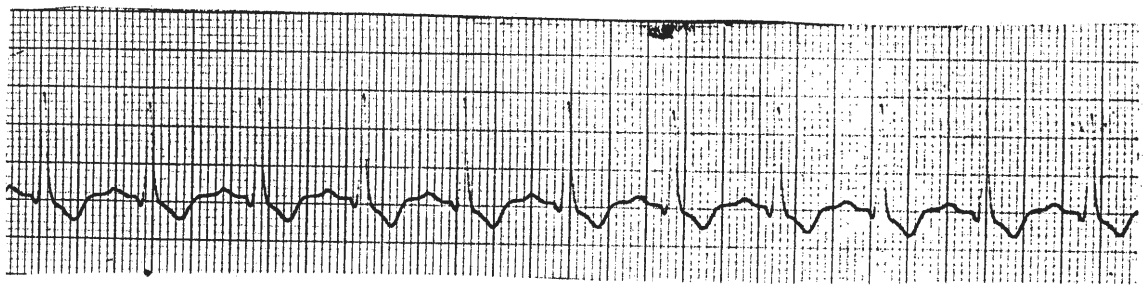


10 minutes



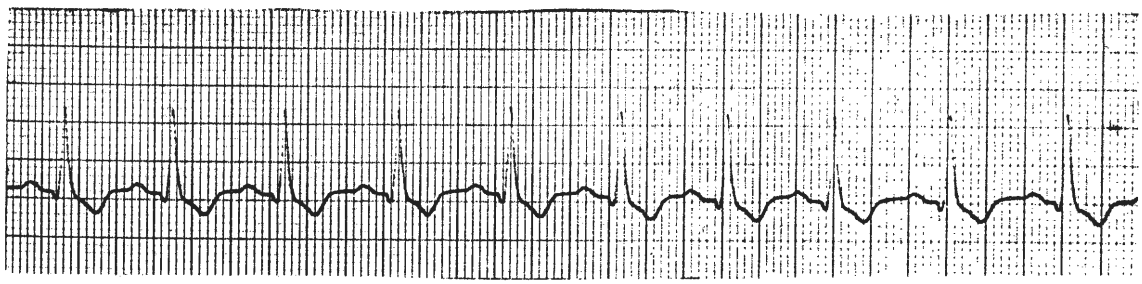
15 minutes

Patient 6

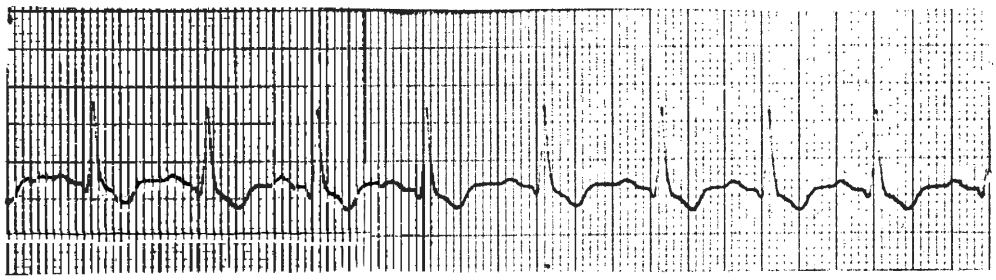


20 minutes

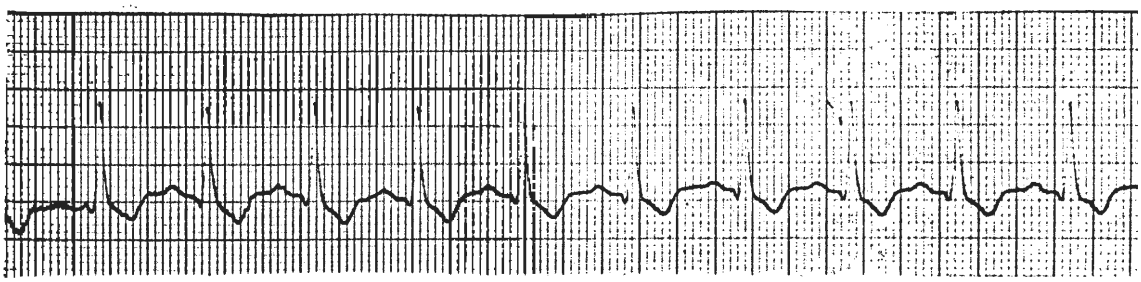
Bath Completed



1 minute

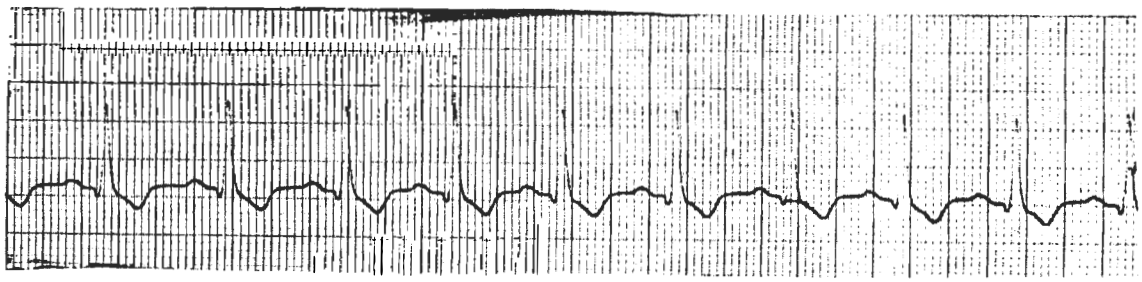


2 minutes

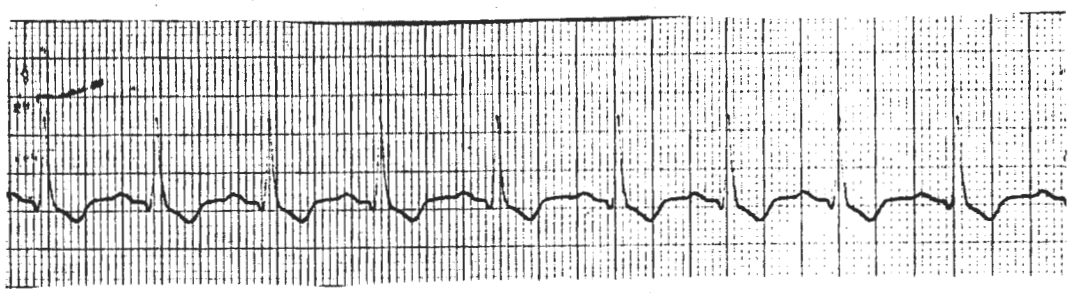


3 minutes

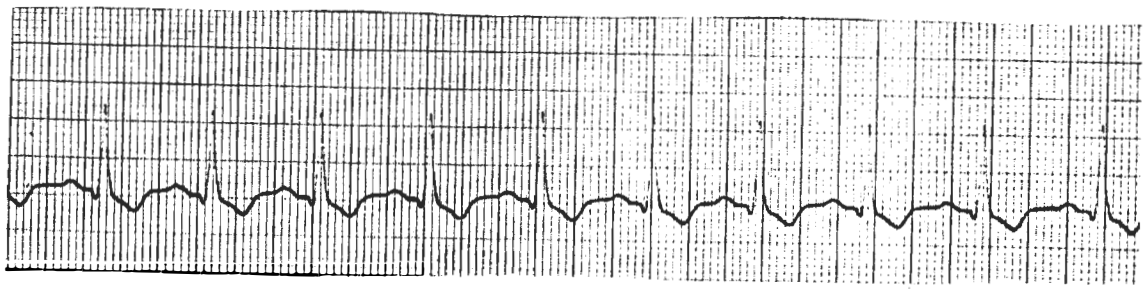
Patient 6



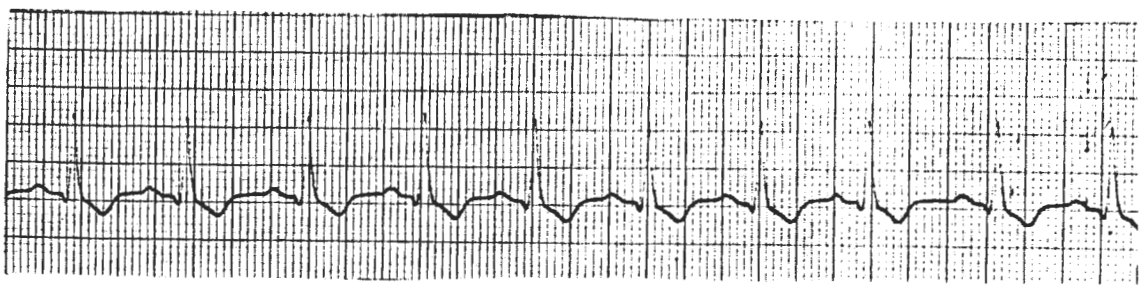
4 minutes



5 minutes

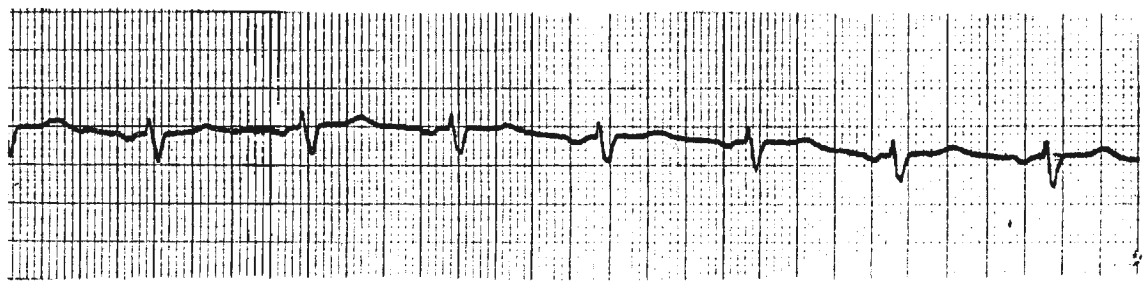


10 minutes

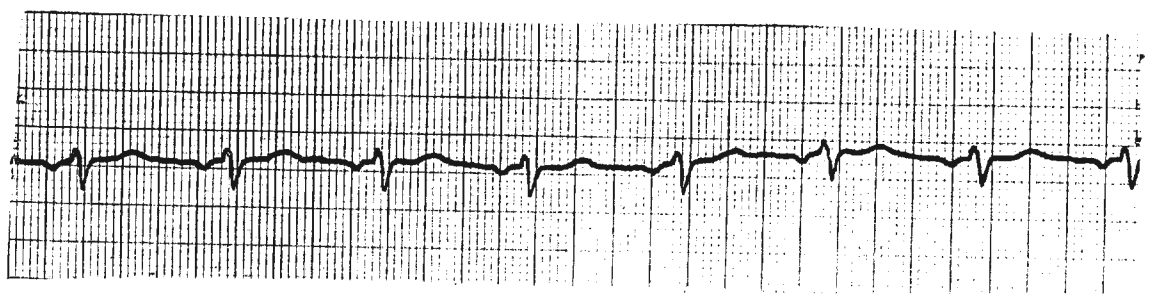


15 minutes

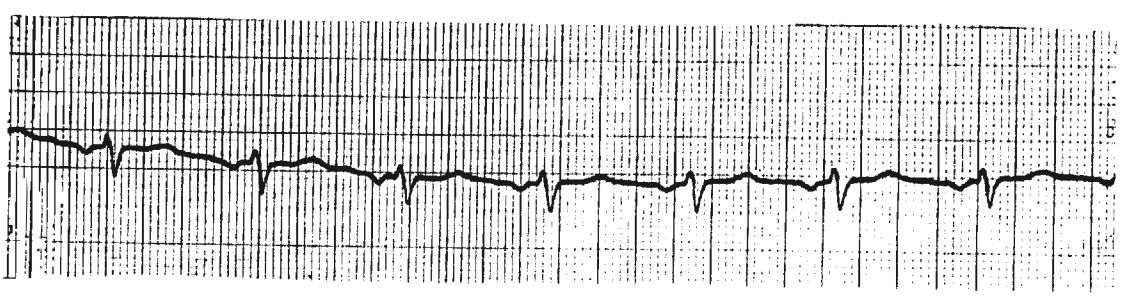
Patient 7 Monitor lead V_1 Medication _____



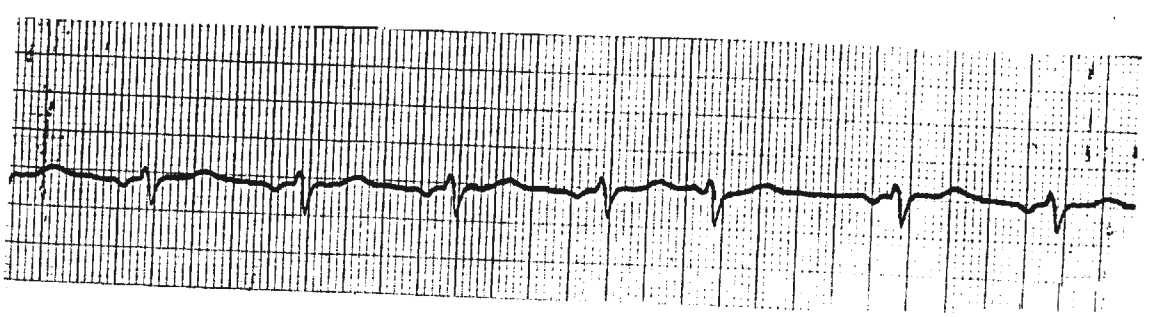
Baseline



1 minute

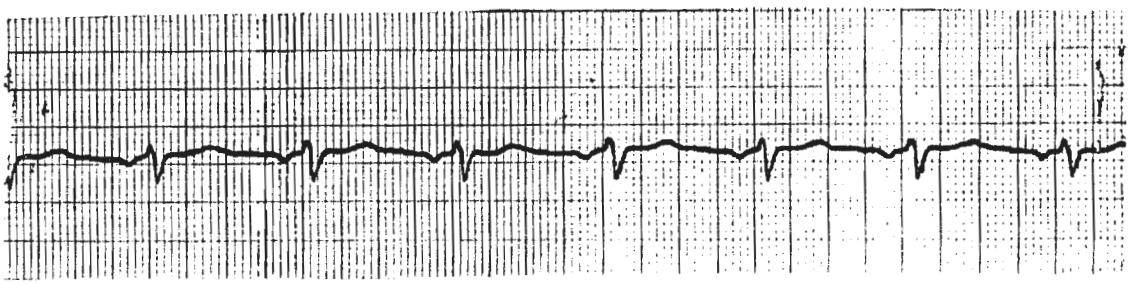


2 minutes

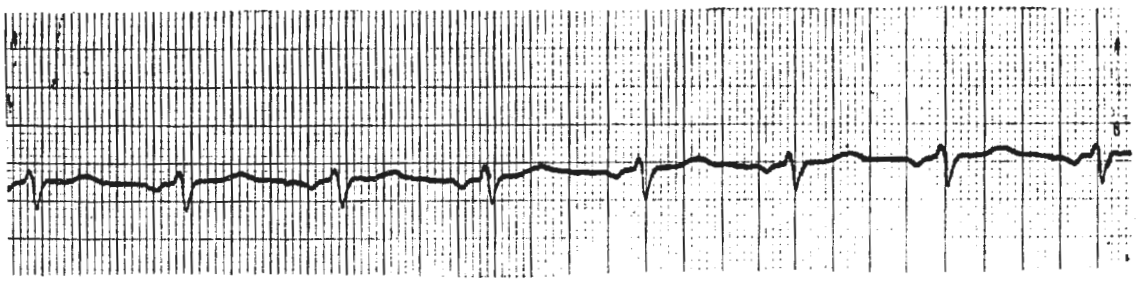


3 minutes

Patient 7



4 minutes



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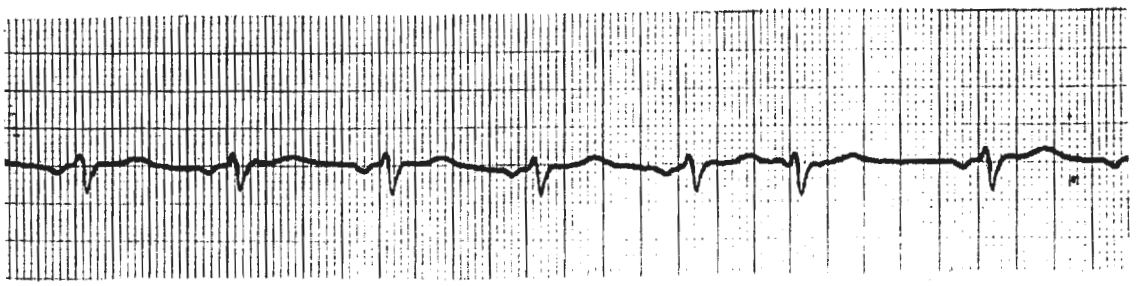
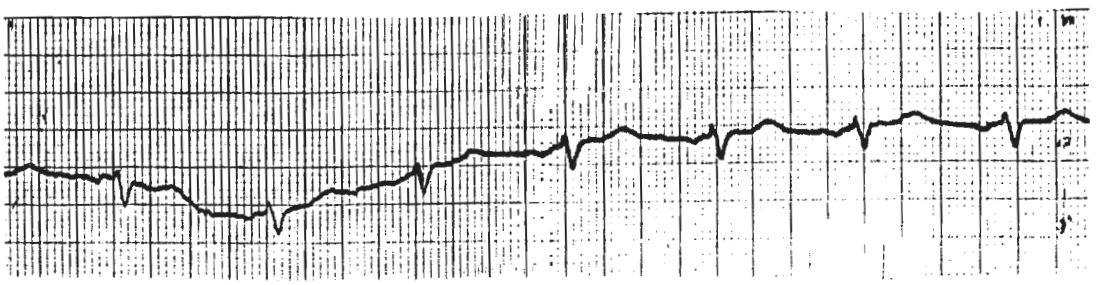
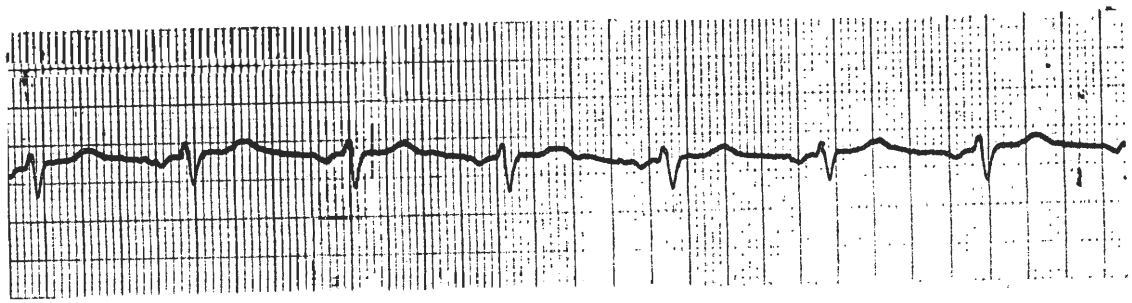


10 minutes



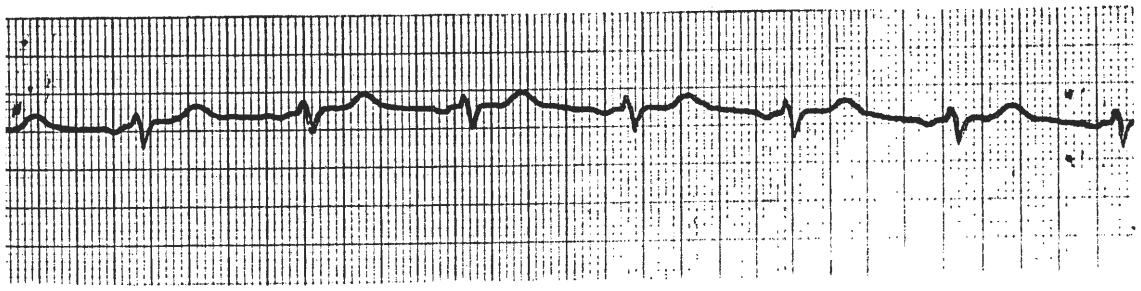
15 minutes

Patient 7



20 minutes

Bath Completed



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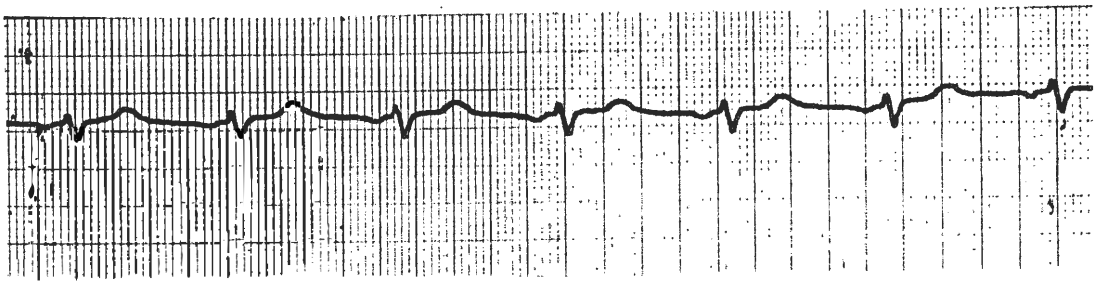

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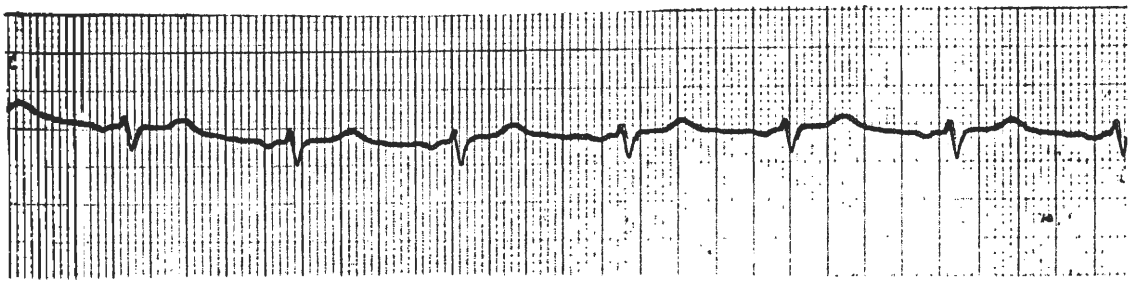
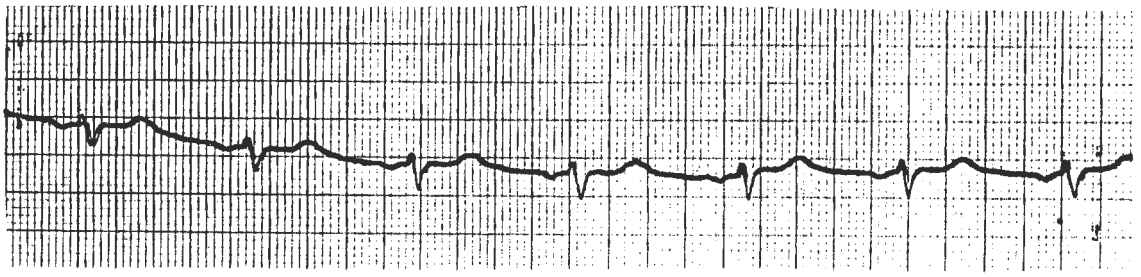
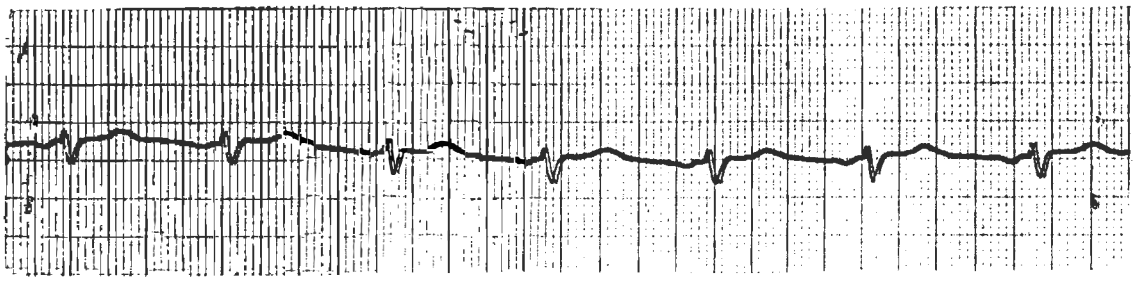


3 minutes

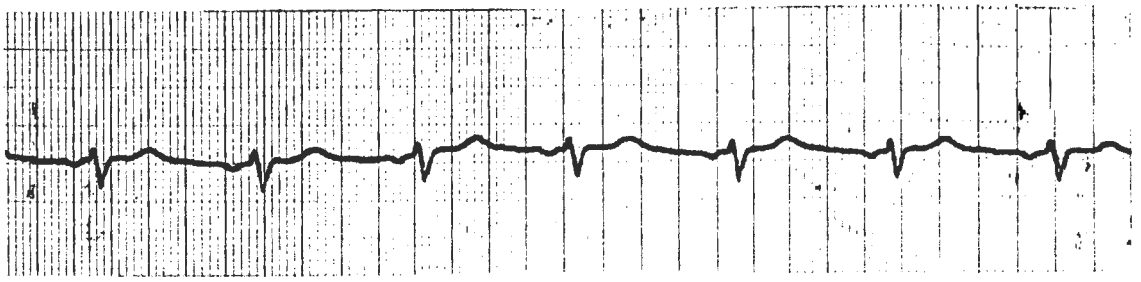
Patient 7



4 minutes



5 minutes



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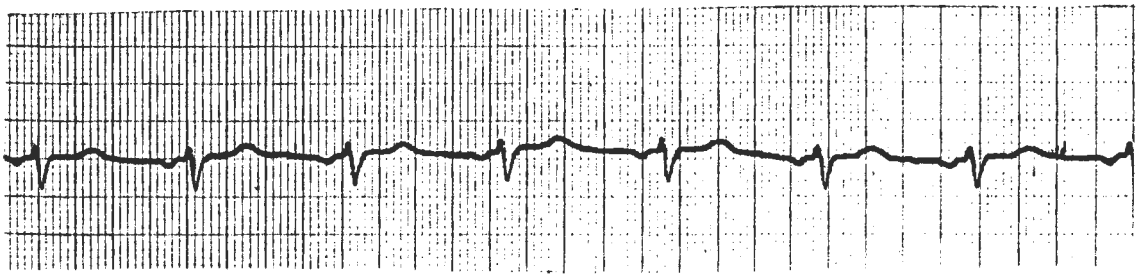
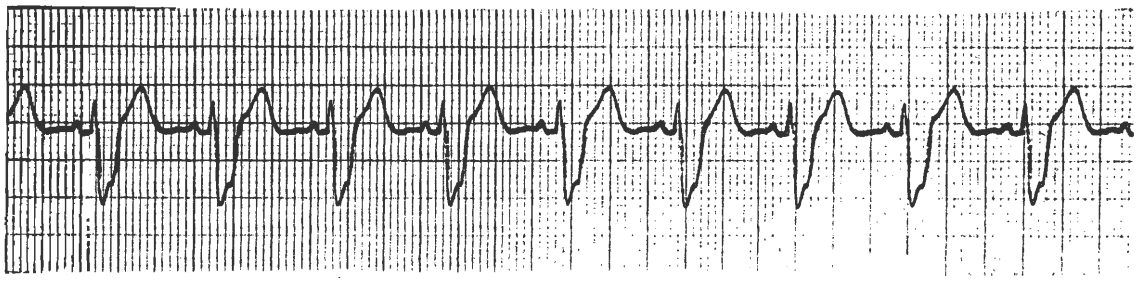
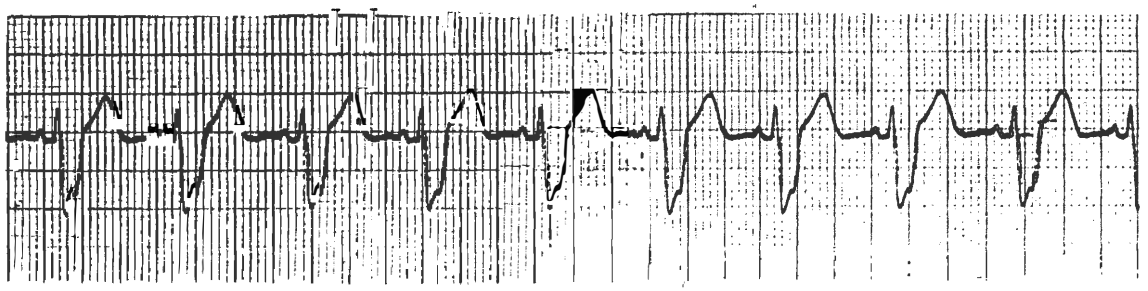


15 minutes

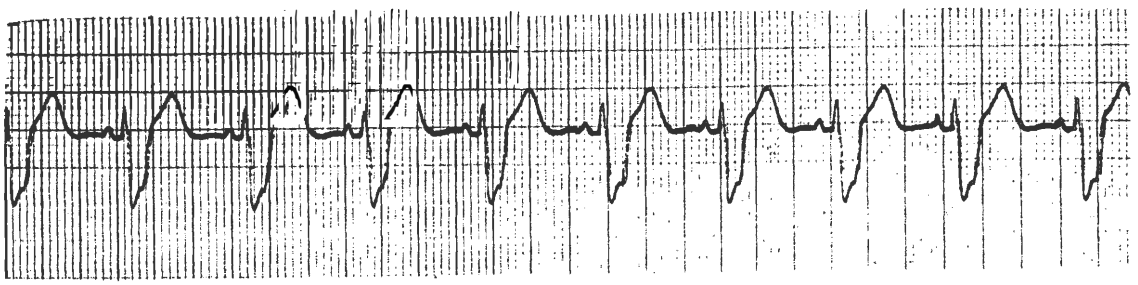
Patient 8 Monitor lead V_1 Medication _____



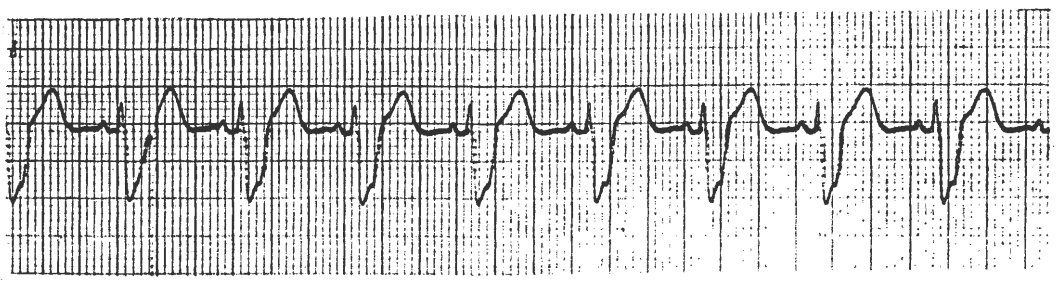
Baseline



1 minute



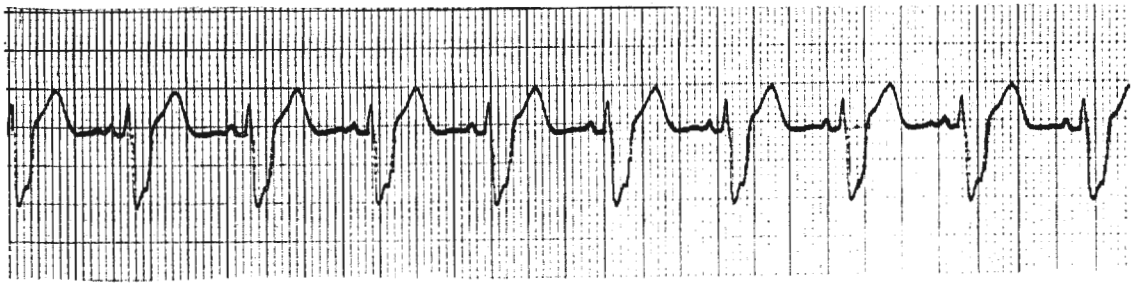
2 minutes



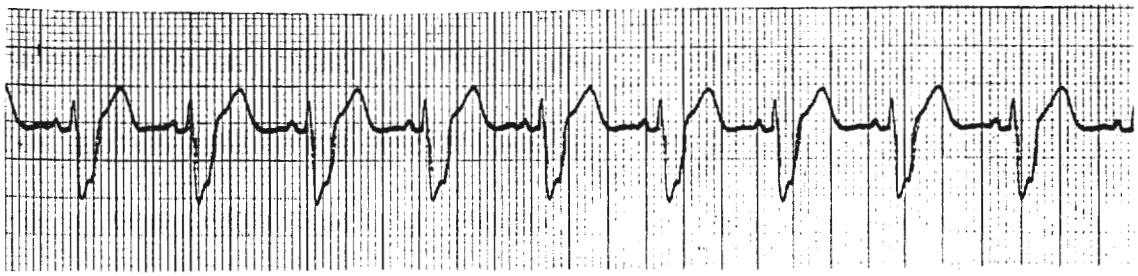
3 minutes

Patient 8

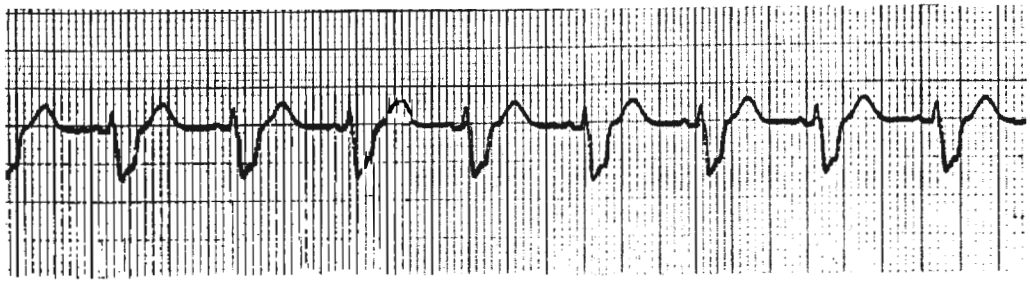
4 minutes



5 minutes

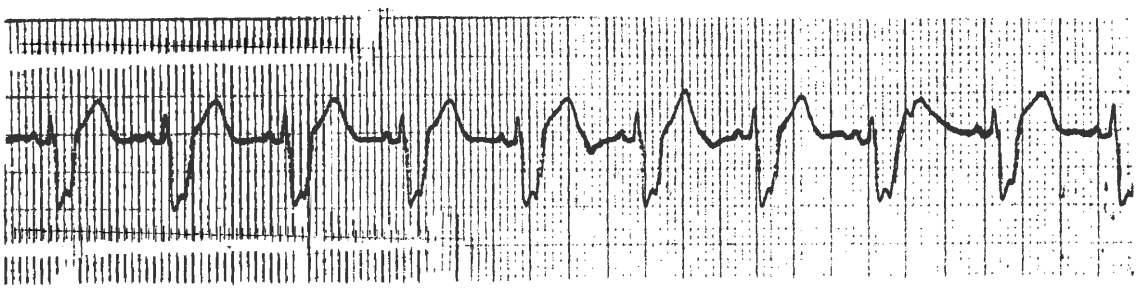


10 minutes



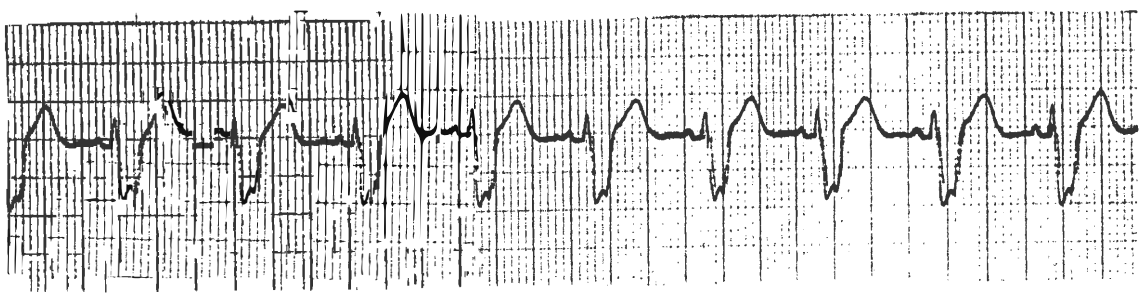
15 minutes

Patient 8

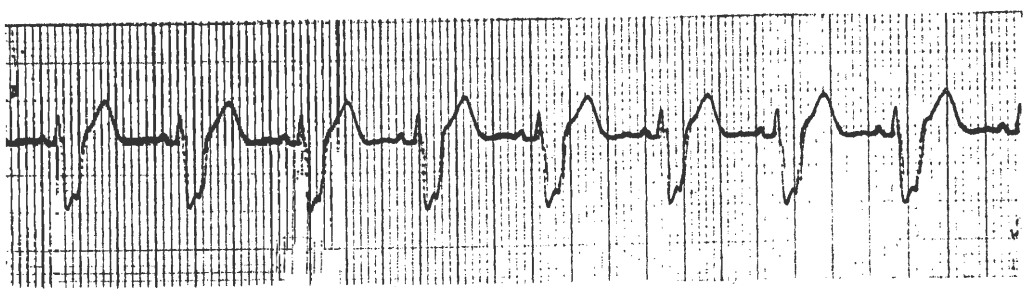


20 minutes

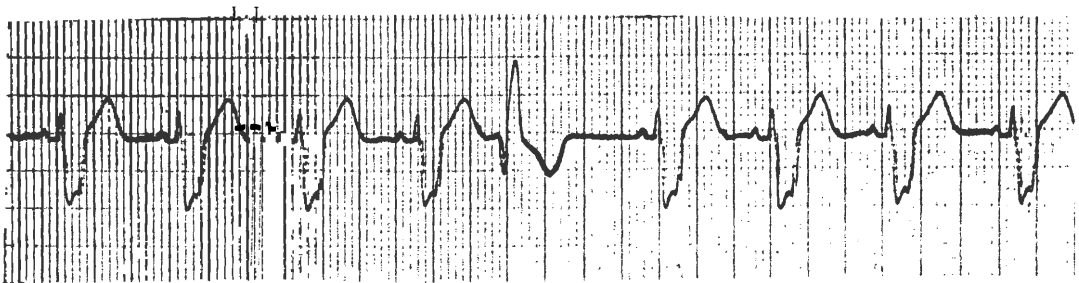
Bath Completed



1 minute

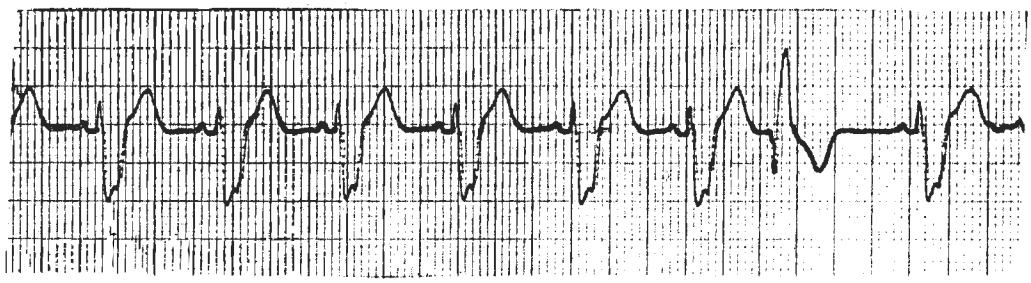


2 minutes

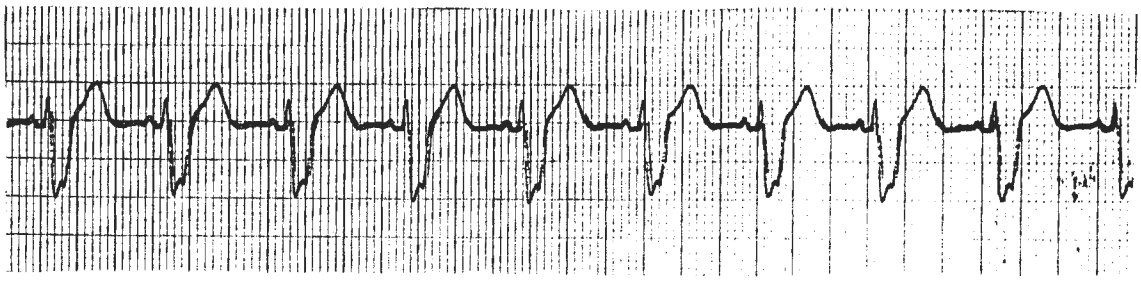


3 minutes

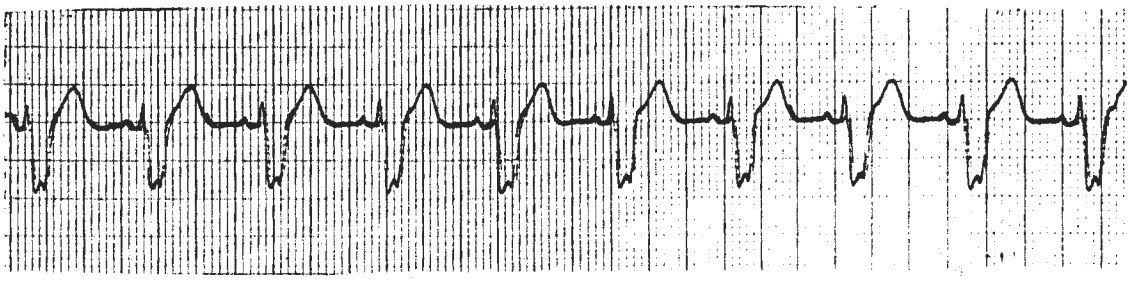
Patient 8



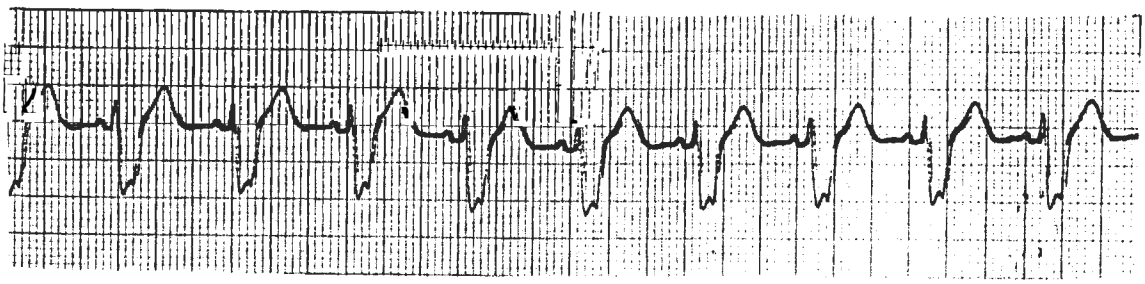
4 minutes



5 minutes

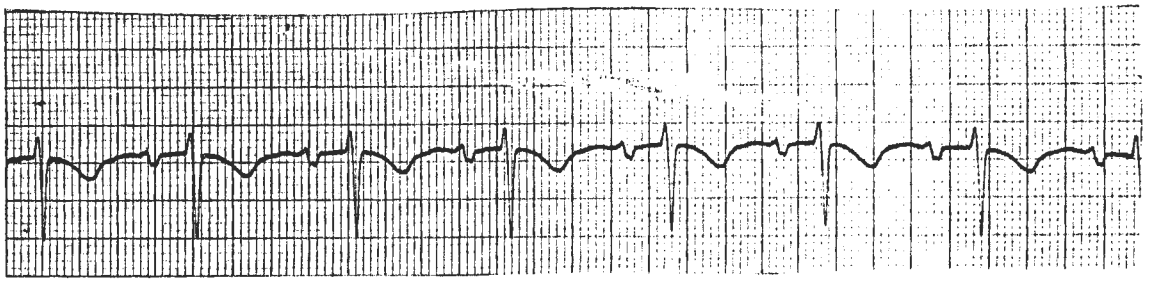


10 minutes

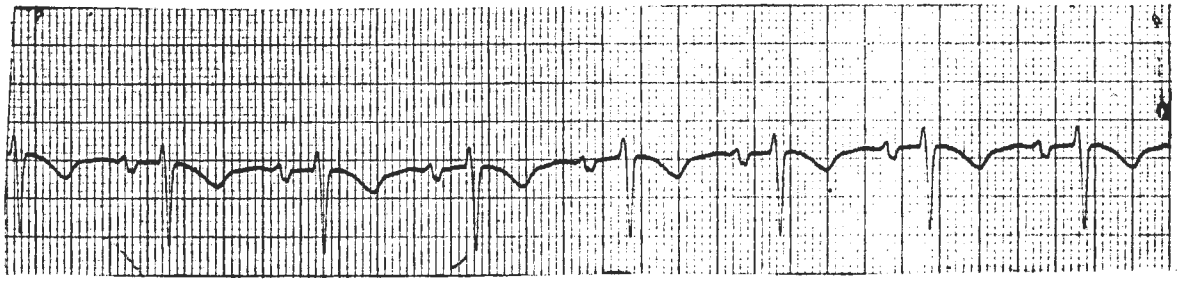


15 minutes

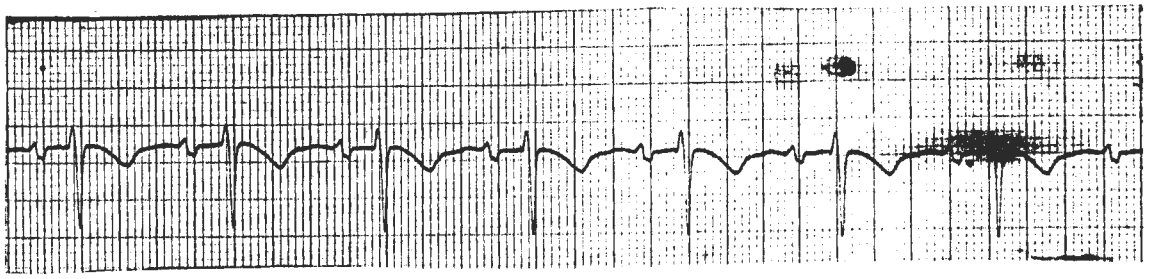
Patient 9 Monitor lead V_1 Medication _____



