FIVE CASES OF CARDIOVASCULAR DISEASES
seen in a General Medical Ward
during a clinical attachment

------------------

1. Multiple cardiac arrests and resuscitation ............. Mrs Hannah Stoddart
2. Management of myocardial infarction .................... Mr Hugh Chapman
3. Investigation of hypertension ............................ Fr Dermot O'Sullivan
4. Aortic stenosis and incompetence ....................... Mr Walter Brotherstone
5. Sudden death due to cardiac asystole .................... Mr David Watt

submitted for
The Wightman Prize in Clinical Medicine

by

Vadakumkaraputhenpurayil Abraham Kurien

June 1963
COMPLAINTS AND THEIR DURATION

1. Angina of effort for over 18 months
2. Heavy period during the last 5 days
3. Severe chest pain for three hours before admission

HISTORY OF PRESENT ILLNESS

This patient who has had angina during the greater part of the last two years was admitted in March 1962 to the Royal Infirmary when she had an episode of severe retrosternal pain. At that time a clinical diagnosis of myocardial infarct was made, but electrocardiographic evidence was not present. She was put on long term anticoagulant therapy at that time. Her Phenindione dosage schedule is 75mgms - 75mgms - 100mgms - 75mgms - 75mgms ....... on successive days. This has kept her prothrombin activity well below normal. When she attended the 'prothrombin clinic' on 15th May 1963, her prothrombin activity was 17% of normal.

During the 15 months she has been on anticoagulant therapy, she has had angina of effort as well as occasional attacks of pain, not related to effort. She has also had intermittent swelling of the ankle and nocturnal dyspnoea.

On Friday, 24th May 1963, she started a heavy period with passage of clots, after amenorrhoea from last Christmas. On her own initiative she stopped her phenindione for two days. She had to go to bed as her angina got worse. She saw her doctor on the 26th May and he gave her a dose of ergot.

During the night previous to admission, she was awakened by a dull ache in her back which spread to her chest. By early morning, the pain began to get worse. Her doctor saw her at 8.30 A.M. and gave her 30 mgms of morphine. The pain became a severe retrosternal pain and persisted for almost three hours till admission. During this episode, she had profuse cold sweats and vomited once. The pain at the time of admission was retrosternal, going through to the back and down the left arm.

PAST MEDICAL HISTORY

Predisposed to 'bronchitis' in the winter
No rheumatic fever - no illness of note
FAMILY HISTORY

Father - died of myocardial infarct
Mother - hypertension and heart disease

Husband, who is an engineer, is alive and well
3 children - all alive and well

EXAMINATION

General
Looks quite fit, but complains of a lot of pain, of the following distribution

Good colour, perhaps slightly cyanosed, but not clinically shocked - no lymphadenopathy or goitre
Breasts normal

CVS
Pulse 82 per minute - runs of regular beats, with occasional dropped beats
BP 140/100 mm Hg
JVP not raised
Apex beat not palpable
Heart sounds - normal in all areas - occasional extrasystole
No ankle or sacral oedema
Peripheral pulses present
Pain in the neck, arm and over praecordium

RS
Trachea central - slight cyanosis
Expansion poor, but equal
Breath sounds vesicular in all areas - faint crepitations in the apex of both lower lobes - bases of lower lobes clear

AS
Mouth clean
Abdomen slightly obese
Liver 2 fb enlarged and tender
Speen not palpable - no other masses or areas of tenderness
Normal bowel sounds

CNS
No obvious abnormality, but full examination not carried out because the patient is in severe pain.

DIAGNOSIS AND TREATMENT ON ADMISSION

Myocardial Infarct
Morphine 10 mgms and 50 mgms of chlorpromazine at 11.25 A.M. Phenindione to be continued after prothrombin activity is assessed.

