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Cardiac Arrythmias in Acute and Chronic Renal Failure ✿

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Cardiac arrythmias are frequent complication in acute and chronic renal failure but the exact incidence of arrythmias remain the subject of considerable debate.

Cardiac arrythmias are mostly of supra – ventricular tachycardia, particulary paroxismal auricular premature contraction, auricular tachycardia and auricular fibrillation which may occur at any stage of the course of the disease, if B.U.N. remains elevated for over a long period of time.

In many instances some specific causative factors can be found, among those, electrolyte imbalances, sustained hypertention, focal degenration of myocardium, pericarditis, and digitalisation appear to be the most potentially dangerous cause of arrythmias.

We have therefore studied cardiac arrythmias in series of patients with acute and chrohic renal failure of varying ethiology with the purpose of identifying the incidence and most common causes of these arrythmias.

Twenty patients (8 with acute renal failure and 12 with chronic renal failure.) were the subject of our studies.

The evaluation of cardiac arrythmias was suported by appropriate physical examination, serial E.C.G. and chest X rays, daily determination of electrolytes and cardiac muscle biopsy or post mortem examination of the heart of the patients immidiately after death.

In our series of patients, the following causes were held responsible for their cardiac arrythmias:

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1- Persistent superimposed infection which were quite common in our patients.

2- Hyperkalemia, Acidosis, anemia and sustained hypertension.

3- Electrolytes imbalances.

4- Digitalization of the patients for their cardiac failure.

5- Focal Degeneration of myocardium which existed in all of our patients.

In brief, we discuss 6 of our patients with acute and chronic renal failure who died immidiately after their cardiac arrythmias supervened and we were able to perform post mortem examinations. The rest survived with immidiate ouabain therapy and other therapeutic measures.

Case No. 1: Miss Z.A. a 13 years old girl was first admitted in April 1966 in complete anuria 24 hours after she ingested some amount of unknown toxic material in a suiccidal attemp.

On physical and laboratory examinations: a soft systolic mur mur was heard on mitral area, B. P. was 165/58 mm Hg. Her B.U.N. was reported 6,5 Gm/Lit and Na:141 mEqu., K:6mEqu., Ca: 7,5 mGm%, P:4,3mGm% and CO2: 13 mEqu.

Patient's urine examination contained trace of albumin and granular and hyaline casts. Chest X-ray demonstrated enlarged heart in all diameters.

Peritoneal dialysis was carried out and successful corrections of electrolytes imbalances and B.U.N. was obtained.

On the ninth day of her hospitalization, B.U.N. arose to 7Gm/Lit and patient developed auricular fibrillation confirmed by E.C.G. Patient expired 24 hours later,in spite of immidiate ouabain therapy.

Pathology: Cardiac muscle biopsy revealed: Areas of focal degeneration of myocardium.

Case No. 2: Mr. E. M. a 32 years old male developed sudden onset of acute renal failure with unknown etiology in March 28, 1966, and was immidiately sent to our service after being in anuria for 48 hours.

On physical and laboratory examinations, a grade I systolic mur mur was heard on the apex of the heart. His B.P. was 180/110 mmHg. Eye ground revealed vast areas of hemorrhage of the retina. His B.U.N. was

reported 5,6 Gm/Lit. Na: 152 mEqu., K: 7,5 mEqu., Ca: 8,5 mgm% P:4,8 mgm% and CO2: 11 mEqu. Immidiate peritoneal dialysis was carried out for 36 hours. B.U.N. dropped to 3,5 Gm/Lit. and electrolytes were reported approximately within normal limits after the completion of dialysis. On the forth day of his hospitalization, he developped high fever and was treated with broad expectrum antibiotics, his B.U.N. soon reached 6,5 Gm/Lit and second peritoneal dialysis was carried out. A week later, he again developped fever, pericardial friction rub and supra-ventricular tachycardia. Ouabain was soon administered and marked improvement was achieved within 24 hours.. A week later patient developed sudden onset of massive gastro-intestinal hemorrhage and became comatous and died on the following day.

Pathology: Post mortem examination of the heart revealed:

1. Areas of fibrinous pericarditis.
2. Left ventricular hypertrophy.
3. Focal degeneration of myocardium.

Case No. 3: Mrs. Z.M. a 26 year old female was first admitted to our hospital in acute renal faillure. 36 hours after having a septic abortion.