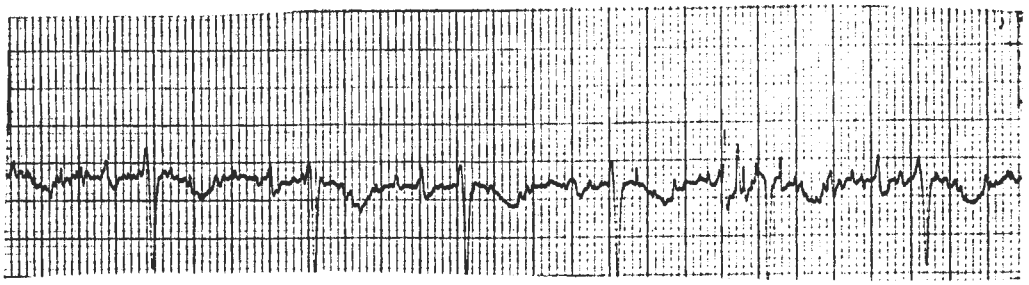
Baseline



1 minute

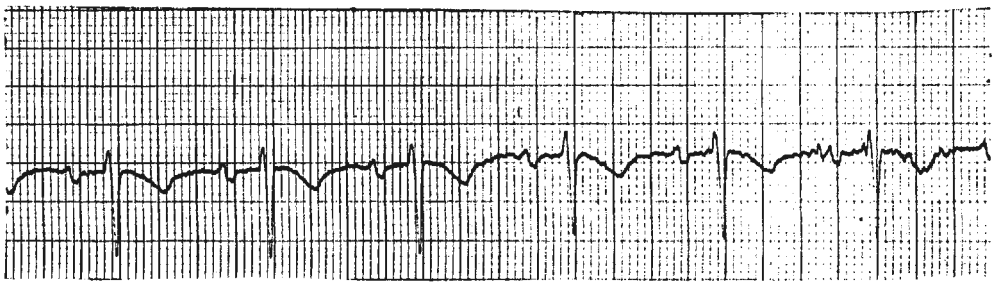


2 minutes

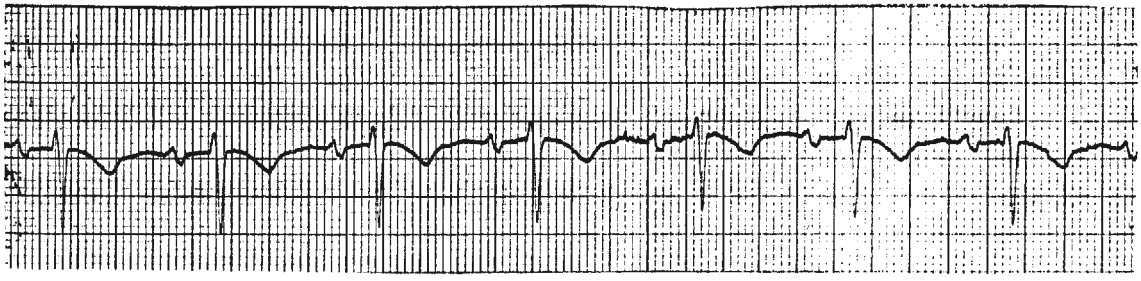


3 minutes

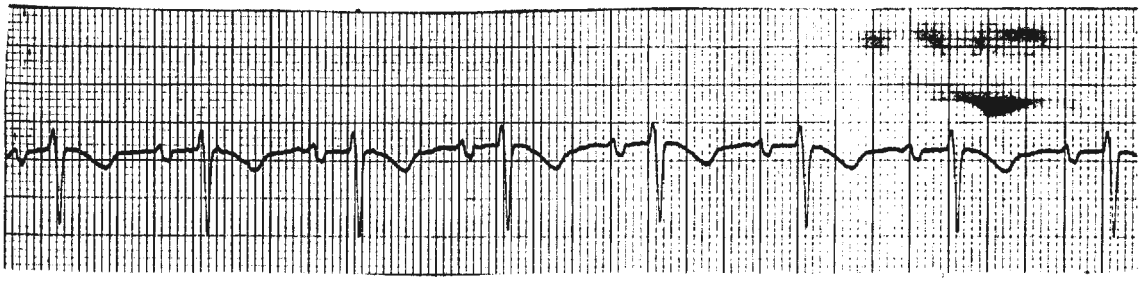
Patient 9



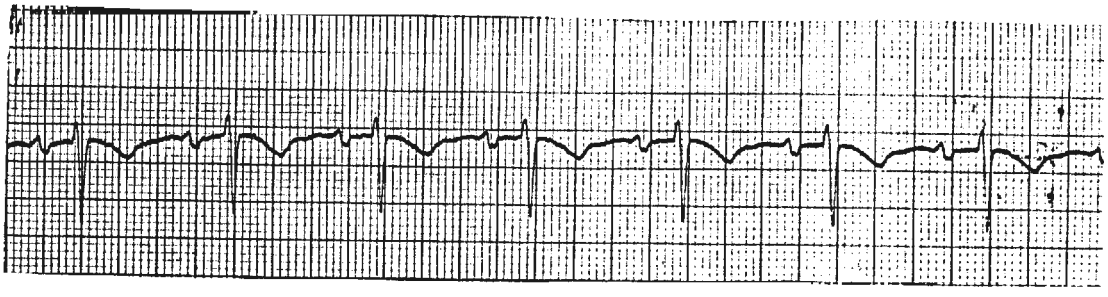
4 minutes



5 minutes

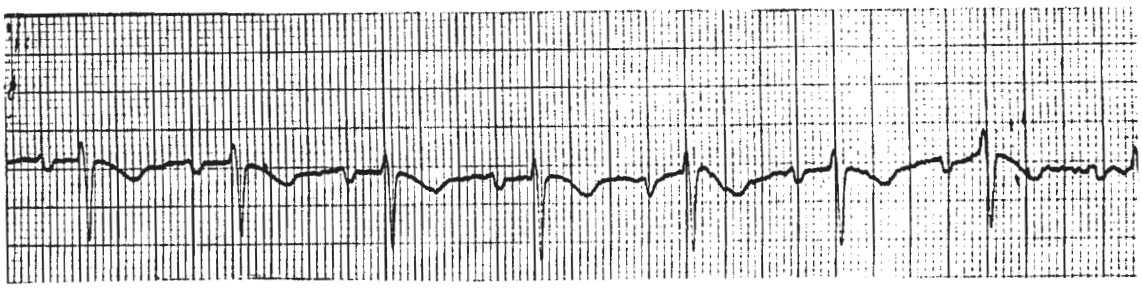


10 minutes



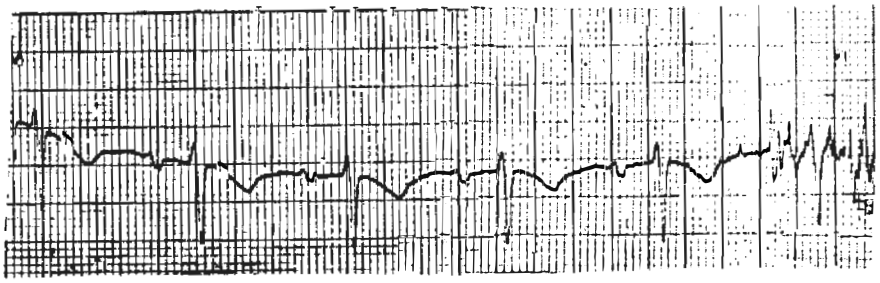
15 minutes

Patient 9

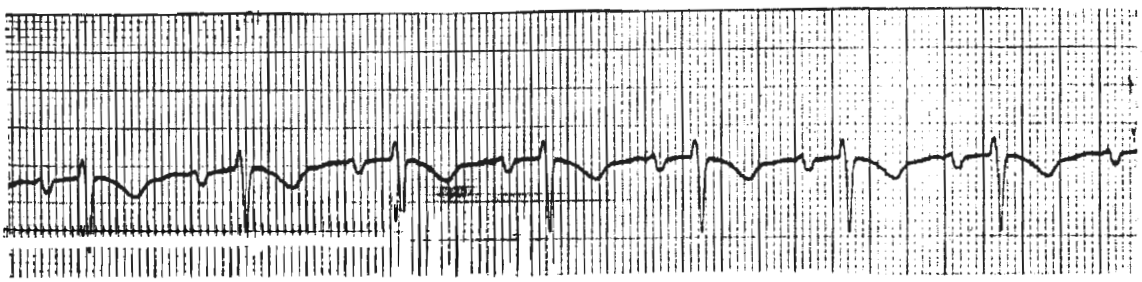


20 minutes

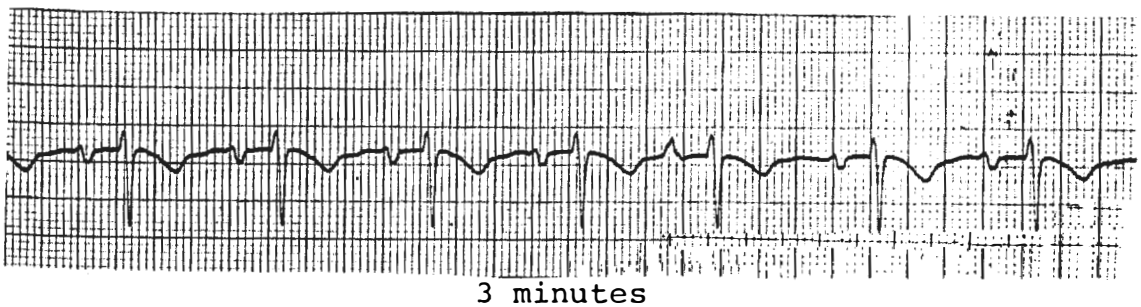
Bath Completed



1 minute

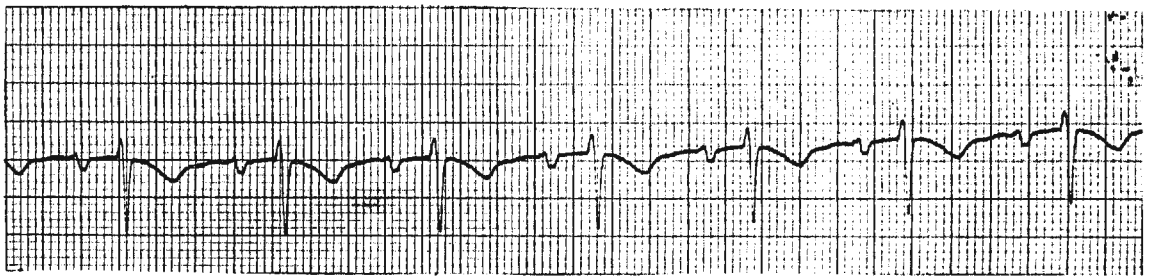


2 minutes

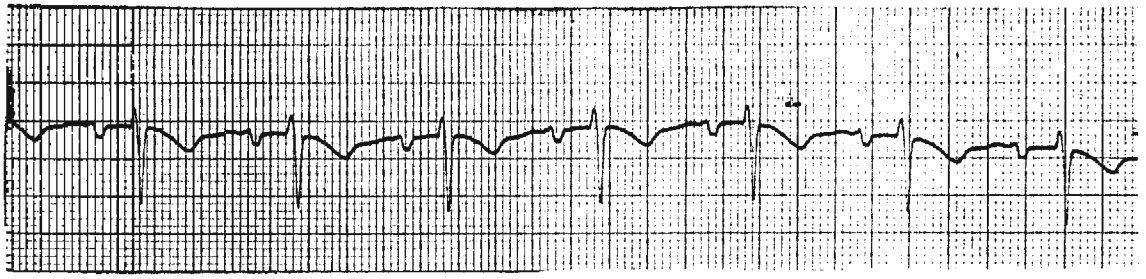


3 minutes

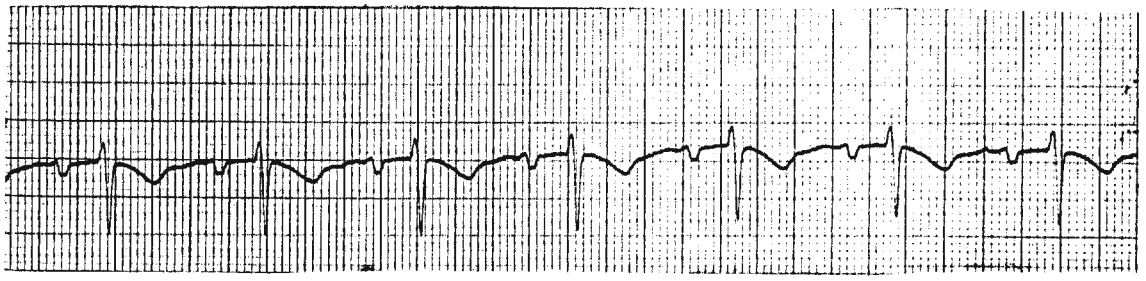
Patient 9



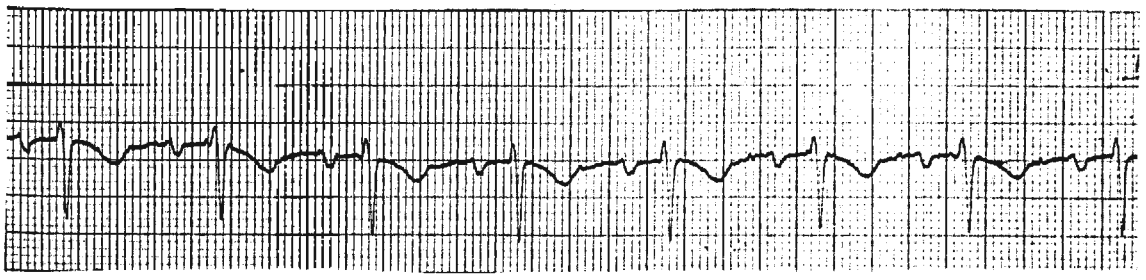
4 minutes



5 minutes

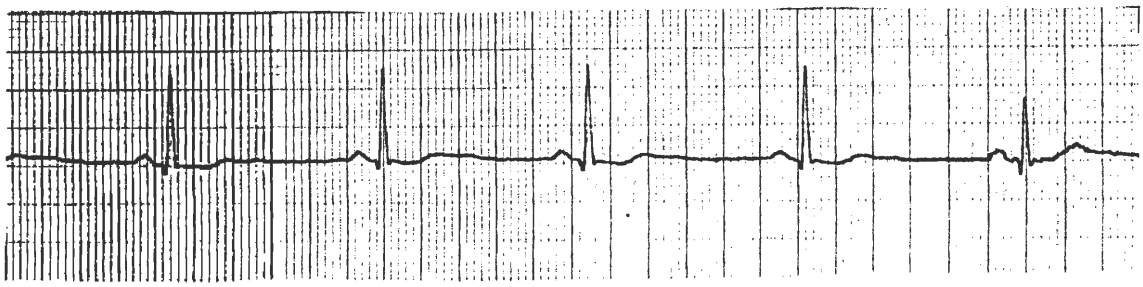


10 minutes

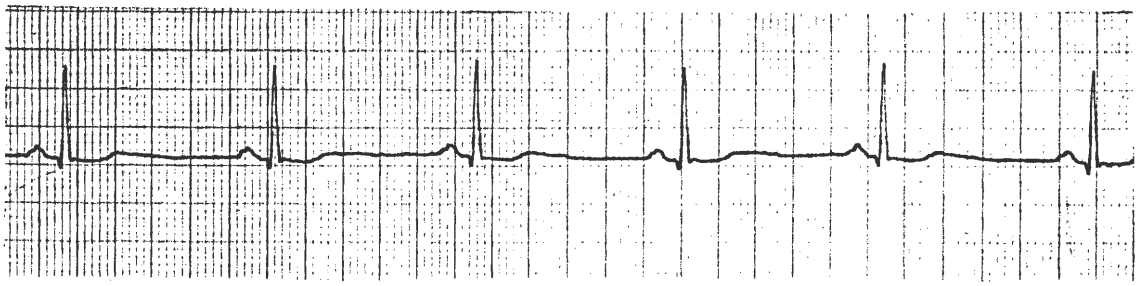


15 minutes

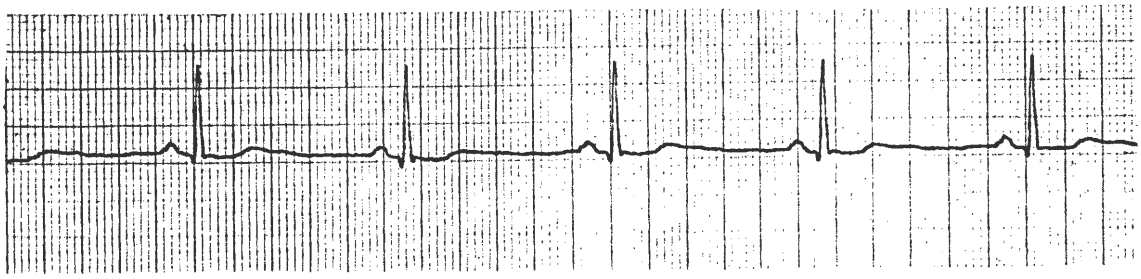
Patient 10 Monitor lead II Medication Pronestyl



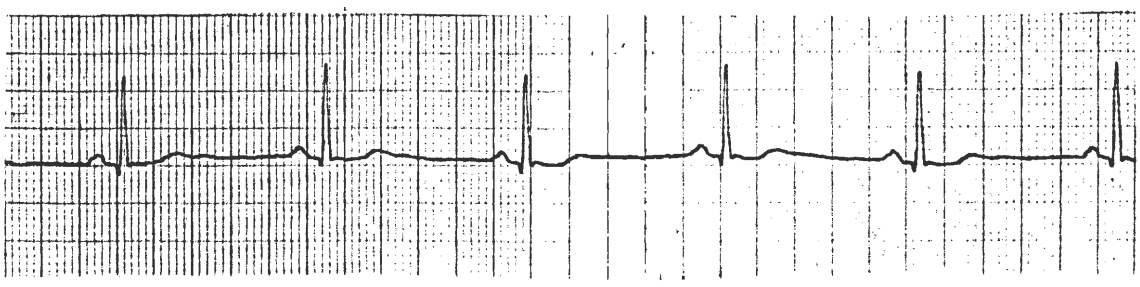
Baseline



1 minute

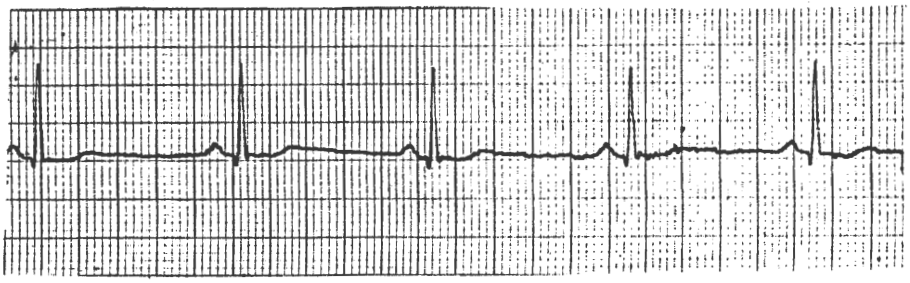


2 minutes

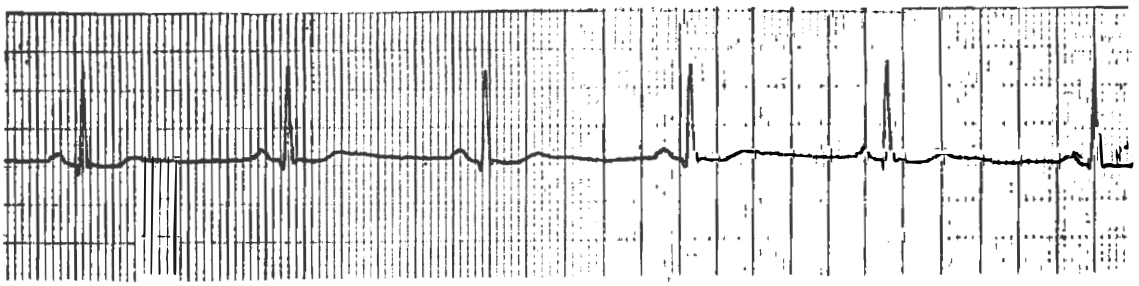


3 minutes

Patient 10



4 minutes



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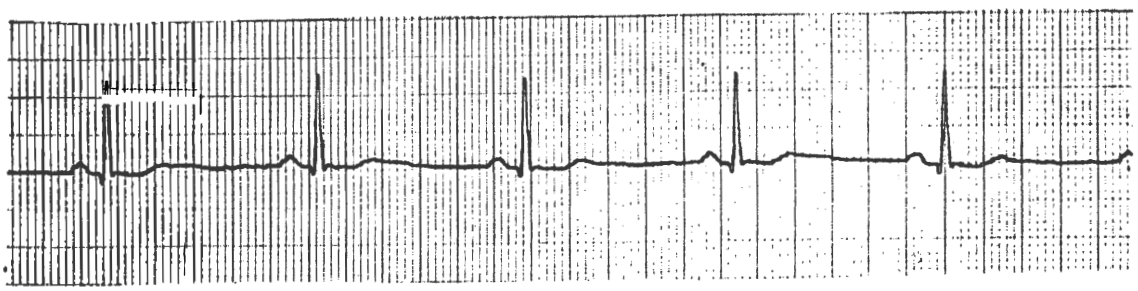

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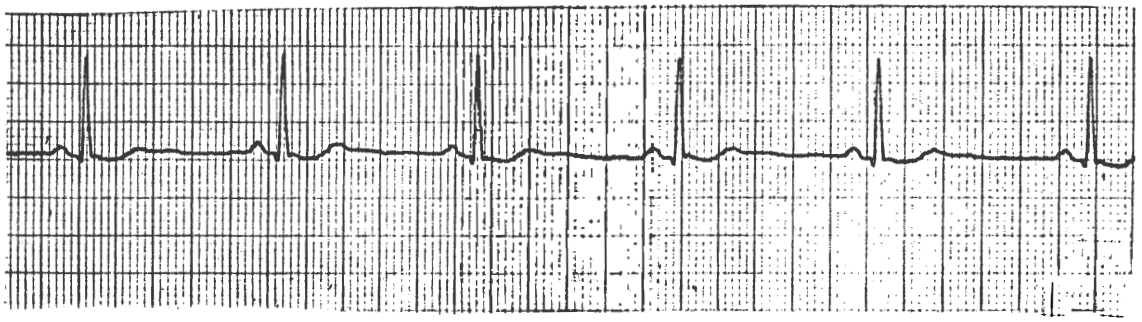
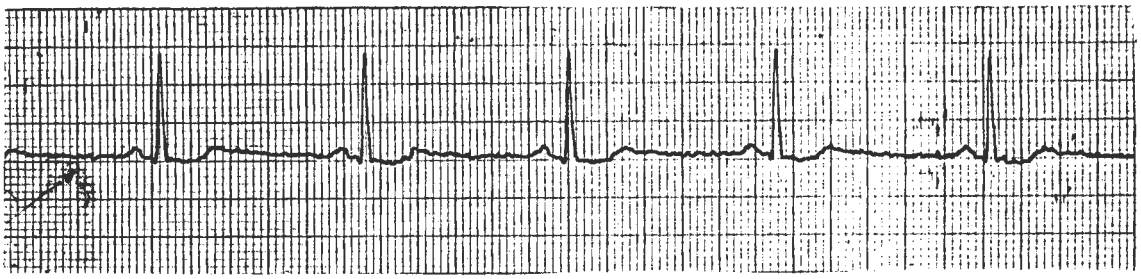


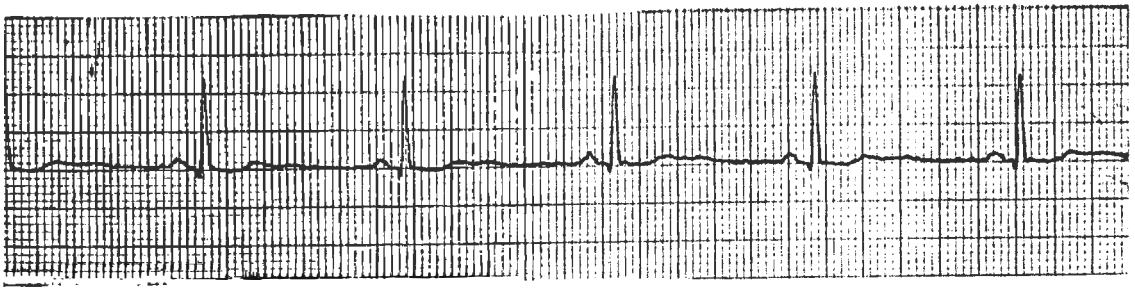
15 minutes

Patient 10

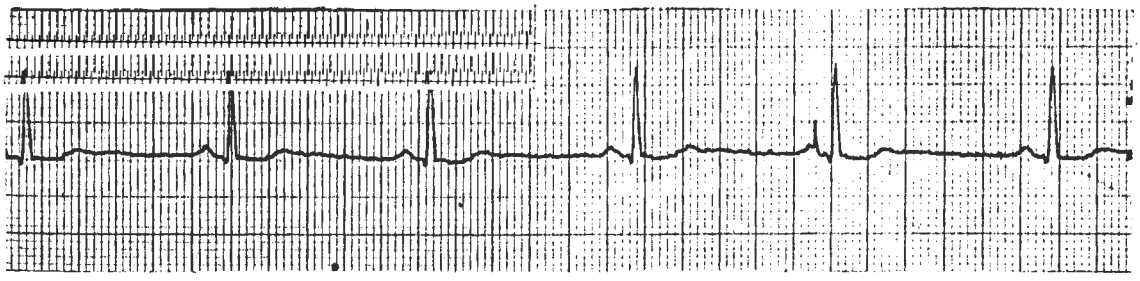


20 minutes

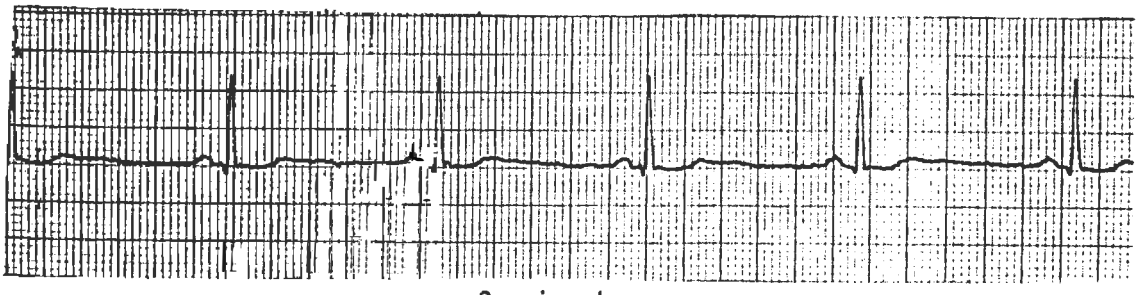
Bath Completed



1 minute

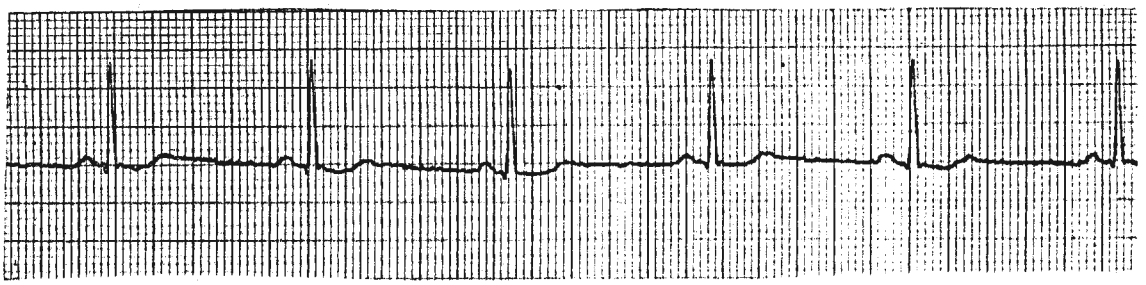


2 minutes

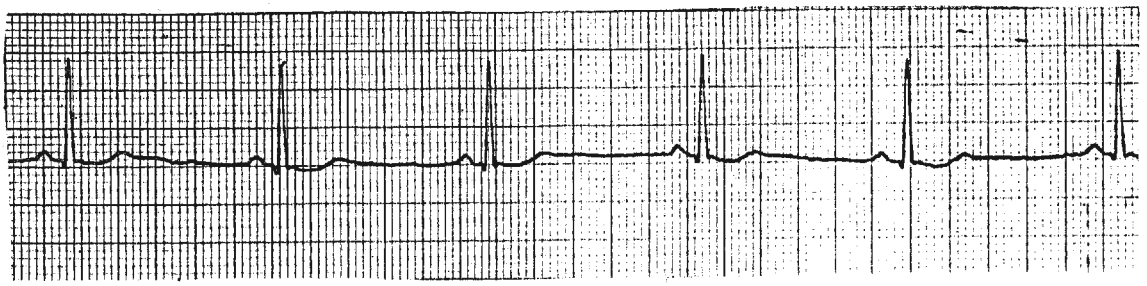


3 minutes

Patient 10



4 minutes



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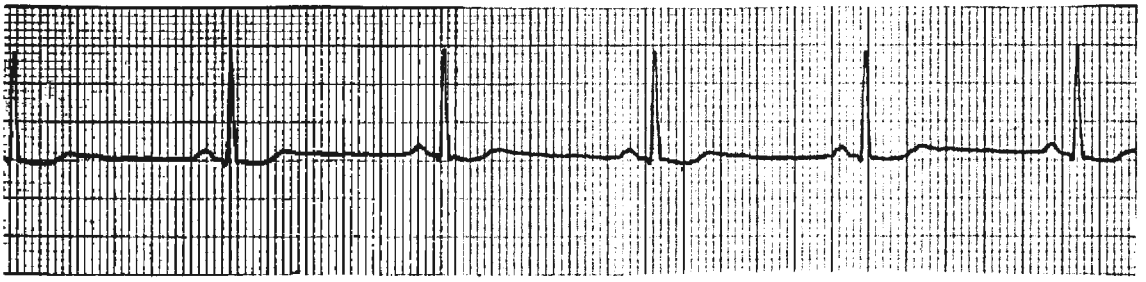

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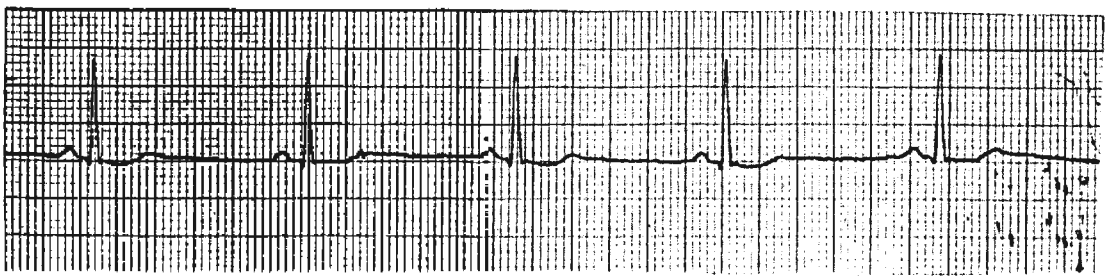
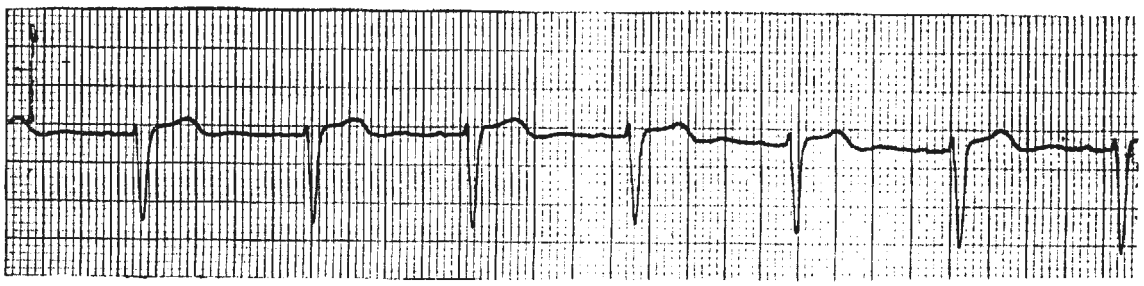
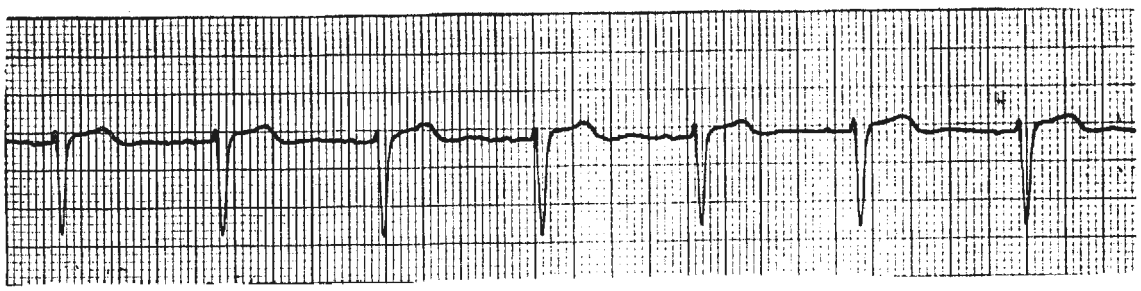


15 minutes

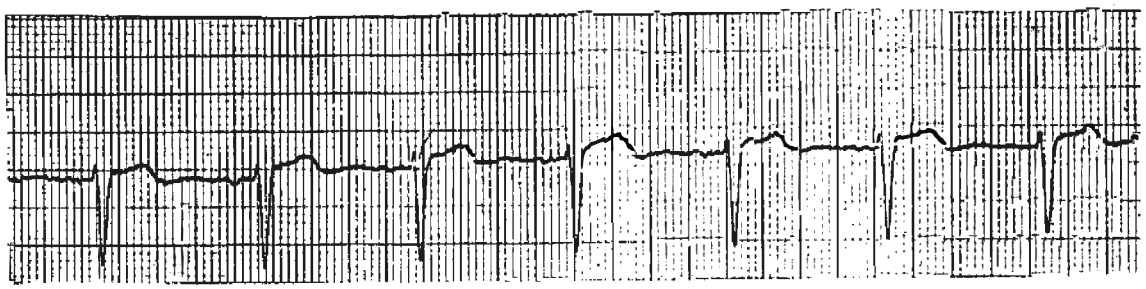
Patient 11 Monitor lead V_1 Medication _____



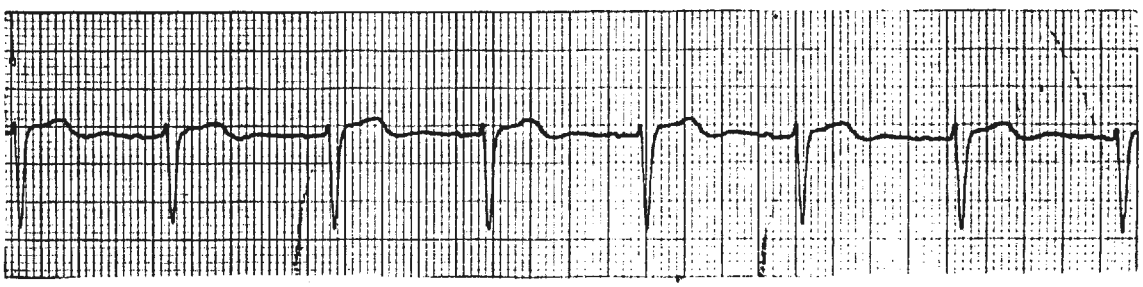
Baseline



1 minute

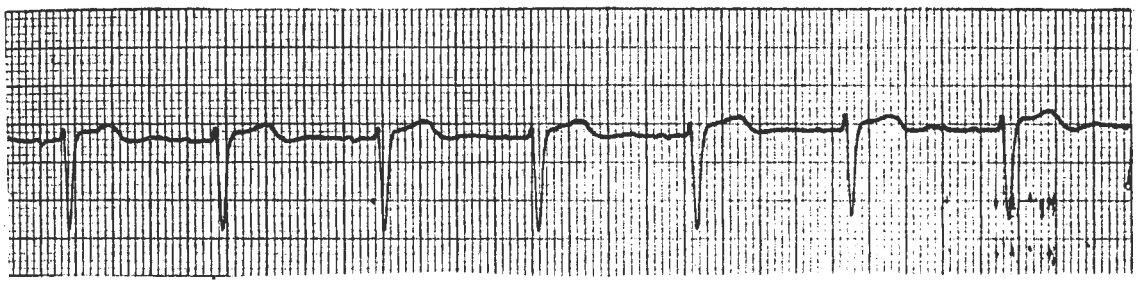


2 minutes

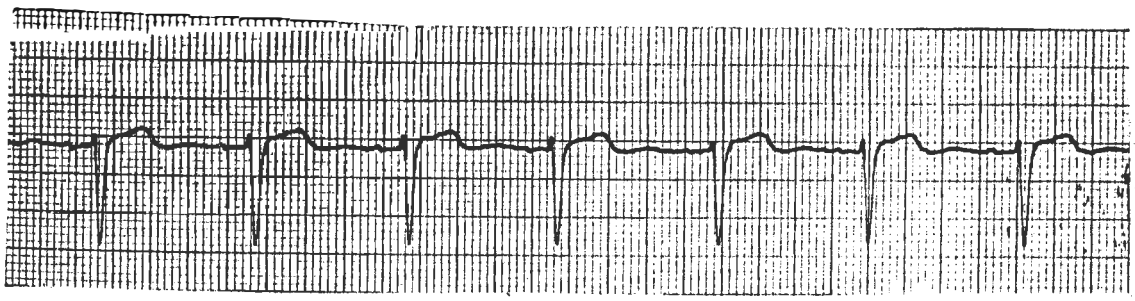


3 minutes

Patient 11



4 minutes



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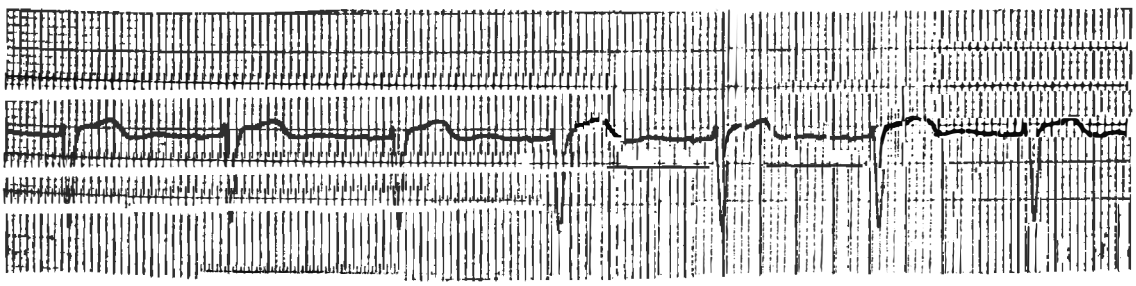
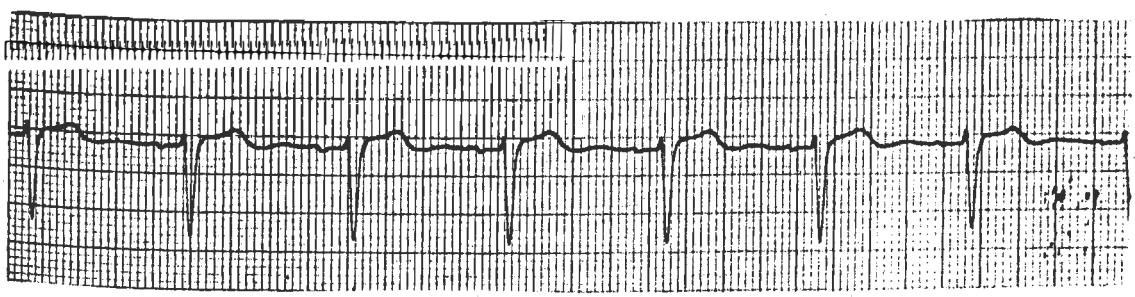
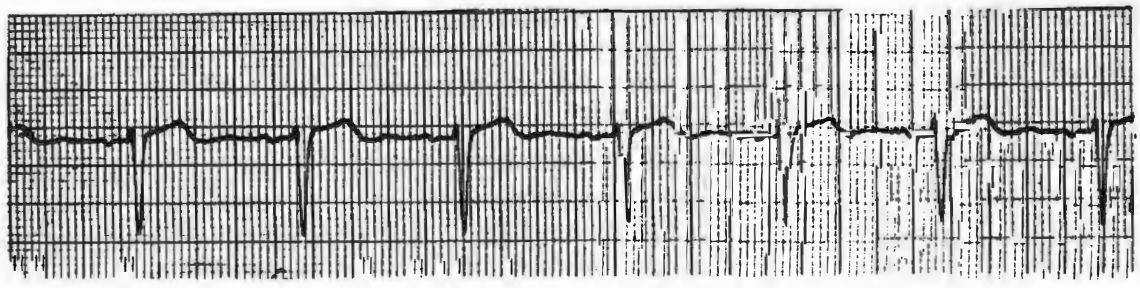


10 minutes



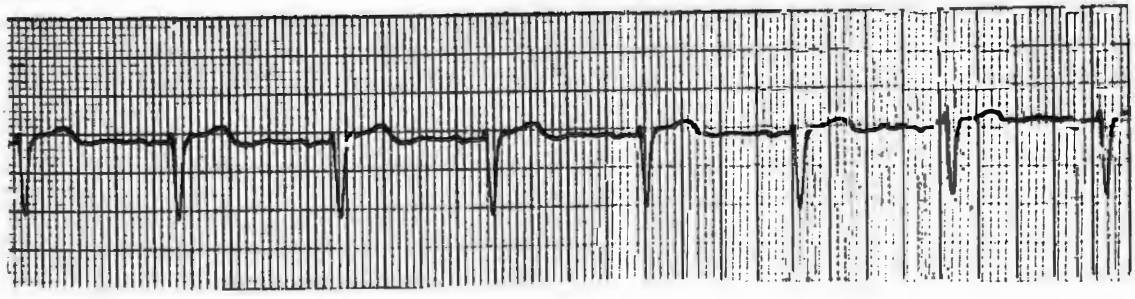
15 minutes

Patient 11

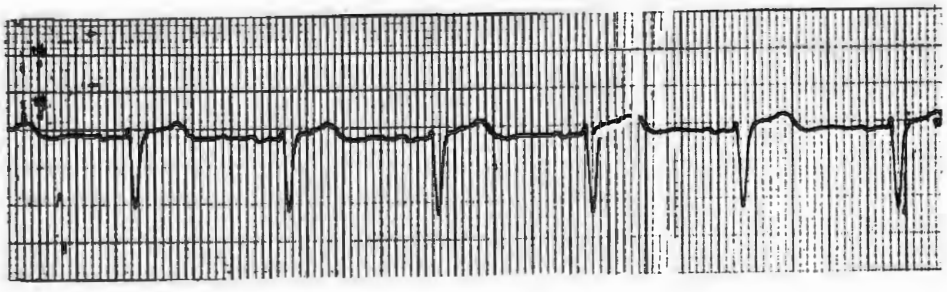


20 minutes

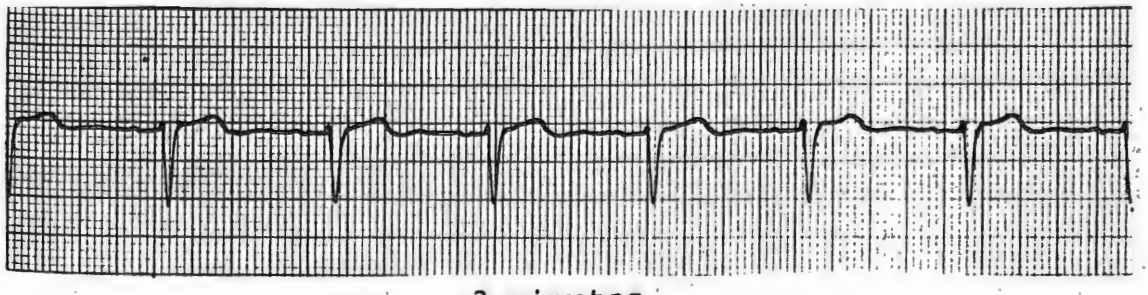
Bath Completed



1 minute

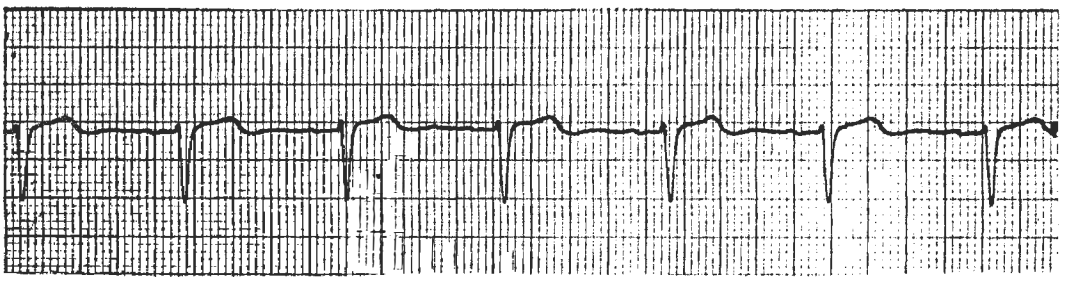


2 minutes

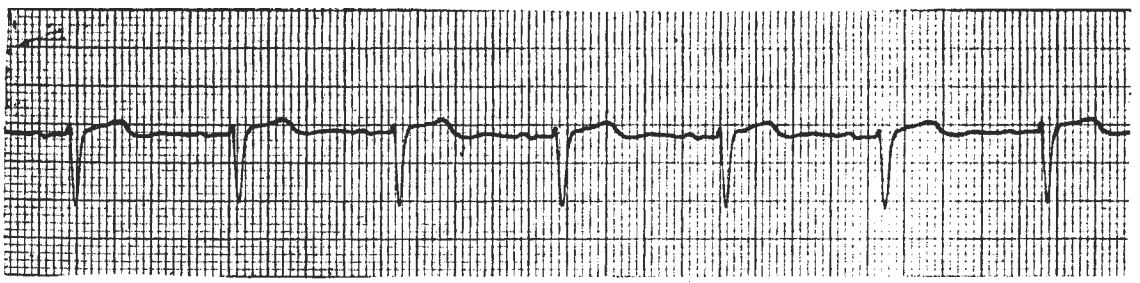


3 minutes

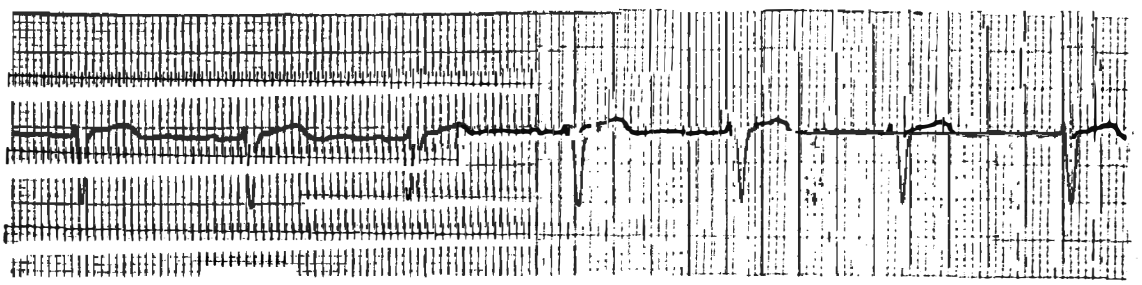
Patient 11



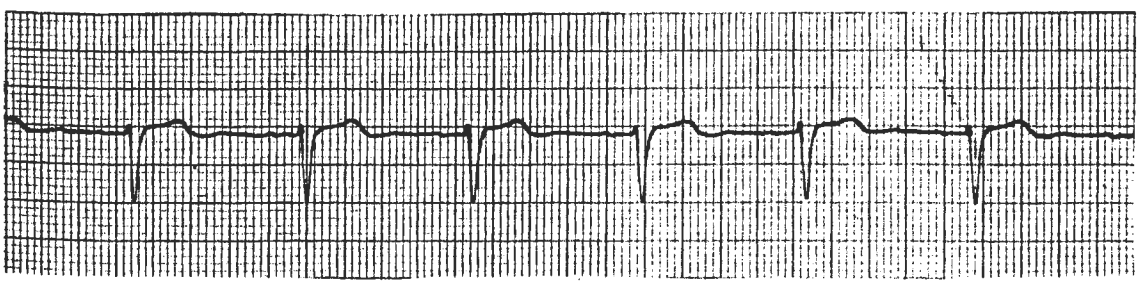
4 minutes



5 minutes

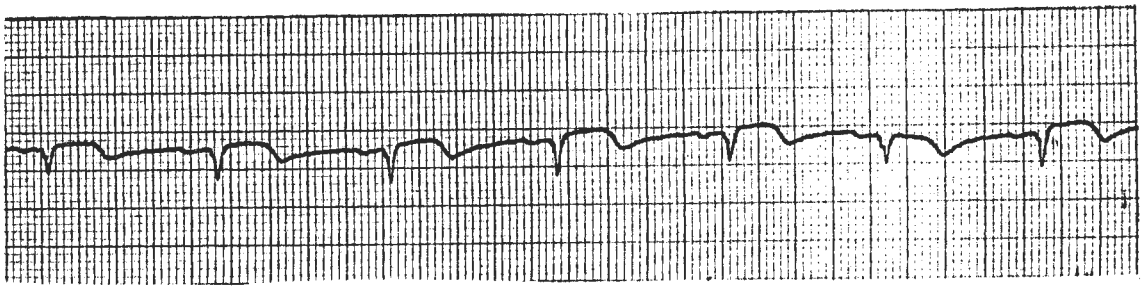


10 minutes

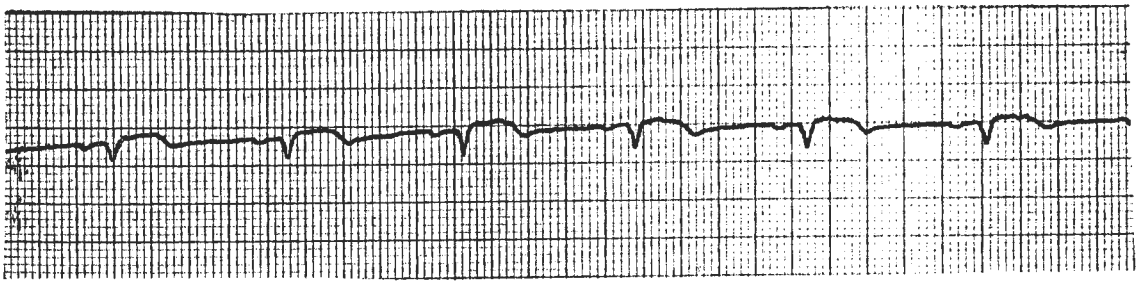


15 minutes

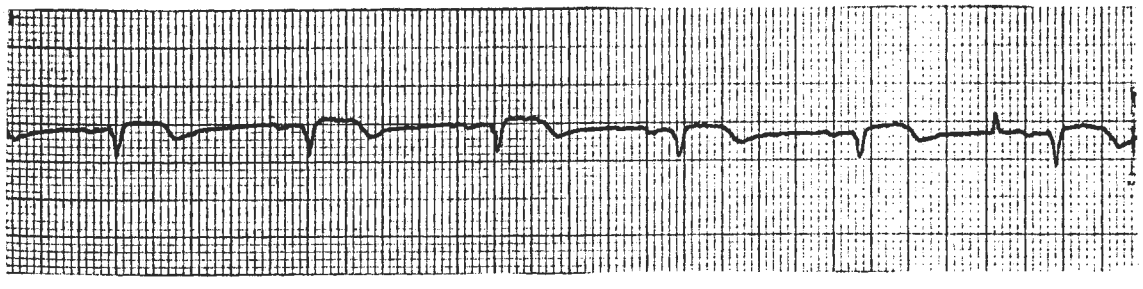
Patient 12 Monitor lead V_1 Medication _____