PROGRESS NOTES

When the patient was seen about an hour after admission, it was noticed that she was beginning to show signs of cardiac failure. The pulse rate had risen to 110 per minute with extra systoles. The JV pressure which was not raised on admission was 1 1/2 inches up at this time. Blood pressure had fallen to 130/80 mm Hg. However there were no clinical signs of shock, and the patient's extremities were warm. There were a few fine crepitations at the bases of the lungs. There was an unexplainable slight cyanosis on her lips and it was wondered whether, in view of the fact that she had taken large numbers of nitrite tablets, this was a methemoglobin cyanosis. The total clinical impression at that time was that the patient had had a severe myocardial infarction with gradually decreasing LV output.

At 1.15 P.M. the doctors were called to the patient's bedside, because she was seen to be in a convulsion. Palpation of the carotid pulses and auscultation of the heart made the diagnosis too obvious. The patient was in CARDIAC ARREST.

External massage started - Brook's Airway used
Intubated at 1.40 P.M. and external massage continued
ECG showed ventricular tachycardia and fibrillation
The patient's pupils were constricted, suggesting that adequate oxygenation was being maintained - the pulses were palpable

Defibrillated at 1.55 using 400 V current
Ventricular tachycardia followed

Digoxin 1 mgm intravenously
Hydrocortisone 200 mgms intravenously
Procaine amide 1.5 Gm intravenously in drip
Isoprenaline 0.1 mgm intravenously

BP 70/? mm Hg - pulse easily palpable - pink extremities
Patient awake - still ventricular tachycardia by ECG
Procaine amide 500 mgm IVI

Sinus rhythm re-established at 3.00 p.m.
P H of blood 7.064 - intravenous Sodiumbicarbonate
Nurse left to watch patient at bedside

CARDIAC ARREST II at 5.00 P.M.
External cardiac massage - oxygen by intratracheal tube.
BP fallen to 40mm Hg systolic
Metaraminol 5 mgm i.v.
Hydrocortisone 100 mgm intravenous injection
Normal rhythm

ventricular tachycardia

ventricular fibrillation

massage

idioventricular rhythm after defibrillation

2:1 block

3:1 block

"coupling"

Normal sinus rhythm restored
Respirations:

Pulse and Blood Pressure:

Temperature:

Time

Remarks:

Mrs. Hannah Stoddart

HALF-HOURLY CHART

CARDIAC ARREST 1

CARDIAC ARREST 2

CARDIAC ARREST 3

CARDIAC ARREST 4

CARDIAC ARREST 5

OXYGEN

Hydrocortisone 100 mg IV

Hydrocortisone 300 mg IV

Cardiac output 2.46 cm

"Acetamin" 10 mg IV

"Acetamin" 10 mg IV

"Acetamin" 10 mg IV

"Acetamin" 10 mg IV

Digoxin 0.25 mg IV
Mrs Hannah Stoddart

HALF-HOURLY CHART

30.5.1963

<table>
<thead>
<tr>
<th>Time</th>
<th>AM 2</th>
<th>AM 3</th>
<th>AM 4</th>
<th>AM 5</th>
<th>AM 6</th>
<th>AM 7</th>
<th>AM 8</th>
<th>AM 9</th>
<th>AM 10</th>
<th>AM 11</th>
<th>AM 12</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Temperature:</th>
</tr>
</thead>
<tbody>
<tr>
<td>30.5.1963</td>
</tr>
<tr>
<td>105</td>
</tr>
<tr>
<td>104</td>
</tr>
<tr>
<td>103</td>
</tr>
<tr>
<td>102</td>
</tr>
<tr>
<td>101</td>
</tr>
<tr>
<td>100</td>
</tr>
<tr>
<td>99</td>
</tr>
<tr>
<td>98</td>
</tr>
<tr>
<td>97</td>
</tr>
<tr>
<td>96</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pulse and Blood Pressure:</th>
</tr>
</thead>
<tbody>
<tr>
<td>30.5.1963</td>
</tr>
<tr>
<td>40</td>
</tr>
<tr>
<td>50</td>
</tr>
<tr>
<td>60</td>
</tr>
<tr>
<td>70</td>
</tr>
<tr>
<td>80</td>
</tr>
<tr>
<td>90</td>
</tr>
<tr>
<td>100</td>
</tr>
<tr>
<td>110</td>
</tr>
<tr>
<td>120</td>
</tr>
<tr>
<td>130</td>
</tr>
<tr>
<td>140</td>
</tr>
<tr>
<td>150</td>
</tr>
<tr>
<td>160</td>
</tr>
<tr>
<td>170</td>
</tr>
<tr>
<td>180</td>
</tr>
<tr>
<td>190</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiration:</th>
</tr>
</thead>
<tbody>
<tr>
<td>30.5.1963</td>
</tr>
<tr>
<td>10</td>
</tr>
<tr>
<td>20</td>
</tr>
<tr>
<td>30</td>
</tr>
<tr>
<td>40</td>
</tr>
<tr>
<td>50</td>
</tr>
<tr>
<td>60</td>
</tr>
<tr>
<td>70</td>
</tr>
<tr>
<td>80</td>
</tr>
<tr>
<td>90</td>
</tr>
</tbody>
</table>