On physical and laboratory examinations, a soft functional systolic mur mur was heard on the apex of the heart and sinus tachycardia was detected on E.C.G. B.P.; 180/113 and eye ground was reported normal.

Peritoneal dialysis was carried out immidiately for 48 hours because of B.U.N. was 7,5 Gm/Lit. Ca: 7,2 mGm%, P. 4,5 mGm%, K: 6,35 mEqu. Na: 137 mEqu. and CO2: 13 mEqu. On the 7th day of hospitalization, E.C.G. revealed L.V.H. and on the ninth day, B.P. dropped to 135/65 mmHg. and protodiastolic gallop was noted on cardiac auscultation. Ouabain was administered immidiately and all symptoms temporarily disappeared within 24 hours, and B. P. remained stable around 130/60 mmHg.

On the llth day, patient developped attacks of generalized seisures due to cerebral edema and died.

Pathology: Post mortem examination of the hear revealed:

1. Enlarged heart mostly on the left side.
2. Focal degeneration of myocardium.
3. Pericarditis. (Fibrinous).

Case No. 4: Mrs. N.Kh. a 34 years old house wife who was known to be a case of chronic nephritis for the past 8 years was admitted with marked anemia and poor general condition.

On physical and laboratory examinations, B.P. was 220/120 mmHg. Hb: 6%, Hct: 16%, B.U.N. 60 mgm%, Na:152mEqu., Ca: 6,3mgm%, Ca:6,8 mgm%, CO2: 10mEqu. E.C.G. on admission revealed sinus tachycardia and L.V.H. with occaisional premature contractions. on cardiac auscultation, a marked friction rub was noted. Because of poor general condition of the patient, peritoneal dialysis was suggested but was refused by relatives of the patient.

On the same evening, patient developed pulmonary edema which was treate with immidiate digitalization. On the sixth day of her admission, she developped massive hematemesis and E.C.G. revealed auricularflutter–fibrillation. Patient expired on the following day.

Pathology: Post mortem examination of the heart revealed:

1. Pericarditis.
2. Cardiac hypertrophy.
3. Edema of myocardium and areas of focal degeneration of myocardium.

Case No. 5: Mr. E.A. a 22 years old male who developped sudden onset of acute nephritis due to unknown etiology was admitted to our service on June 5,1966 ten days after the onset of his illness.

On physical and laboratory examinations: B.U.N. was 6,5 Gm/Lit and Na: 142mEqu., K: 4,5mEqu., Ca: 7,8mgm% and CO2: llmEqu. Urine contained trace of albumin with a few W.B.C., R.B.C. and hyalin casts. E.C.G. revealed sinus tachycardia and on cardiac auscultation, a soft systolic mur mur was heard. B.P. was revealed: 195/112 mmHg.

During a month of his hospitalization, he was under treatment with peritoneal dialysis. B.U.N. raised to 6Gm/Lit and patient went into a deep coma. E.C.G. revealed L.V.H. and ventricular premature contractions. Patient died 2 days later.

Pathology: Post mortem examination of the heart revealed:

1. Uremic pericarditis.
2. Cardiac hypertrophy.
3. Edema and focal degeneration of myocardium.

Case No. 6: Miss M.K. a 14 years old female was admitted to our service in a semi–comatous state. She was known to have been suffering from subacute glomerulo–nephritis, for the past four years.

On physical and laboratory examinations: her B.U.N. was 4,5 Gm/Lit and Na: 155 mEqu., K: 4,2mEqu., Ca: 8mgm%, P: 5.2mgm%and CO2: 10mEqu. B.P. was 160/80 mmHg. and a soft systolic mur mur was heard on mitral area. E.C.G. revealed sinus tachycardia. Patient's relatives refused to have peritoneal dialysis done and therefore, symptomatic treatment was carried out for 22 days. On the night of her 23rd day of admission, she developed generalized seizures. Eye ground reveal papille edema. Soon she developed complete arrythmia with multi–focal extrasystole, and died 2 days later.