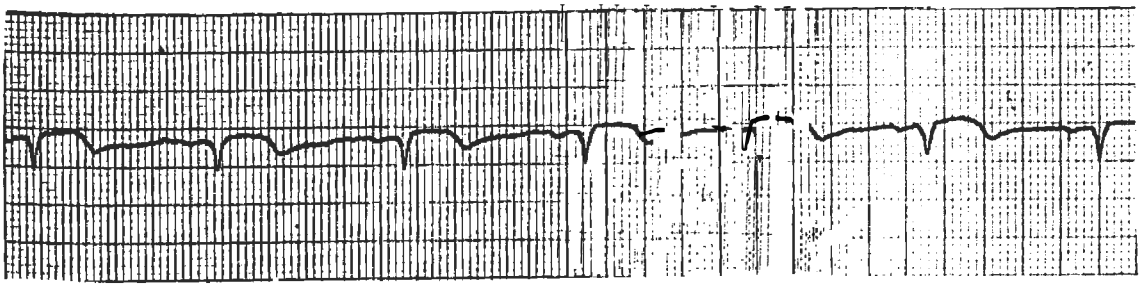
Baseline



1 minute

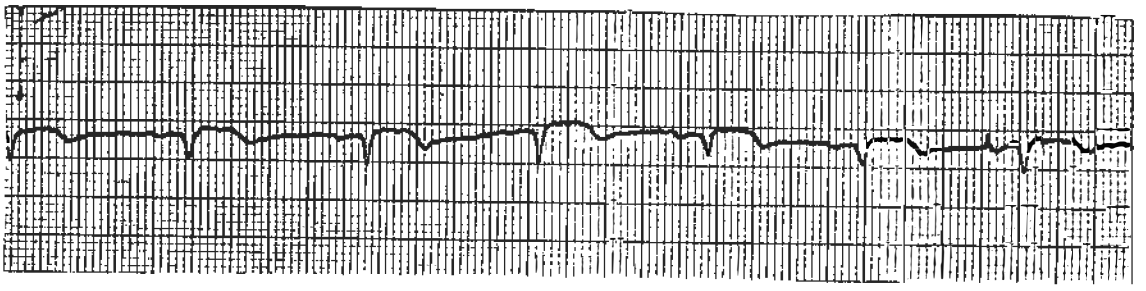


2 minutes

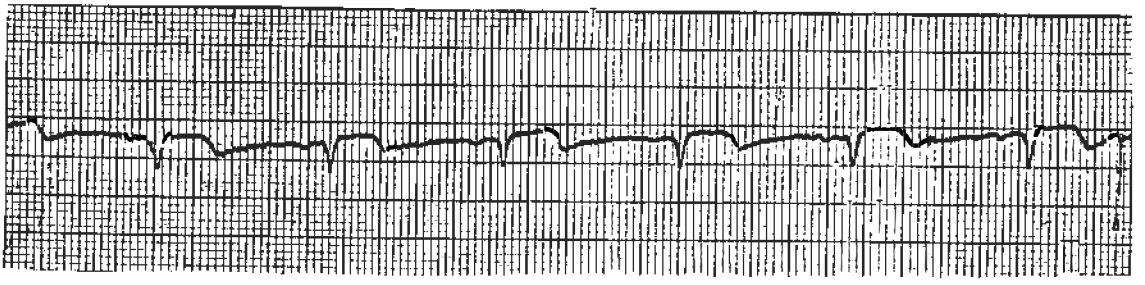


3 minutes

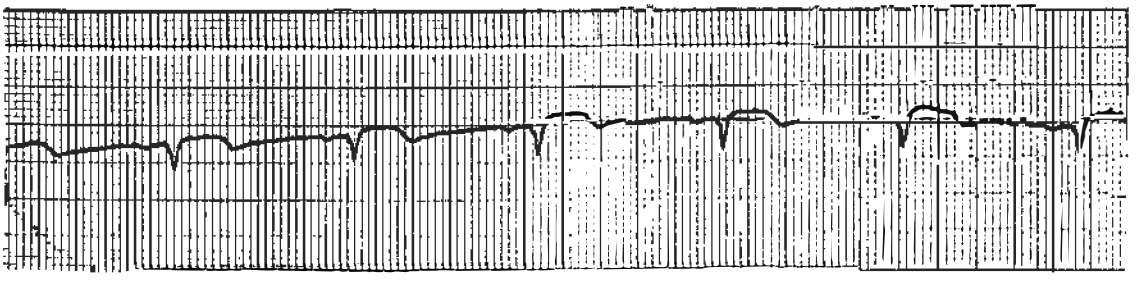
Patient 12



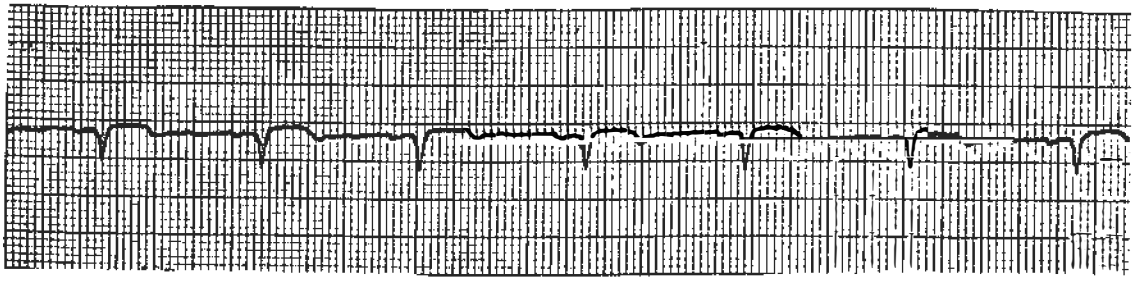
4 minutes



5 minutes

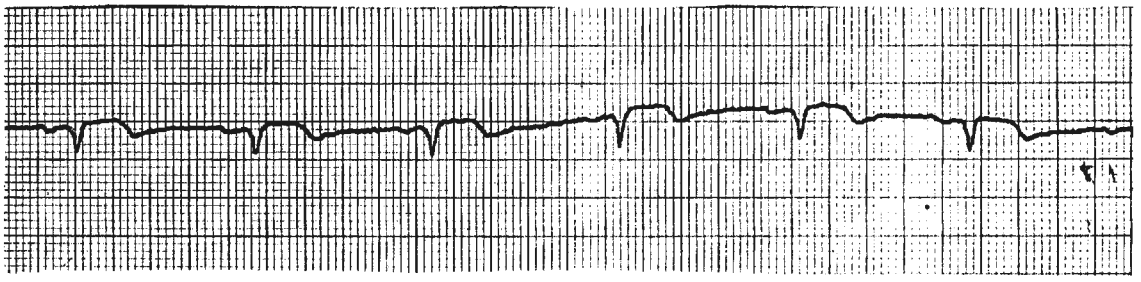


10 minutes



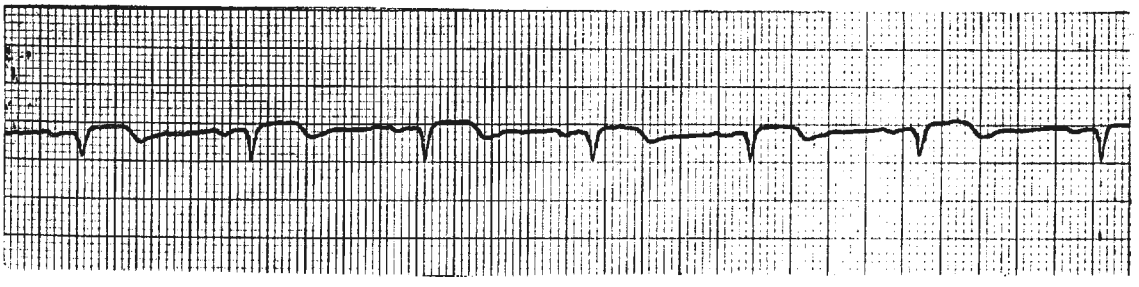
15 minutes

Patient 12

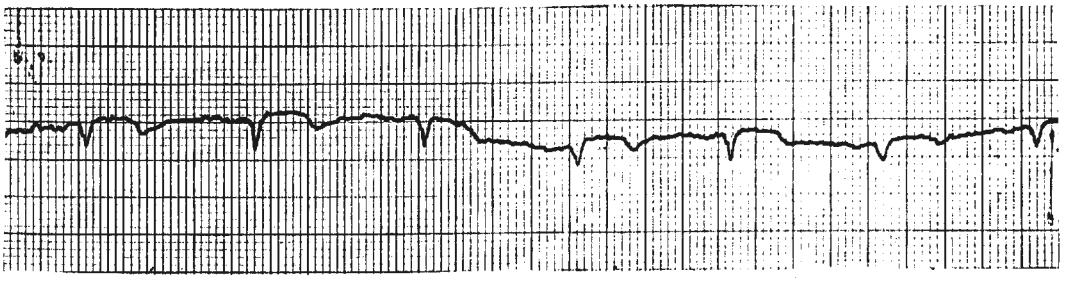


20 minutes

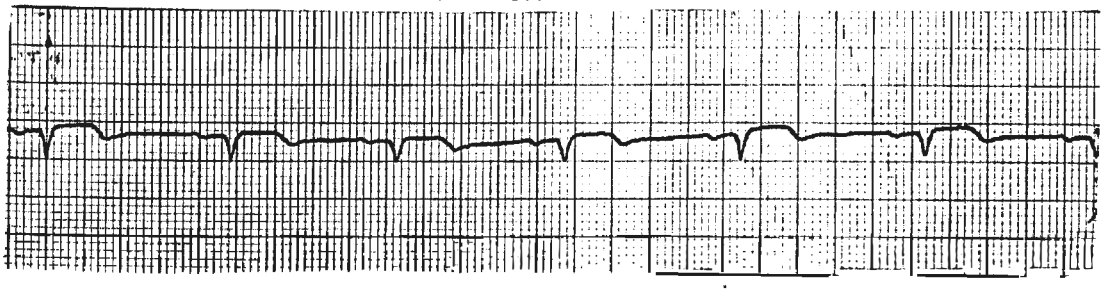
Bath Completed



1 minute

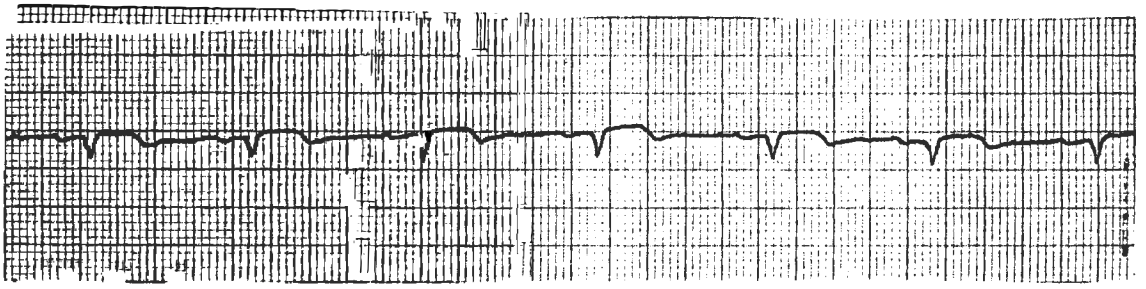


2 minutes

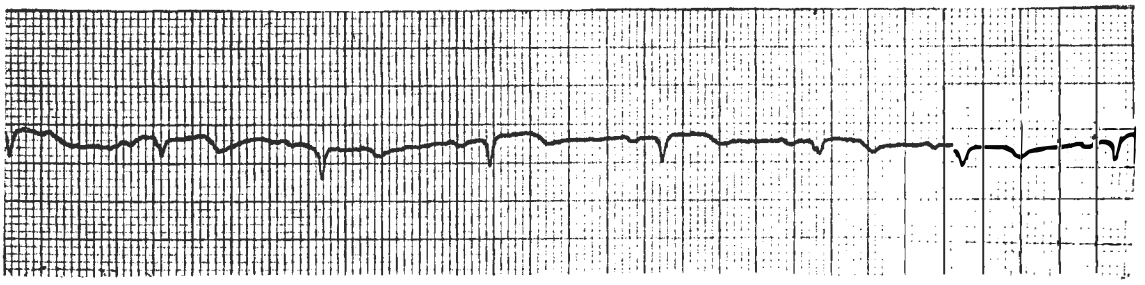


3 minutes

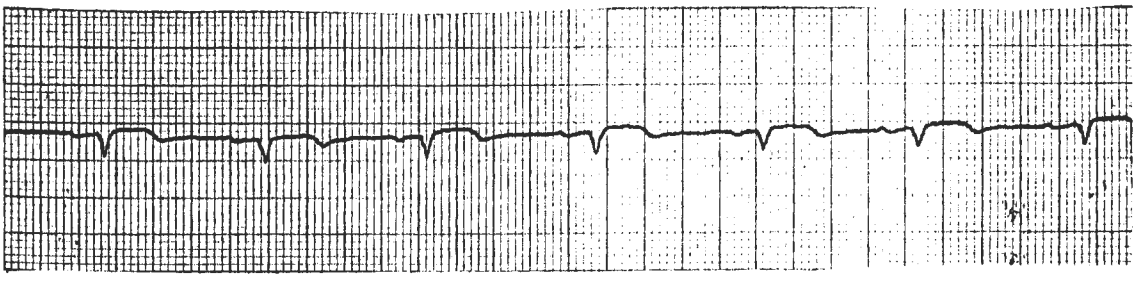
Patient 12



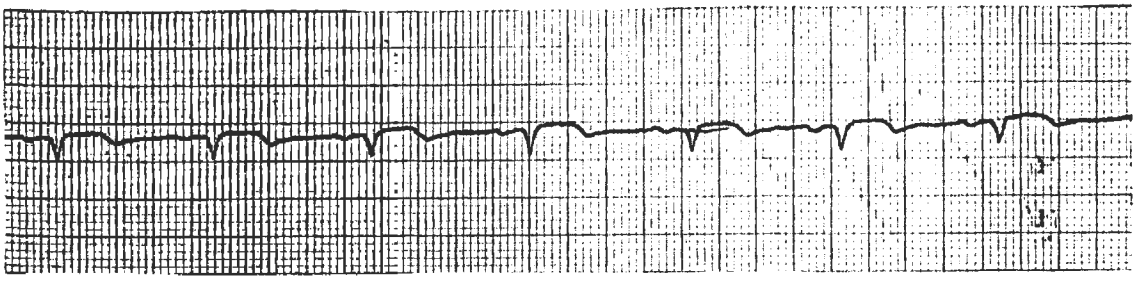
4 minutes



5 minutes

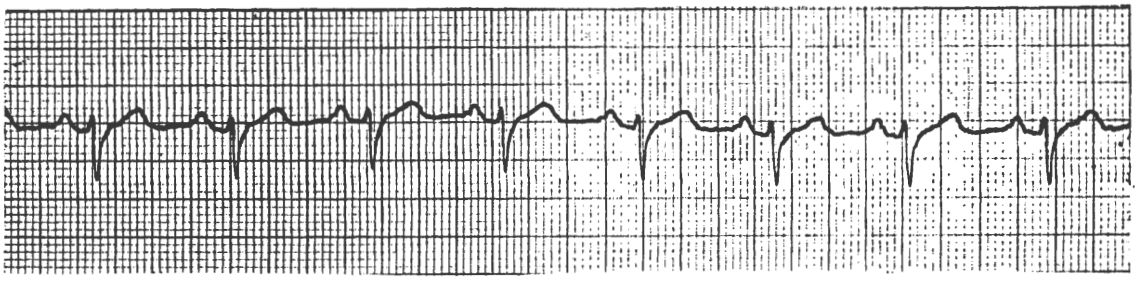


10 minutes

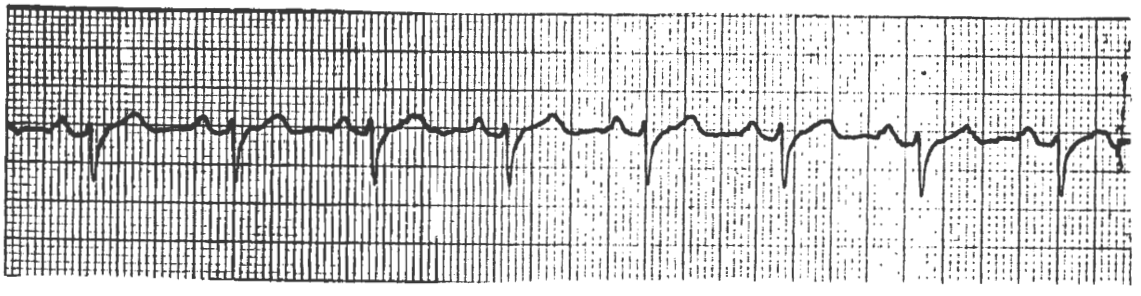


15 minutes

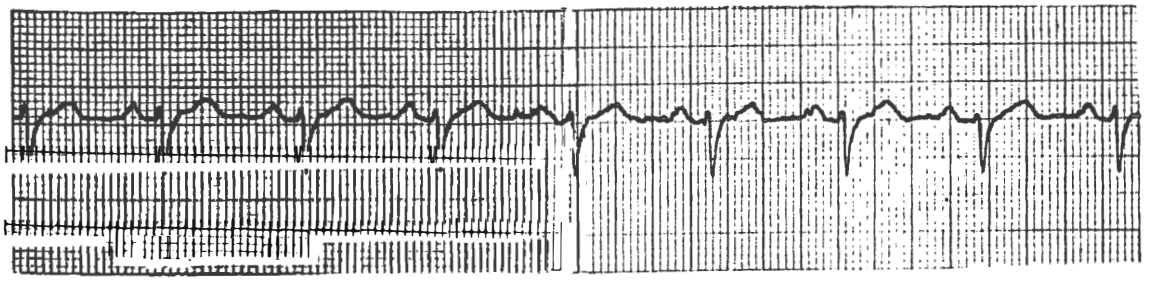
Patient 13 Monitor lead II Medication Lanoxin



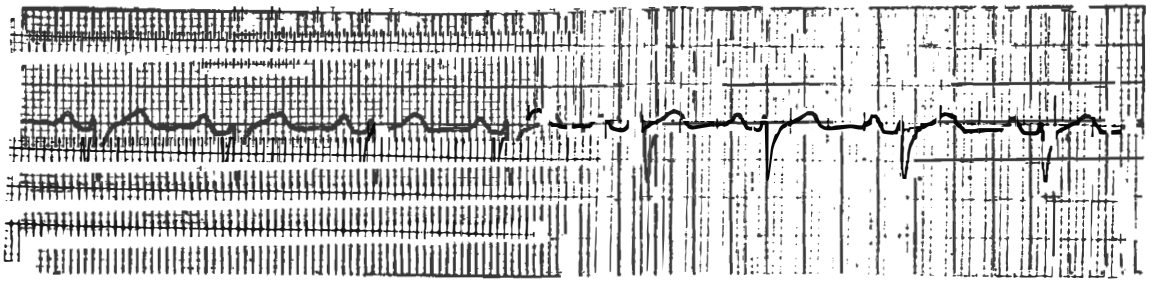
Baseline



1 minute

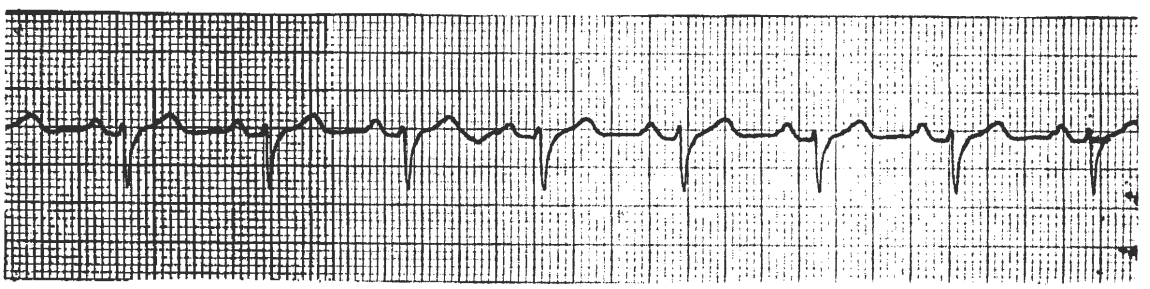


2 minutes

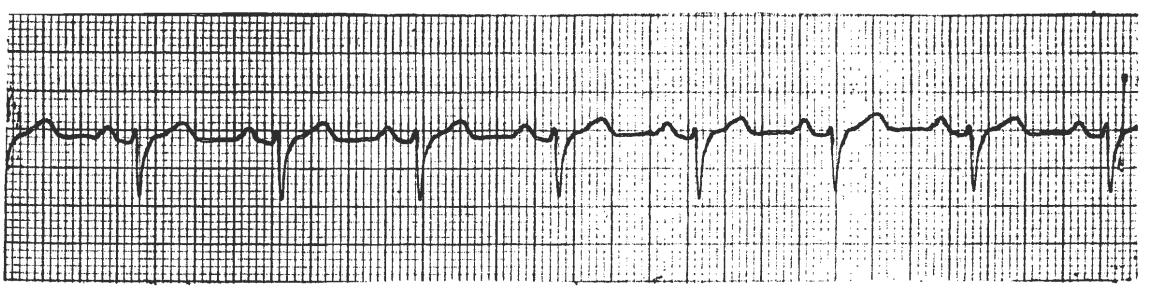


3 minutes

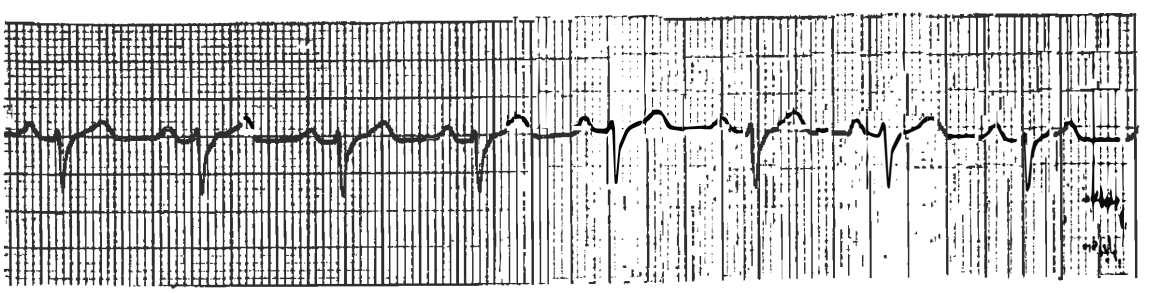
Patient 13



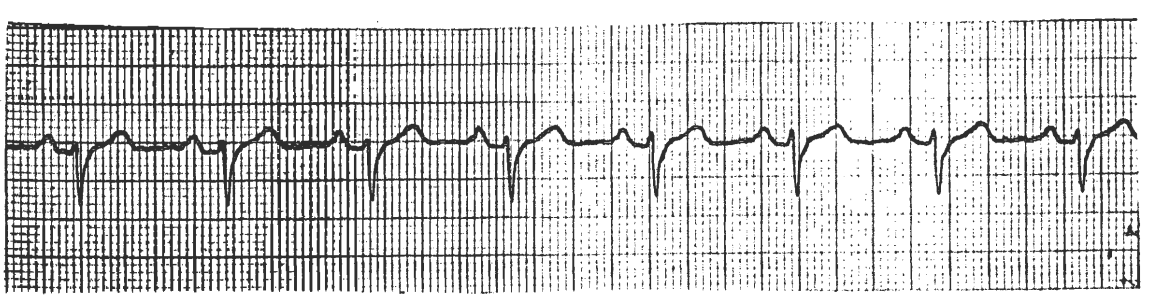
4 minutes



5 minutes

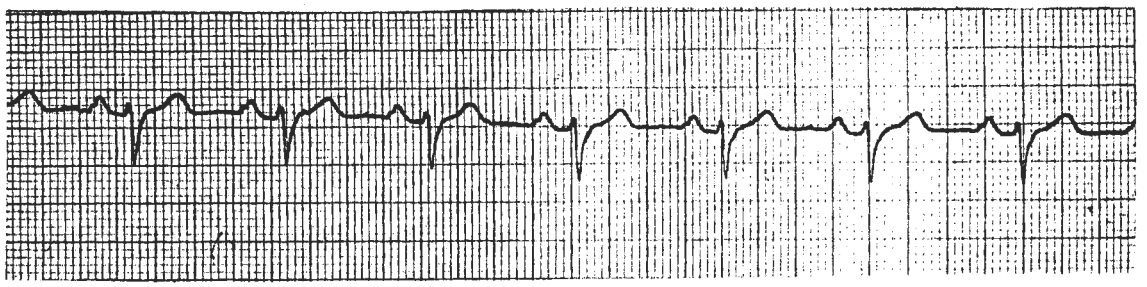


10 minutes



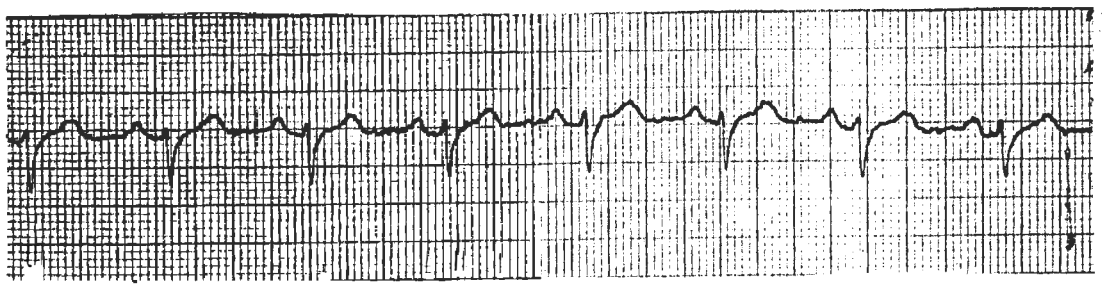
15 minutes

Patient 13

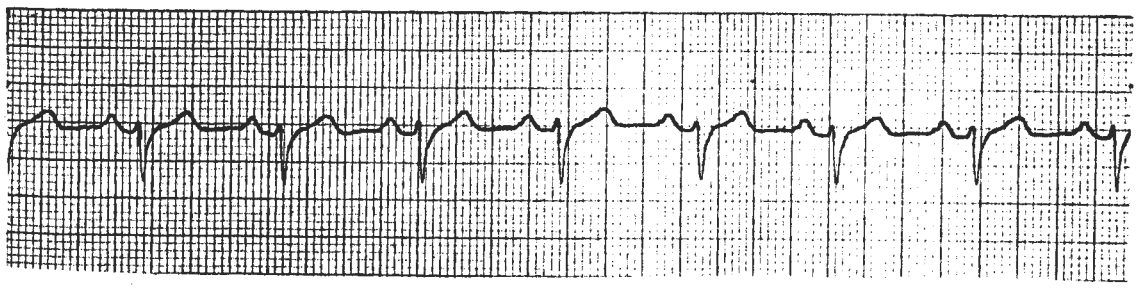


20 minutes

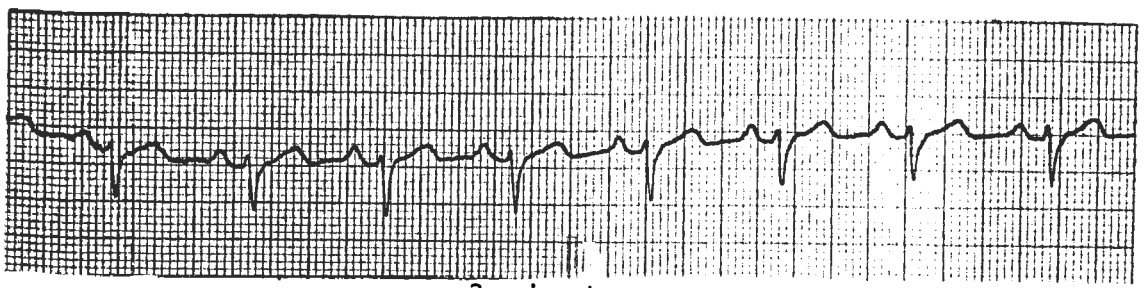
Bath Completed



1 minute

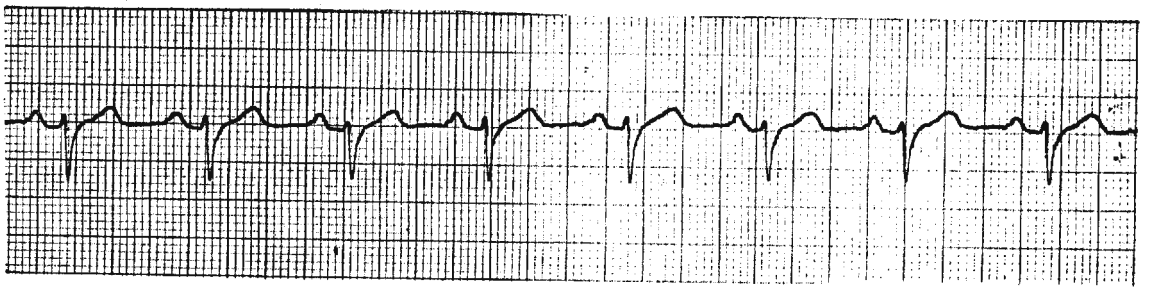


2 minutes

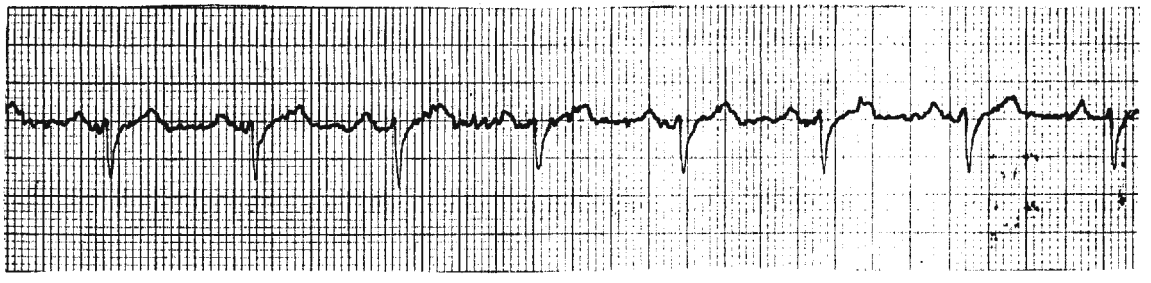


3 minutes

Patient 13



4 minutes



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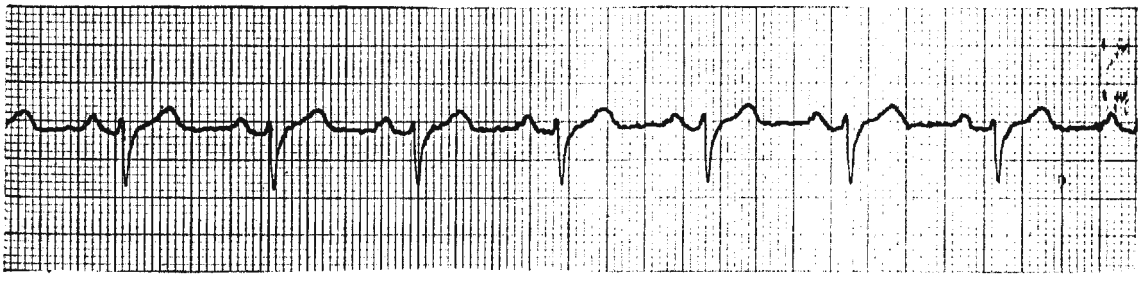
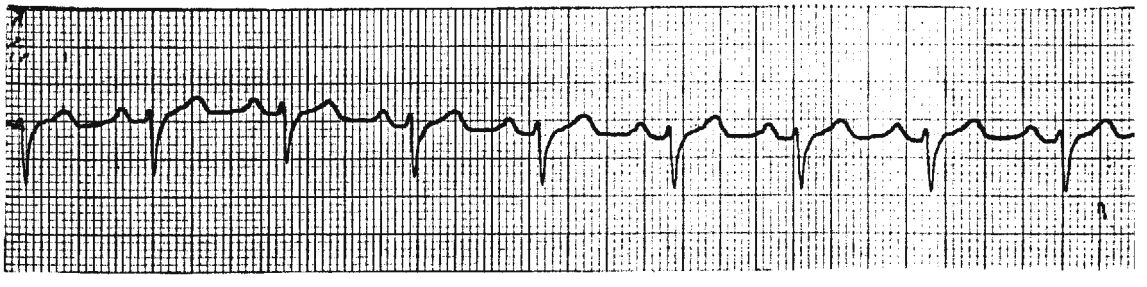
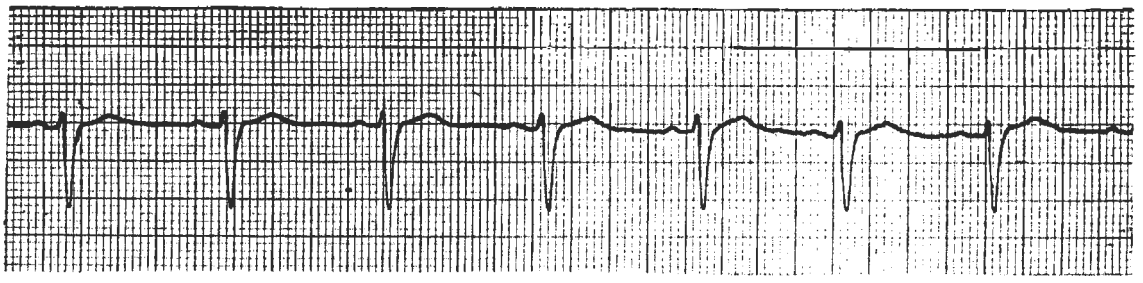


10 minutes

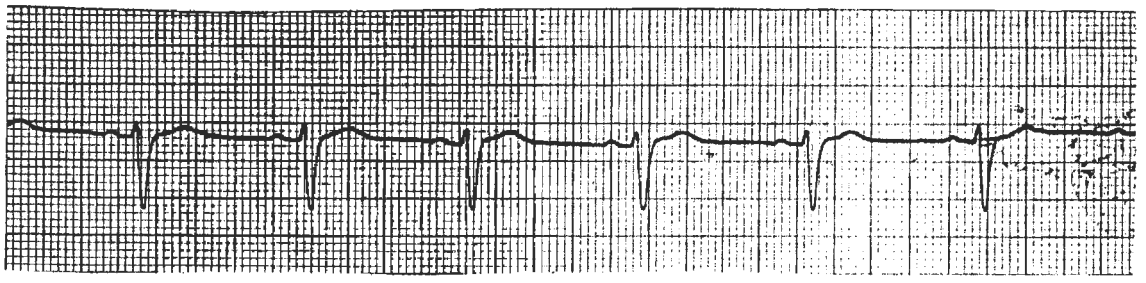


15 minutes

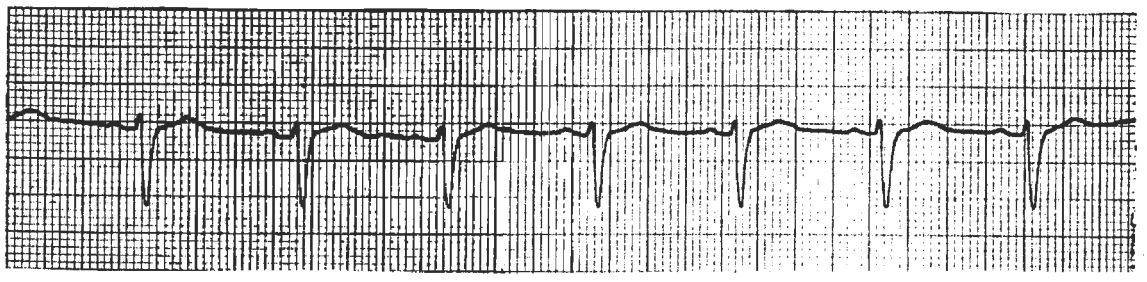
Patient 14 Monitor lead V_1 Medication _____



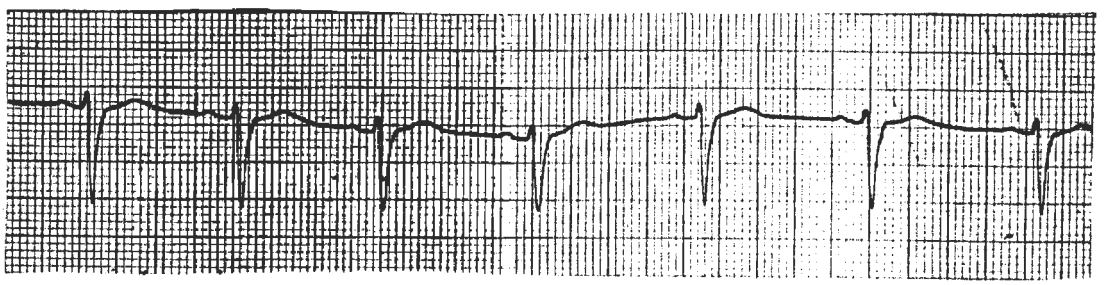
Baseline



1 minute

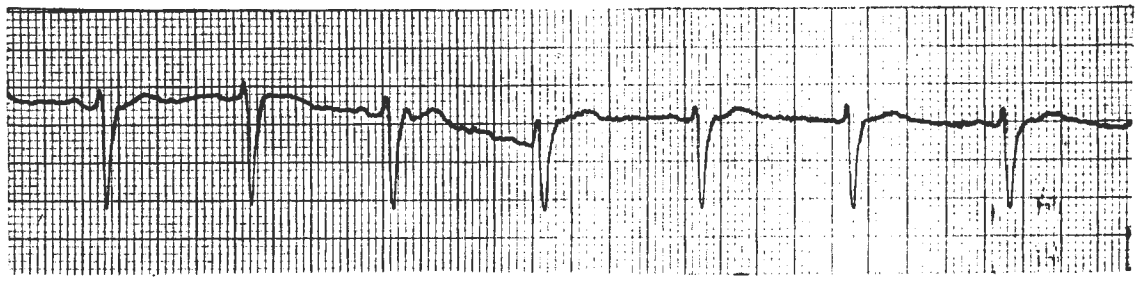


2 minutes

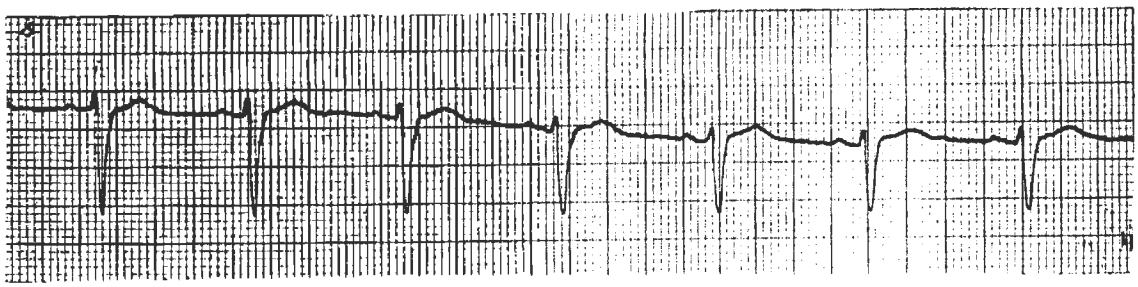


3 minutes

Patient 14



4 minutes



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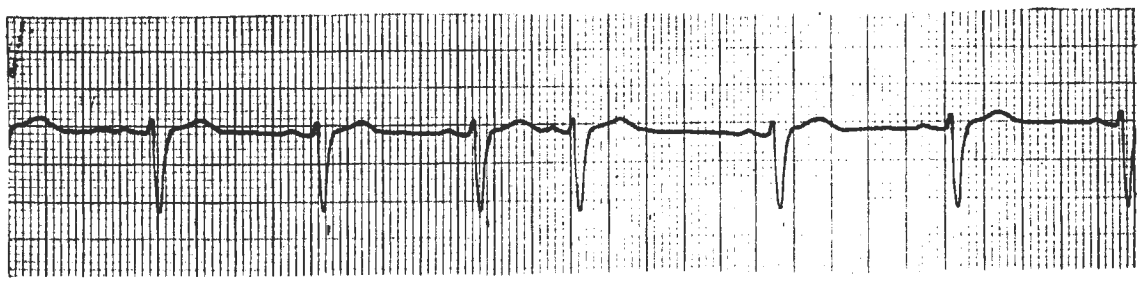
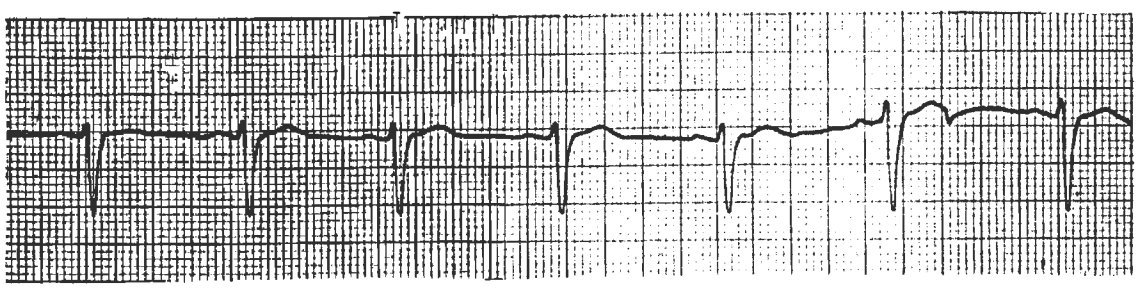
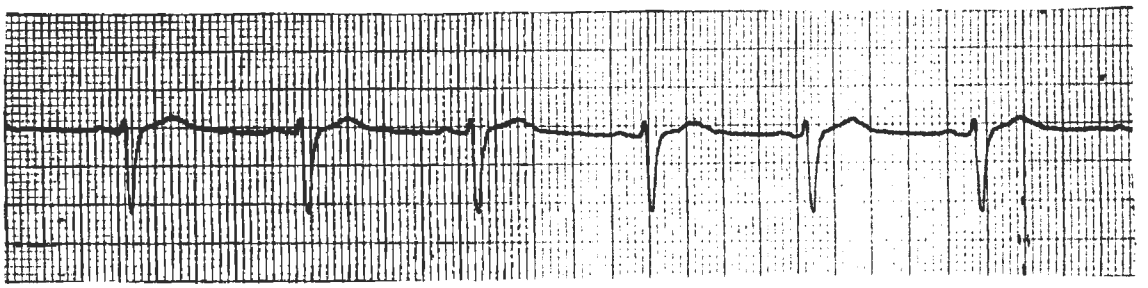


10 minutes



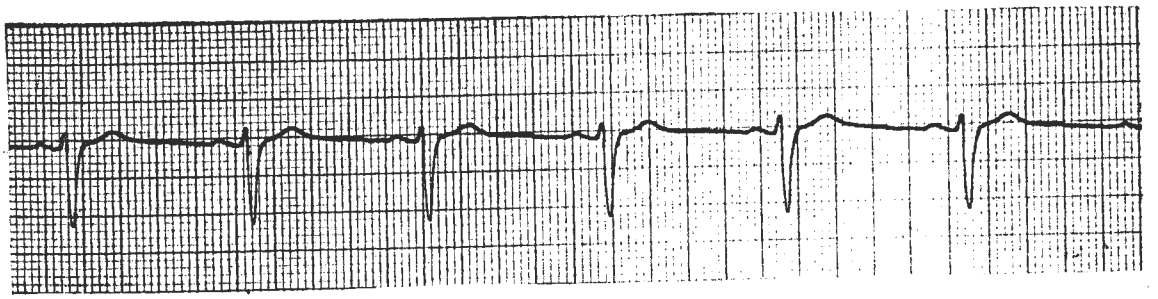
15 minutes

Patient 14

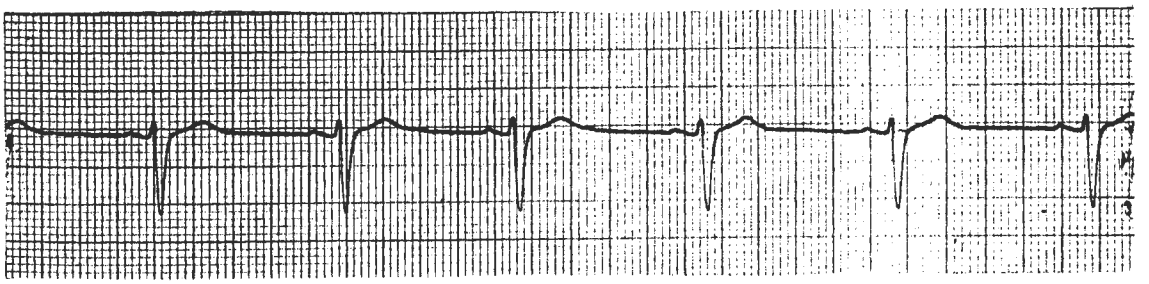


20 minutes

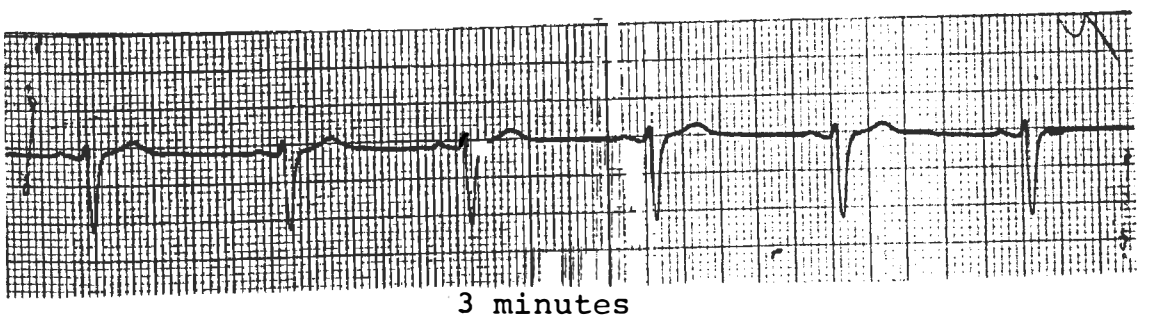
Bath Completed



1 minute

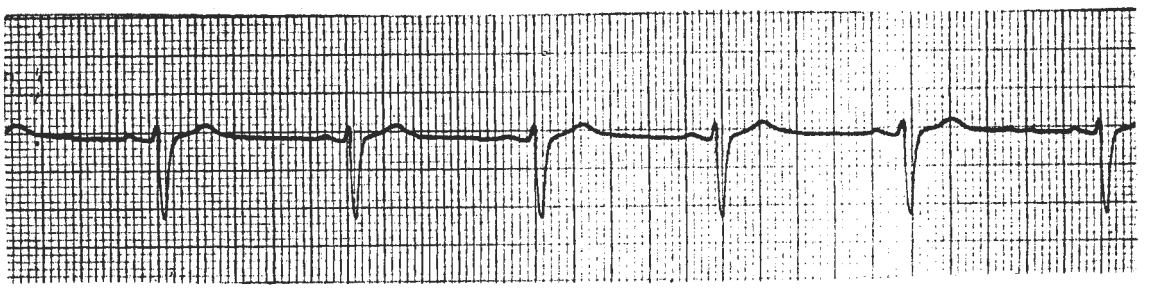


2 minutes

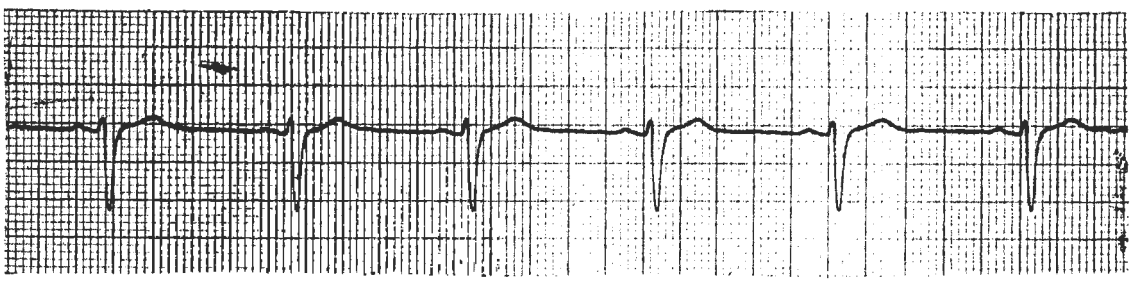


3 minutes

Patient 14



4 minutes



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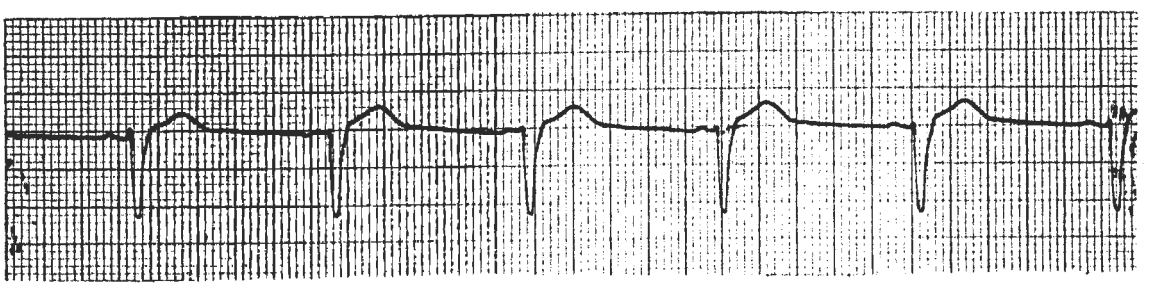

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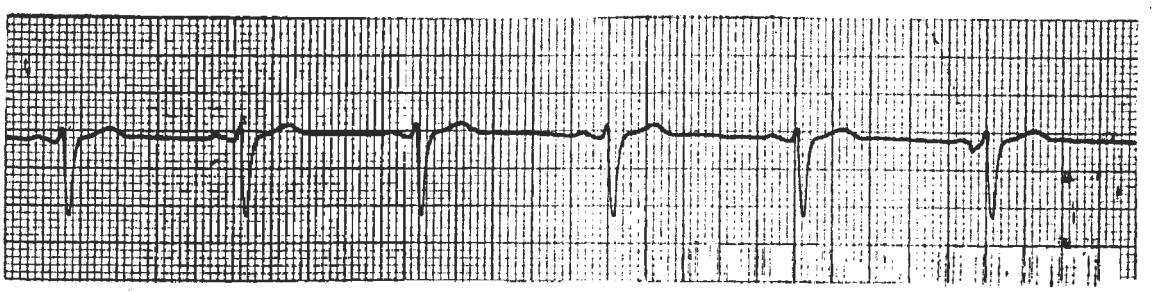
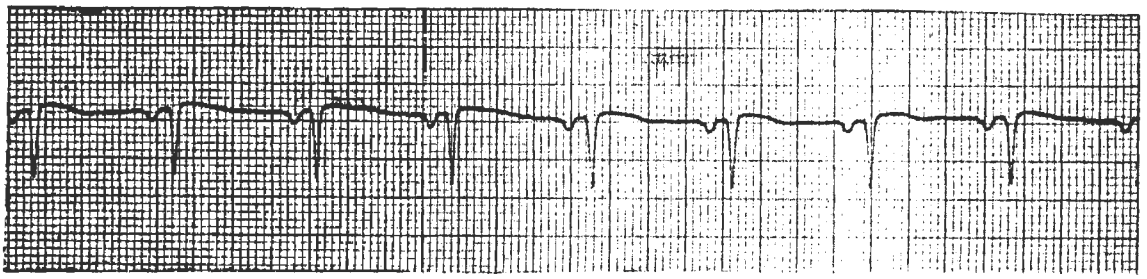
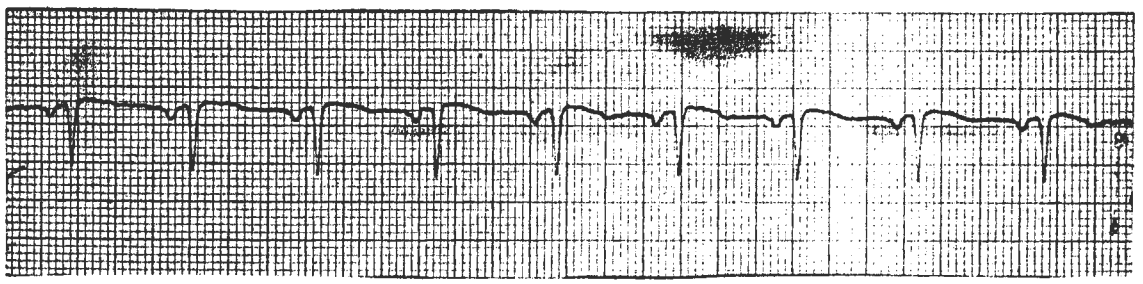