Remarks:
- Awake
- Go to bed
- Tired
- Cold
- Sleep
- Tired
- Mean value
- Mean value
- Mean value
- Mean value
- Mean value
- Mean value
- Mean value
- Mean value
- Mean value

Graph:
- Arrows indicating changes in vital signs
CARDIAC ARREST III at approx. 5.45 PM - ventricular fibrillation
Patient brought back to ventricular tachycardia after complete heart block picture, by external massage and oxygen therapy
Hydrocortisone 300 mgm injected intravenously
Sinus rhythm restored

CARDIAC ARREST IV at approx. 6.15 PM
Again external massage - complete heart block - ventricular tachycardia - sinus rhythm
Quinidine drip with 2.4 Gms per 500 mls

CARDIAC ARREST V at approx. 6.40 PM
External cardiac massage
Aramine 20 mgm intravenously
Hydrocortisone 300 mgms by intravenous injection

7.00 P.M. 
A nasogastric tube was introduced instead of the endotracheal tube because the patient was conscious between attacks and intolerant of the tube

9.00 P.M. 
150 mgms of Sodiumbicarbonate and 300 mgms of hydrocortisone were added to the drip containing Quinidine

Patient in good shape - no further arrests since 7.00 P.M. 
A further dose of Digoxin 0.25 mgm given IMI at 1.00 A.M. 
Patient awake at 2.00 A.M. and at 4.15 A.M. Speaking coherently
Mersalyl, 2ml given IMI at 4.00 A.M. Hydrocortisone 100 mgm IVI at 6.00 A.M and Heparin 12,500 units IVI at 7.00 A.M.
Maintained overnight by 2 hourly injections of Metaraminol

10.00 A.M. 
urethral catheter passed - producing some urine
patient remained well overnight - well oriented - pink all over warm in the extremities, but hypotensive at 70 mm Hg systolic

30th May 1963
Digoxin 0.25 mgm IMI every day
Heparin 10,000 units six hourly
IV fluids: 20% laevulose
Parenterovite

31st May 1963
Cortisone acetate 50 mgms 4 hourly
Quinidine sulphate 200 mgm 6 hourly

1st June 1963
ACTH 60 IU twice daily for two days
Ampicillin 500 mgm stat and 250 mgms 6 hourly