Pathology: post mortem examination of the heart revealed:

1. L. V. H.
2. Areas of focal degeneration of myocardium.

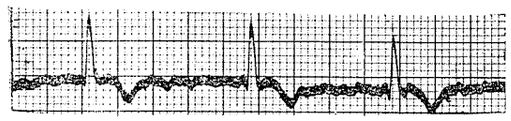
Comment and Discussion

Cardiac arrythmias are frequent complication in acute and chronic renal failure. These arrythmias are of the type of supra–ventricular tachycardia, particulary, paroxismal auricular tachycardia and fibrillation and may well acount for sudden unexplained deaths in patients who appear to have been doing well. Acidosis, high blood urea nitrogen, anemia, sustained hypertention, infection, focal degeneration of myocardim and changes in the ionic composition of the extracellular fluid, with their consequence on the response to digitalis appear to be responsible

In spite of what appears in literature, focal degeneration in uremia is the most potentially dangerous cause of arrythmias and death due to cardiac arrythmias in uremic patients

Intra–venous route of digitalis administration should be avoided in patients with acute and chronic renal failure who developed cardiac failures, pulmonary edema, or arrythmias since they appear to be potentially dangerous, and will initiate or aggravate cardiac arrythmias.

we suggest, in emergency instances, ouabain to be used instead of digitalis. We found it quite safe with dramatic results in most of our patients.



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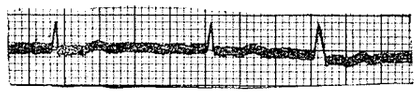

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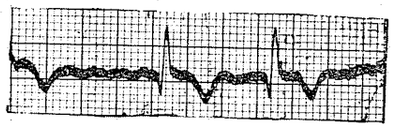

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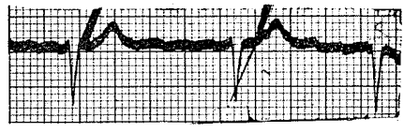

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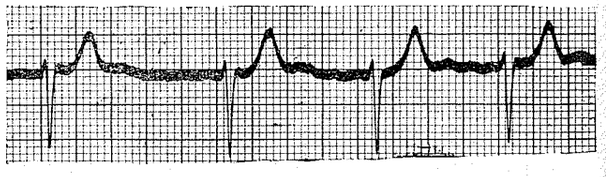

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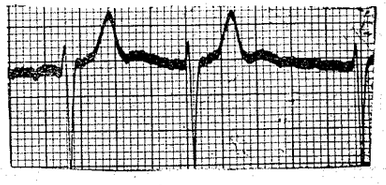

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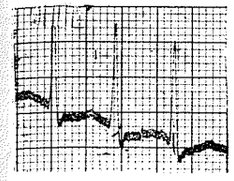

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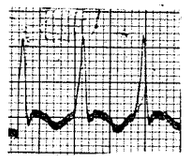

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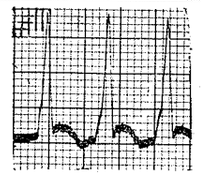

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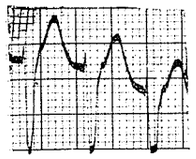

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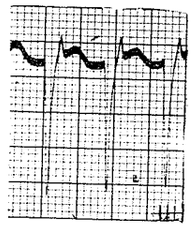

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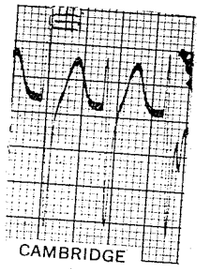

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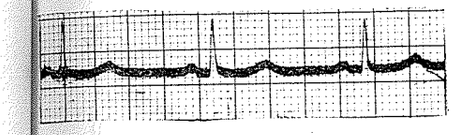
"BEFORE OUABAINE THERAPY"



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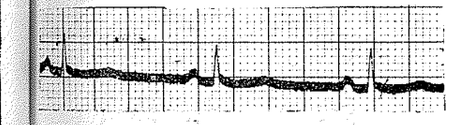

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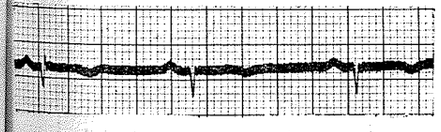

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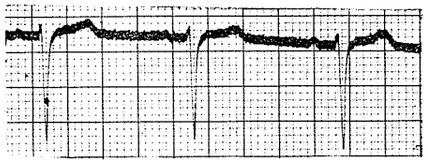