15 minutes

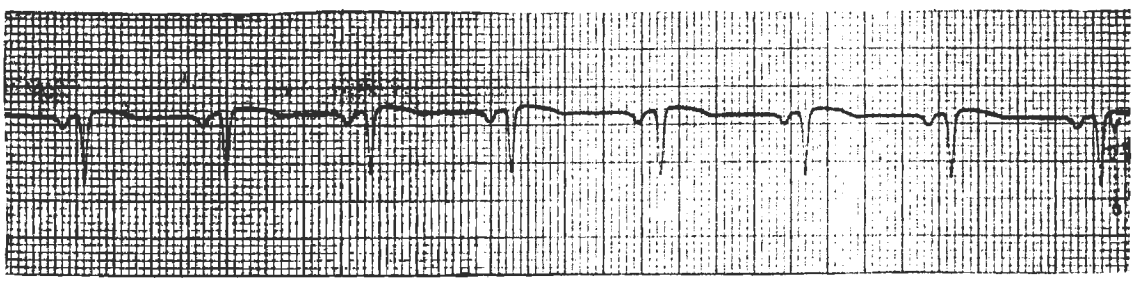
Patient 15 Monitor lead V_1 Medication Pronestyl



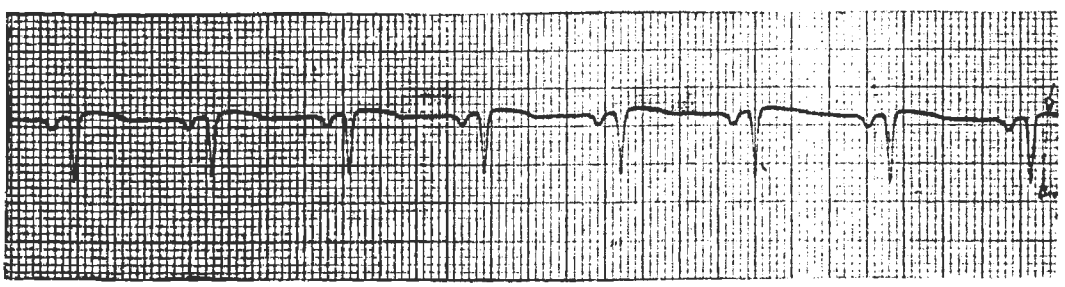
Baseline



1 minute

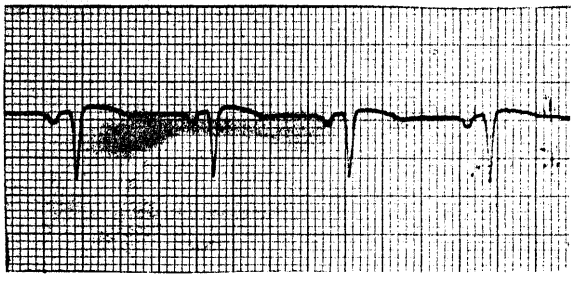


2 minutes

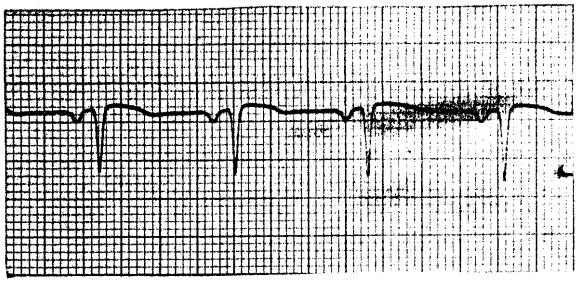


3 minutes

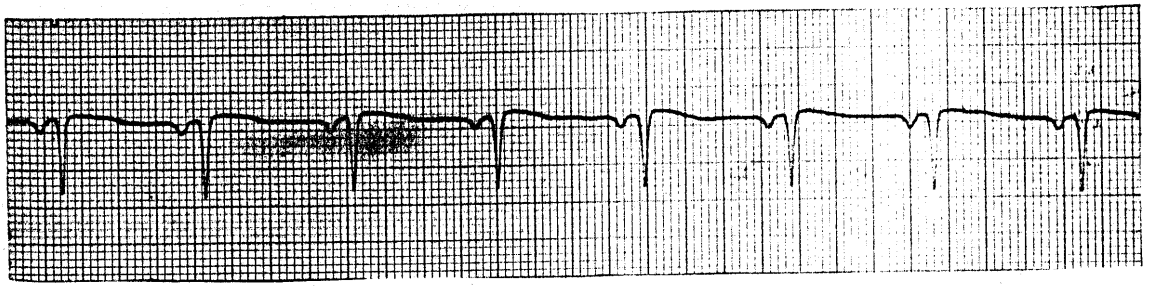
Patient 15



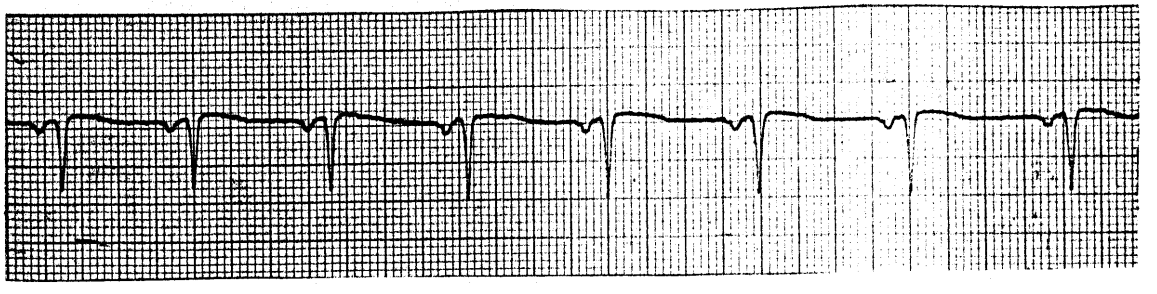
4 minutes



5 minutes

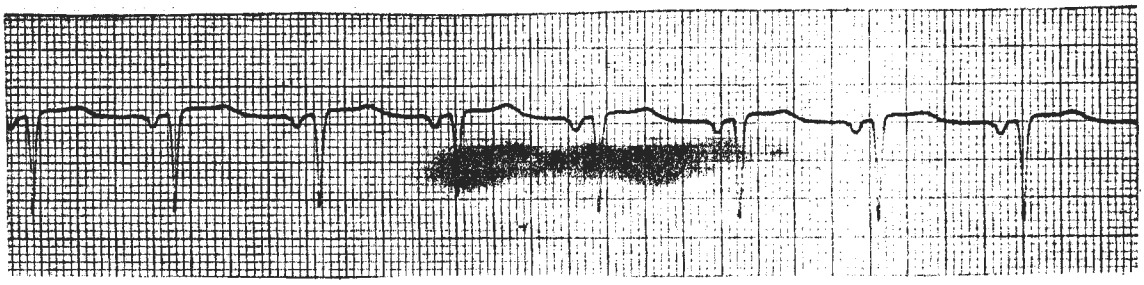


10 minutes



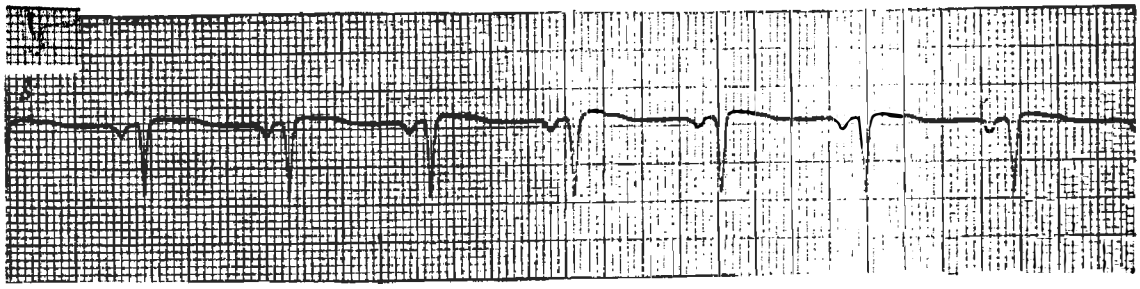
15 minutes

Patient 15

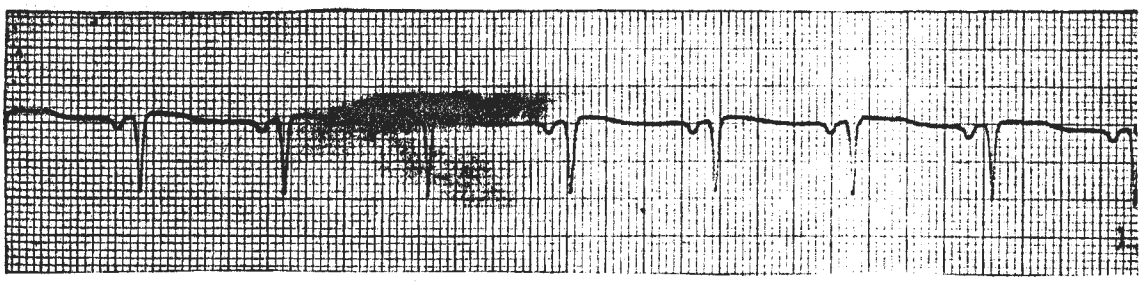


20 minutes

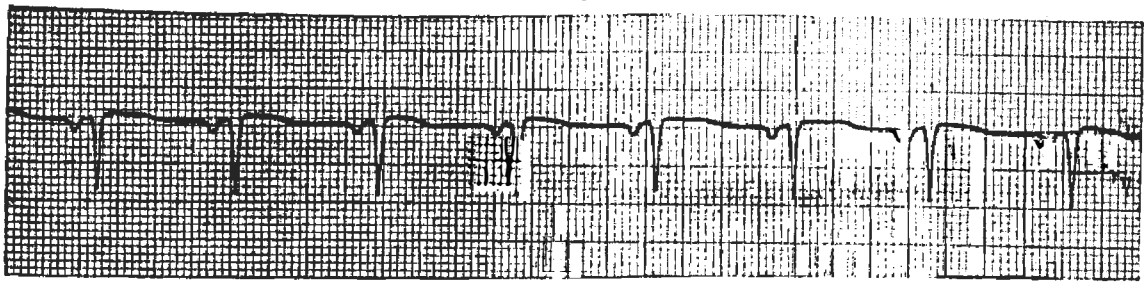
Bath Completed



1 minute

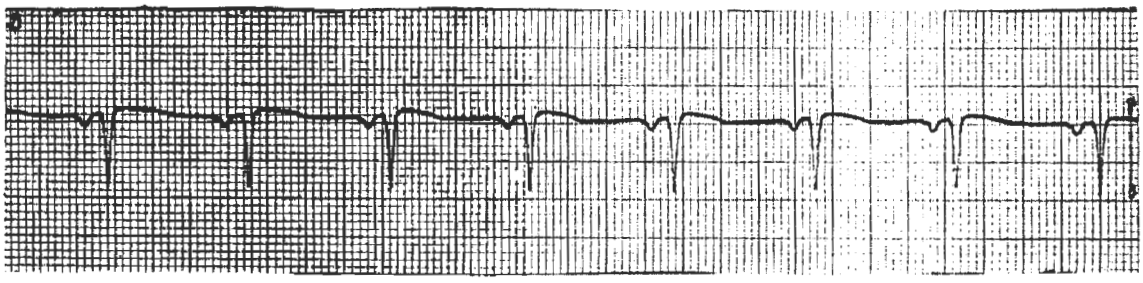


2 minutes

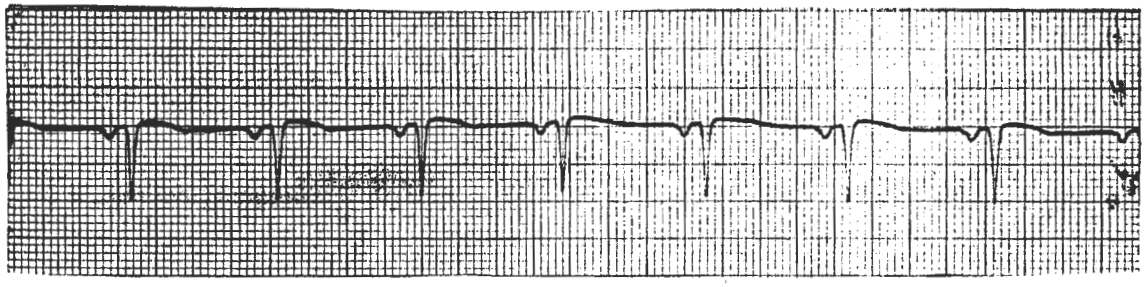


3 minutes

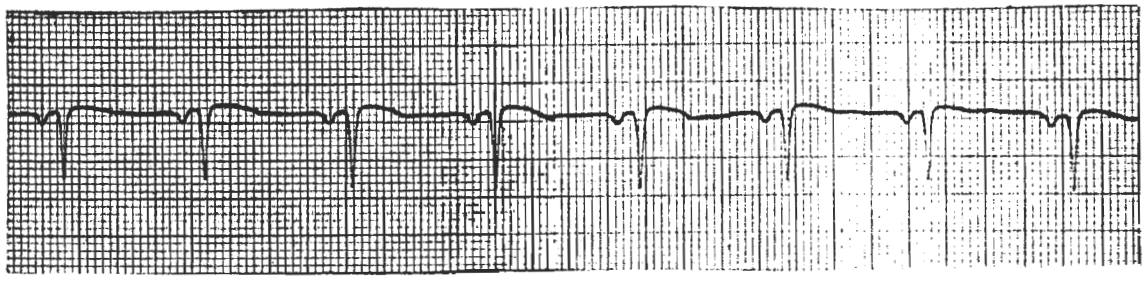
Patient 15



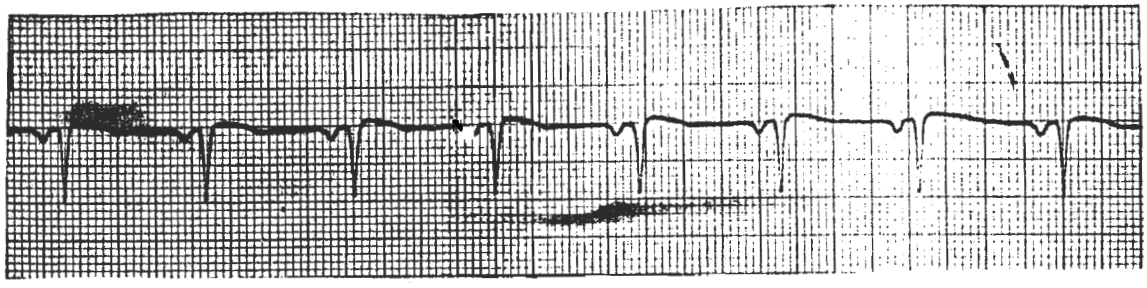
4 minutes



5 minutes

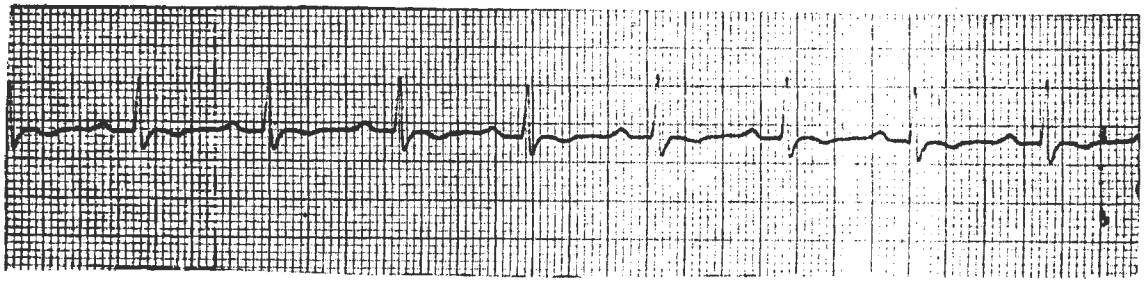


10 minutes

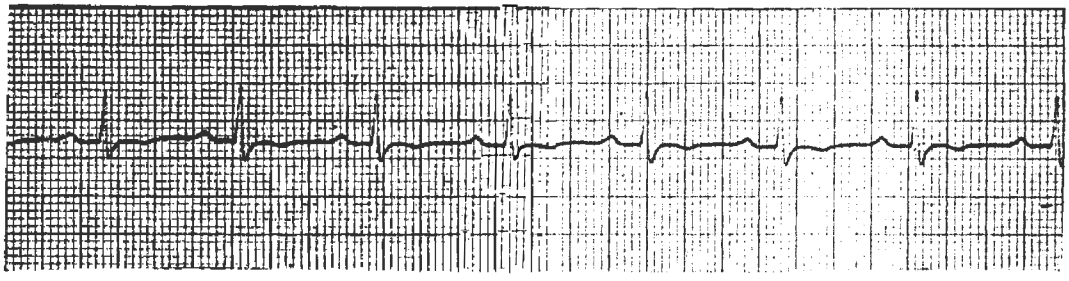


15 minutes

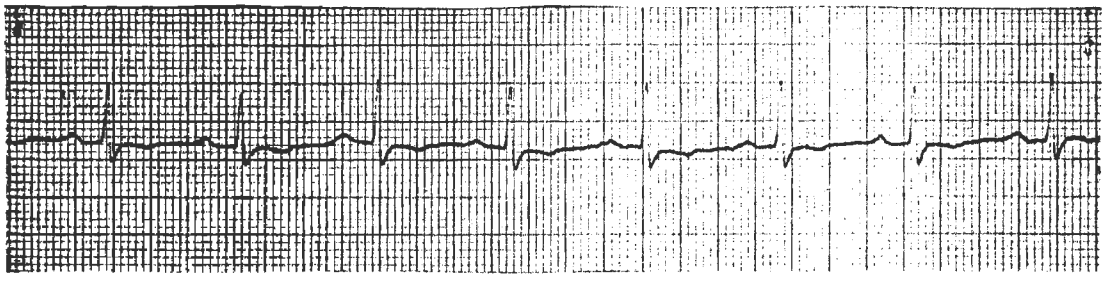
Patient 16 Monitor lead II Medication _____



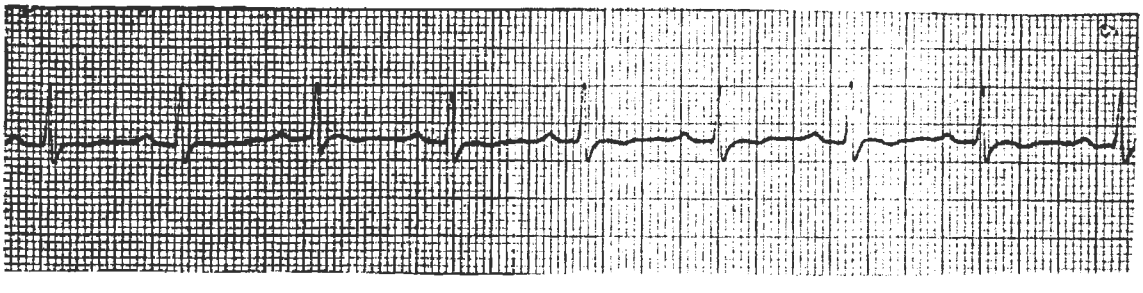
Baseline



1 minute

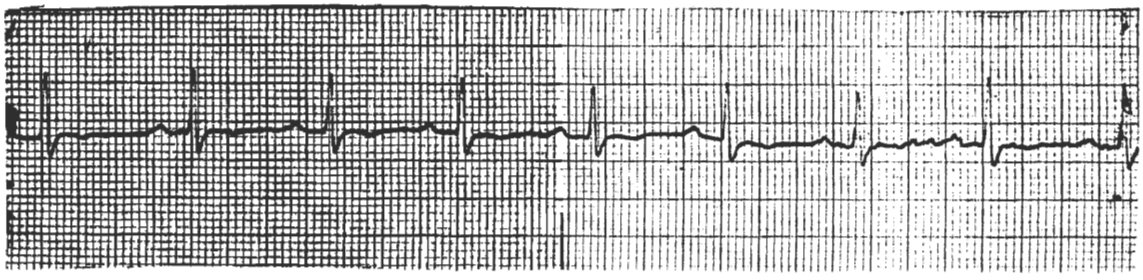


2 minutes

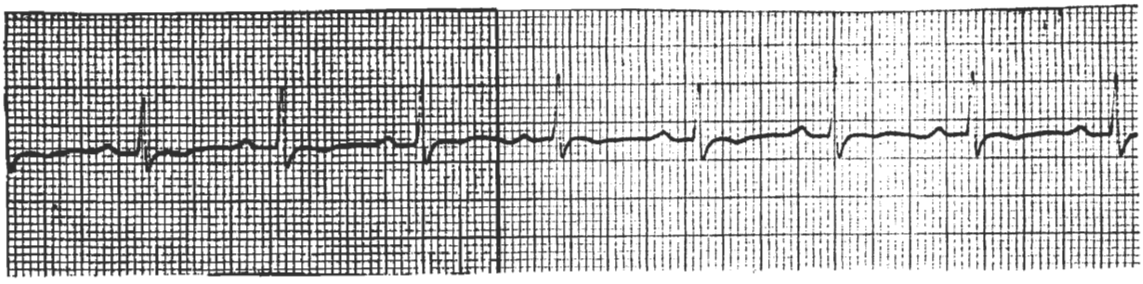


3 minutes

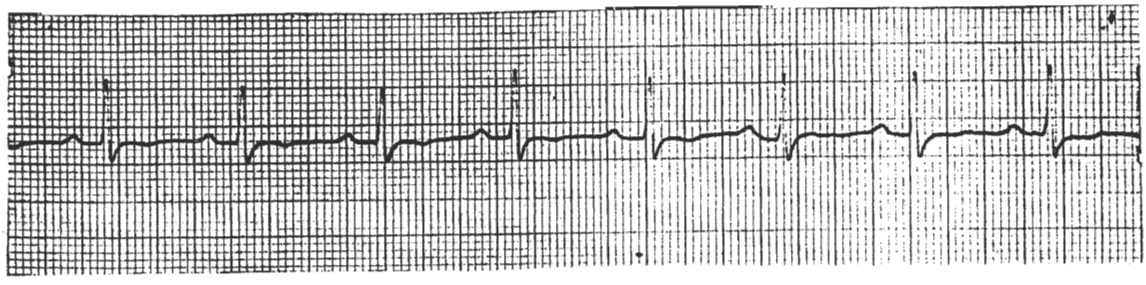
Patient 16



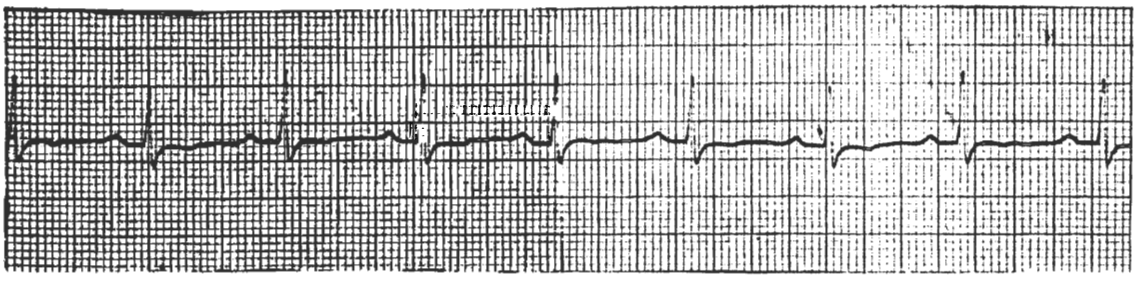
4 minutes



5 minutes

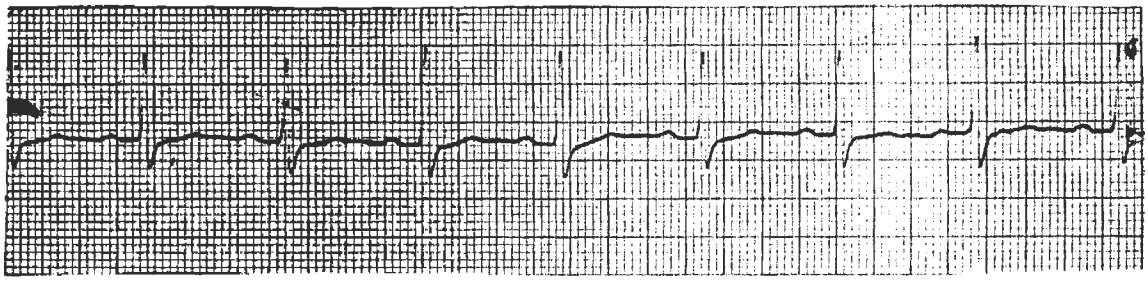


10 minutes



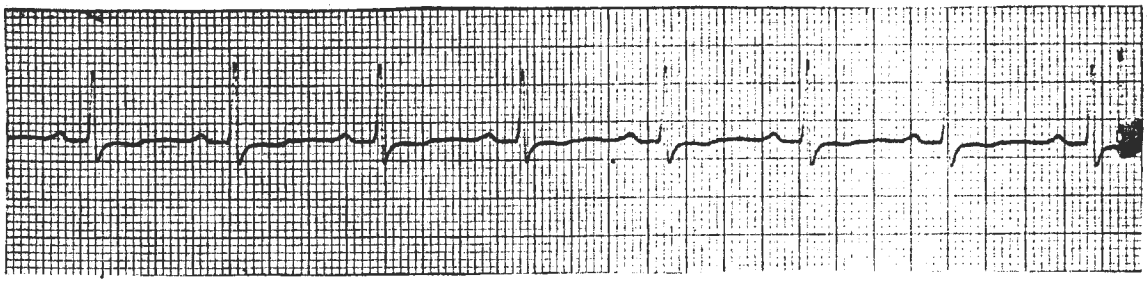
15 minutes

Patient 16

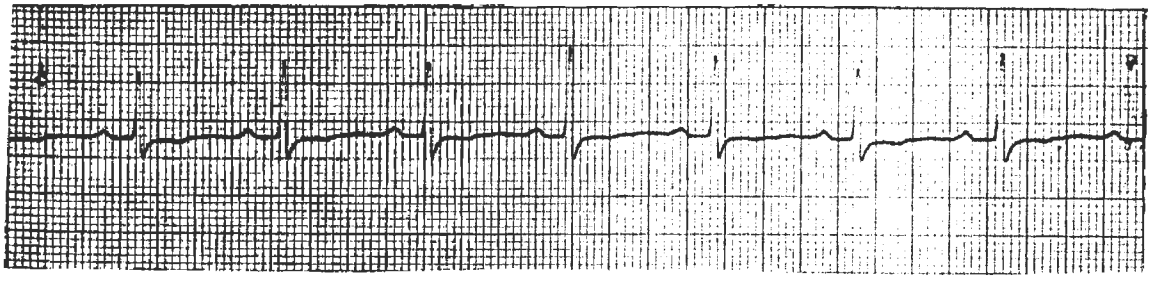


20 minutes

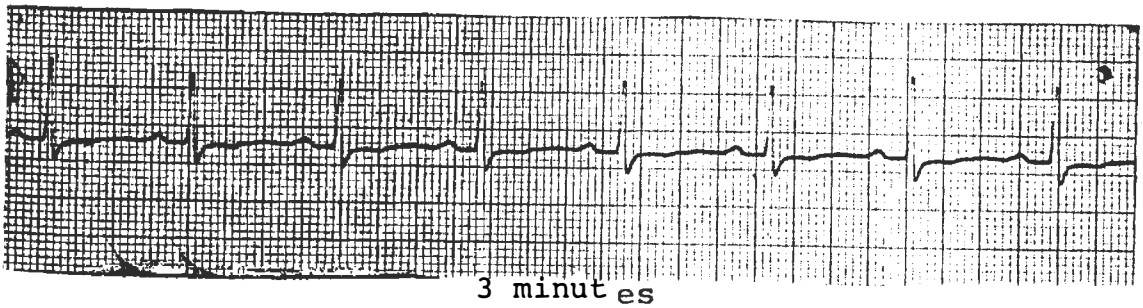
Bath Completed



1 minute

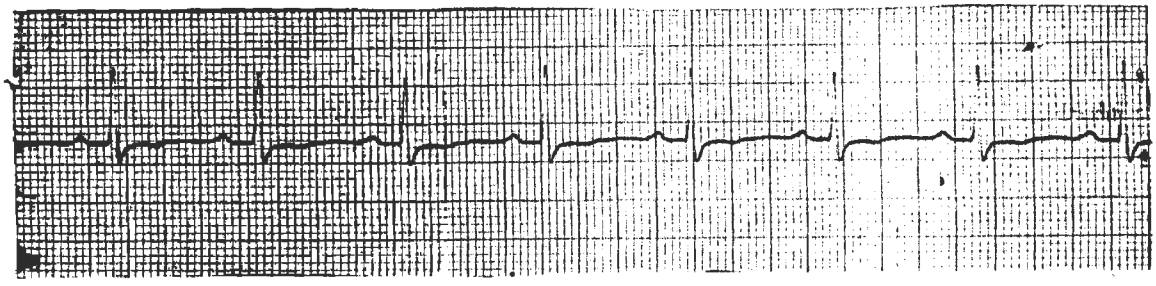


2 minutes

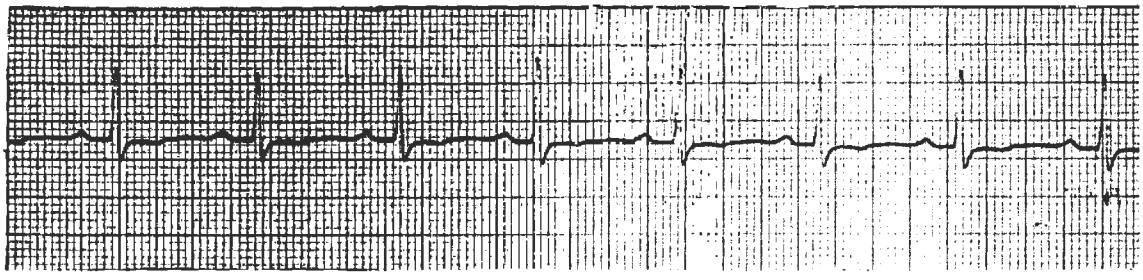


3 minutes

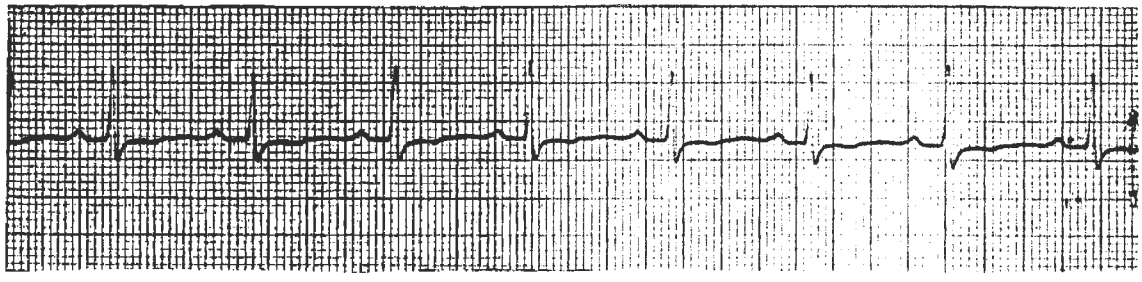
Patient 16



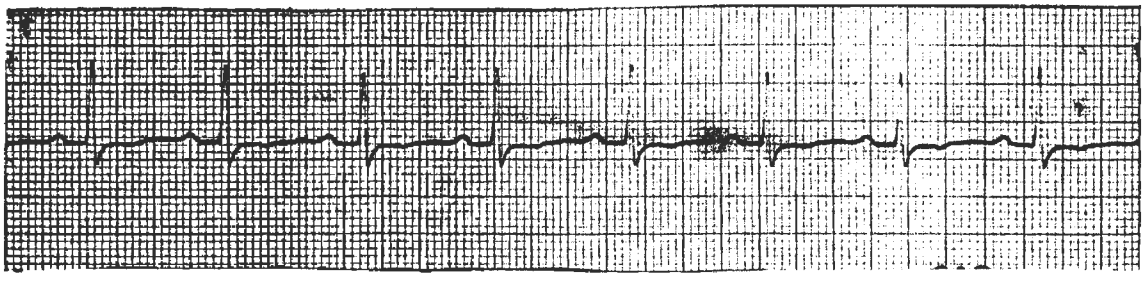
4 minutes



5 minutes

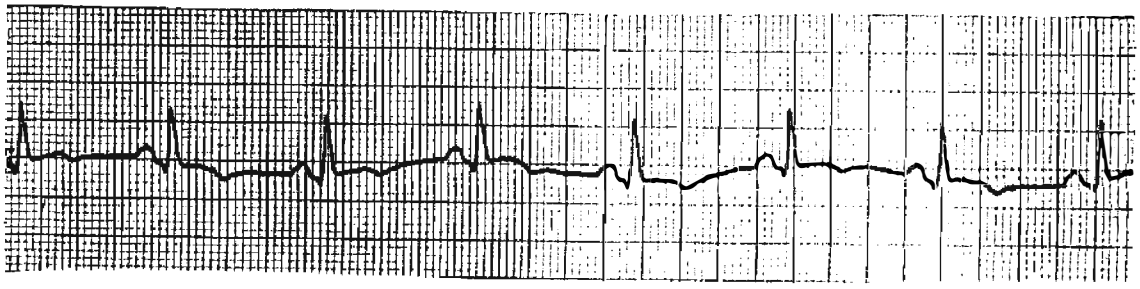


10 minutes

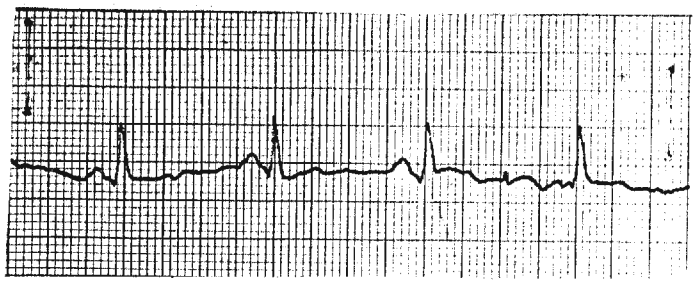


15 minutes

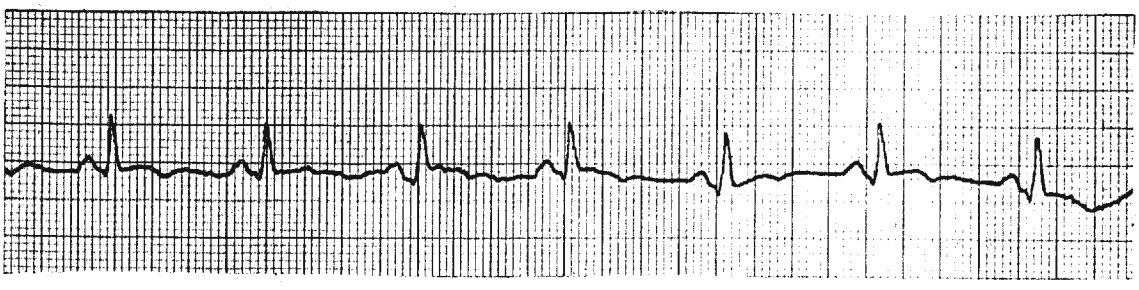
Patient 17 Monitor lead II Medication Inderal



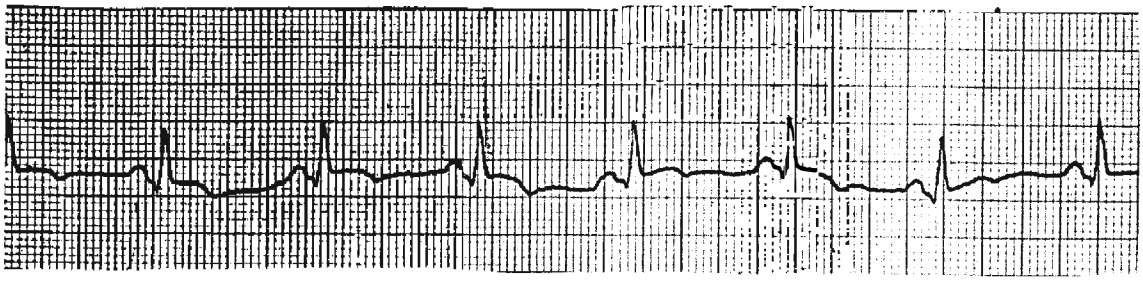
Baseline



1 minute

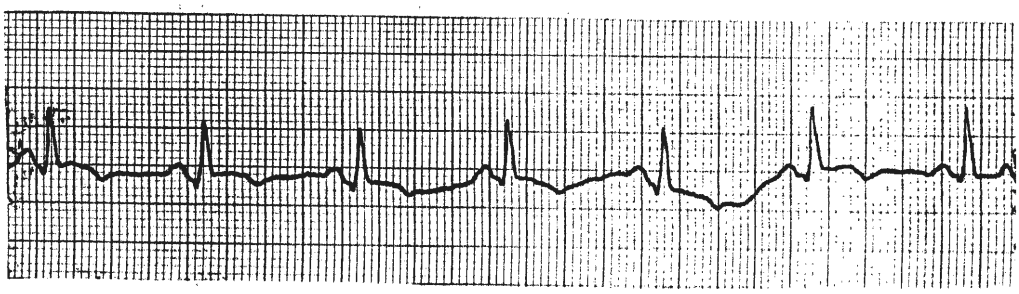


2 minutes

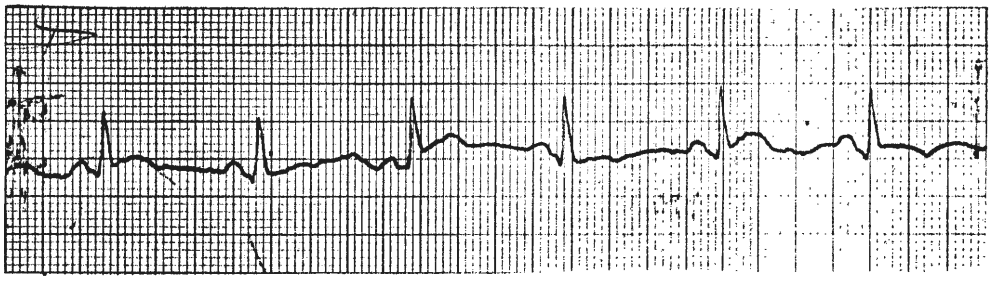


3 minutes

Patient 17



4 minutes



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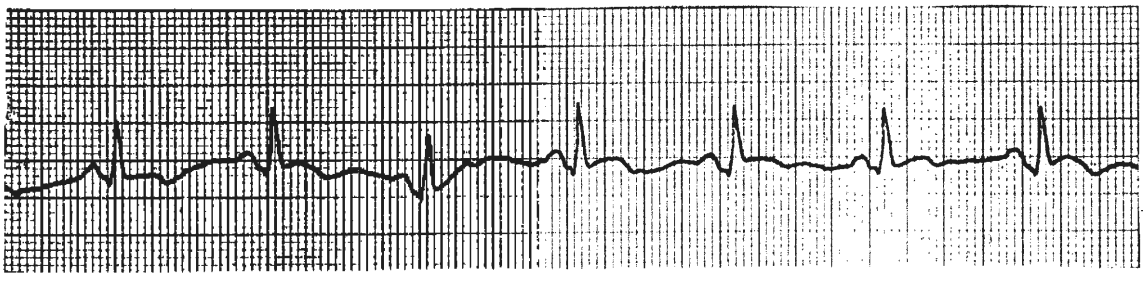
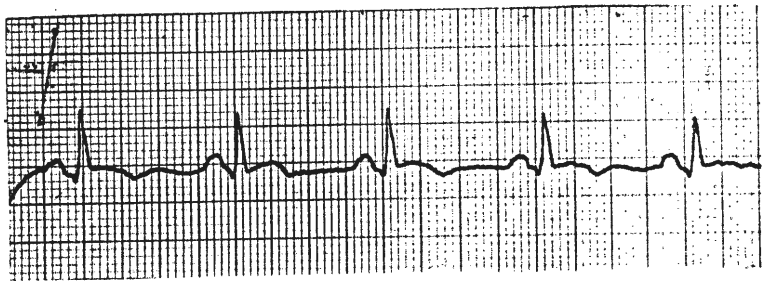
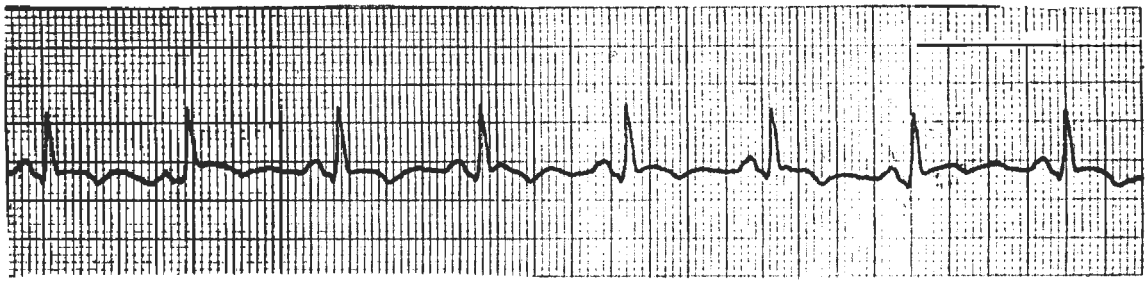


10 minutes



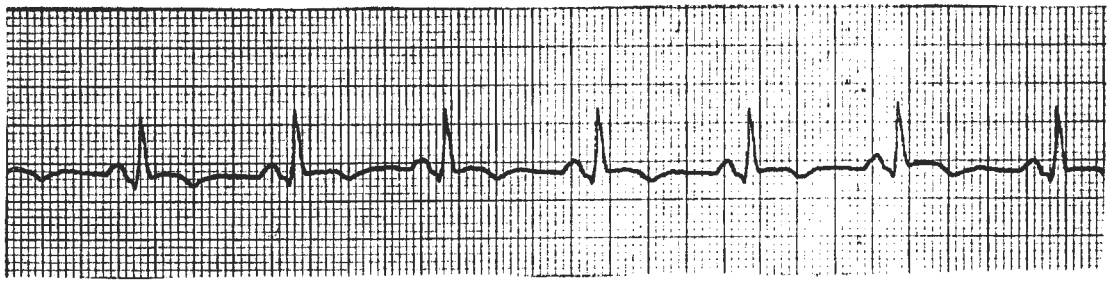
15 minutes

Patient 17



20 minutes

Bath Completed



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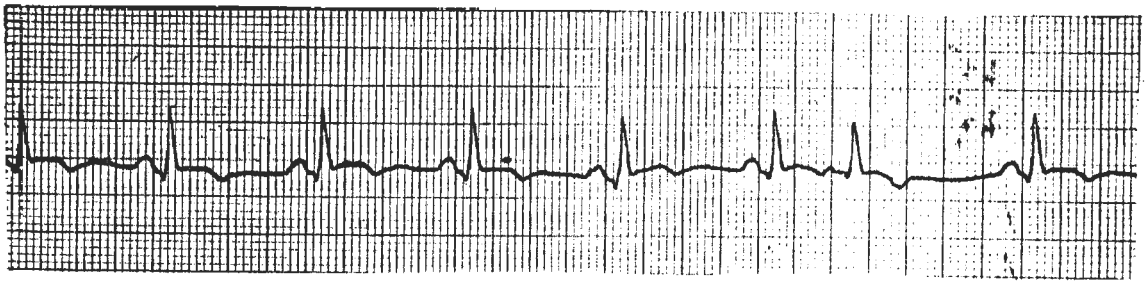
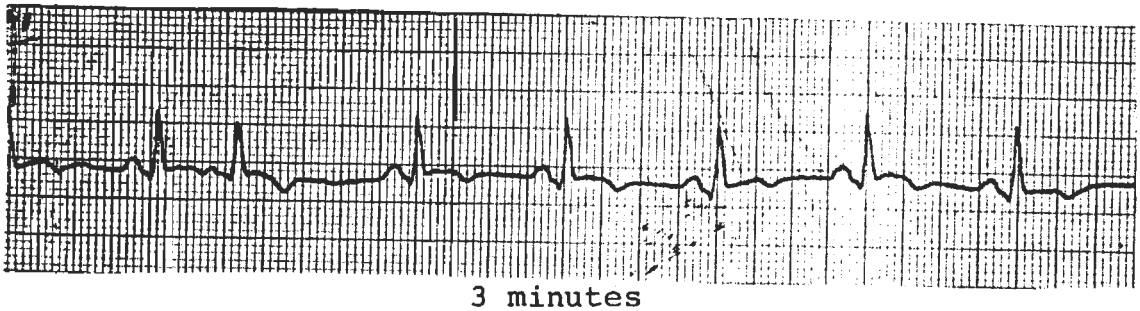
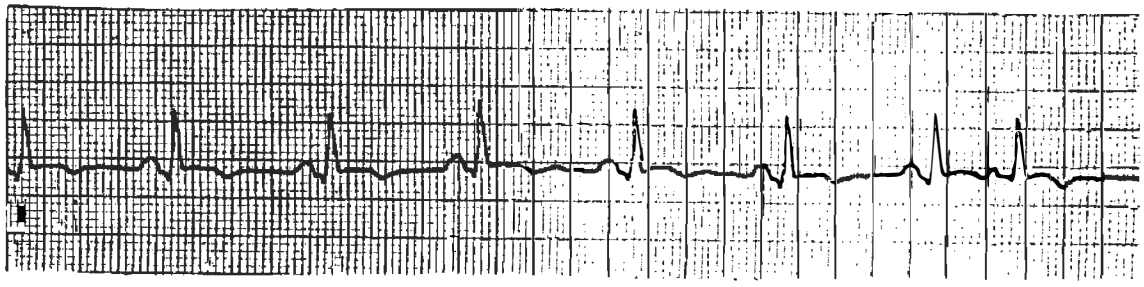


2 minutes



3 minutes

Patient 17



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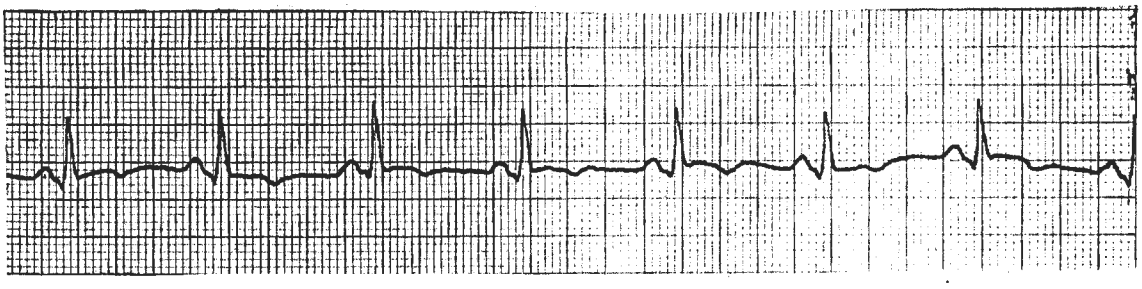
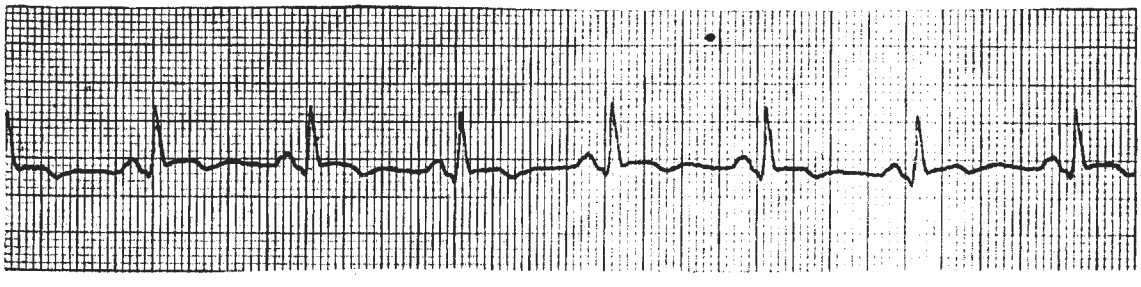
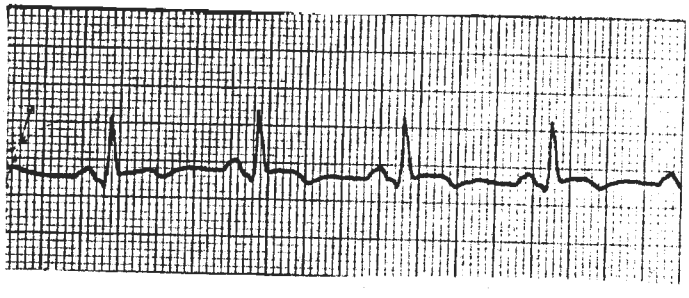