2nd June 1963
Stop cortisone
Soluble penicillin 1 mega unit a day

3rd June 1963
Stop quinidine
Digoxin orally
## INVESTIGATIONS

### Blood

<table>
<thead>
<tr>
<th>Date</th>
<th>Hb</th>
<th>PCV</th>
<th>MCHC</th>
<th>WBC</th>
<th>N</th>
<th>L</th>
<th>M</th>
<th>B</th>
<th>ESR</th>
</tr>
</thead>
<tbody>
<tr>
<td>29th</td>
<td>93%</td>
<td>48%</td>
<td>29%</td>
<td>17,100</td>
<td>85%</td>
<td>11%</td>
<td>3%</td>
<td>1%</td>
<td>24mm/hr</td>
</tr>
<tr>
<td>30th</td>
<td>92%</td>
<td>48%</td>
<td>29%</td>
<td>17,100</td>
<td>85%</td>
<td>11%</td>
<td>3%</td>
<td>1%</td>
<td>20mm/hr</td>
</tr>
<tr>
<td>31st</td>
<td>85%</td>
<td>48%</td>
<td>29%</td>
<td>17,100</td>
<td>85%</td>
<td>11%</td>
<td>3%</td>
<td>1%</td>
<td>42mm/hr</td>
</tr>
<tr>
<td>3rd</td>
<td>65%</td>
<td>48%</td>
<td>29%</td>
<td>17,100</td>
<td>85%</td>
<td>11%</td>
<td>3%</td>
<td>1%</td>
<td>120mm/hr</td>
</tr>
<tr>
<td>4th</td>
<td>94%</td>
<td>48%</td>
<td>29%</td>
<td>17,100</td>
<td>85%</td>
<td>11%</td>
<td>3%</td>
<td>1%</td>
<td>28mm/hr</td>
</tr>
<tr>
<td>5th</td>
<td>99%</td>
<td>50%</td>
<td>30%</td>
<td>13,300</td>
<td>71%</td>
<td>21%</td>
<td>5%</td>
<td>1%</td>
<td>85mm/hr</td>
</tr>
</tbody>
</table>

### Biochemistry

<table>
<thead>
<tr>
<th>Date</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>HCO₃</th>
<th>Urea</th>
<th>SGOT</th>
<th>SHBD</th>
</tr>
</thead>
<tbody>
<tr>
<td>29th</td>
<td>132</td>
<td>5.2</td>
<td>99</td>
<td>22.3</td>
<td>78</td>
<td>109 U</td>
<td>745 U</td>
</tr>
<tr>
<td>30th</td>
<td>134</td>
<td>5.0</td>
<td>94</td>
<td>22.3</td>
<td>158</td>
<td>118 U</td>
<td>975 U</td>
</tr>
<tr>
<td>31st</td>
<td>128</td>
<td>5.2</td>
<td>88</td>
<td>21.0</td>
<td>249</td>
<td>211</td>
<td></td>
</tr>
<tr>
<td>1st</td>
<td>132</td>
<td>4.2</td>
<td>25.9</td>
<td>140</td>
<td>122</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Prothrombin Activity

<table>
<thead>
<tr>
<th>Date</th>
<th>Prothrombin time - Patient</th>
<th>Prothrombin time - control</th>
<th>Prothrombin activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>29th</td>
<td>15 secs</td>
<td>11 secs</td>
<td>36%</td>
</tr>
<tr>
<td>30th</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31st</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4th</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5th</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Other investigations

- **29th May** -
  - Buffer base: 32
  - CO₂: 16
  - pCO₂: 57
  - pH: 7.084

- **30th May** -
  - pH: 7.33
  - O₂ Saturation: 85%
  - Cholesterol: 251 mg/dL

- **4th June** -
  - Prothrombin activity: 35%

- **5th June** -
  - Prothrombin activity: 26%
Bacteriology

30th May - Blood culture - no growth

31st May - Catheter urine - small number of pus cells
few mixed epithelial cells
enterococci and Esch. Coli

Puradantin S: Ampicillin S

Midstream urine - very occasional pus cells
few sq. epithelial cells
contaminants only

1st June - Blood culture - no growth

sputum - large number of pus cells with few
disintegrating Gm negative bacilli
which failed to grow on culture

2nd June - Sputum - moderate numbers of pus cells with
few Gm positive cocci which failed
to grow on culture - no acid or alcohol
fast bacilli were seen

Midstream urine - few pus cells, mixed epithelial
cells and Esch. Coli and entero-
cocci

Combined sensitivities - SUR: PR: SR: CS: AR: TR:
Ampicillin S

24 hour Urinary Input - Output Chart

<table>
<thead>
<tr>
<th></th>
<th>Input</th>
<th>Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>29th</td>
<td>800ml</td>
<td>200ml</td>
</tr>
<tr>
<td>30th</td>
<td>1,000ml</td>
<td>410ml</td>
</tr>
<tr>
<td>31st</td>
<td>880ml</td>
<td>800ml</td>
</tr>
<tr>
<td>1st</td>
<td>1,160ml</td>
<td>1,600ml</td>
</tr>
<tr>
<td>2nd</td>
<td>2,810ml</td>
<td>2,180ml</td>
</tr>
</tbody>
</table>
Electrocardiograms

29.5.1963 - Sinus rhythm - rate 83/min. - PR interval 0.16 sec. The P waves are tall and peaked in lead LL suggesting right atrial hypertrophy. There is ST depression in leads I and V₄ to V₆ with diphasia of the T waves. These changes are due to ischemia, but there is as yet no evidence of infarction.