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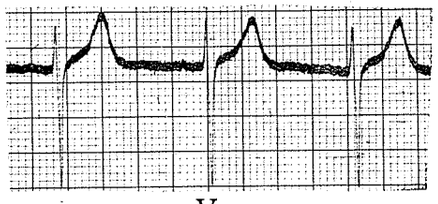

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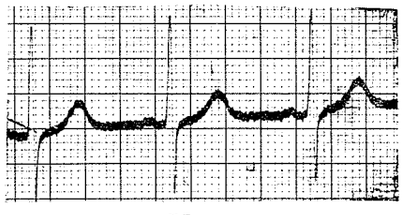

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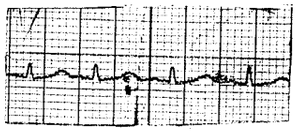

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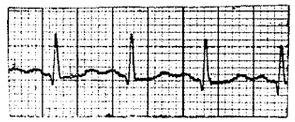

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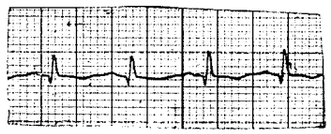

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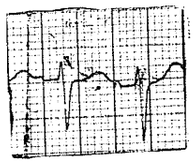

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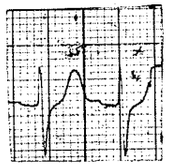

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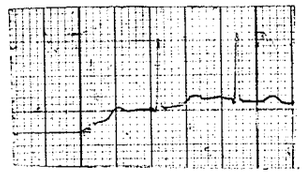

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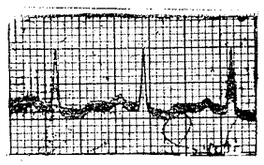

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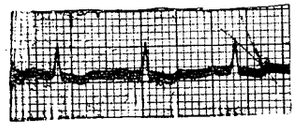

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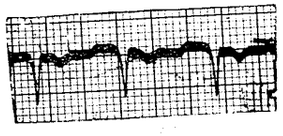

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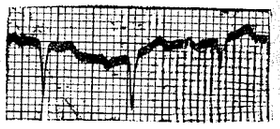

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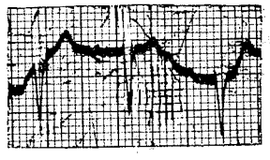

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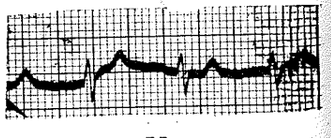

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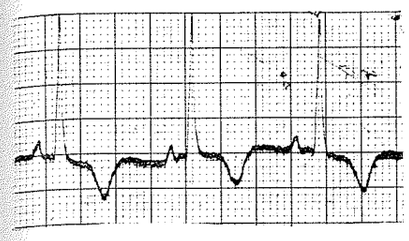
"BEFORE DEATH"

CASE No 3



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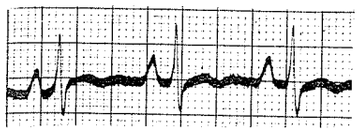

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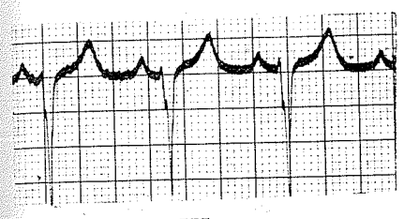

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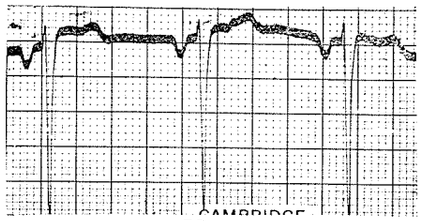

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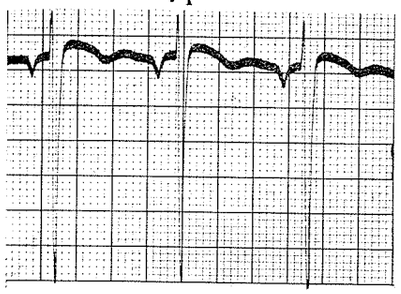