5 minutes

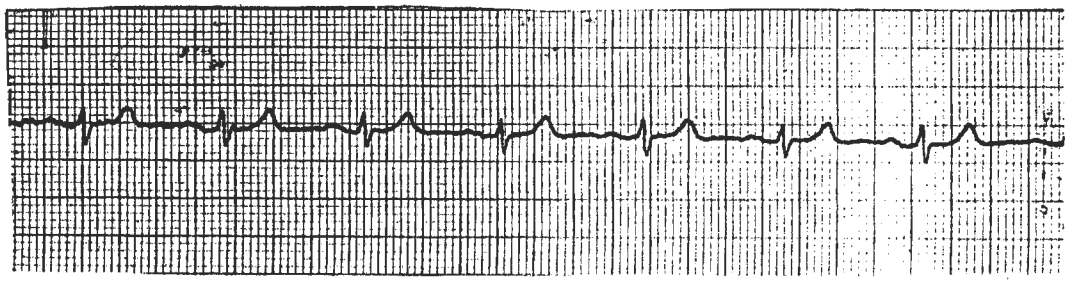


10 minutes

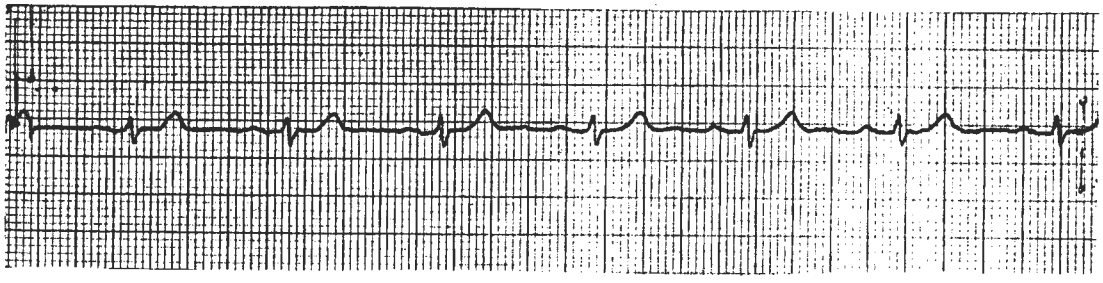


15 minutes

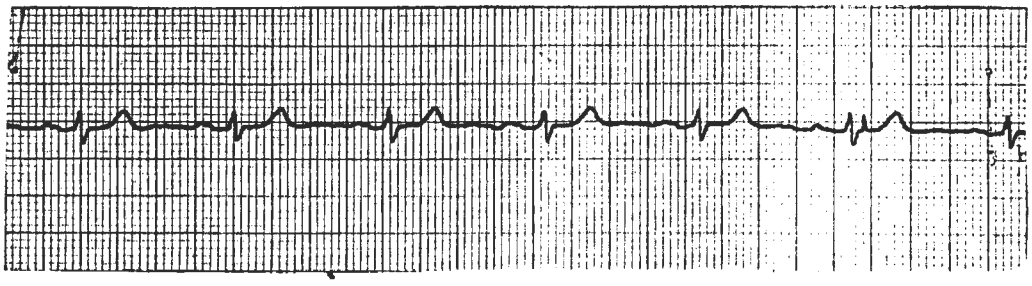
Patient 18 Monitor lead II Medication _____



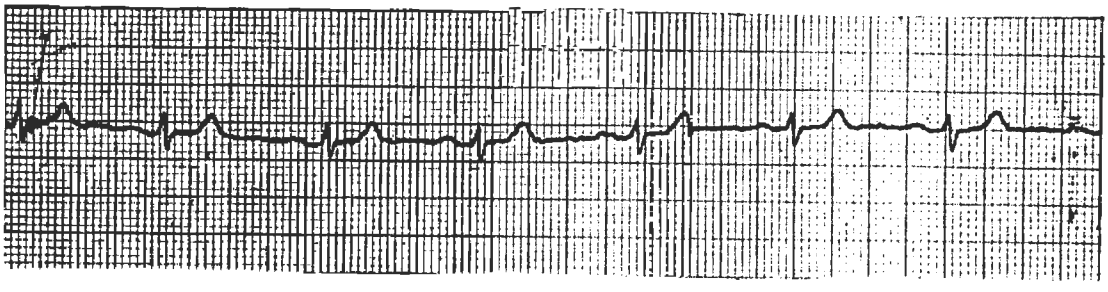
Baseline



1 minute

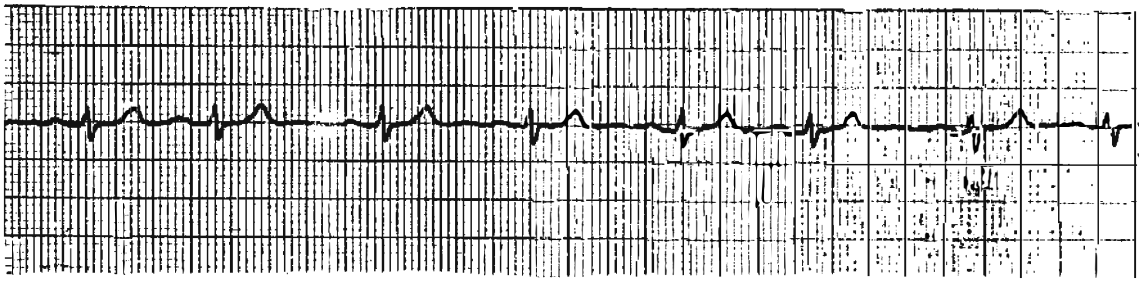


2 minutes

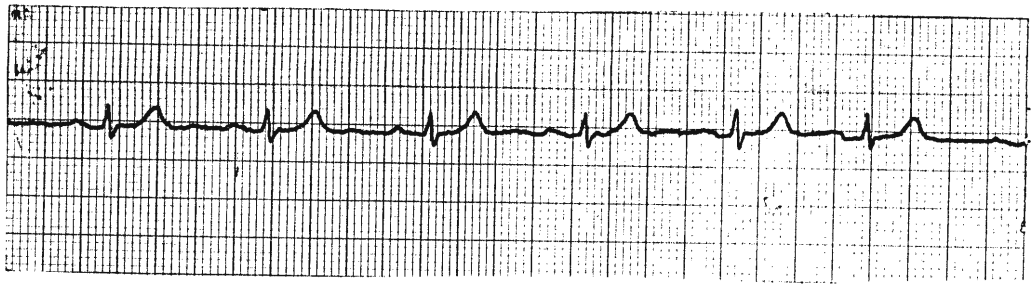


3 minutes

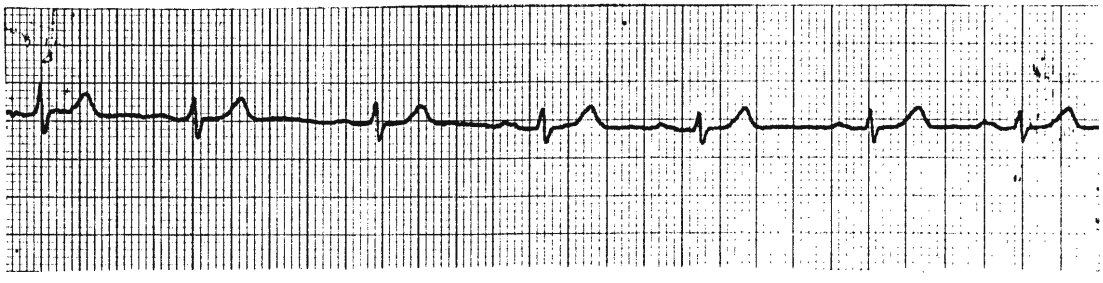
Patient 18



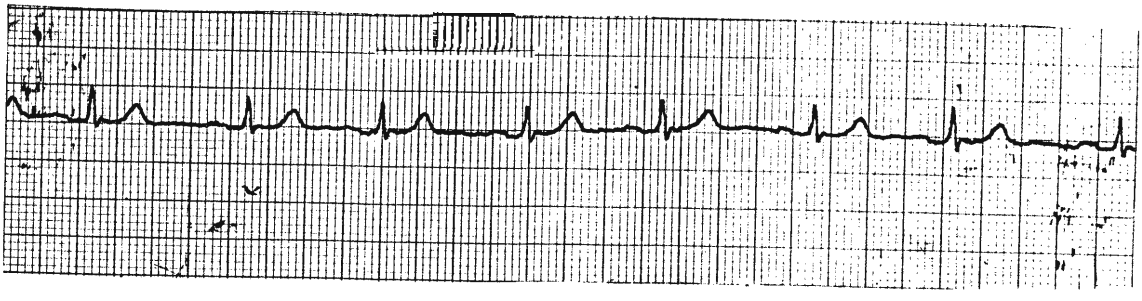
4 minutes



5 minutes

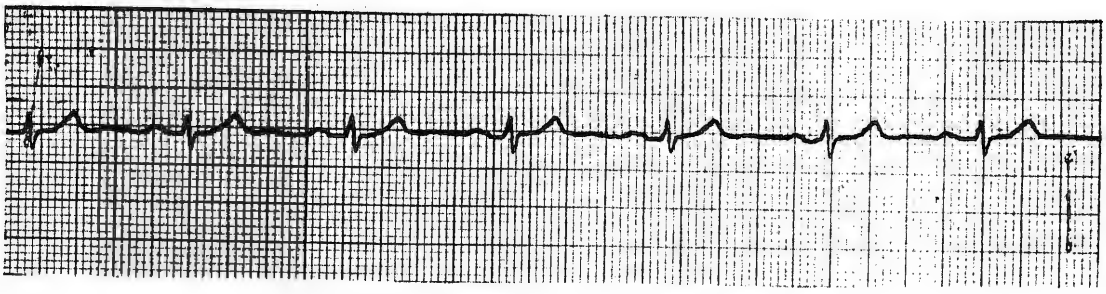


10 minutes



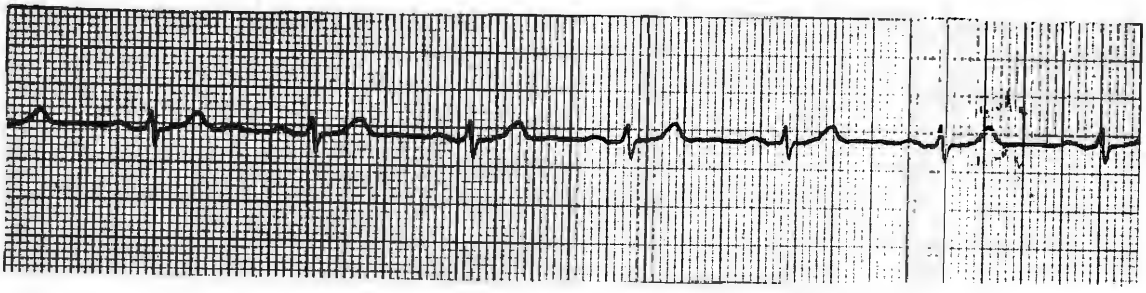
15 minutes

Patient 18

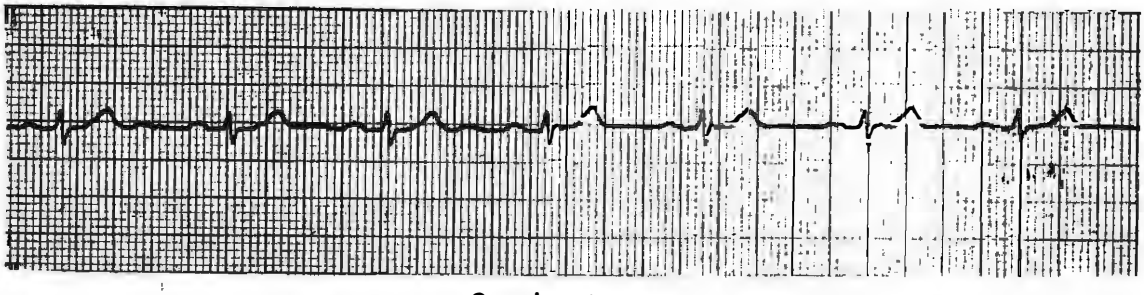


20 minutes

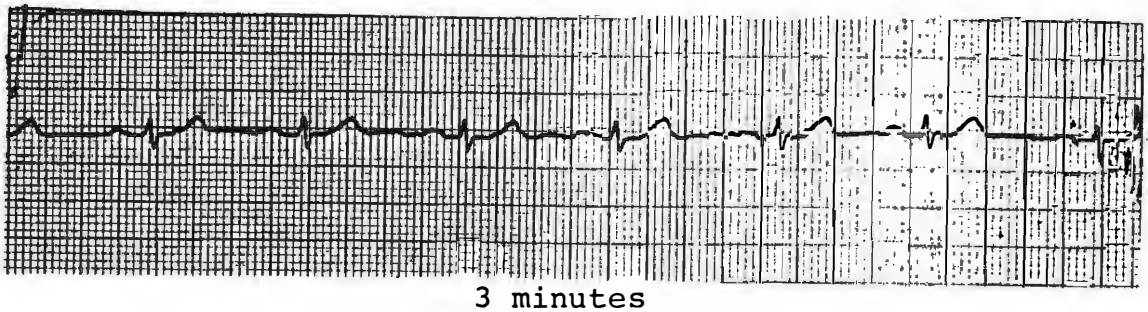
Bath Completed



1 minute

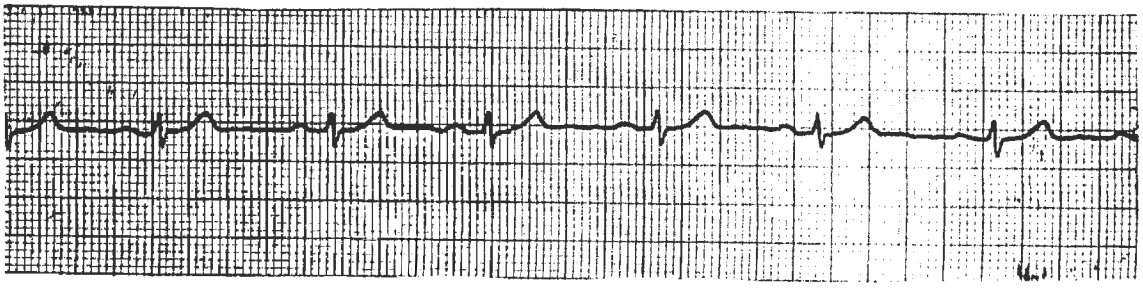


2 minutes

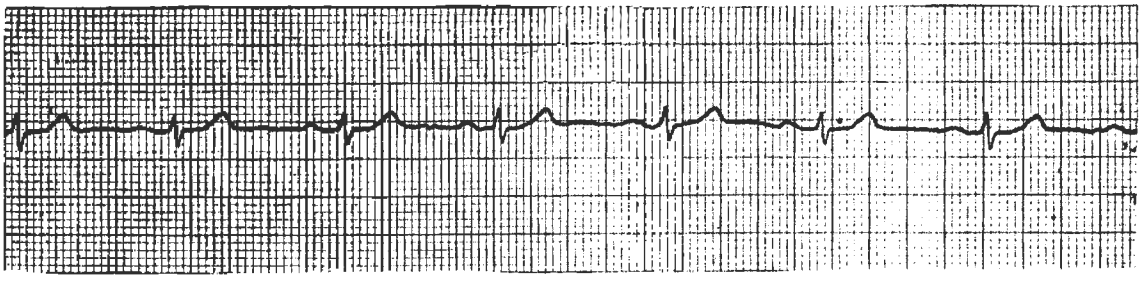


3 minutes

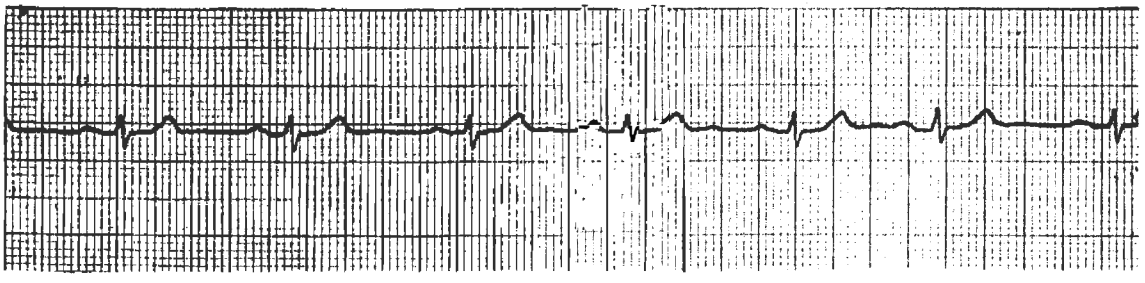
Patient 18



4 minutes



5 minutes



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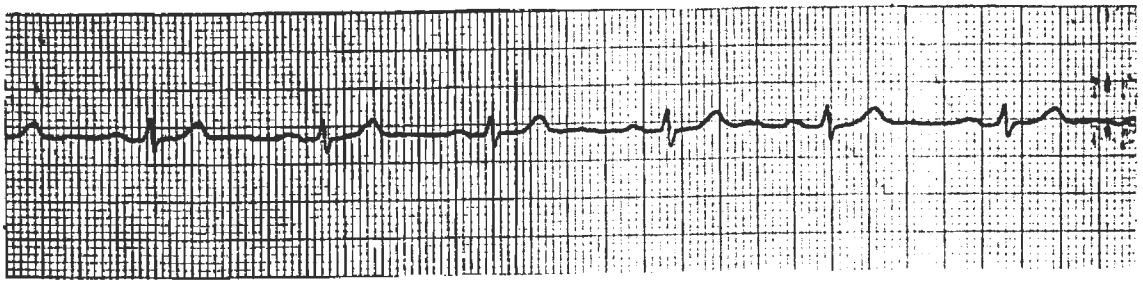
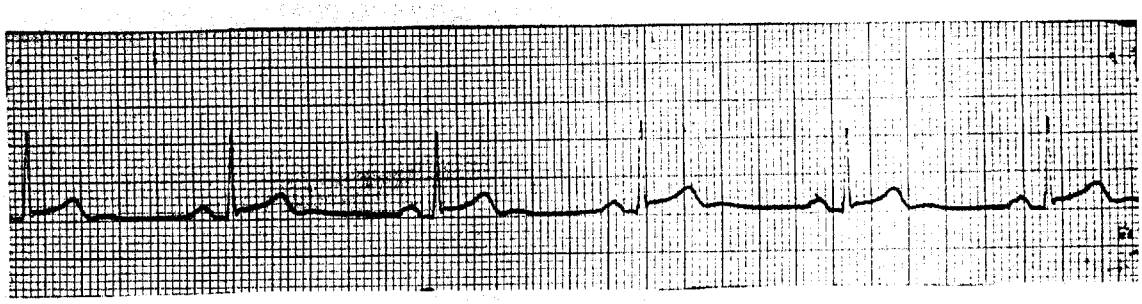
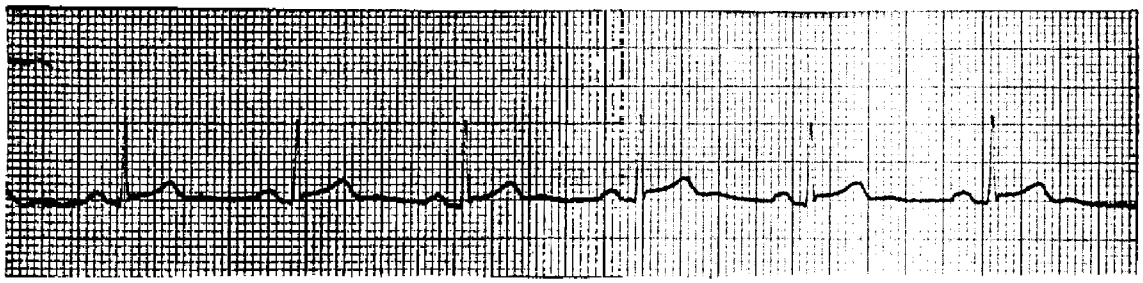


15 minutes

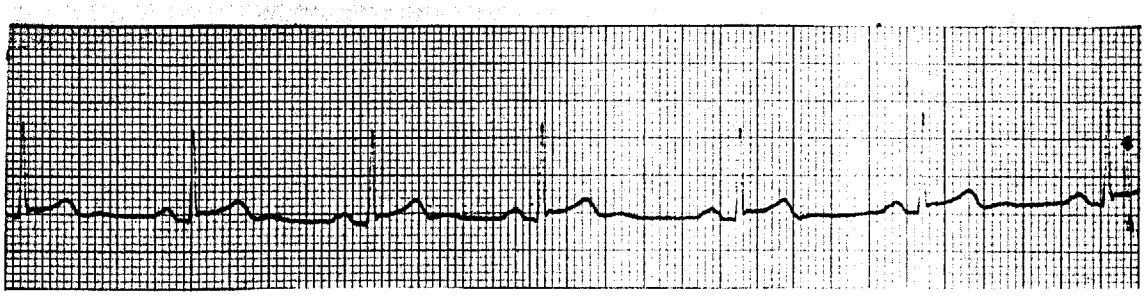
Patient 19 Monitor lead II Medication _____



Baseline



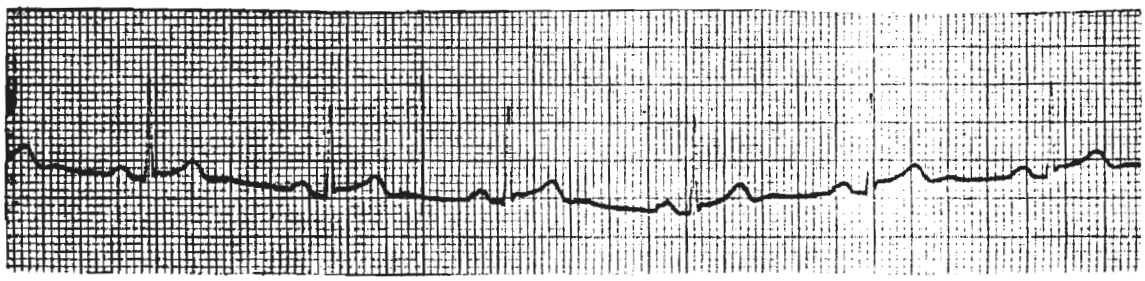
1 minute



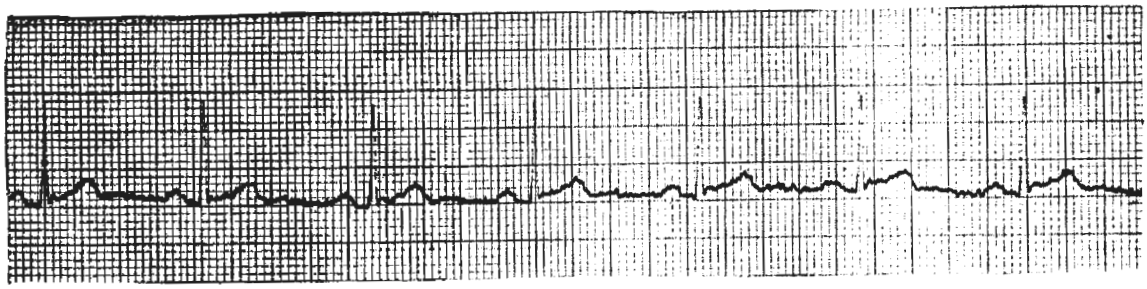
2 minutes

3 minutes

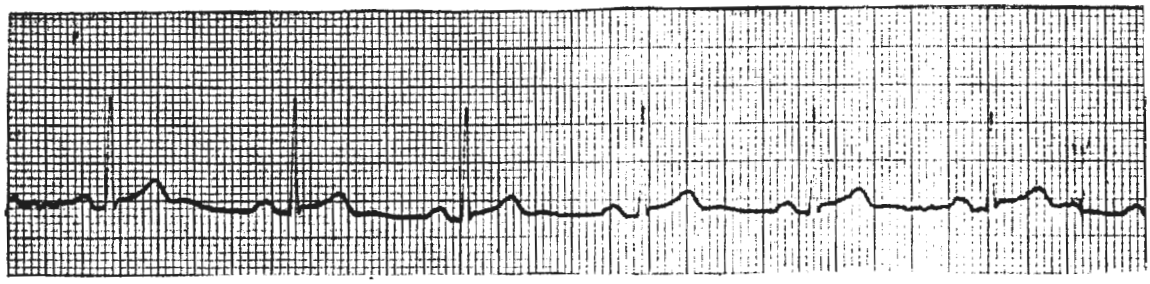
Patient 19



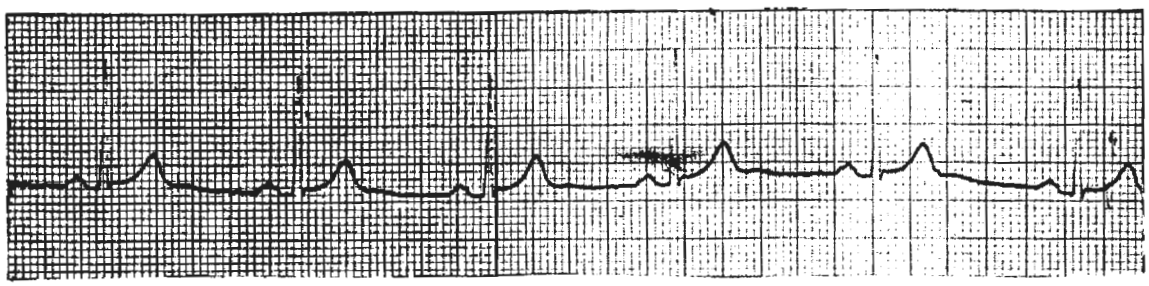
4 minutes



5 minutes

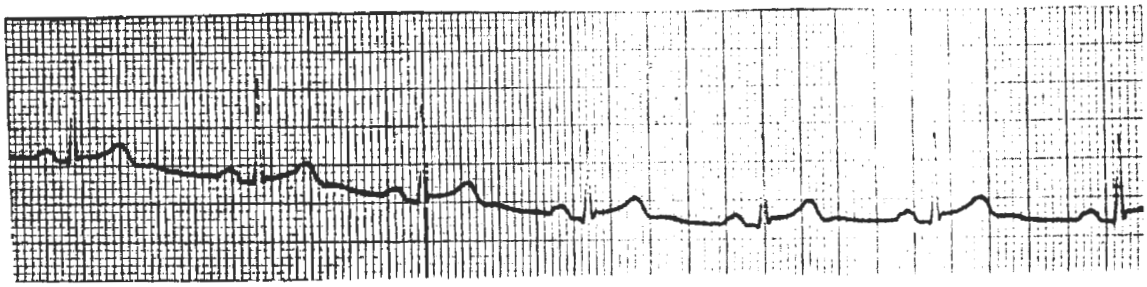


10 minutes



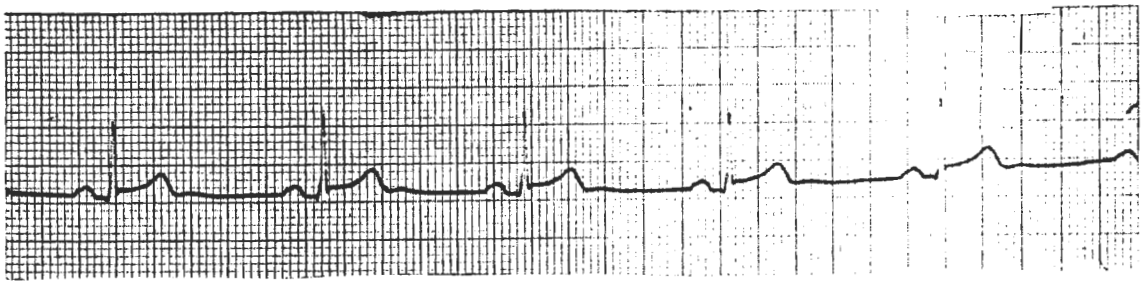
15 minutes

Patient 19



20 minutes

Bath Completed



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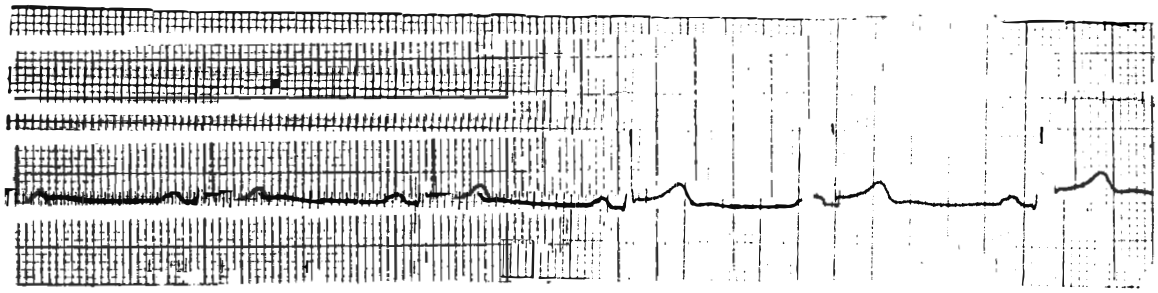
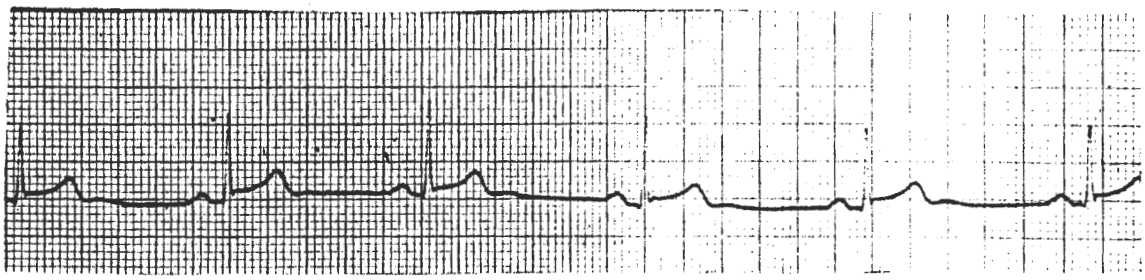
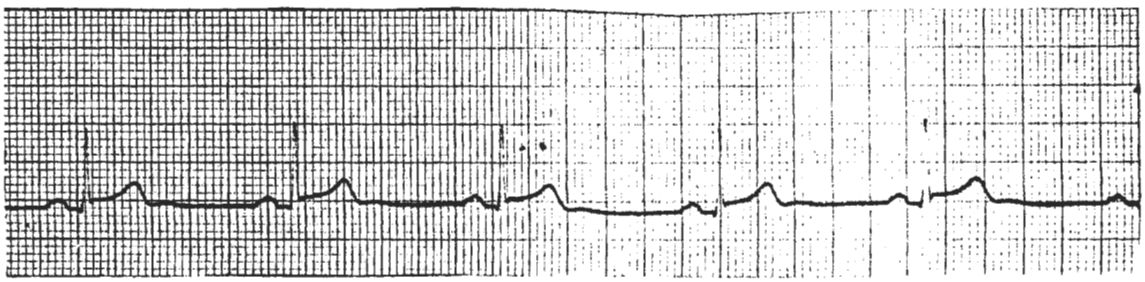


2 minutes

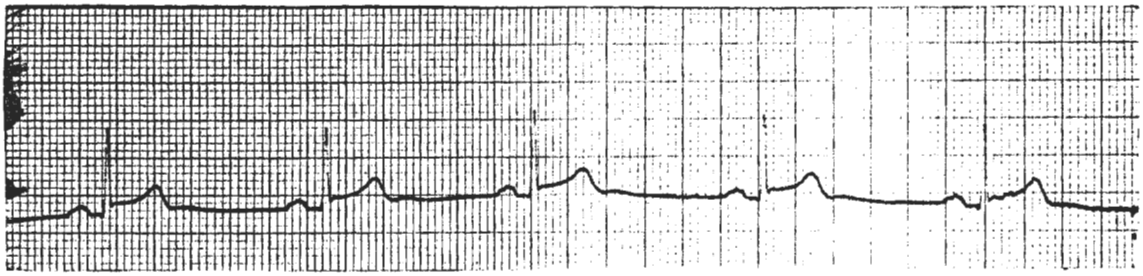


3 minutes

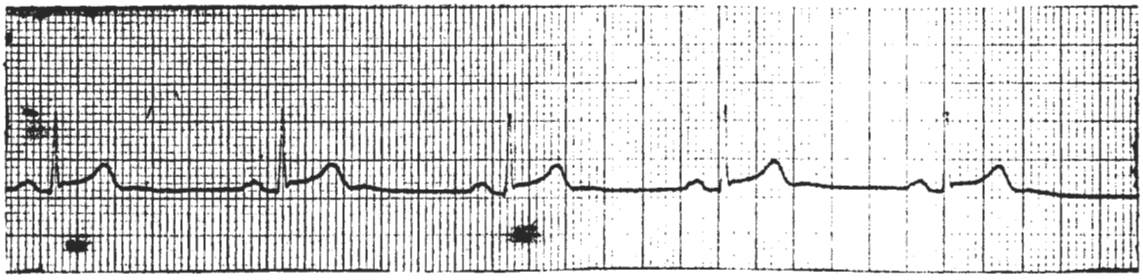
Patient 19



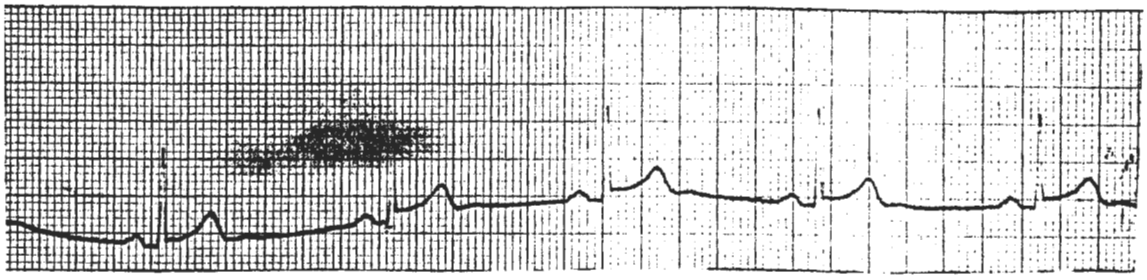
4 minutes



5 minutes

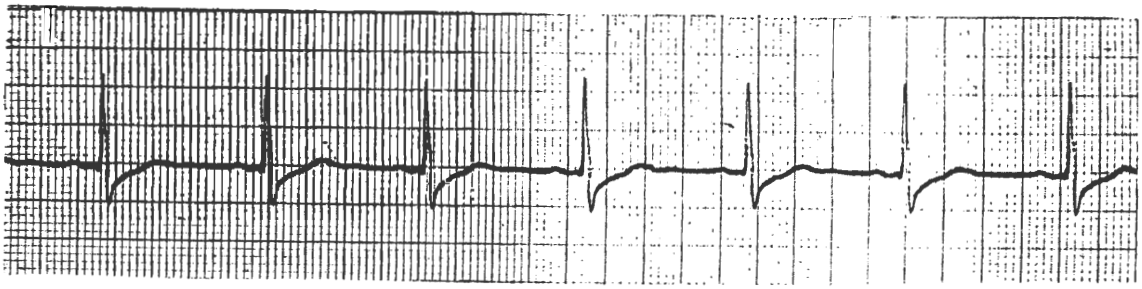


10 minutes

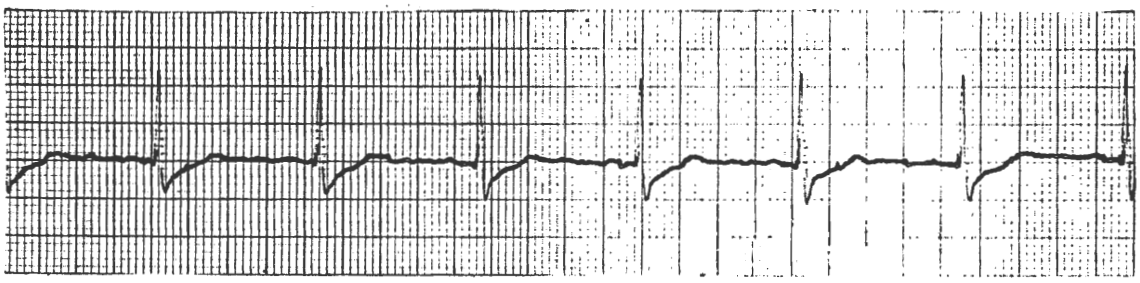


15 minutes

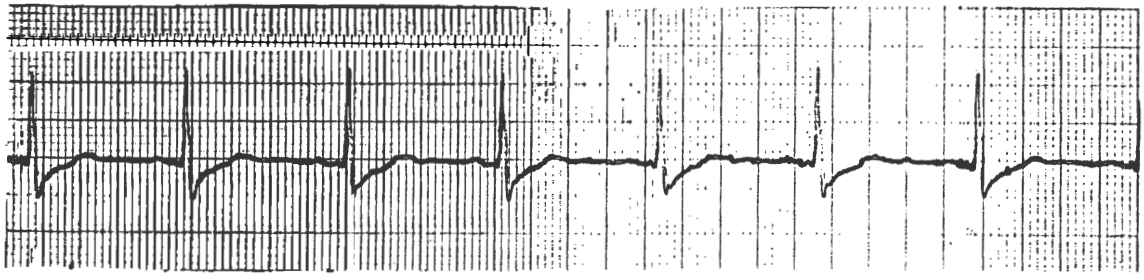
Patient 20 Monitor lead II Medication _____



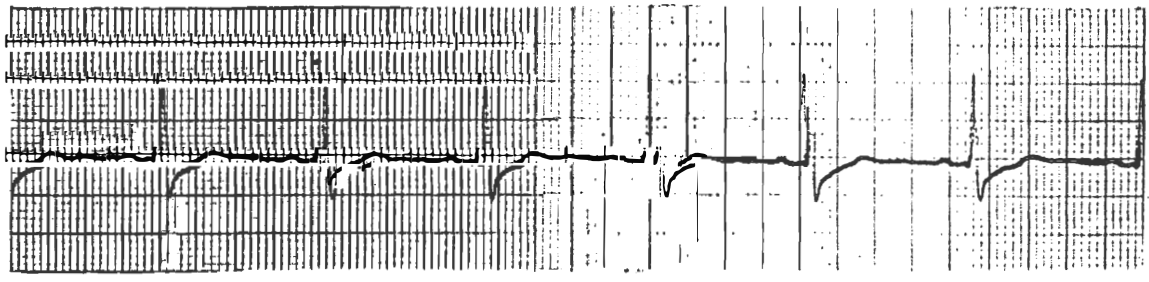
Baseline



1 minute

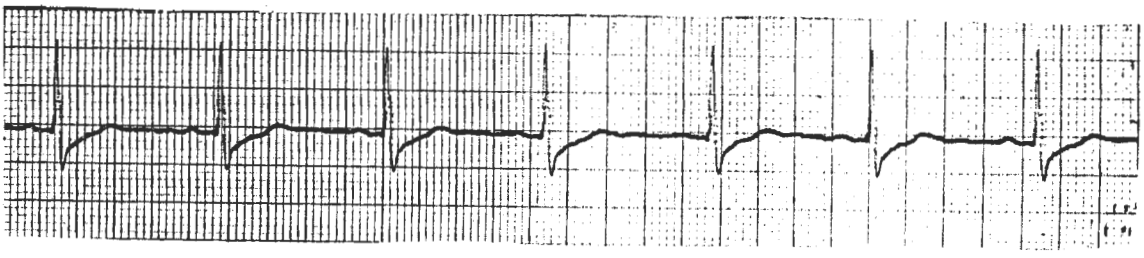


2 minutes

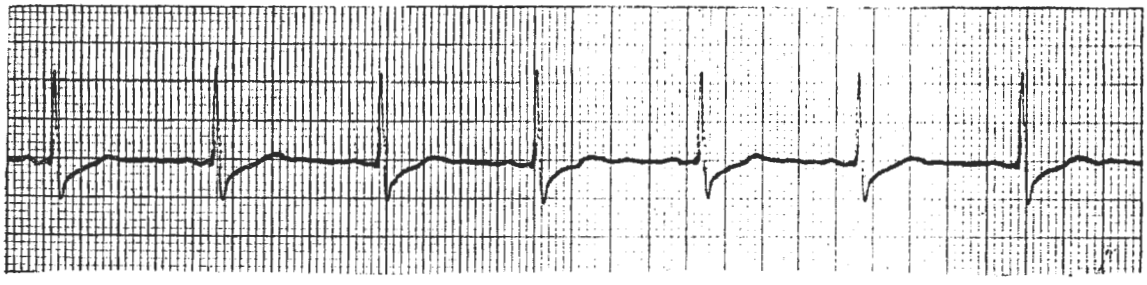


3 minutes

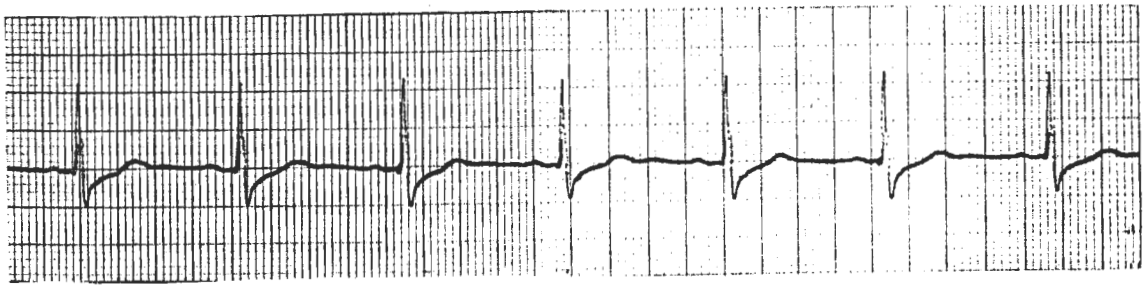
Patient 20



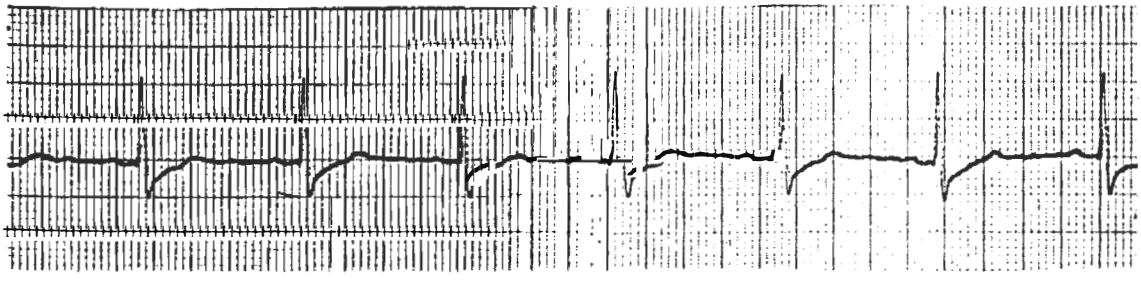
4 minutes



5 minutes

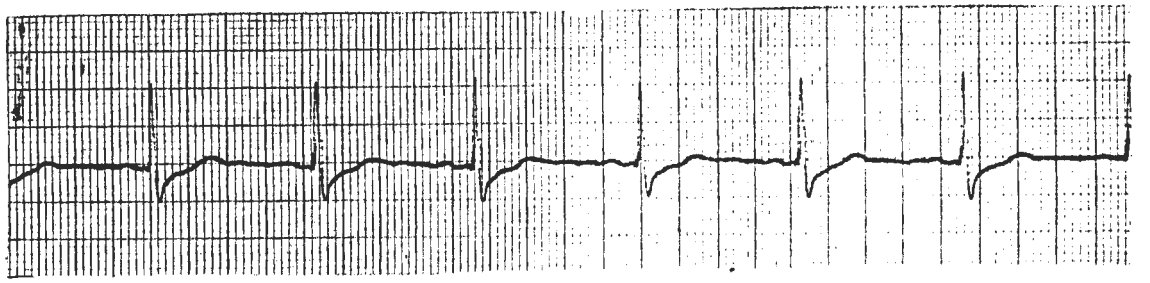


10 minutes



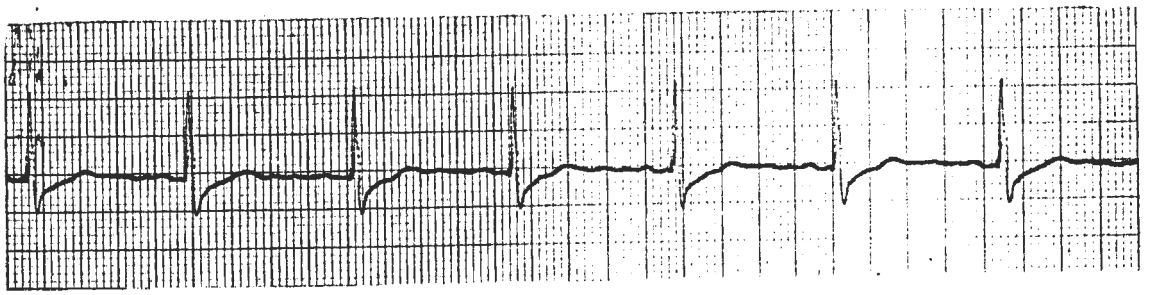
15 minutes

Patient 20

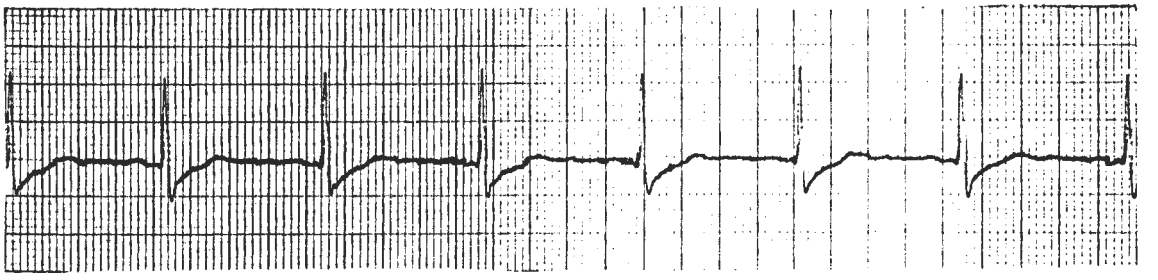


20 minutes

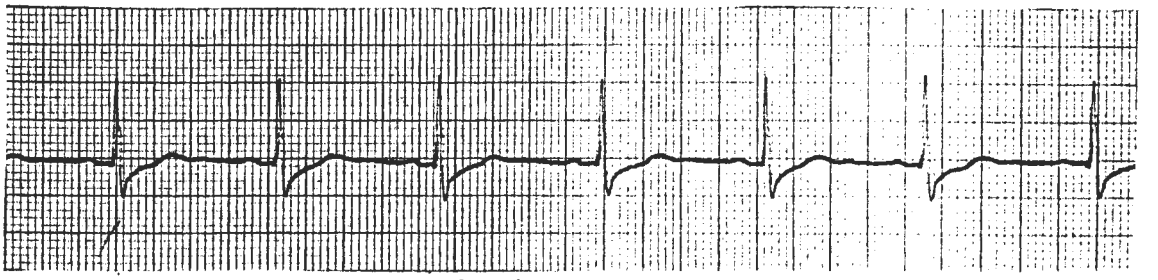
Bath Completed



1 minute

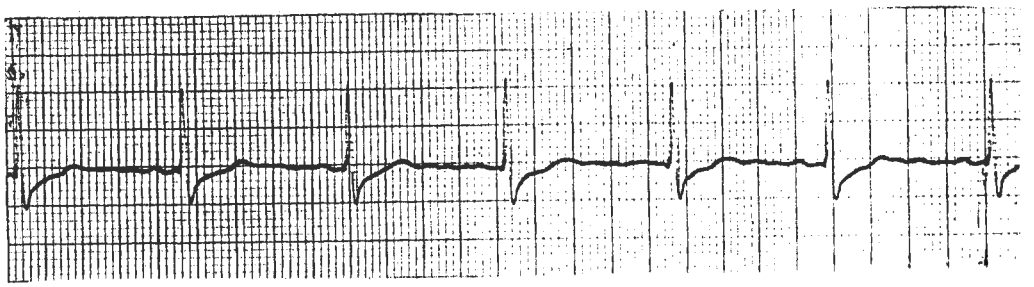


2 minutes

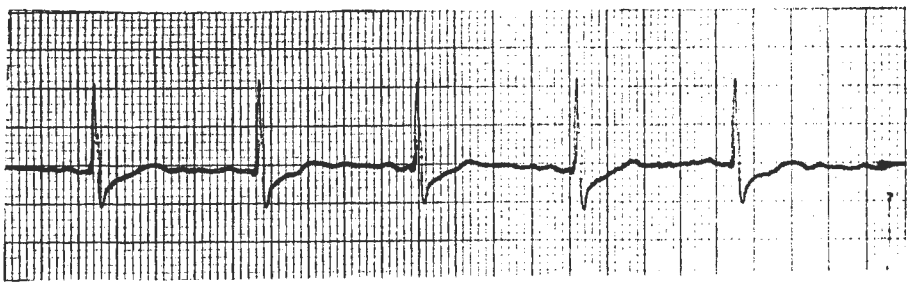


3 minutes

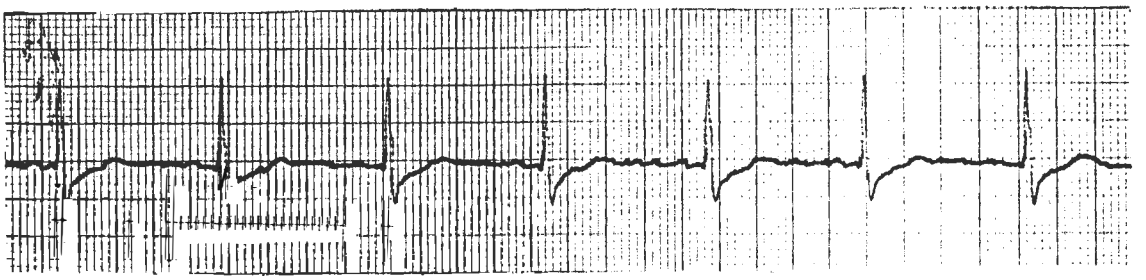
Patient 20



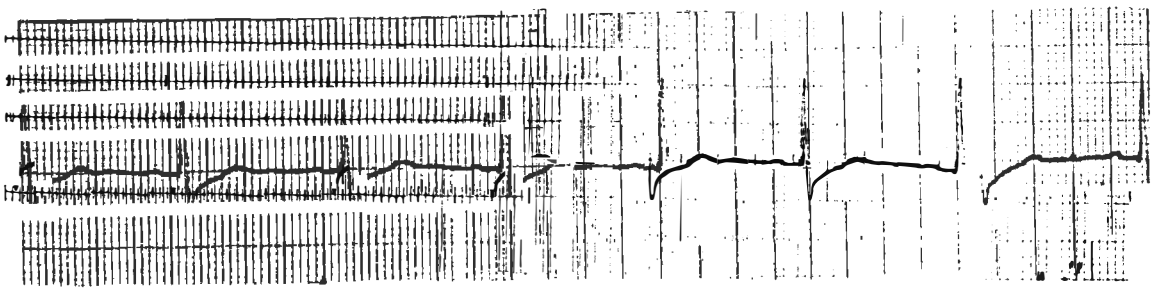
4 minutes



5 minutes

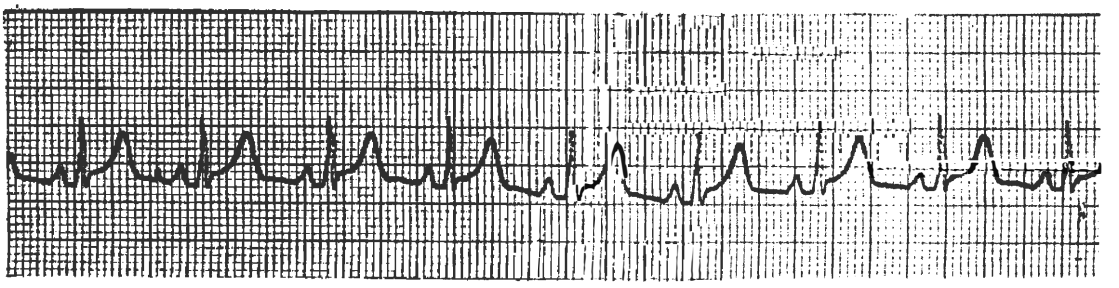


10 minutes

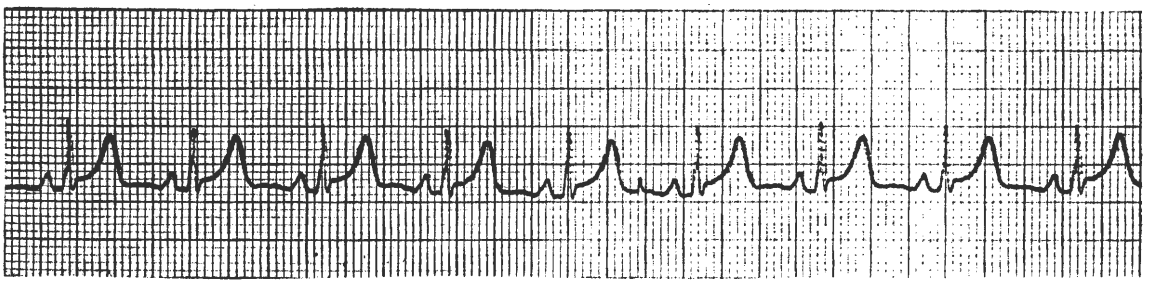


15 minutes

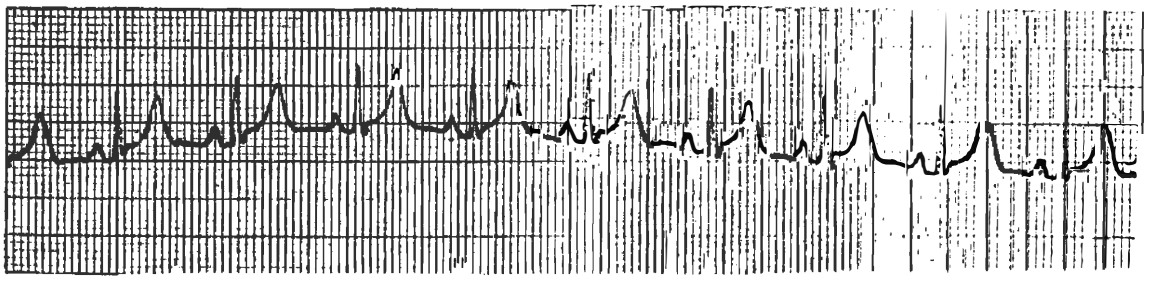
Patient 21 Monitor lead II Medication _____