29.5.1963 - Comparison with the previous record shows significant change. Most of the record shows sinus tachycardia with a heart rate of 107/min. and PR interval of 0.16 sec. There appears to be a developing posterior infarct with marked contralateral ischemia. There are frequent ventricular extrasystoles.

short strips of lead II have been recorded showing a ventricular tachycardia and ventricular fibrillation.

1.6.1963 - Comparison with previous record of 29.5.63 shows significant change. There are now broad Q waves with ST elevation and terminal inversion of the T waves in lead III with ST depression persisting in leads I and aVL. and the developments of Q waves in aVF. These changes are indicative of recent transmural posterior myocardial infarction, although it is surprising that aVF does not have the changes in V₃ in respect of ST elevation.

3.6.1963 - Comparison with previous record of 1.6.63 shows slight change. Q waves persist as the initial deflection in V₃ but there is now no ST elevation in this lead where the T waves are now flat. There has been no significant change in aVF.

Further follow-up records will be necessary to confirm the presence of posterior transmural myocardial infarction.

4.6.1963 - Comparison with the record of 1.6.63 shows slight changes only. There is a broad Q with ST elevation in lead III and aVF but the ST elevation is less marked than it was previously. There is now also a broad Q wave in lead II with T wave inversion. The pattern is therefore clearly that of a posterior transmural myocardial infarction.

There is now less ST depression in lead I and aVL, but ST depression persists in leads V₂-V₆.
29th May - Chest - There is no obvious abnormality of the cardiac shadow - there is slight increase in lung field vascularity in the perihilar regions - otherwise clear

30th May - Chest - Shows slight collapse of the left lower lobe - no obvious abnormality in the cardiac shadow

4th June - Chest - The lung fields appear fully expanded and the cardiac shadow is normal
No fractured ribs seen
DISCUSSION

This is the dramatic story, rare in medical history, of a woman who has survived FIVE CARDIAC ARRESTS - all of them confirmed by ECG - after a severe myocardial infarct. At the time of writing, over a week after that exciting and strenuous afternoon, the patient shows no signs of mental or psychological defects which often result in decerebrate patients, brought back to life by external cardiac massage. This story clearly illustrates the effectiveness of external cardiac massage when carried out immediately after cardiac arrest and that perhaps at least theoretically, there seems to be no limit to the number of times a person can be resuscitated without the danger of decerebration, if equipment, personnel and careful management are available. It is also a tribute to the earnestness and devotion of the younger members of the medical and nursing profession.

When the patient was brought into the hospital, it was obvious from her history and the agony of her retrosternal pain that she had had a myocardial infarct. Though it seems most likely that she would have had an infarct at this time in any case, certain events during the week before admission seem to have precipitated matters considerably. After nearly seven months of amenorrhoea, she had a torrential haemorrhage per vaginum. It is reasonable to assume that her being on anticoagulant therapy has a definite part to play in this. She stopped Phenindione for two days, consulted her doctor who to stop her heavy periods gave her a 'small dose of ergot'. She was in the hospital within 24 hours with a history of over three hours of continuous and severe retrosternal pain.

There was very little to suggest, from the clinical picture on admission, that she would develop the serious complications that followed. She seemed generally fit, and her pulse was normal except for occasional extrasystoles. Her blood pressure was well maintained. In retrospect one wonders if the fairly high blood pressure were not really the peak that one usually sees before the phase of hypotension begins.