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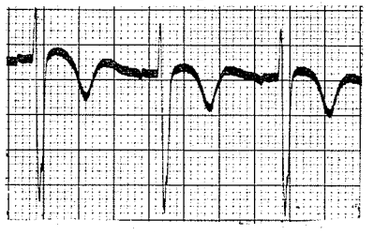
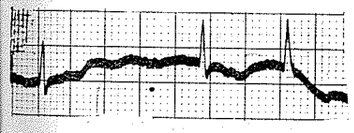
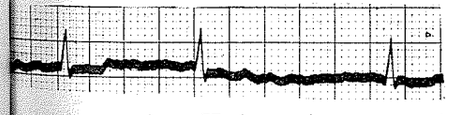


V_3

"ON ADMISSION"



I



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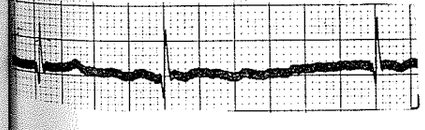

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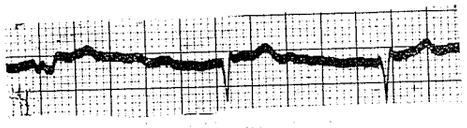
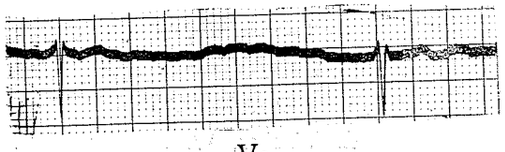


V_1



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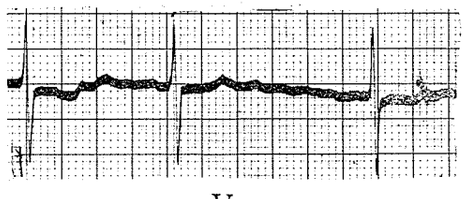

V_3

"BEFORE DEATH"

CASE No 4

Summery

Cardiac arrythmias are frequent complications in acute and chronic renal failure and they may well account for sudden unexplained death in these patients. Based upon our recent study, we strongly beleive that among other causative factors, focal degeneration of myocardium is the commonest and the most potentially dangerous cause of cardiac arrythmias. We suggest in emergency instances, ouabain to be used instead of digitalis which appears to be quite safe with dramatic results in cardiac arrythmias of these group.

Resumé

Les arythmies Cardiaques à l'uremie aigue et chronique sont relativement fréquantes, et souvent l'apparition de l'arythmie nous explique les morts sulutes de les malades. Sur la base de les études nous pensons que la raison la plus fréquante et la plus dangereuse de l'apparition des arythmies cardiaques chez les malades et la dégeneressance focal du myocarde et afin de traitement on choisit ouabaine qui est plus efficae par rapport à Digitale.

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LE MONGOLISME EN IRAN ✡

Par

Dr. Djalal Brimani ✡✡

Etude du Mongolisme en Iran ✡✡✡

Nous avons étudié un groupe de 14 mongoliens, examinés au Centre du Guidage Infantil à TEHERAN se divisant en 8 garçons et 6 filles.

Les origines raciales et religieuses des parents de ces enfants sont diverses:

Chrétienne dans un cas, juive dans 2 cas, musulmane pour le reste.

1- Les facteurs liés à la péristase:

Nous avons résumé dans les tableaux suivants l'âge des malades et de leurs parents lors de la naissance de nos mongoliens:

a) L'âge des enfants :

L'âge des enfants	de 2 à 5 ans	de 5 à 10 ans	de 10 à 15 ans
Nombre de cas	3	6	5

b) L'âge maternel:

Comme nous voyons dans le tableau ci - dessous, l'âge des mères au moment de l'accouchement du mongolien dépasse souvent 30 ans.

L'âge maternel	de 20 à 30 ans	de 30 à 40 ans	plus de 40 ans
Nombre de cas	6	5	3

(✡) Travail du Centre du Guidage Infantil, attaché à la Société Iranienne de la Protection de l' Enfance.

(✡✡) Chef de Clinique Neurologique à la Faculté de Médecine de Téhéran.

(✡✡✡) Les observations de ces enfants nous ont été confiées grâce à l'obligeance du Docteur DAFTARI, Directeur Général de la Société Nationale de la Protection de l'Enfance; du Docteur DJAFARIAN, Directeur du Centre du Guidage Infantil, et du Docteur MANSOUR, Directeur du Centre des Observations Psychologiques, auxquels je présente tous mes remerciements.