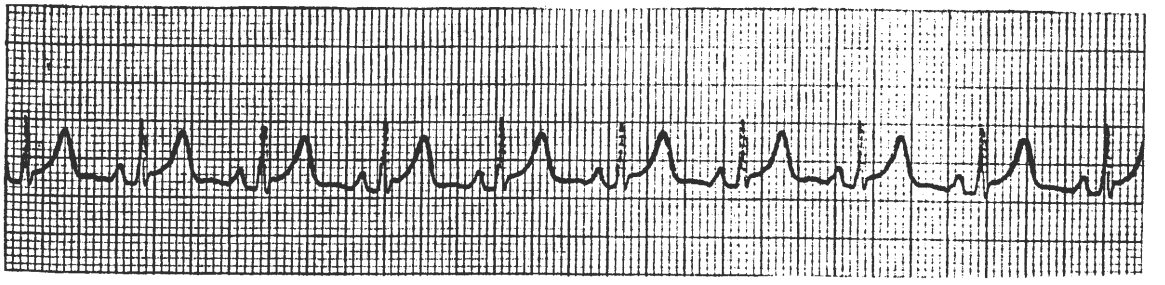
Baseline



1 minute

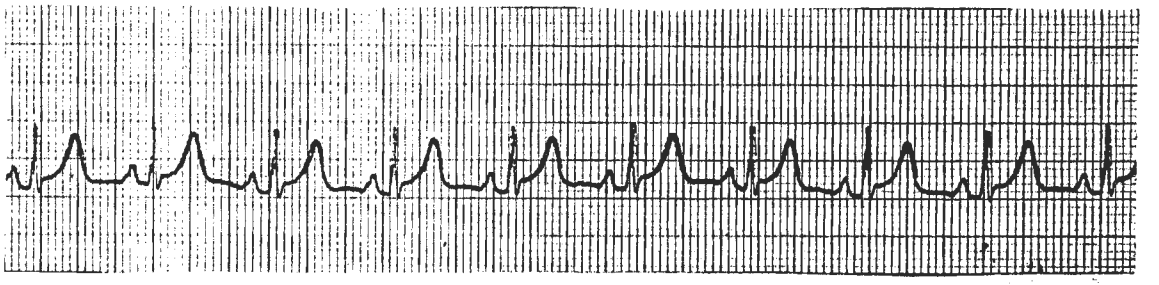


2 minutes

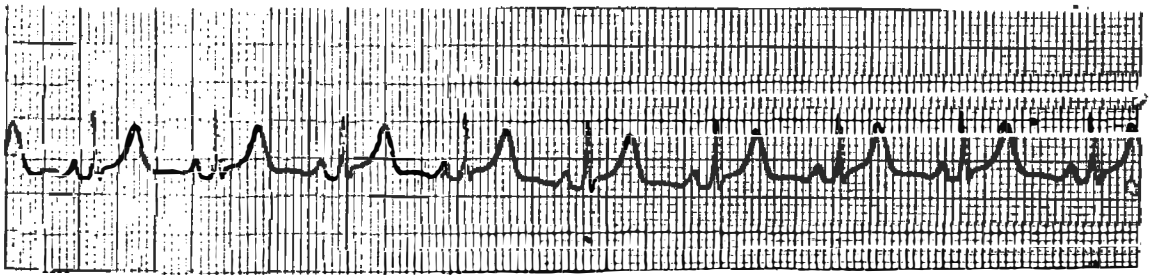


3 minutes

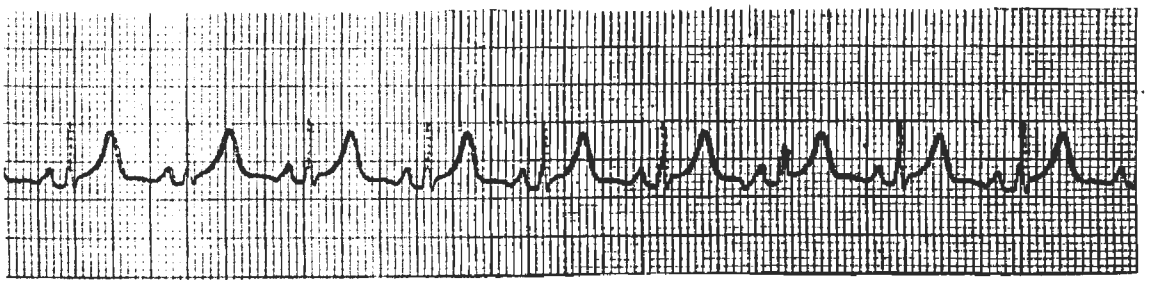
Patient 21



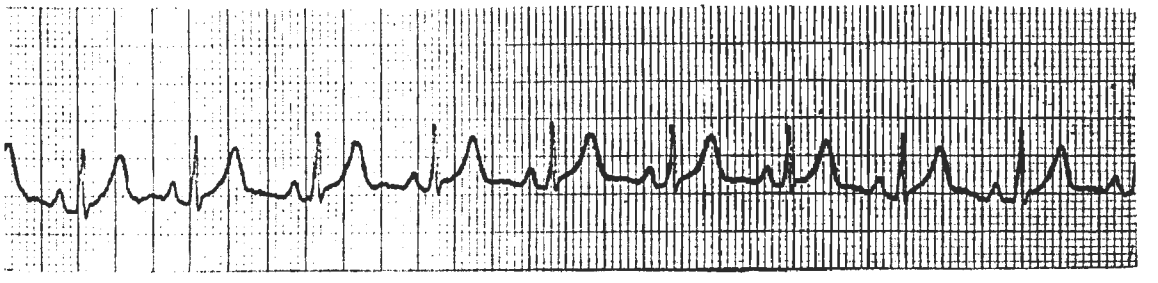
4 minutes



5 minutes

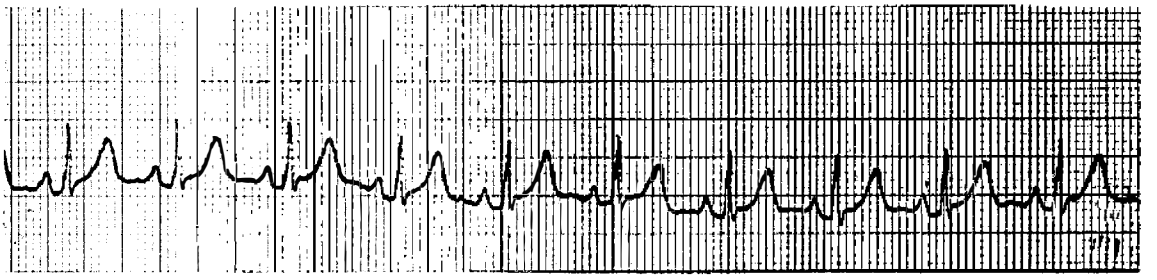


10 minutes



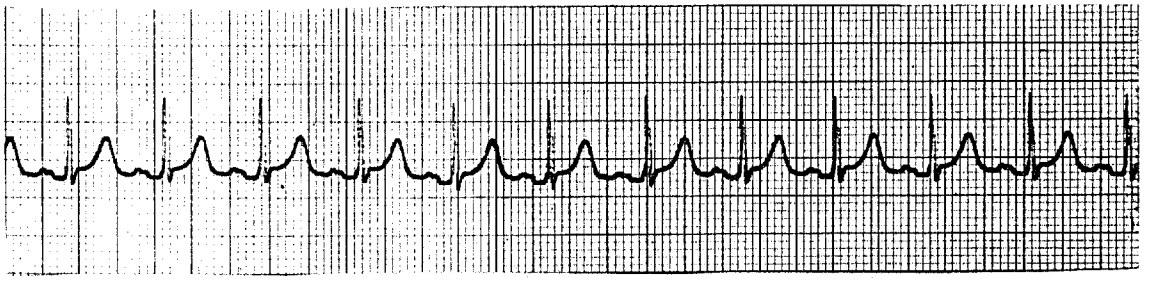
15 minutes

Patient 21

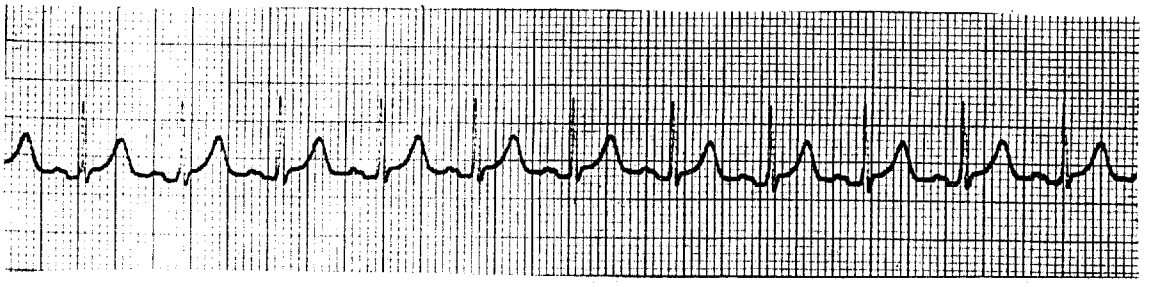


20 minutes

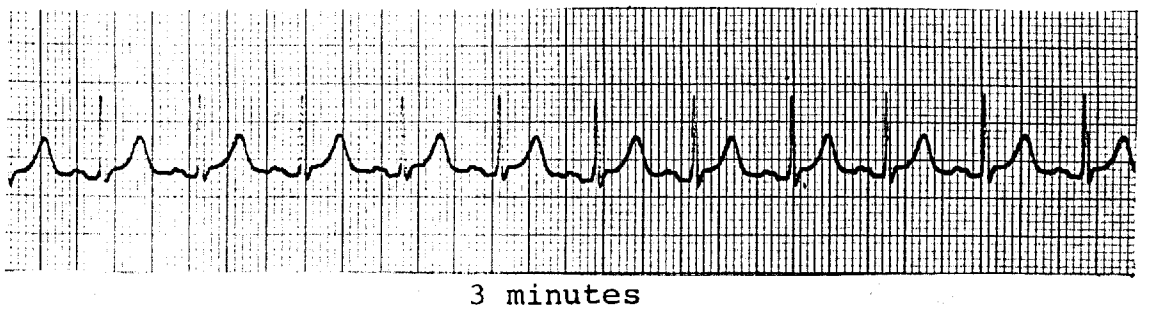
Bath Completed



1 minute

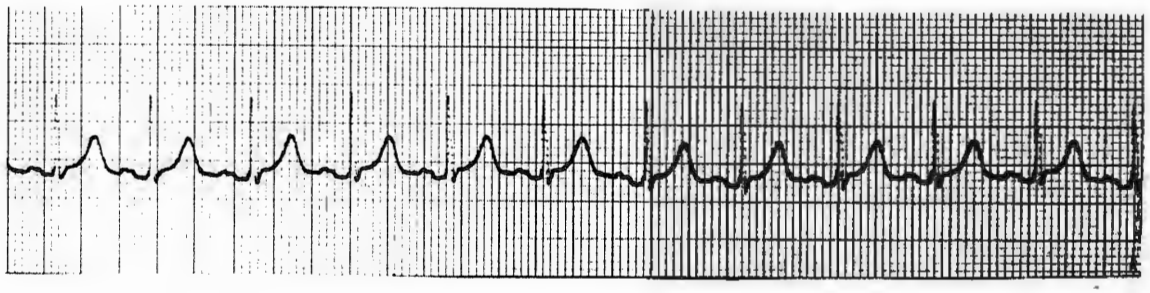


2 minutes

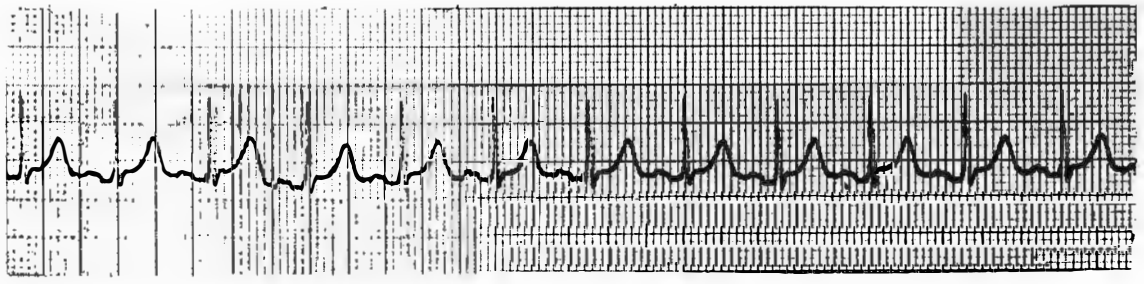


3 minutes

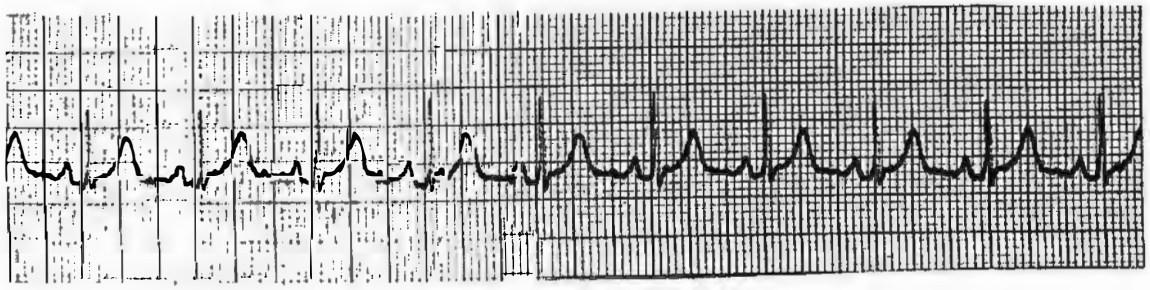
Patient 21



4 minutes



5 minutes



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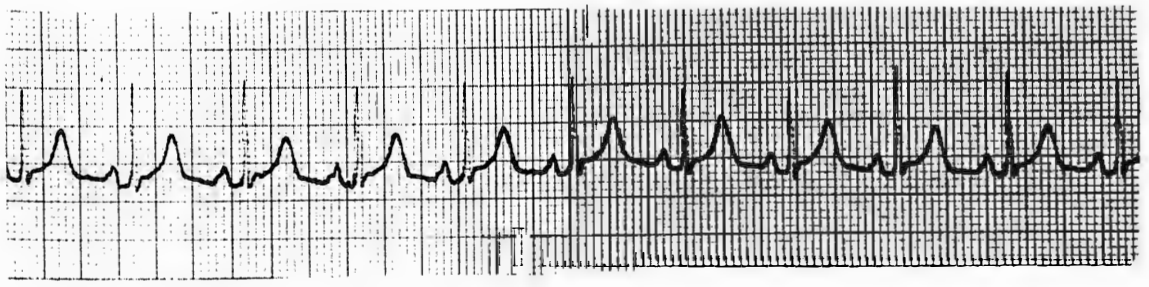
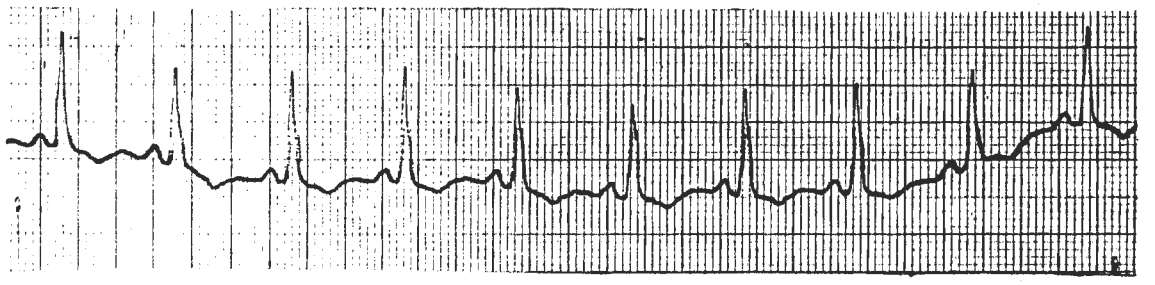
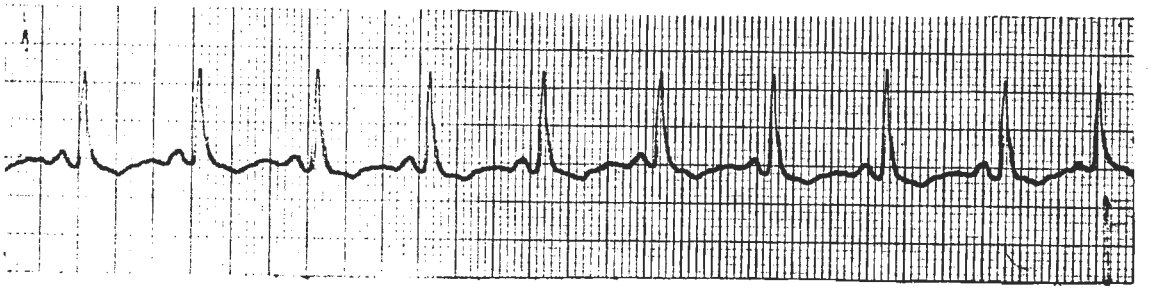


15 minutes

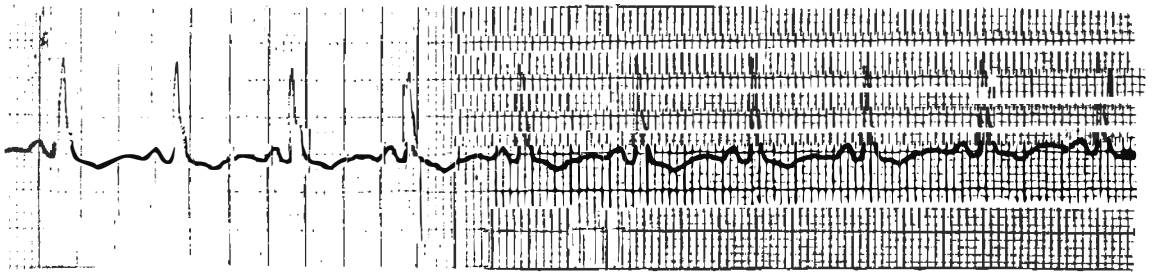
Patient 22 Monitor lead II Medication Lanoxin



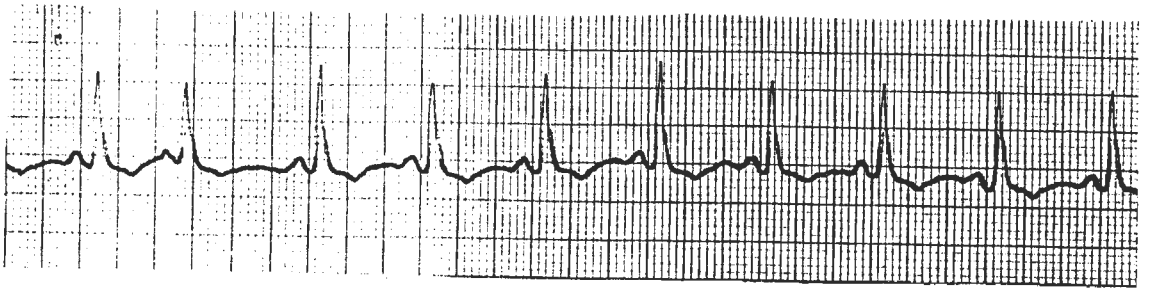
Baseline



1 minute

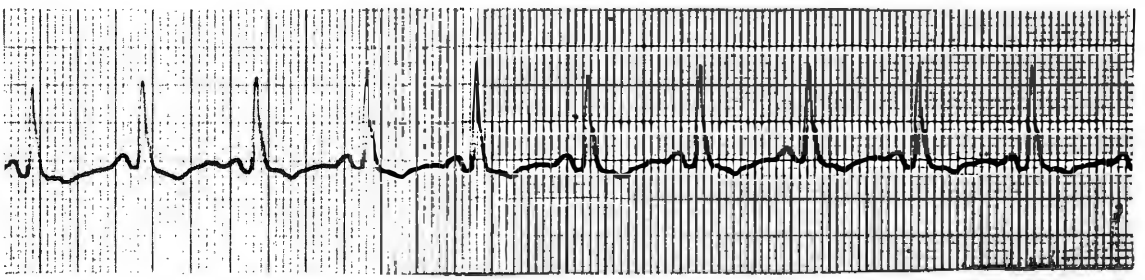


2 minutes

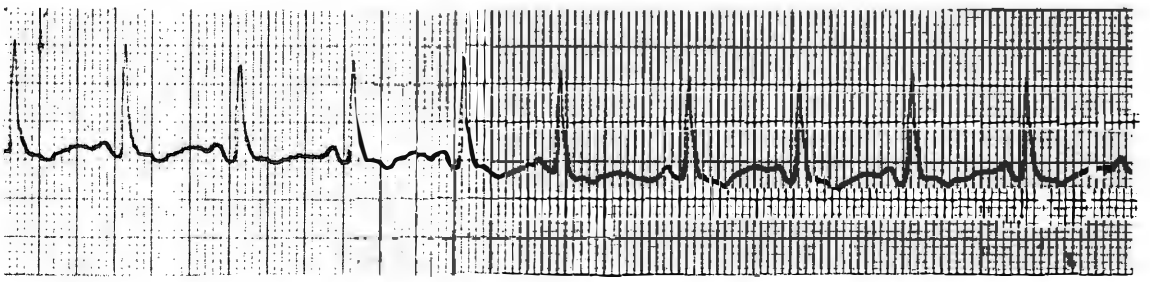


3 minutes

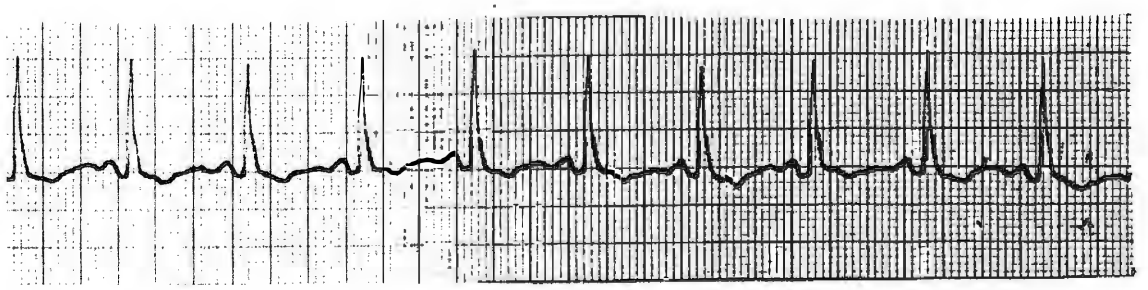
Patient 22



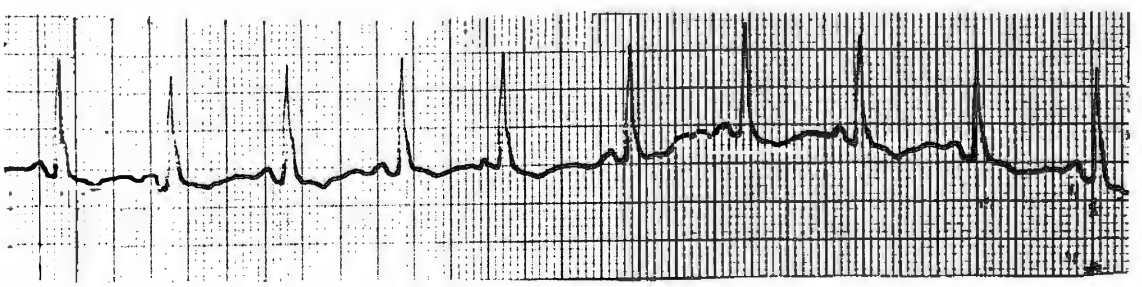
4 minutes



5 minutes

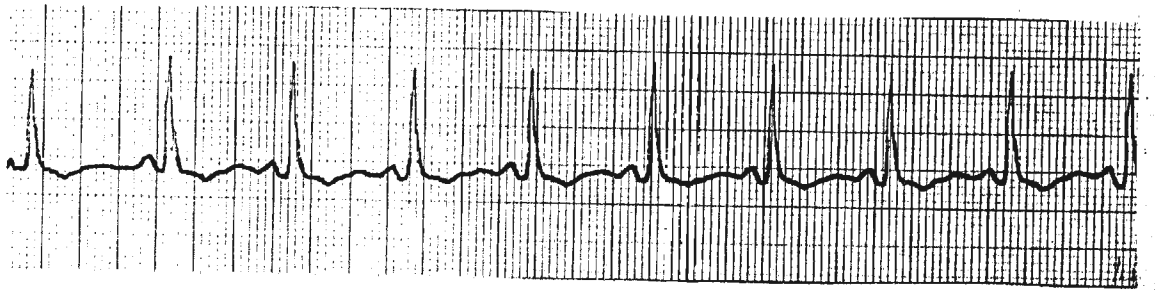


10 minutes



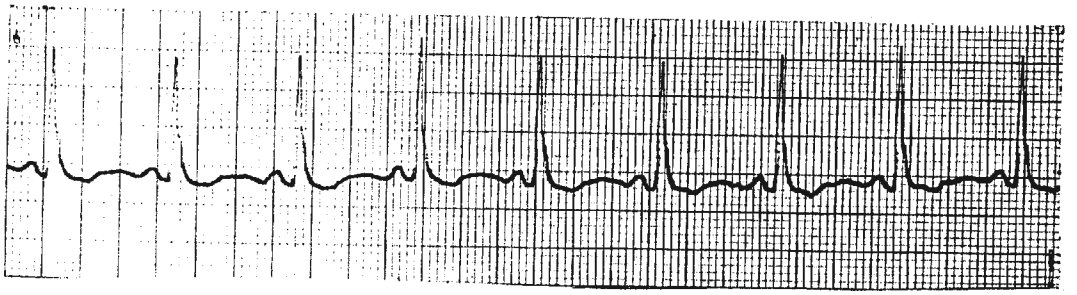
15 minutes

Patient 22

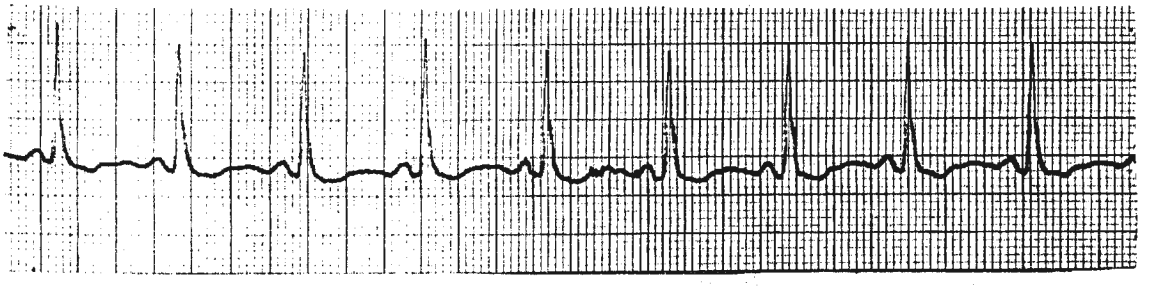


20 minutes

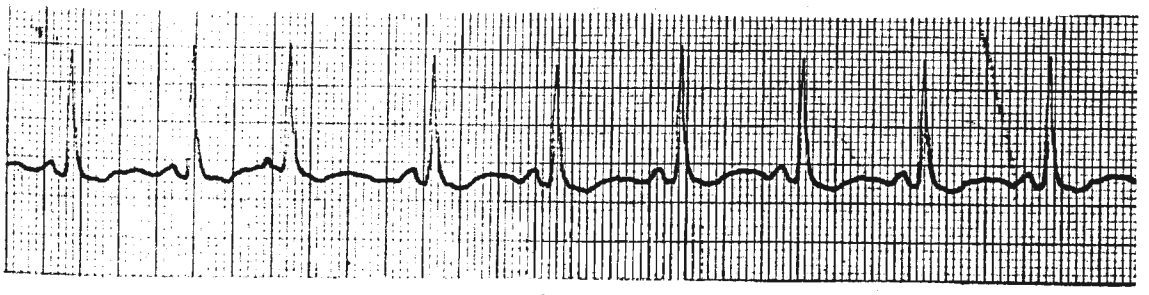
Bath Completed



1 minute

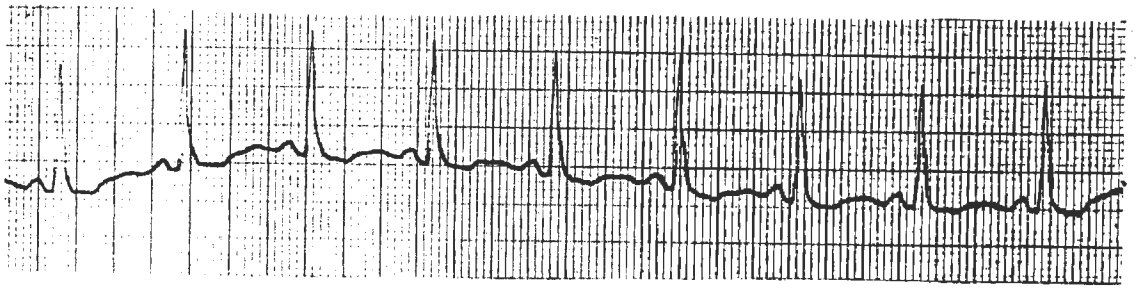


2 minutes

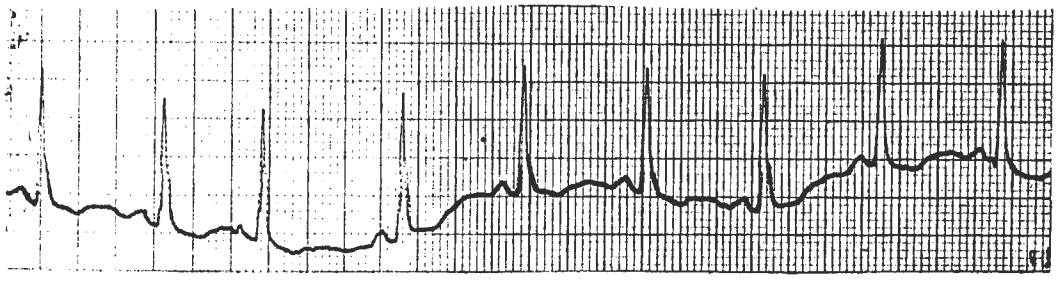


3 minutes

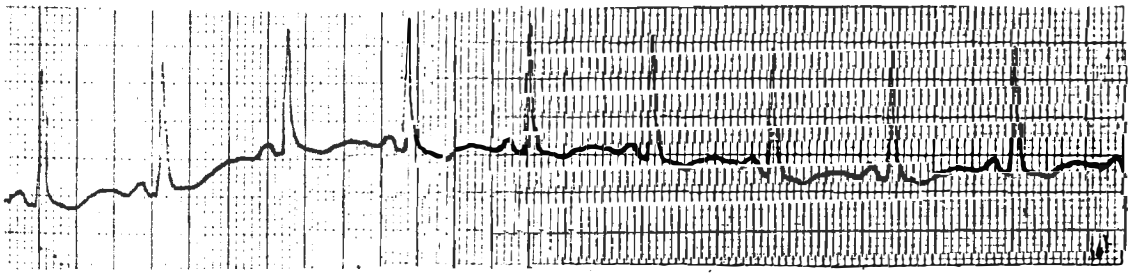
Patient 22



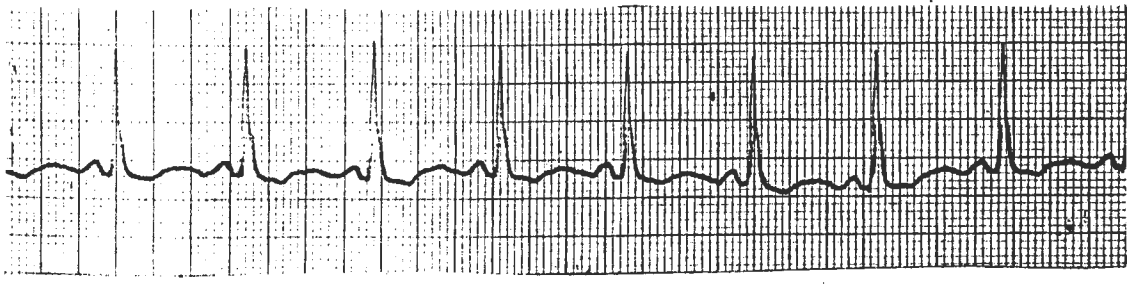
4 minutes



5 minutes

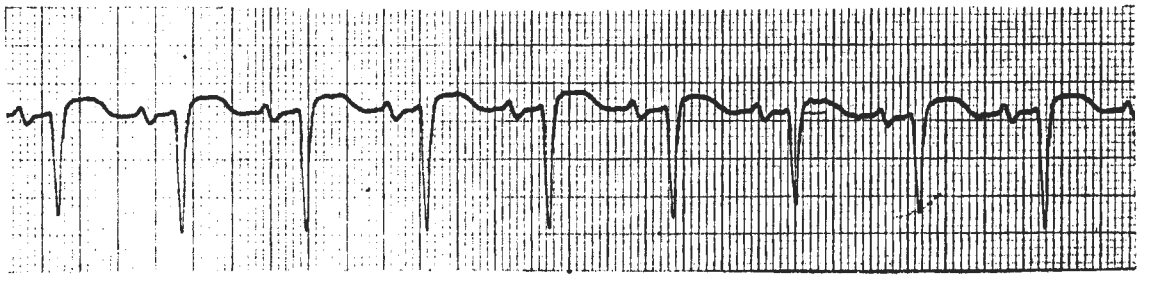


10 minutes

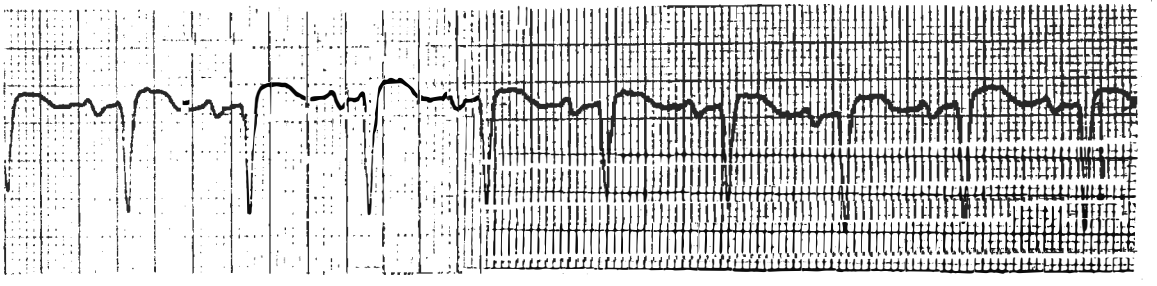


15 minutes

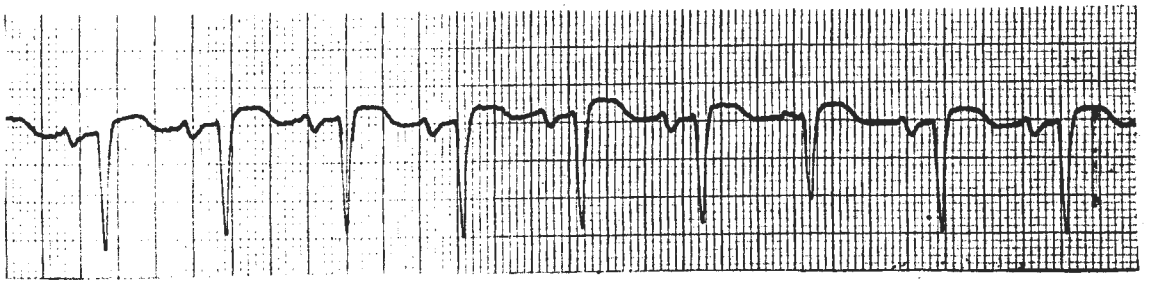
Patient 23 Monitor lead V_1 Medication _____



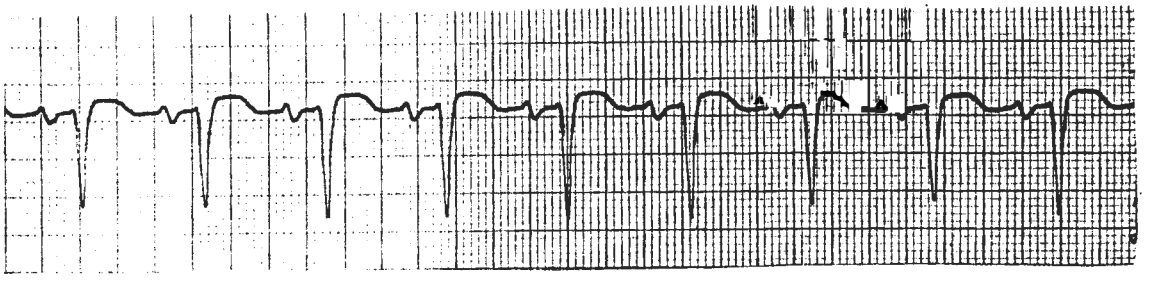
Baseline



1 minute

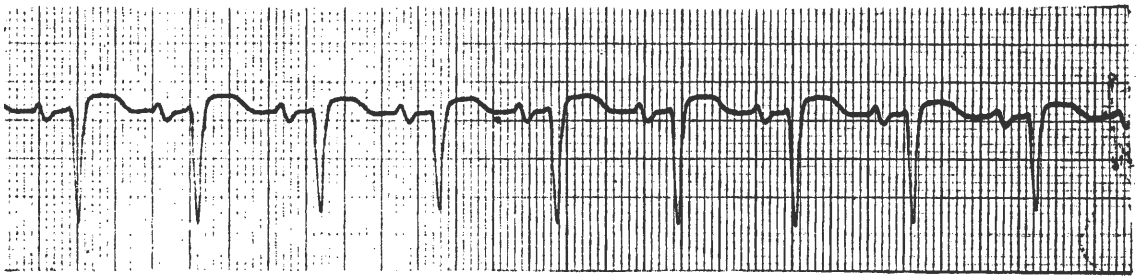


2 minutes

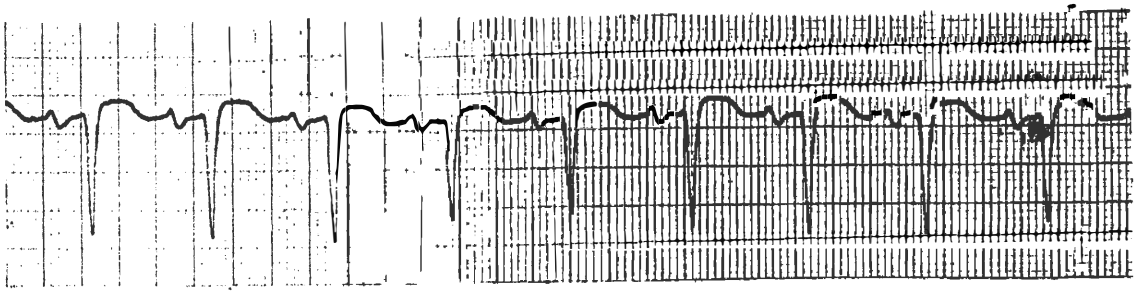


3 minutes

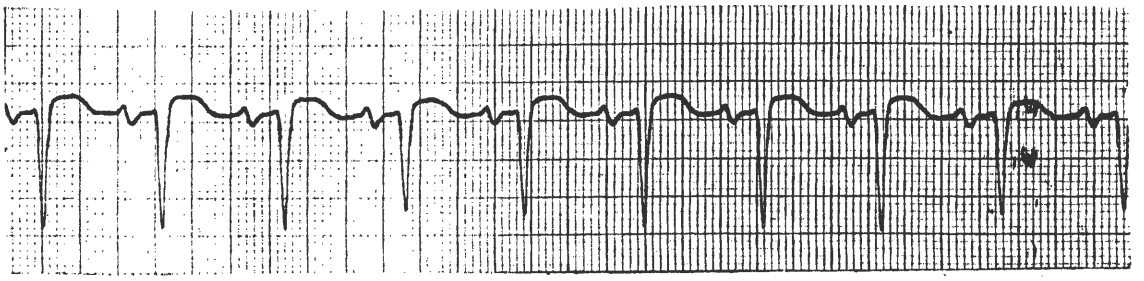
Patient 23



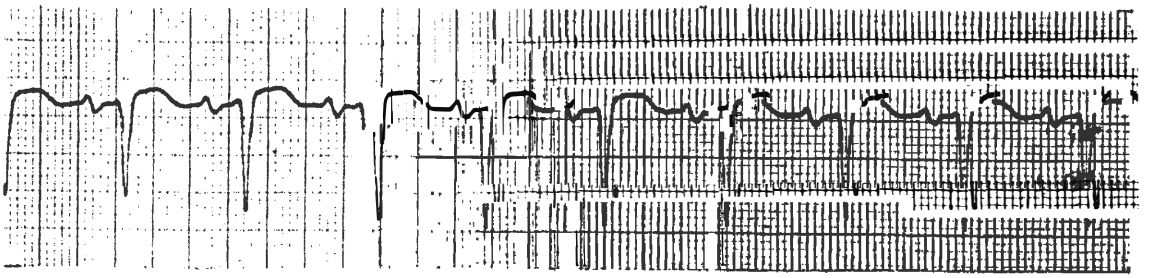
4 minutes



5 minutes

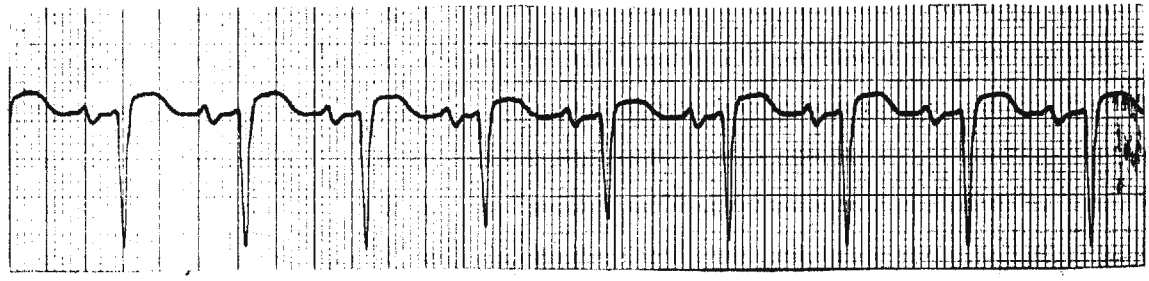


10 minutes



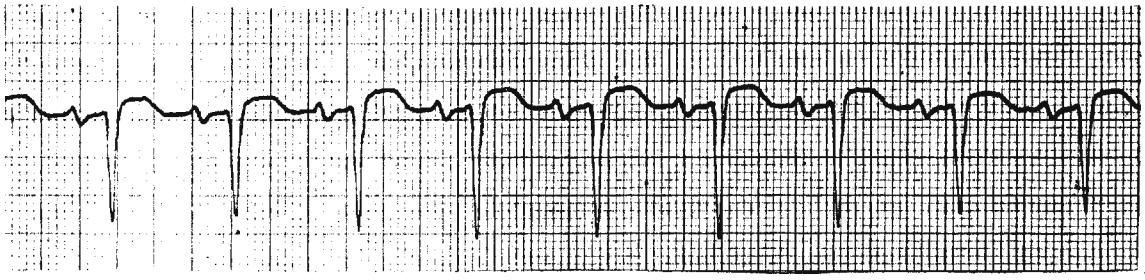
15 minutes

Patient 23

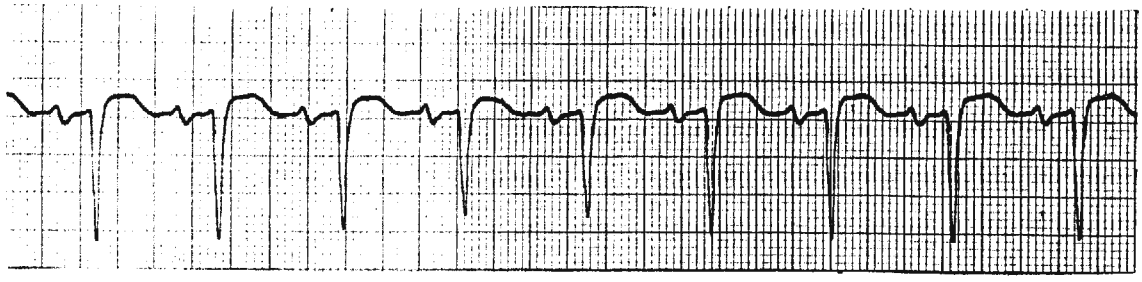


20 minutes

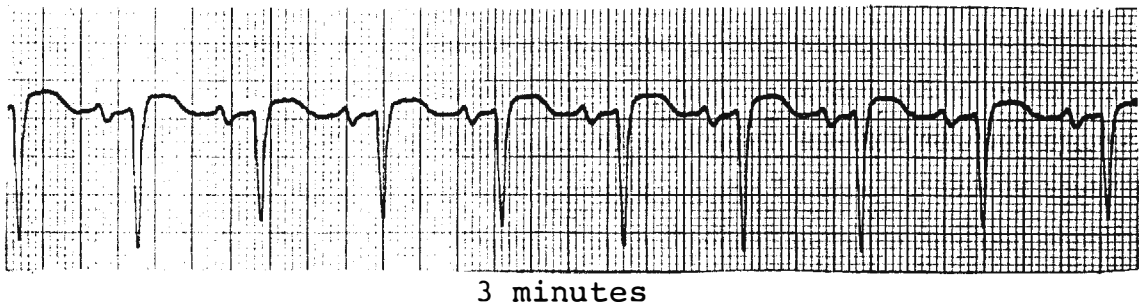
Bath Completed



1 minute

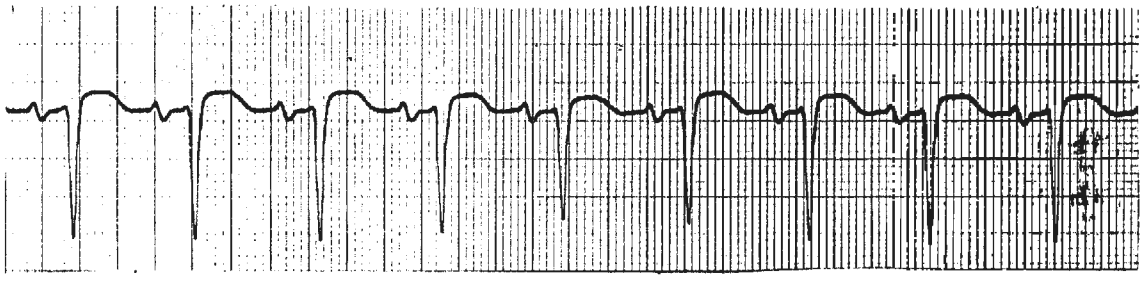


2 minutes

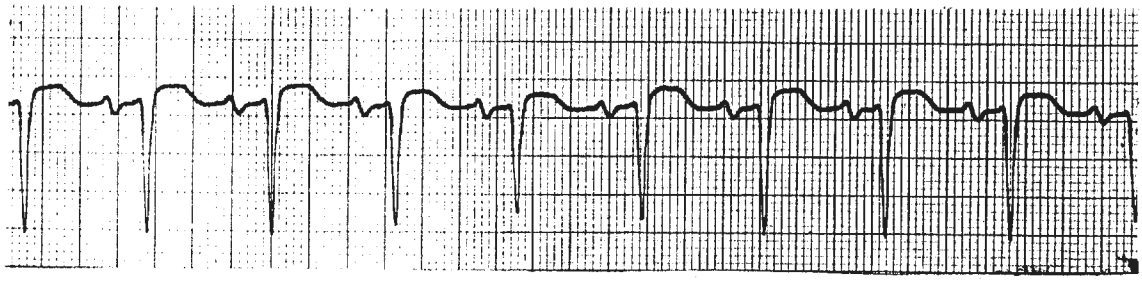


3 minutes

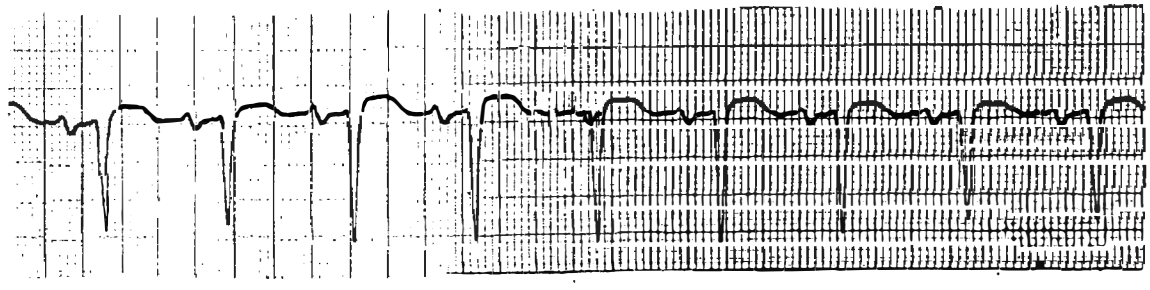
Patient 23



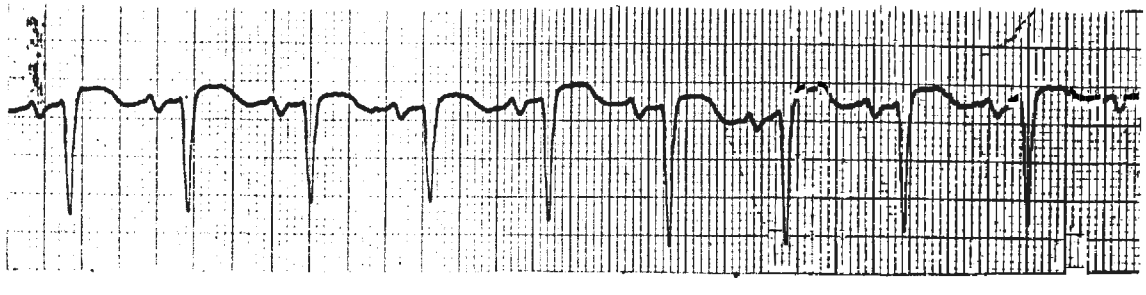
4 minutes



5 minutes

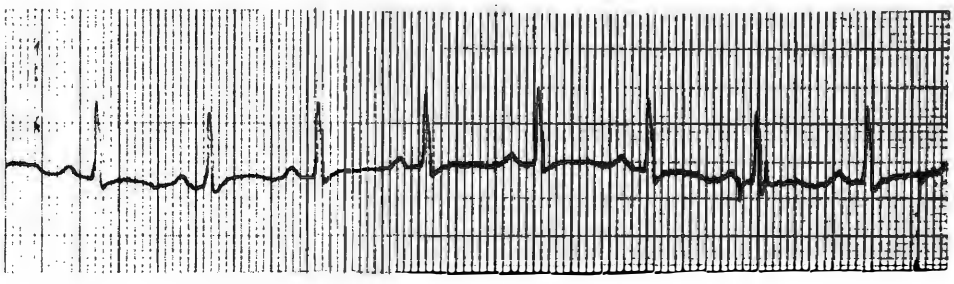


10 minutes

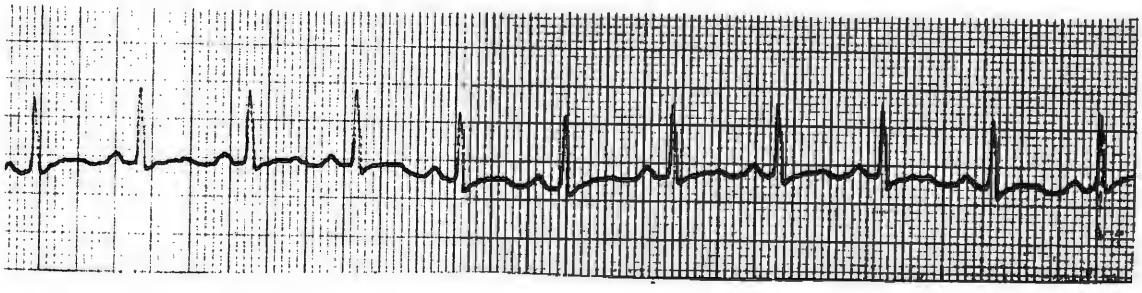


15 minutes

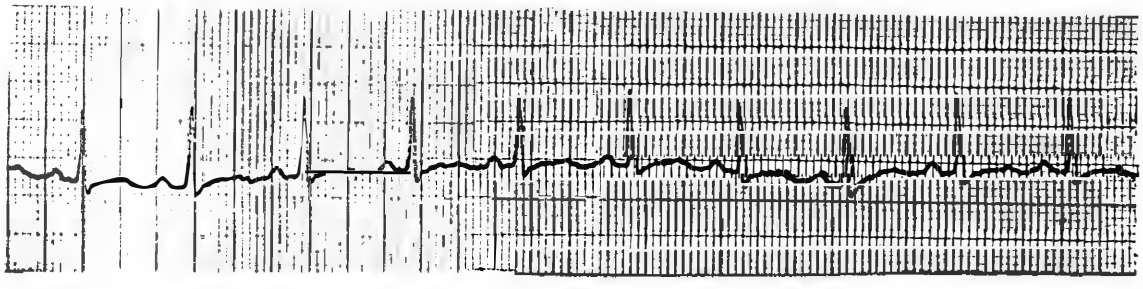
Patient 24 Monitor lead II Medication _____