The patient had her first arrest at 1.15 P.M. and because there was immediate aid available, external massage was begun in less than 30 seconds. Defibrillation at 400 volts stopped the heart, which started again beating with the pattern of ventricular tachycardia. The most reasonable explanation of the cardiac arrest seemed to be impairment of atrioventricular or ventricular conduction due to oedema in the infarcted area of the myocardium. Steroid therapy in the form of Hydrocortisone intravenously in huge doses was indicated and immediately given. It also seemed rational therapy to depress the hyperexcitability of the myocardium by procaine amide given intravenously. Digoxin had to be given because the signs of failure were noted even before the cardiac arrest occurred.
Sinus rhythm was re-established only after over 1½ hours. It was noted that the pH of blood had fallen to the very dangerous level of 7.084. This can be explained as a result of the anaerobic oxidation that must have taken place during the time of the cardiac arrest. It was essential that this be corrected quickly and efficiently and so sodium bicarbonate was given intravenously.

Four cardiac arrests followed one after another at about half hourly intervals from about 5.00 P.M. External massage was started immediately in every instance because medical personnel were watching over the patient continuously. Sinus rhythm was restored after each arrest. More hydrocortisone, quinidine, Metaraminol and endotracheal oxygen tide the patient over each crisis.

There were two fears at the back of the minds of the resuscitation team. The first was whether, the patient's cortical functions would be severely affected. This was disproved when in the middle of the night the patient woke up to complain that she was feeling tired and cold! The other was whether the periods of severe hypotension through which she passed might have adversely affected her kidneys. This suspicion seemed to be confirmed when urethral catheterisation produced very little urine and blood urea rose to 249 mgm%. However, the gradual fall in the urea concentration and the rapid increase in urinary volume to normal values suggested that her renal tubules had not been damaged to any great extent.

The patient was started on intravenous heparin, intramuscular digoxin and in view of urinary and chest infection on ampicillin and benzyl penicillin. The area of collapse seen in a chest X-ray cleared up very quickly. Gradually steroids were stopped and ACTH used for 2 days. Quinidine was stopped on the third day. The only drugs she continues to receive are digoxin orally and heparin intravenously. She was given intravenous 20% laevulose for fluid and calories.

At the time of writing the patient is still in the hospital. The prognosis at the moment seems to be completely dependent on the prognosis of a rather extensive posterior myocardial infarct that she has. She seems to be none the worse for the numerous cardiac arrests.

Since the introduction of external cardiac massage as the treatment of cardiac arrest, in preference to internal cardiac massage, more attempts have been made to resuscitate patients who die suddenly. The technique of external massage and the 'kiss of Life' has put cardiac resuscitation into hands of nurses and house officers. This has brought with it the danger that indiscriminate use may be made of this technique to attempt to resuscitate almost every patient who dies in a hospital ward. So the criteria for the use of external massage has had to be defined especially in view of bringing back to 'life' decerebrate men and women. Under no circumstances should external cardiac massage be attempted if more than 4 minutes have passed since the arrest of the heart. This is
maximum period of time for which the CNS which was adequately oxygenated before the arrest can be expected to survive without showing signs of damage. The interval is much shorter in cases where the supply of blood or oxygen to the brain was poor before the arrest. Needless to say this rules out of consideration any case of generalised cerebral arteriosclerosis, cases of chronic bronchitis who are in a continued state of hypoxia. The eagerness to resuscitate any case of cardiac arrest has to be controlled by the age of the person, the general medical state of the person and the prognosis that awaits him/her if he/she were successfully resuscitated.

From this point of view the patient was an ideal case for resuscitation. She is a comparatively young woman of 46, who has been well throughout her life and whose only complaint has been severe angina of exertion. This is the first time she had had an infarct which has been confirmed by ECG. She is the mother of three children and an irreparable loss to the family at this time. Given an opportunity it seems likely that she would recover from her myocardial damage. Above all, on every occasion it was possible to start resuscitative methods within a few seconds of cardiac arrest.