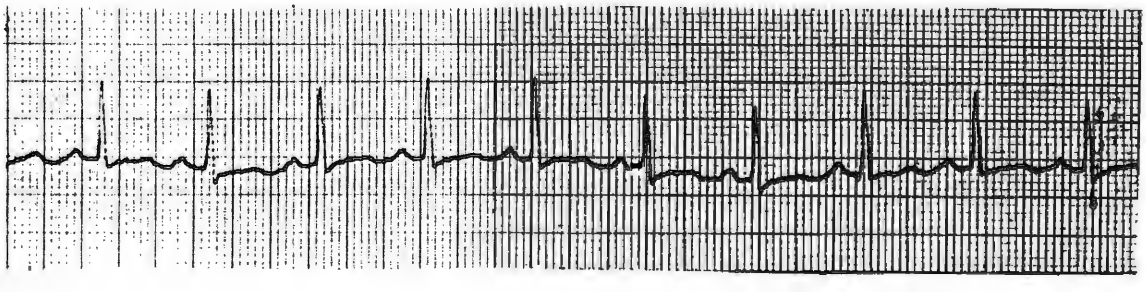
Baseline



1 minute

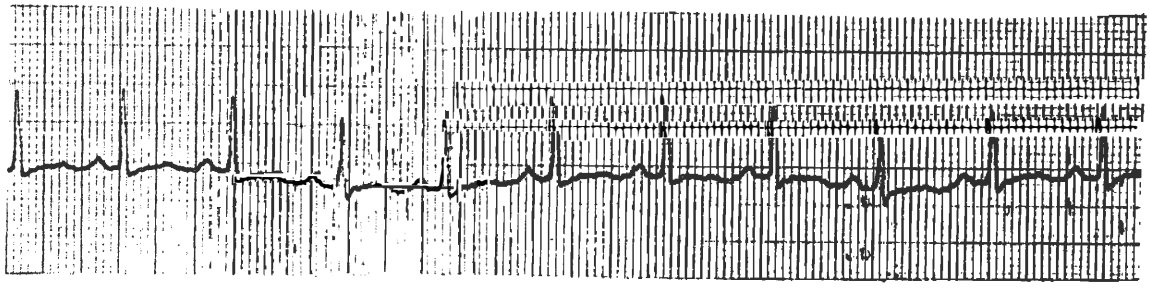


2 minutes

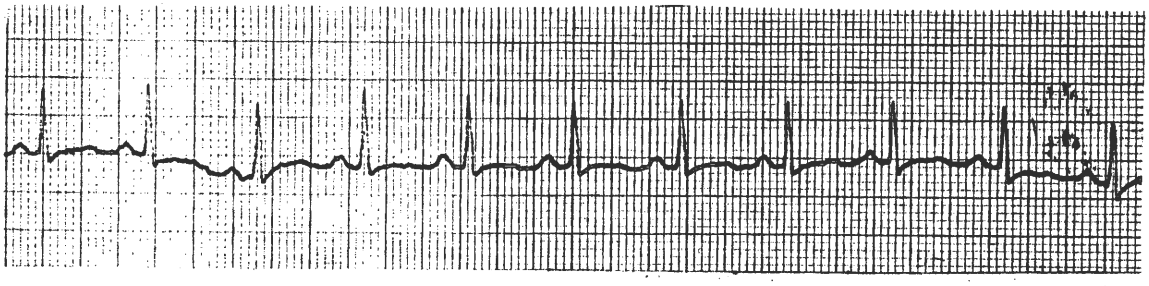


3 minutes

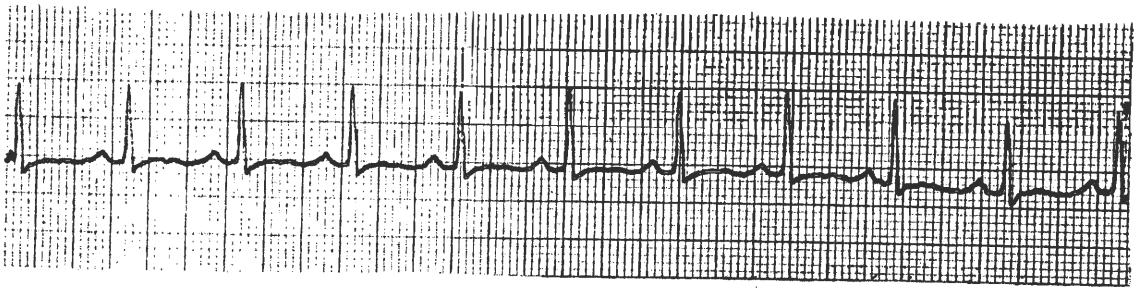
Patient 24



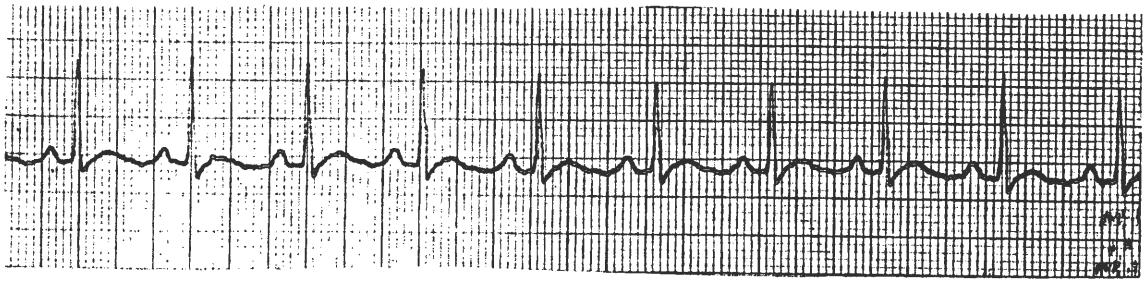
4 minutes



5 minutes

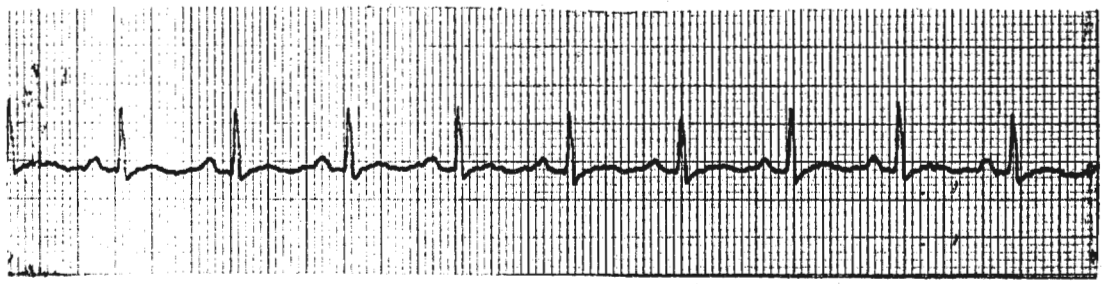


10 minutes



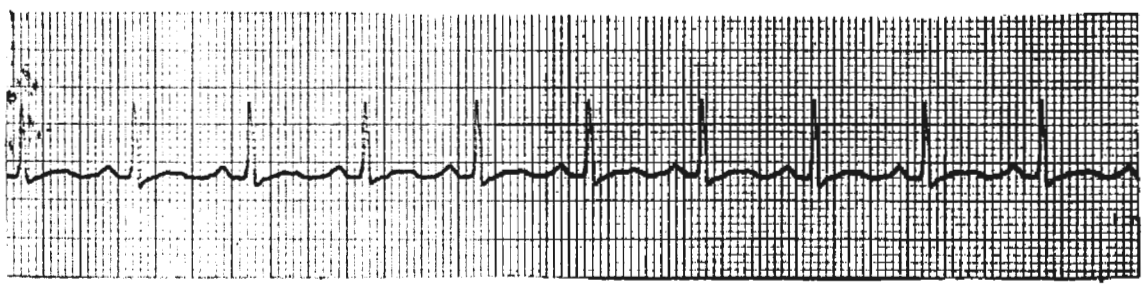
15 minutes

Patient 24



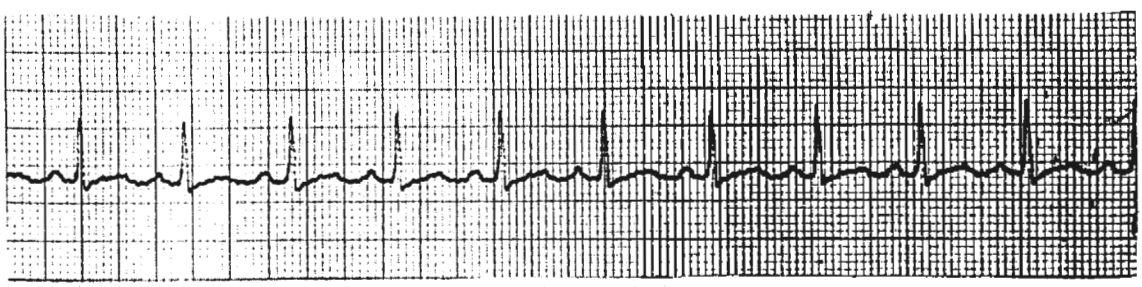
20 minutes

Bath Completed



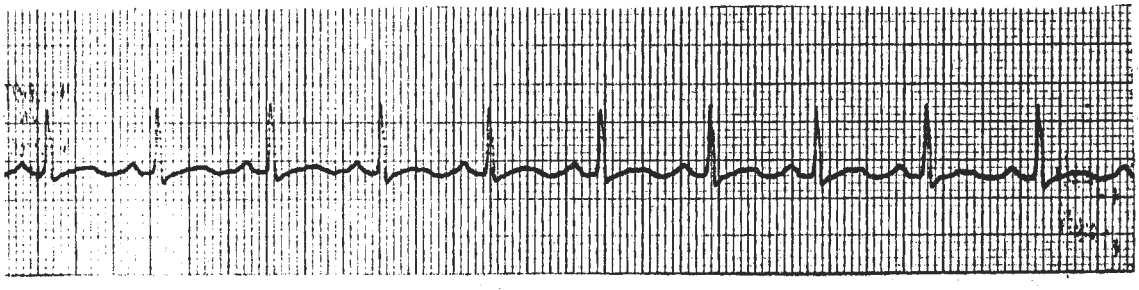
1 minute

2 minutes

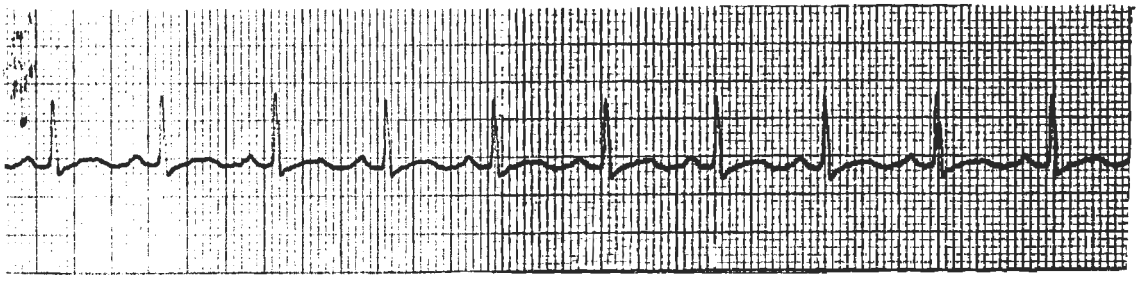


3 minutes

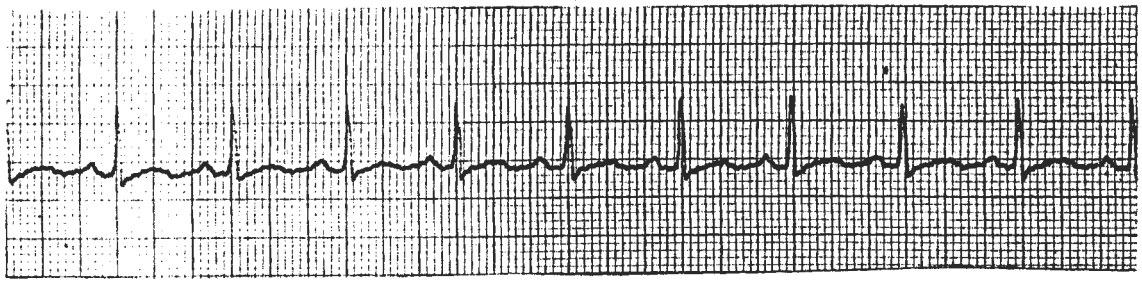
Patient 24



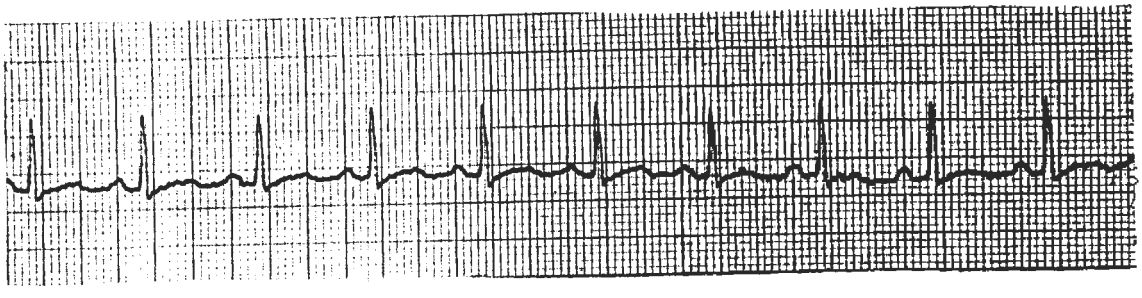
4 minutes



5 minutes

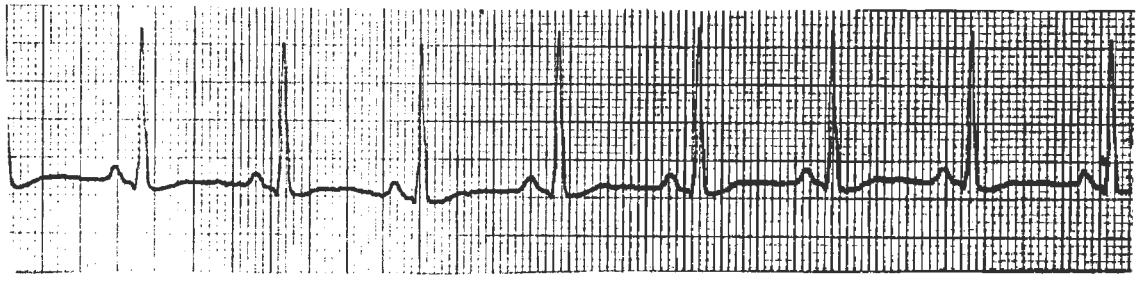


10 minutes

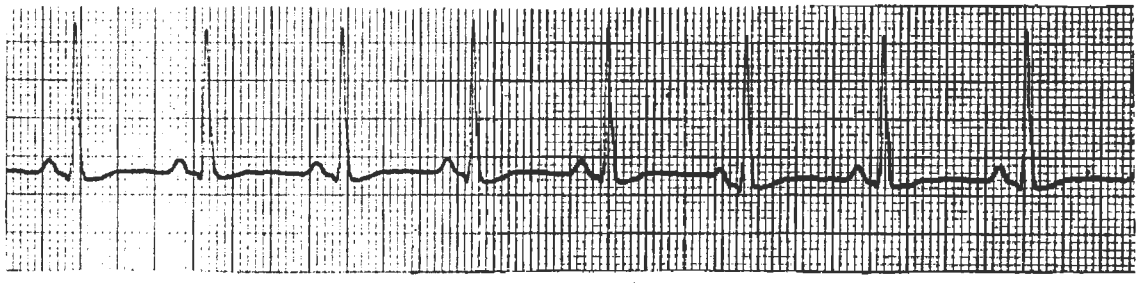


15 minutes

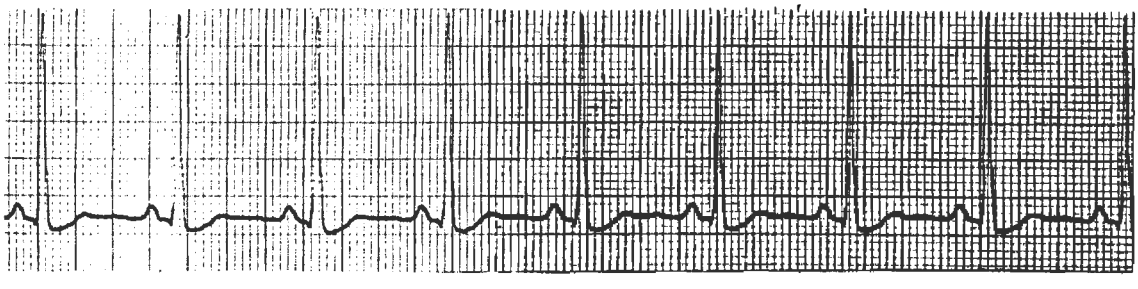
Patient 25 Monitor lead II Medication _____



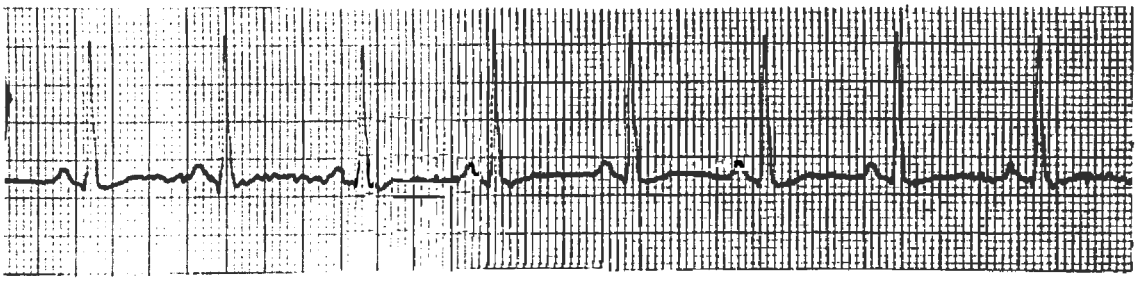
Baseline



1 minute

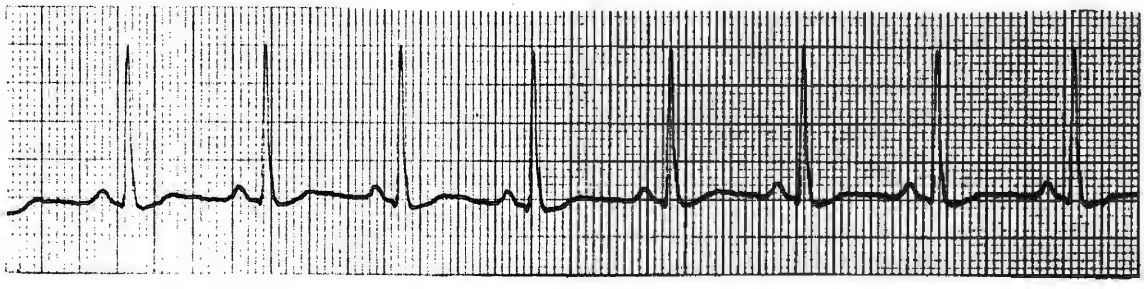


2 minutes

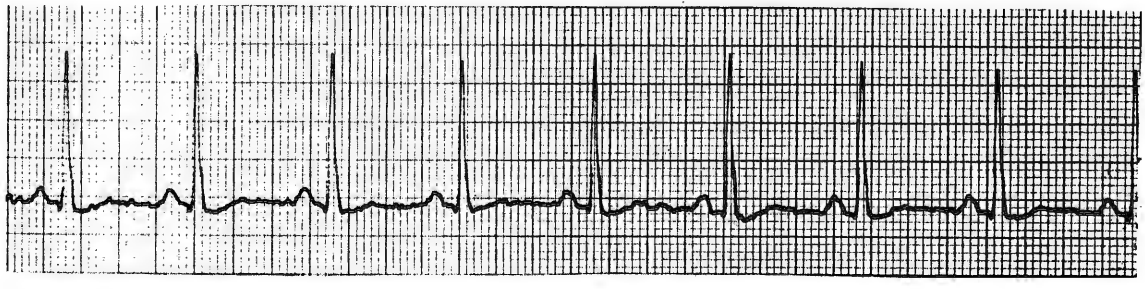


3 minutes

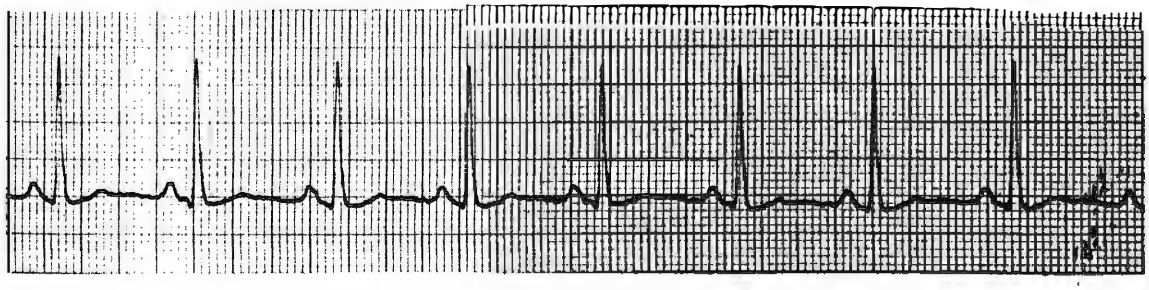
Patient 25



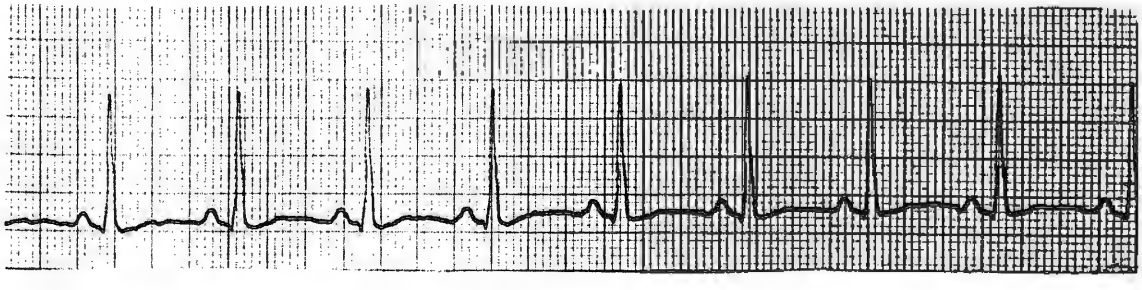
4 minutes



5 minutes

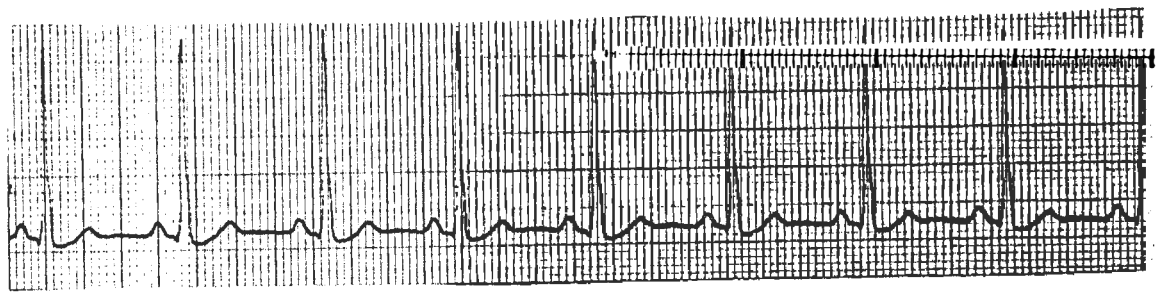


10 minutes



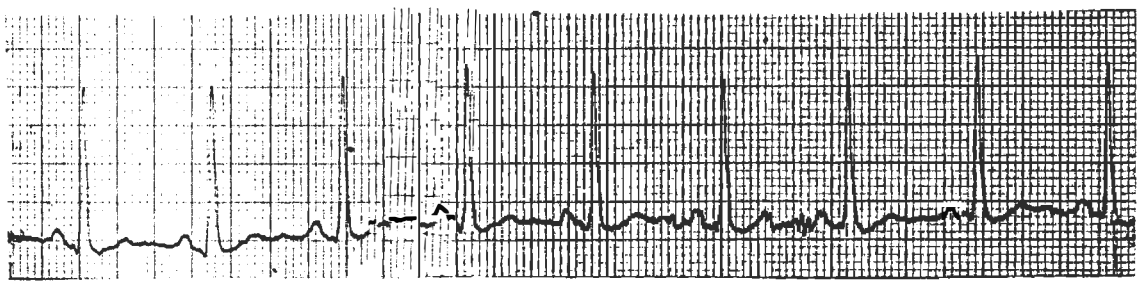
15 minutes

Patient 25

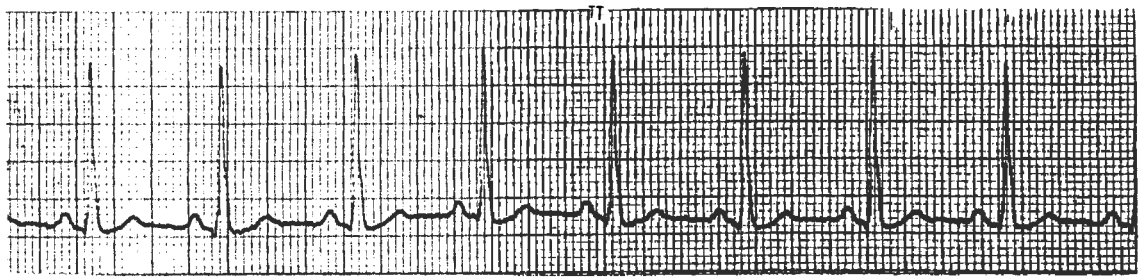


20 minutes

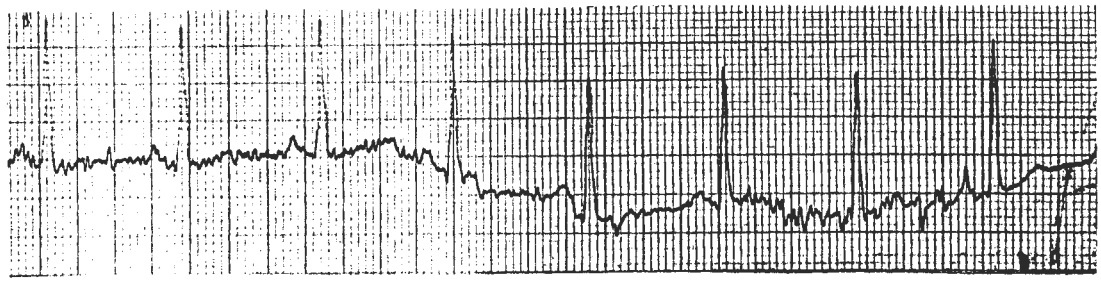
Bath Completed



1 minute

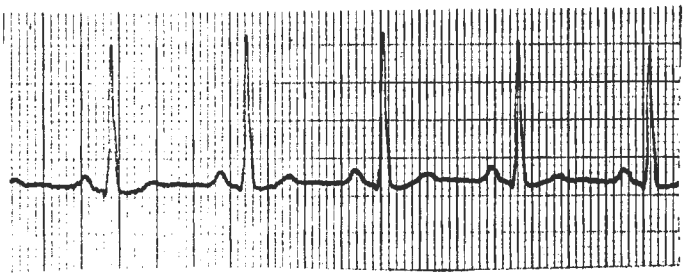


2 minutes

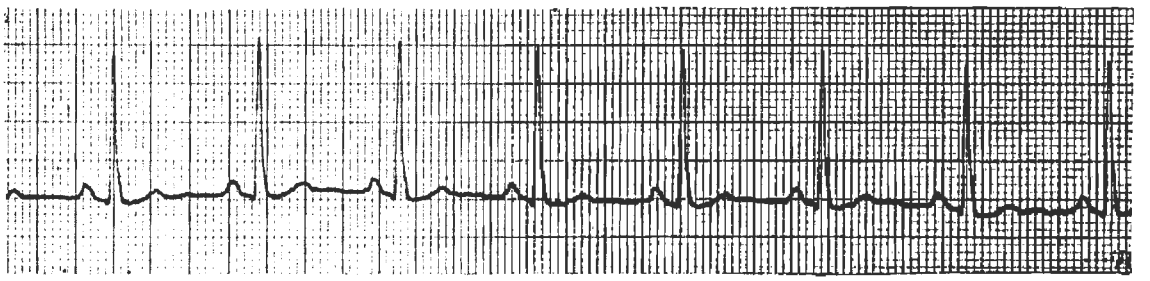


3 minutes

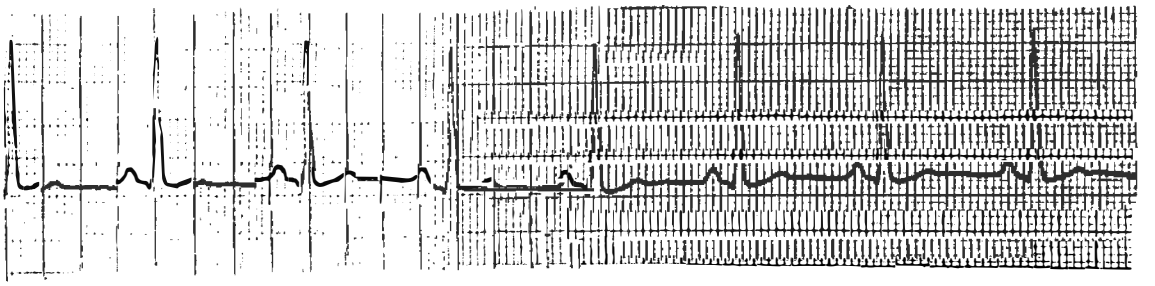
Patient 25



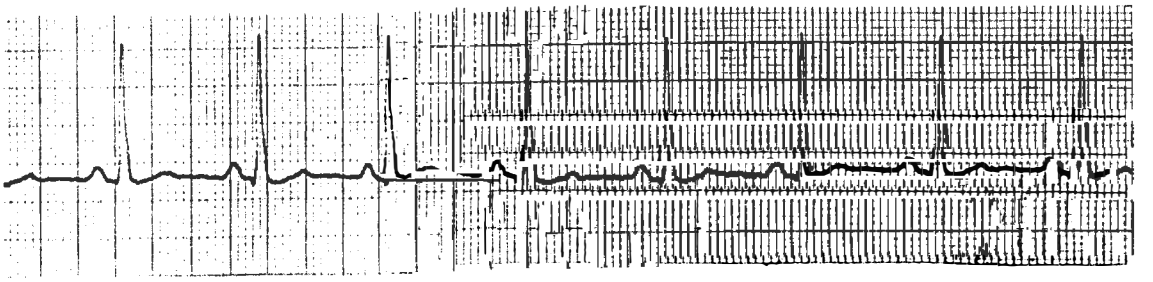
4 minutes



5 minutes

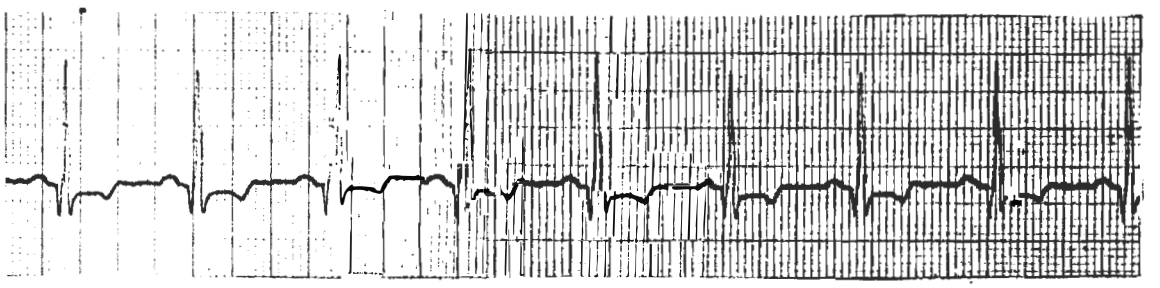


10 minutes

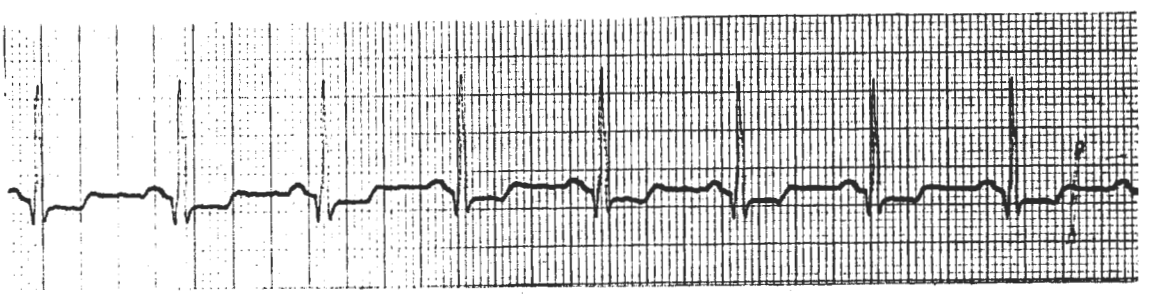


15 minutes

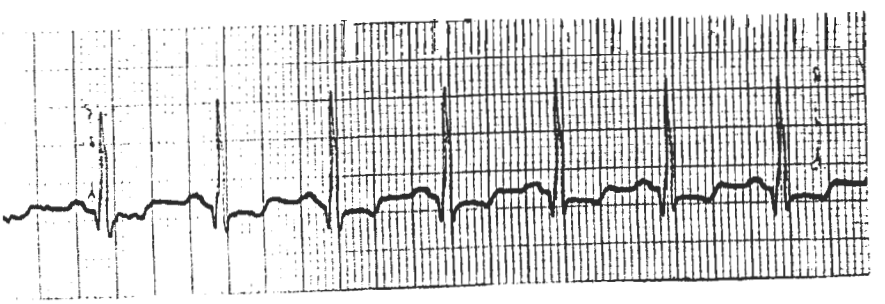
Patient 26 Monitor lead II Medication _____



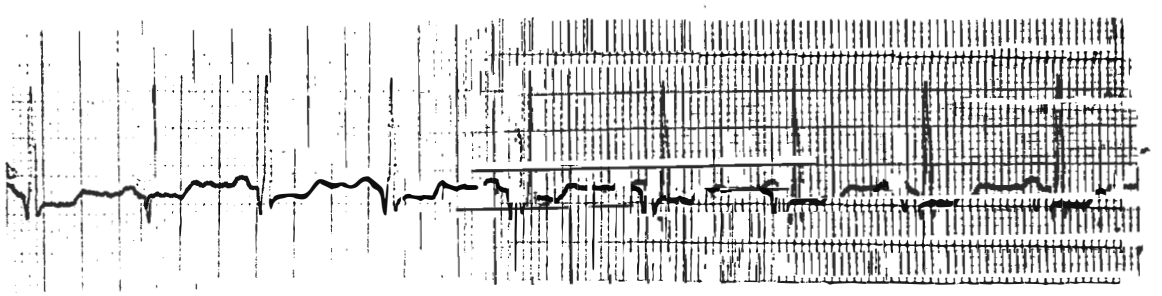
Baseline



1 minute

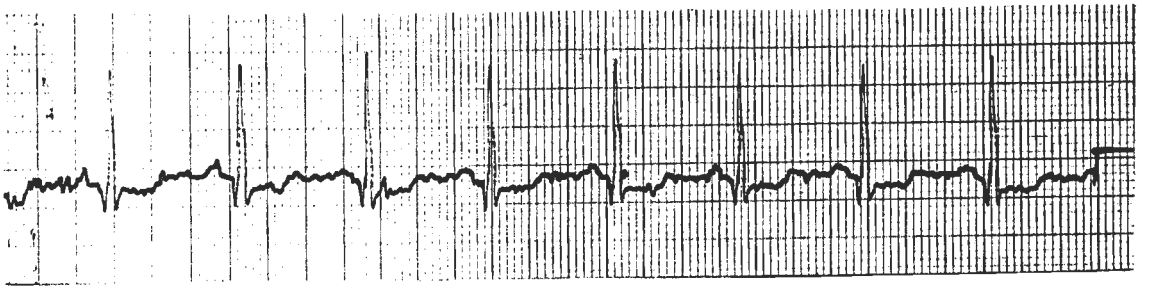


2 minutes

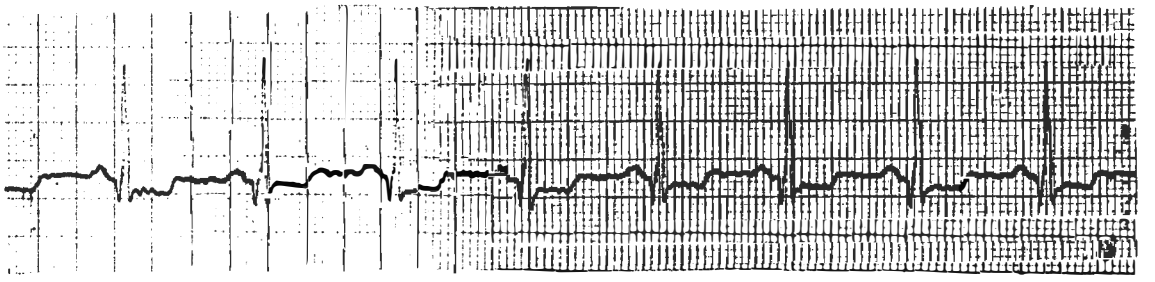


3 minutes

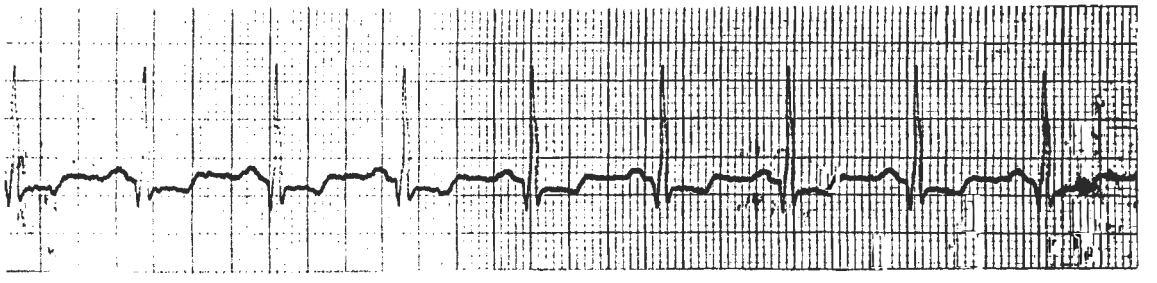
Patient. 26



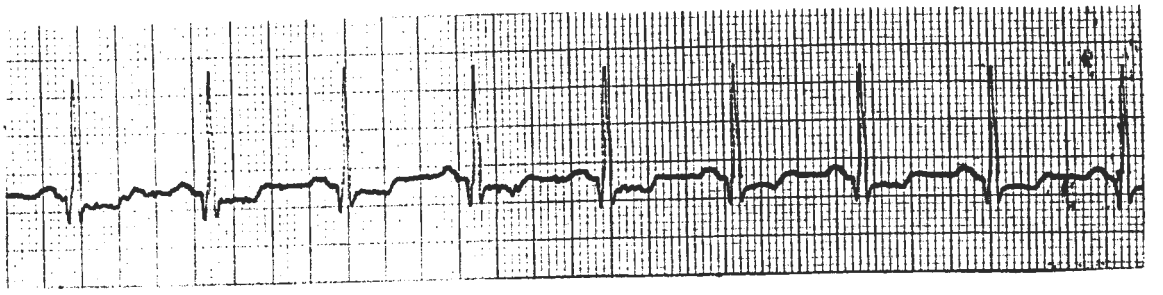
4 minutes



5 minutes

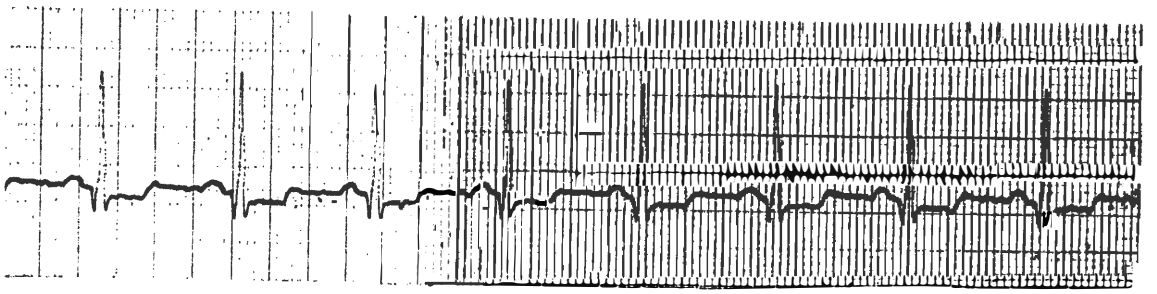


10 minutes



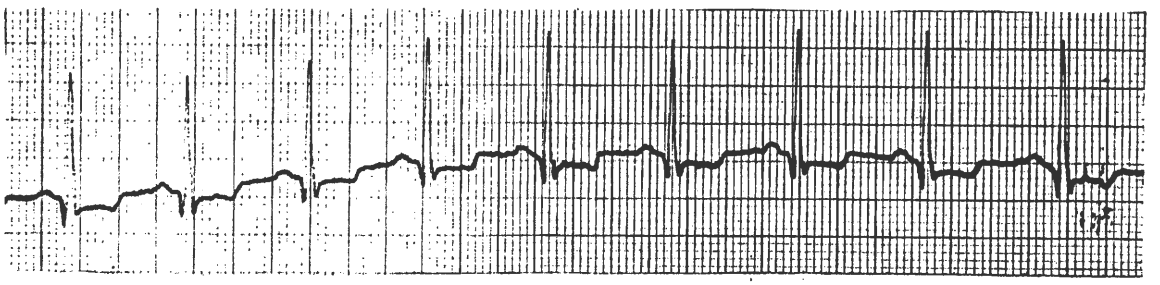
15 minutes

Patient 26

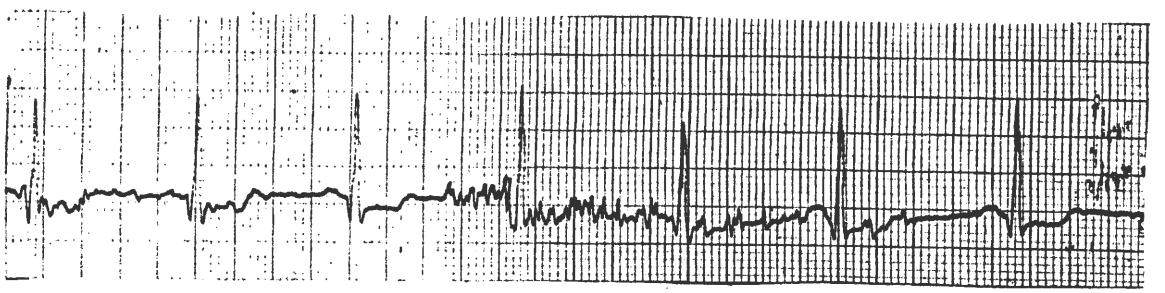


20 minutes

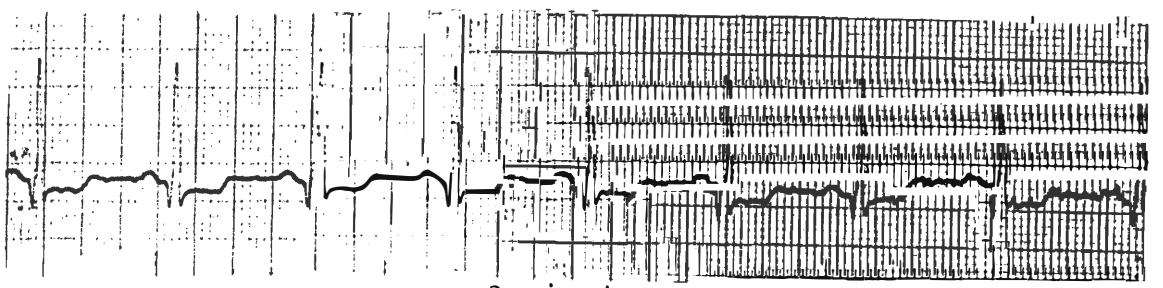
Bath Completed



1 minute

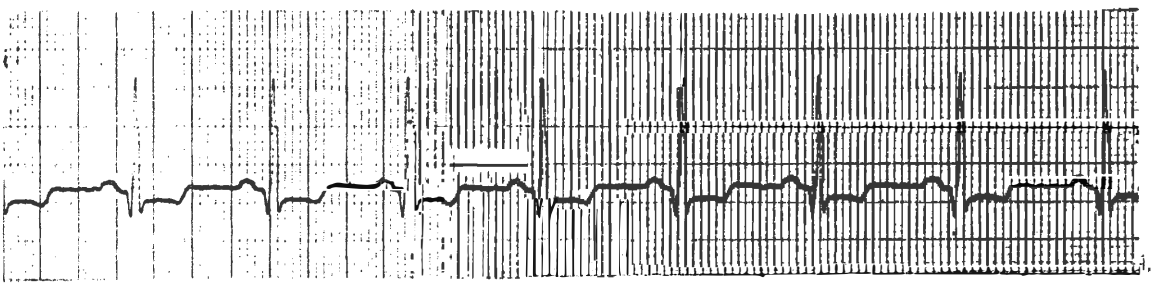


2 minutes

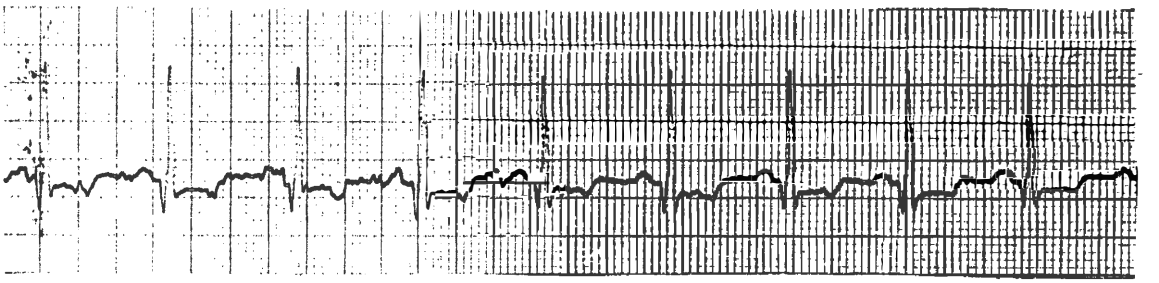


3 minutes

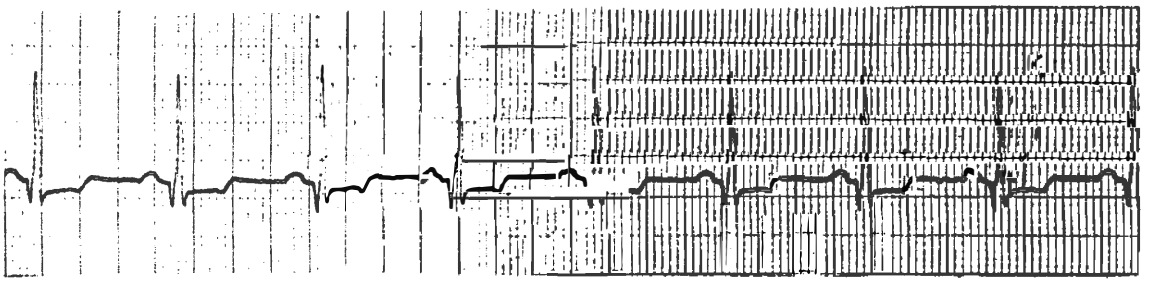
Patient 26



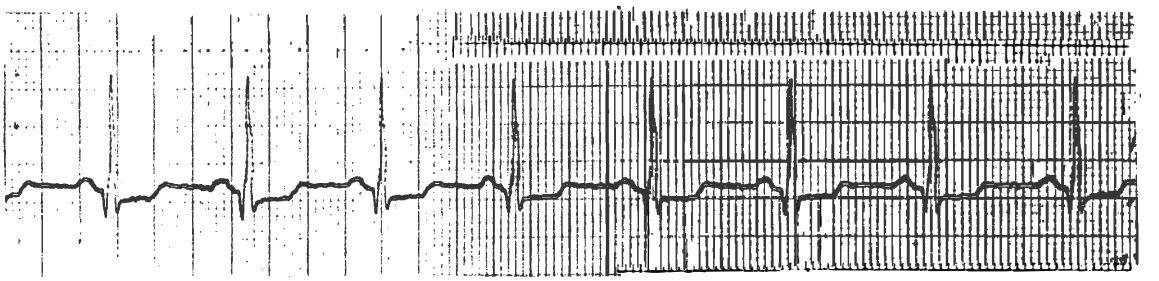
4 minutes



5 minutes

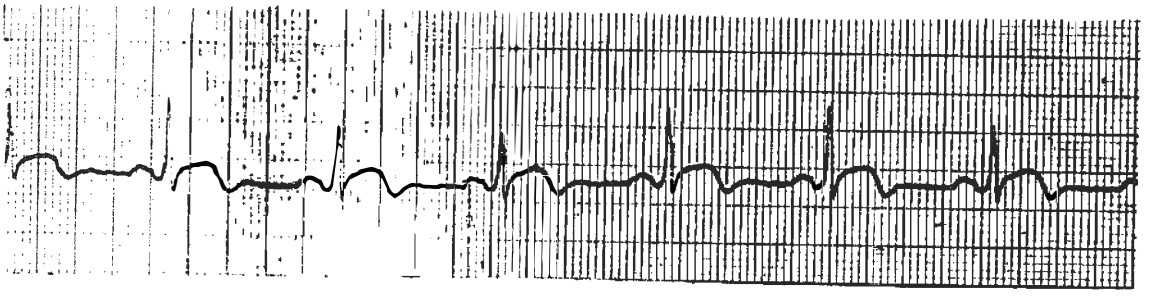


10 minutes

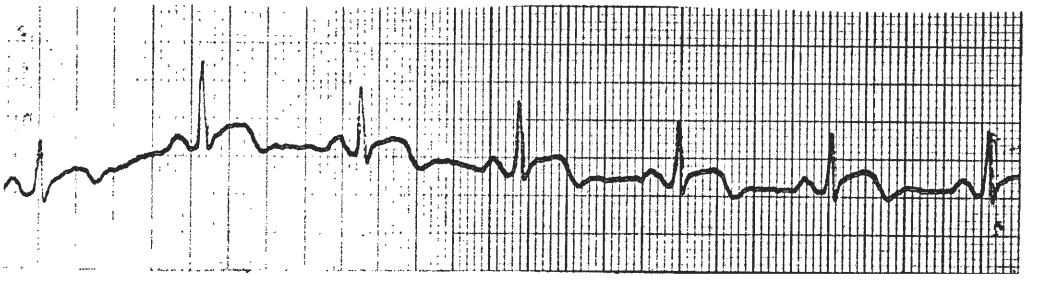


15 minutes

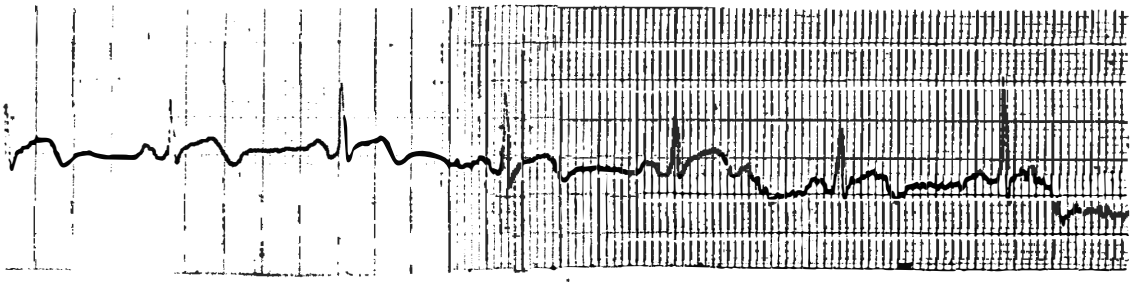
Patient 27 Monitor lead II Medication _____



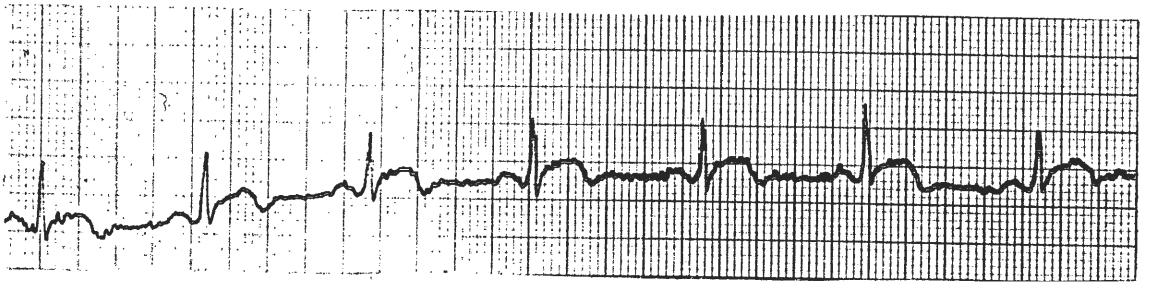
Baseline



1 minute

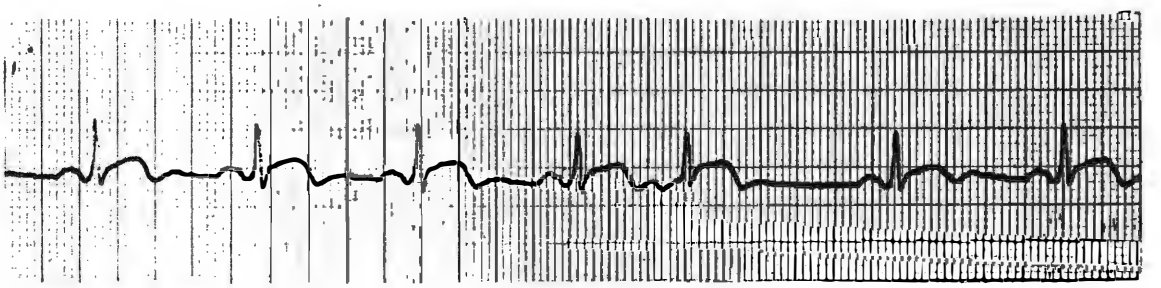


2 minutes

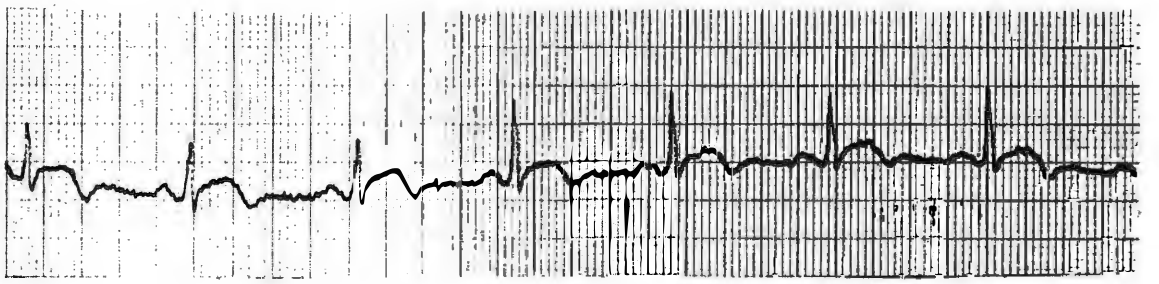


3 minutes

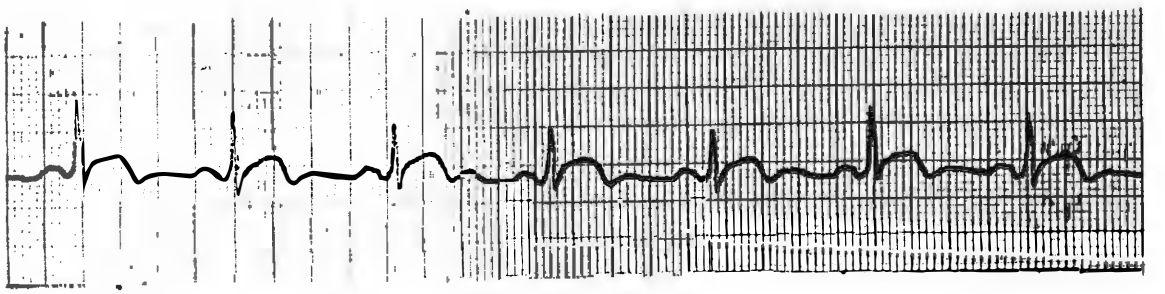
Patient 27



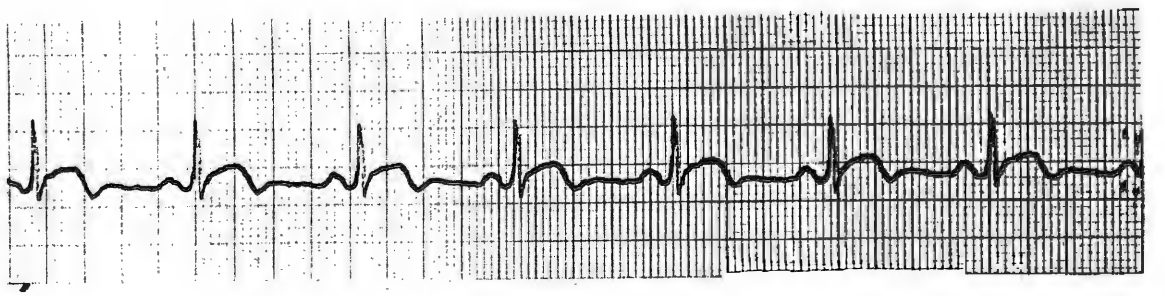
4 minutes



5 minutes

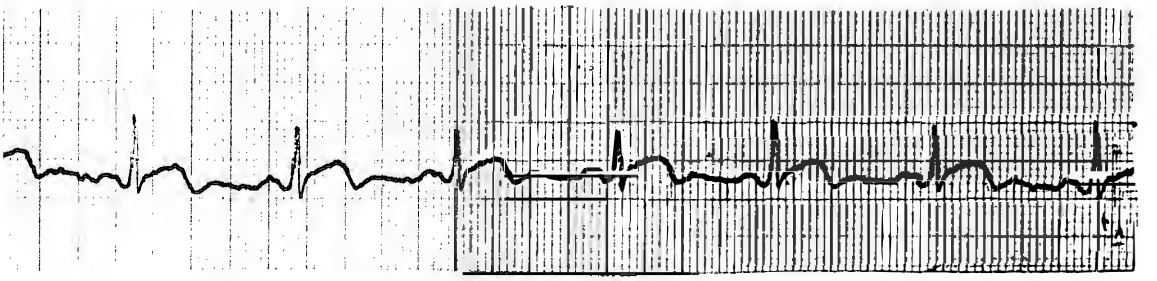


10 minutes



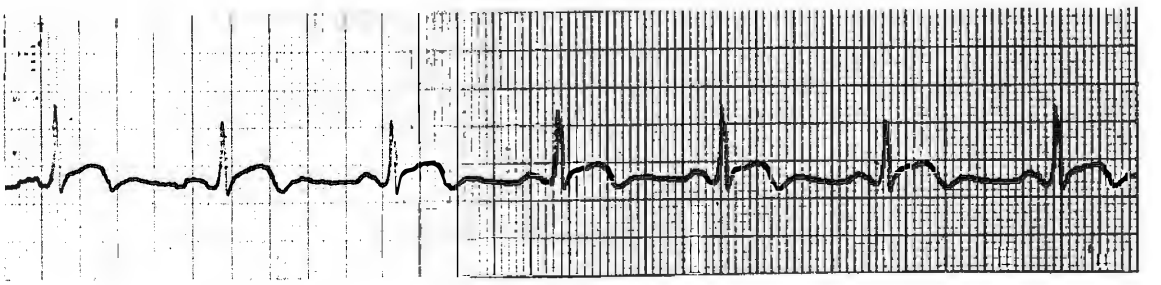
15 minutes

Patient 27

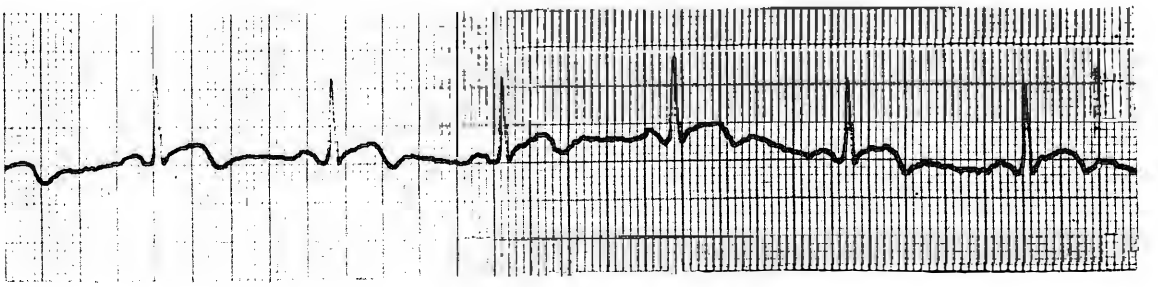


20 minutes

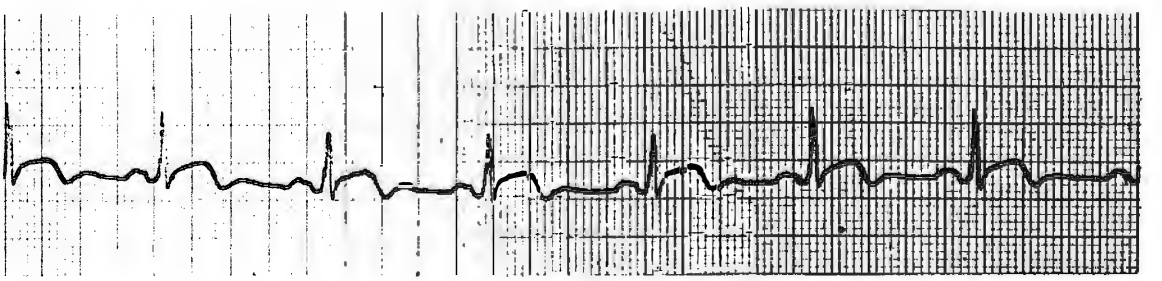
Bath Completed



1 minute

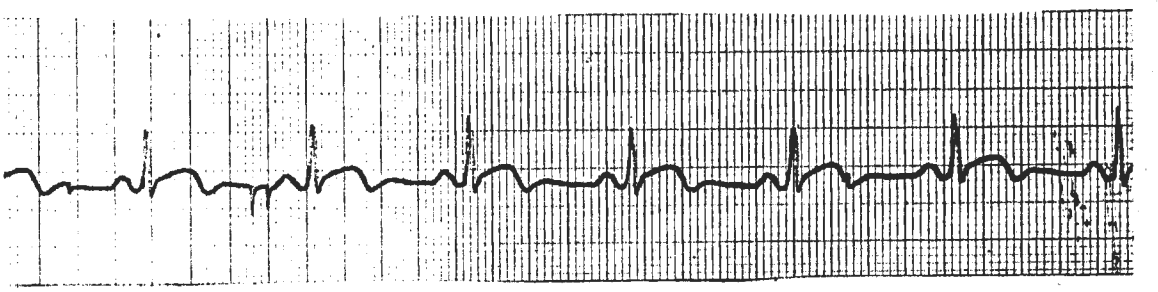


2 minutes

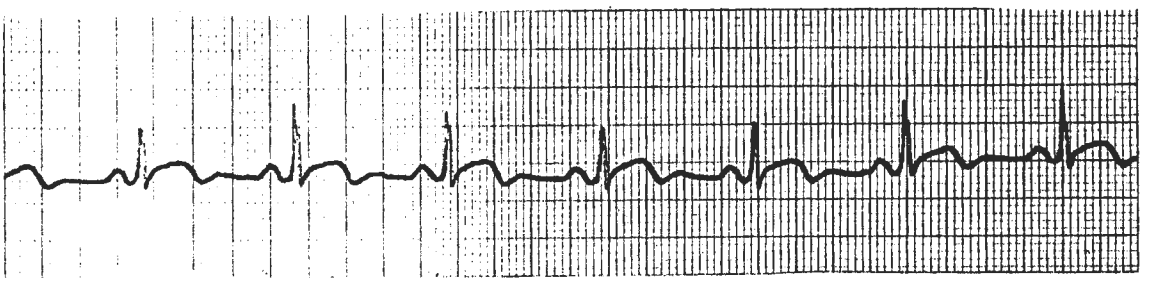


3 minutes

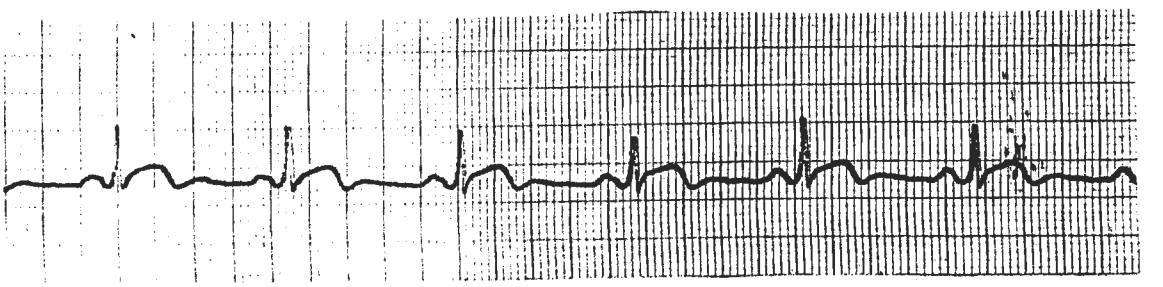
Patient 27



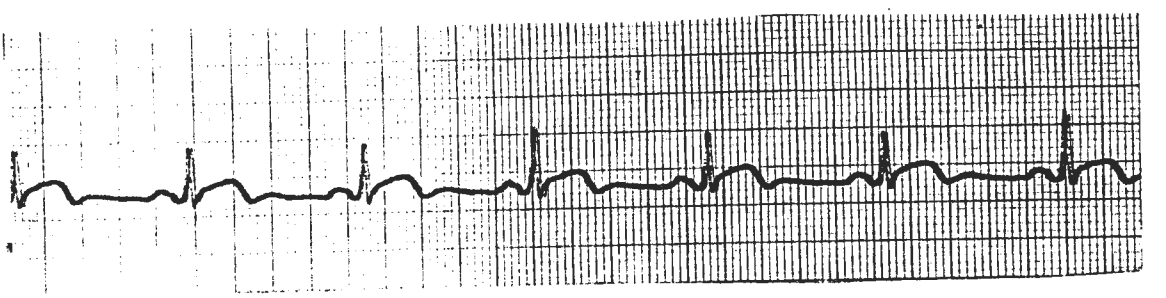
4 minutes



5 minutes

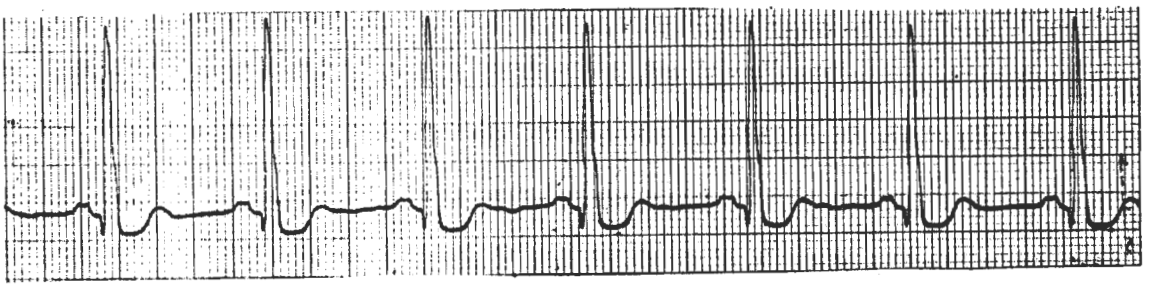


10 minutes

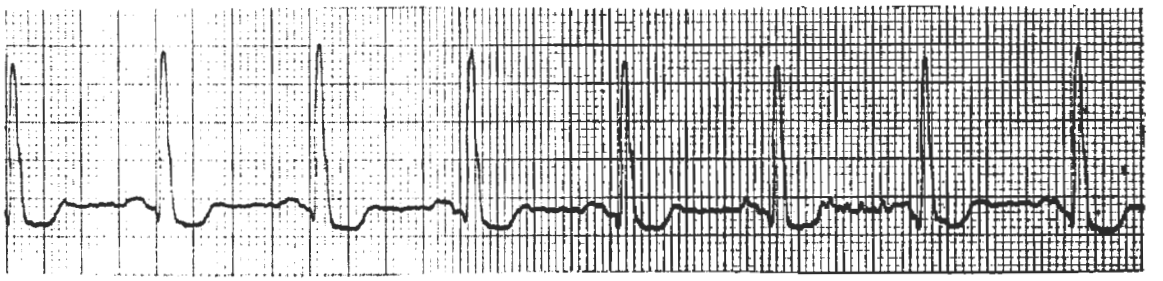


15 minutes

Patient 28 Monitor lead II Medication _____



Baseline



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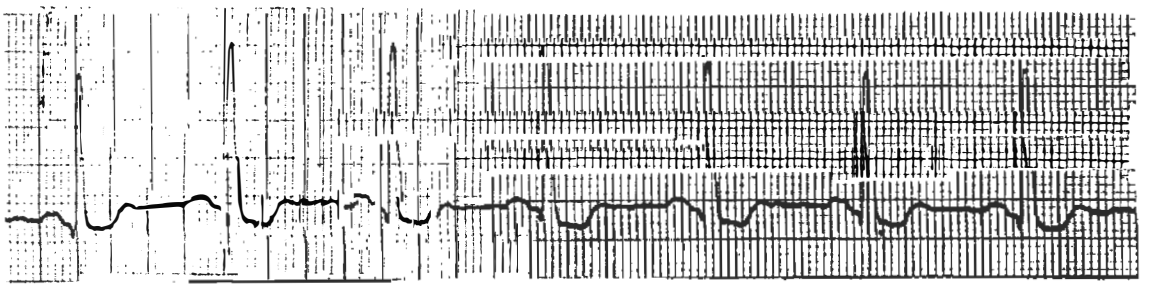

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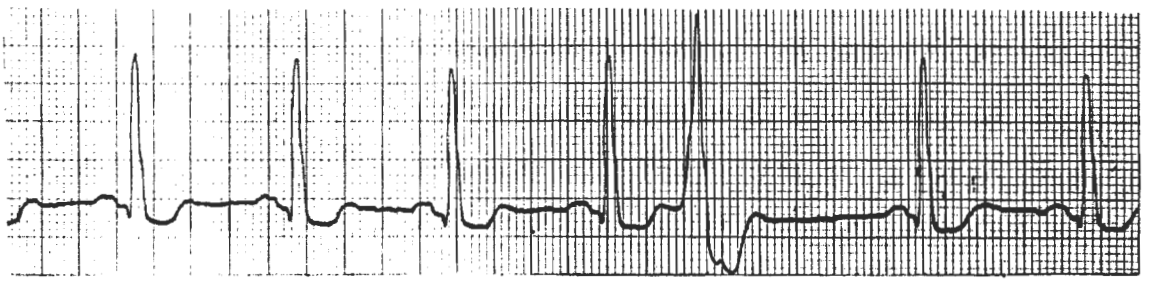
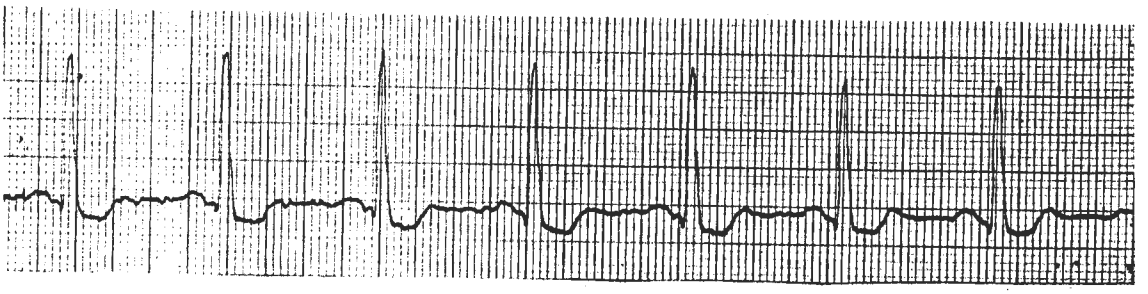


3 minutes

Patient 28



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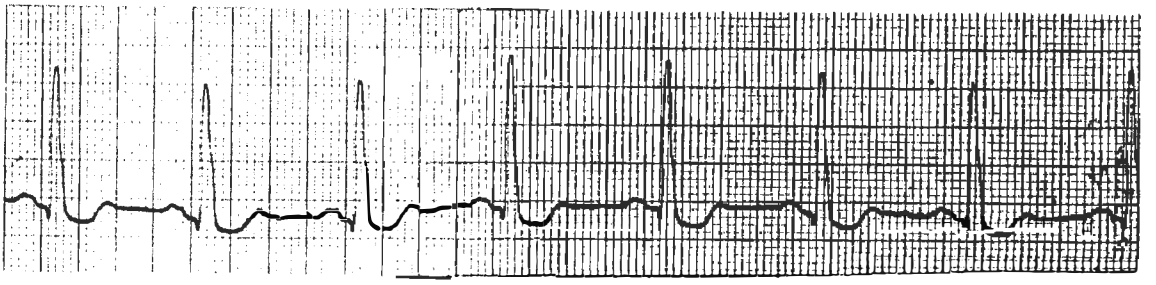

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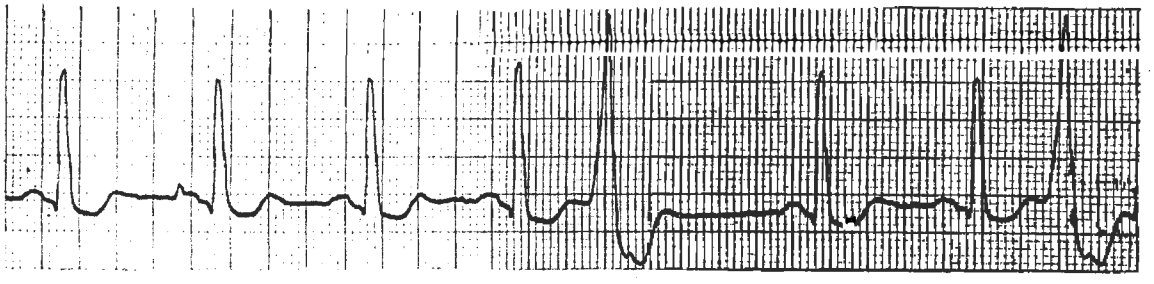

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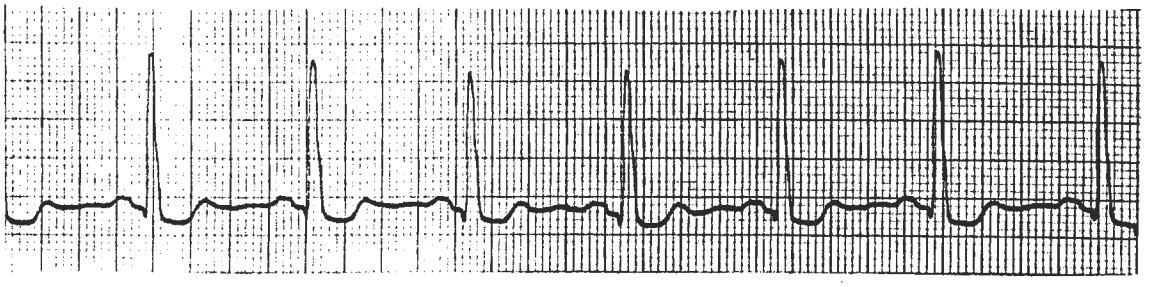

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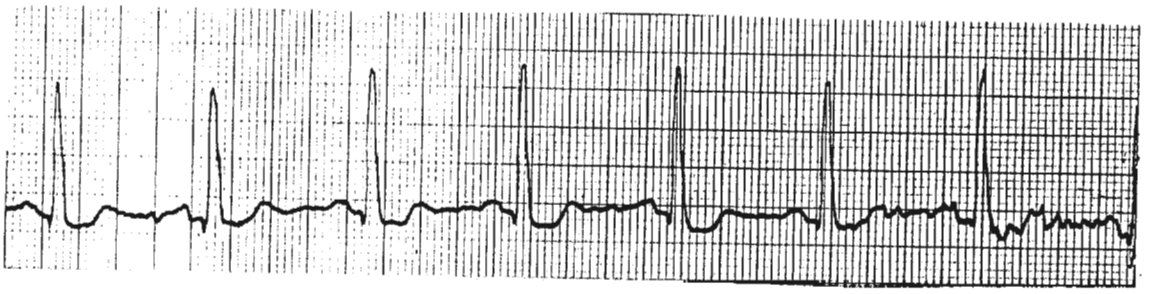

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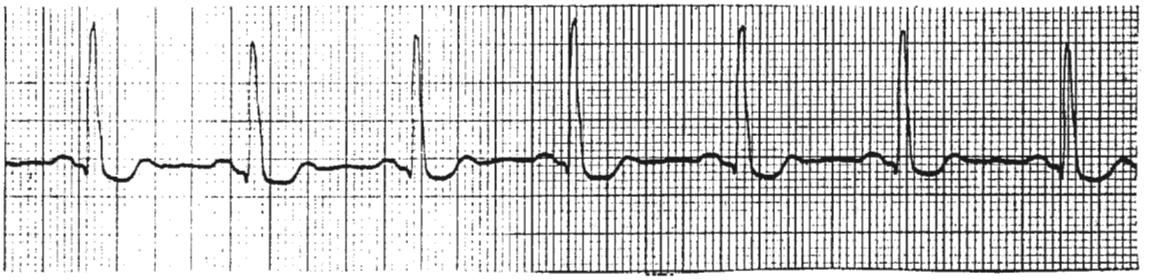

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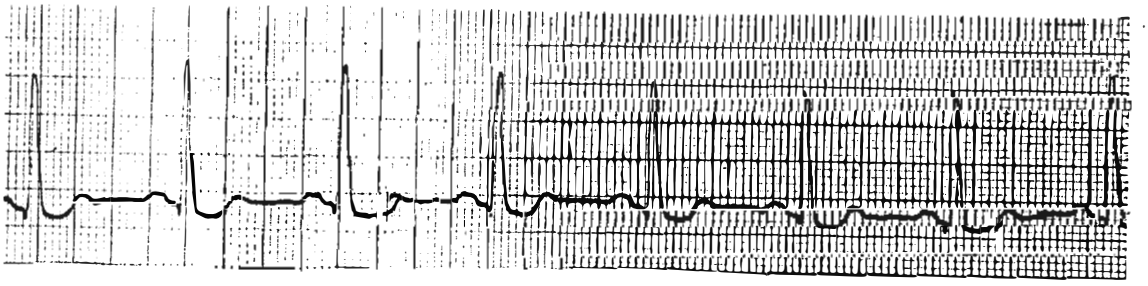

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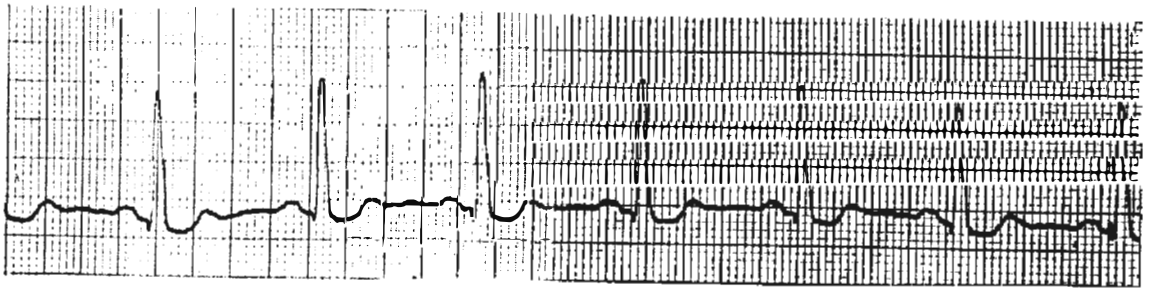

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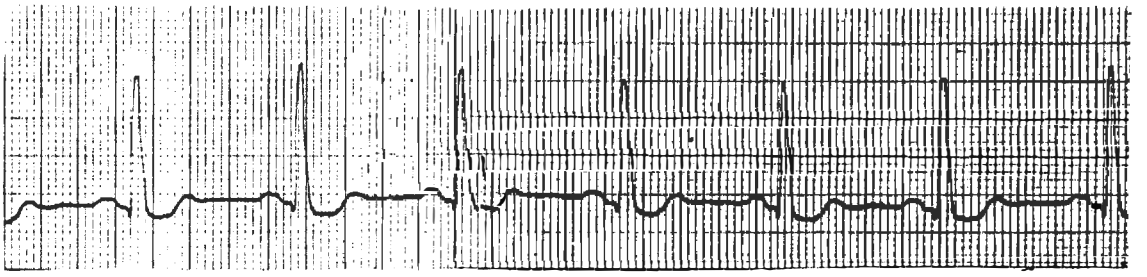

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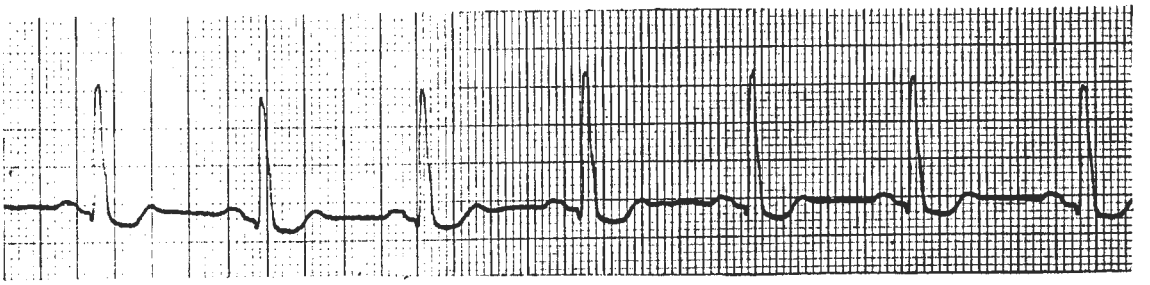

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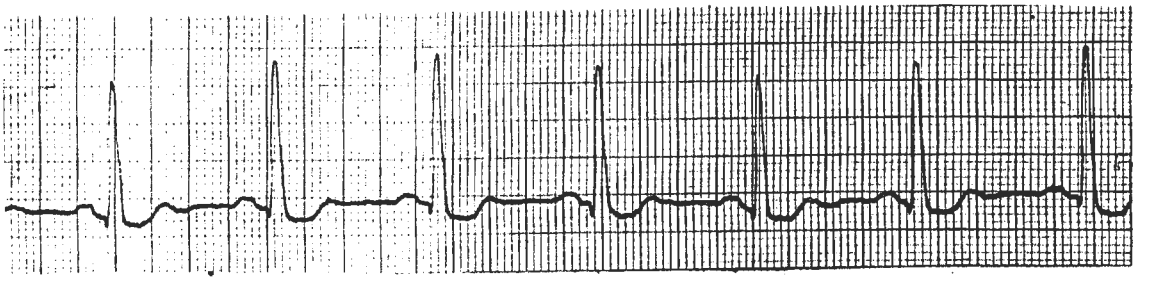

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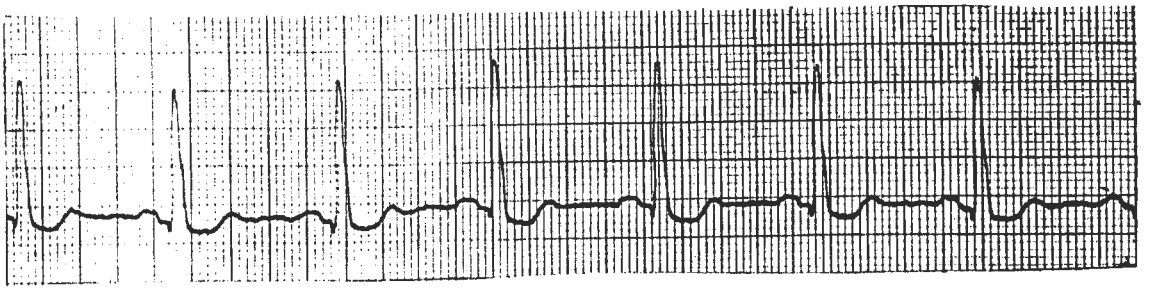

15 minutes

REFERENCES CITED

Abdellah, Faye G., and Levine, Eugene. Better Patient Care Through Nursing Research. New York: MacMillan Company, 1965.

Adams, Crawford W. "Recognition and Evaluation of Cardiogenic Shock." Heart and Lung 2 (November-December 1972): 893-895.

Alder, Henry L., and Roessler, Edward B. Introduction to Probability and Statistics. 4th ed. San Francisco: W. H. Freeman and Company, 1968.

Beland, Irene L., and Passos, Joyce Y. Clinical Nursing: Pathophysiological and Psychosocial Approaches. 3rd ed. New York: MacMillan Company, 1975.

Bloch, Antoine; Maeder, Jean-Pierre; Haissly, Jean-Claude; Elix, Jose; and Blackburn, Henry. "Early Mobilization After Myocardial Infarction." American Journal of Cardiology 34 (August 1974): 152-157.

Brunner, Lillian Sholtis, and Suddarth, Doris Smith. Textbook of Medical-Surgical Nursing. 3rd ed. Philadelphia: J. B. Lippincott Company, 1975.

Bryne, Marjorie L., and Thompson, Lida F. Key Concepts for the Study and Practice of Nursing. St. Louis: C. V. Mosby Company, 1972.

Conn, Howard Franklin; Clohecy, Robert J.; and Conn, Rex B. Current Diagnosis. 4th ed. Philadelphia: W. B. Saunders Company, 1974.

Conn, Susan Elizabeth. Acute Myocardial Infarction Patient's Response to Significant Others. Denton, Texas: Texas Woman's University, 1973.

Conrad, Linda. "The Valsalva Maneuver: A Clinical Inquiry." American Journal of Nursing 71 (March 1971): 553-554.

DeBusk, Robert. "The Value of Exercise Stress Testing." Journal of American Medical Association 232 (June 2, 1975): 956-958.

Dubin, Dale. Rapid Interpretation of EKG's. 3rd ed. Tampa, Florida: Cover Publishing Company, 1974.

DuGas, Beverly Witter. Introduction to Patient Care. 2nd ed. Philadelphia: W. B. Saunders Company, 1972.

Duke, Martin. "Bed Rest and Myocardial Infarction." Connecticut Medicine 37 (September 1973): 461.

Dunn, Halbert L. High Level Wellness. Arlington, Virginia: Beatty, 1973.

Ericsson, Martin; Granath, Arne; Ohlsen, Paul; Sodermark, Tore; and Volpe, Ulderico. "Arrhythmias and Symptoms During Treadmill Testing Three Weeks After Myocardial Infarction in 100 Patients." British Heart Journal 35 (December 1972): 787-790.

Flores, Angeles, and Zohman, Lenore. "Energy Cost of Bedmaking to the Cardiac Patient and the Nurse." American Journal of Nursing 70 (June 1970): 1264-1267.

Frohlick, Edward D. Pathophysiology. Philadelphia: J. B. Lippincott Company, 1972.

Friedewald, Vincent E., Jr. "Maximal-stress, Multiple-lead Exercise Testing: The Significance of ST-segment Changes in the Detection of Coronary Arterial Occlusive Disease." Heart and Lung 5 (January-February 1976): 91-96.

Fuerst, Elinor V.; Wolff, LuVerne; and Weitzel, Marlene H. Fundamentals of Nursing. 5th ed. Philadelphia: J. B. Lippincott Company, 1974.

Geertsen, H. Reed; Ford, Marilyn; and Castle, C. Hilmon. "The Subjective Aspects of Coronary Care." Nursing Research 25 (May-June 1976): 211-214.

Gentry, W. Doyle; Musante, Gerard J.; and Haney, Thomas. "Anxiety and Urinary Sodium/Potassium as Stress Indicators on Admission to a Coronary-Care Unit." Heart and Lung 2 (November-December 1973): 875-877.

Grace, William J. "Guide to the Management of the Complications of Acute Myocardial Infarction." Hospital Medicine 11 (October 1975): 6-27.

Gragg, Shirley Hawke, and Roes, Oliver M. Scientific Principles in Nursing. St. Louis: C. V. Mosby Company, 1974.

Gross, Harry. "Rest and Ambulation in Myocardial Infarction." New York Journal of Medicine (July 1, 1973): 1778-1781.

Gruber, Patricia Ann. "Changes in Cardiac Rate Associated With the Use of the Rectal Thermometer in the Patient with Acute Myocardial Infarction." Heart and Lung 3 (March-April 1974): 288-292.

Guyton, Arthur C. Textbook of Medical Physiology. 5th ed. Philadelphia: W. B. Saunders Company, 1976.

Haskell, William L. "Physical Activity After Myocardial Infarction." American Journal of Cardiology 33 (May 20, 1974): 776-782.

Houser, Doris. "Safer Care for the M.I. Patient." Nursing '74 4 (July 1974): 42-47.

_____ "Ice Water for Myocardial Infarction Patients? Why Not?" American Journal of Nursing 76 (March 1976): 432-434.

Hurst, J. Willis, ed. The Heart. 3rd ed. New York: McGraw-Hill Book Company, 1974.

Jarvis, Carolyn Mueller. "Vital Signs." Nursing '76 6 (April 1976): 31-37.

Joyce, Sister Pamela Mary. "The Effectiveness of Coronary-care Units in Reducing the Mortality of Acute Myocardial Infarction." Heart and Lung 1 (July-August 1972): 487-489.

Lavin, Mary Ann. "Bed Exercise for Acute Cardiac Patients." American Journal of Nursing 73 (July 1973): 1226-1227.

Luckmann, Joan, and Sorensen, Karen Creason. Medical-Surgical Nursing. Philadelphia: W. B. Saunders Company, 1974.

Marriott, Henry J. L. Practical Electrocardiography. 5th ed. Baltimore, Maryland: Waverly Press Inc., 1972.

McCorkle, Ruth. "Effects of Touch on Seriously-Ill Patients." Nursing Research 23 (March-April 1974): 125-132.

McHenry, Paul L.; Morris, Stephen N.; and Jorden, John W. "Stress Testing in Coronary Heart Disease." Heart and Lung 3 (January-February 1974): 83-91.

Meltzer, L. E., and Dunning, A. J. Textbook of Coronary Care. Bowie, Maryland: Charles Press, 1972.

Meltzer, Lawrence E.; Abdellah, Faye G.; and Kitchell, J. Roderick, eds. Concepts and Practices of Intensive Care for Nurse Specialists. 2nd ed. Bowie, Maryland: Charles Press Publishers, Inc., 1976.

Mitchell, Pamela Holsclaw. Concepts Basic to Nursing. New York: McGraw-Hill Book Company, 1973.

Nordmark, Madelyn T., and Rohweder, Anne W. Scientific Foundations of Nursing. 3rd ed. Philadelphia: J. B. Lippincott Company, 1975.

Oliver, M. F. "The Metabolic Response to a Heart Attack." Heart and Lung 4 (January-February 1975): 57-60.

Rogers, Martha E. "Doctoral Education in Nursing." Nursing Forum 5 (February 1975): 75-82.

Romhilt, Donald W., and Fowler, Noble O. "Physical Signs in Acute Myocardial Infarction." Heart and Lung 2 (January-February 1973): 74-79.

Rosenman, Ray H. "Observations on the Pathogenesis of Coronary Heart Disease." Heart and Lung 1 (January-February 1972): 68-73.

Shepro, David; Belamarich, Frank; and Levy, Charles. Human Anatomy and Physiology: A Cellular Approach. New York: Holt, Rinehart, and Winston, 1974.

Sodeman, William A., Jr., and Sodeman, William A. Pathologic Physiology. 5th ed. Philadelphia: W. B. Saunders Company, 1974.

Stude, Carol. "Cardiogenic Shock." American Journal of Nursing 74 (September 1974): 1636-1640.

Talano, James V., and Ronan, James A. "The Effect of Sinus Tachycardia on the Phases of Left Ventricular Systole During Acute Myocardial Infarction in Man." American Heart Journal 87 (April 1974): 428-432.

Thomas, Sue A.; Lynch, James J.; and Mills, Mary E. "Psychosocial Influence on Heart Rhythm in the Coronary-Care Unit." Heart and Lung 4 (September-October 1975): 746-750.

Treece, Eleanor Walters, and Treece, James William. Elements of Research in Nursing. St. Louis: C. V. Mosby Company, 1973.

Wintrobe, Maxwell M.; Thorn, George W.; Adams, Raymond D.; Braunwaol, Eugene; Isselbacher, Kurt J.; and Petersdorf, Robert G., eds. Harrison's Principles of Internal Medicine. 7th ed. New York: McGraw-Hill Book Company, 1974.

Yu, Paul N., Chairman. "Resources for the Optimal Care of Patients with Acute Myocardial Infarction." Circulation 43 (May 1971): A-171-A-180.