One of the big dangers of external cardiac massage is the ease with which the thoracic cage can be damaged due to the application of excessive pressure. The likelihood of this occurrence is multiplied many times when resuscitative procedures have to be employed on a number of occasions within half an hour of each other. It is to the credit of the resuscitation team that not one rib was fractured. Such careful management reduces the chest complications which may otherwise follow. The other main problem of repeated cardiac arrests is the tendency to severe hypotension with its effect on the kidney. As in this case injections of a pressor agent can maintain an adequate systolic pressure, when the myocardium itself is not capable of maintaining it.

It is interesting to speculate on the reasons for these repeated cardiac arrests. It is now believed that most of the cases of myocardial infarcts which die before reaching medical assistance do so because the myocardium goes into asystole or fibrillation due to conduction defects. Extrasystoles after a myocardial are so common that this tendency to hyperexcitability has occasionally to be suppressed by quinidine therapy. The first cardiac arrest therefore seems to be easy of explanation. It has been suggested recently that conduction defects following a myocardial infarct respond to treatment with hydrocortisone because it prevents the inflammatory reaction around the bundle of His. In this particular case the patient received over 1 Gm of hydrocortisone in the first 24 hours. The repeated arrests may therefore be explained as due to conduction defect. On the other hand it is likely that another important factor may be involved. This is the pH of blood. After the first arrest, the pH fell to 7.084 - a very unphysiological level. There was no marked change in K levels in the blood. It seems reasonable to assume that the very low pH may have also had a big
part to play in the production of the arrests. One would like to believe that the correction of this state of affairs by the IV administration of Sodium bicarbonate did help to stabilise the rhythm of the myocardium. There has been no irregularity of rhythm after the last arrest and one hopes that this state of affairs will continue.

The immediate prognosis therefore seems good. She has already been on anticoagulant therapy for over a year. Whether or not a rapid fluctuation of prothrombin activity, caused by the bleeding as well as the stoppage of Phenindione for two days, along with ergot was responsible for this infarct it is difficult to say. However, one thing is clear. The patient needs closer control and evaluation of her anticoagulant therapy. There seems to be a heavy hereditary loading for coronary heart disease in the family. It may be worth investigating the lipid pattern of her blood to see if there is another possible line of treatment.

Irrespective of what may lie ahead of the patient in the next few weeks of her stay in the hospital or the few years of her life she has now left, a tribute must be paid to the excellent and effective resuscitation of this patient, without neurological, psychological, renal damage.

--------------

On the day of admission the patient visited once in the morning and once in the evening. On the first occasion it contained bile, whereas on the second occasion, the visitas contained the food he had just eaten.

Between these two 'attacks' of pain, there is no history of angina or intermittent clausaration.

PAST MEDICAL HISTORY

Right inguinal herniography 1950
Second repair 1955
No tropical illnesses (patient was in Malaya for 3 years, 57-60)

SYSTEM REVIEW

CVS Excrion tolerance only moderate - takes very little exercise - no ankle swelling.

RS Brokes at least 40 cigarettes a day and has done so for considerable time - started cigarette smoking at the age of 11. Has had 'smoker's cough' for 20 years.
HUGH CHAPMAN
4, Hamilton Terrace,
EDINBURGH

Age 43 years
Occupation Warrant Officer
Date of admission 3rd May 1963

COMPLAINTS AND THEIR DURATION

1. Retrosternal pain two days before admission, while at work
2. Retrosternal pain about 4 months ago, while sleeping.

HISTORY OF PRESENT ILLNESS

This man was well until 4 months ago, when he woke up from sleep one night with a gripping retrosternal pain, with severe sweating. He drank a glass of water and the pain disappeared in about half an hour. He thought no more about it until two days before admission, on 1st May, at about 9 AM while at work he developed a similar but more severe pain. The pain left him for a while, but came back at 10 AM with a greater intensity, radiating this time to below his shoulder blades and down his left arm. This pain subsided at about midday. The pain came on again at about 1.30 PM and he decided to stop work and go home. The pain never came on again, but he felt very shaky and tired. He has remained in bed since and on one occasion felt a twinge of pain in his left shoulder on stooping down to pick up his slippers.

On the day of admission the patient vomited once in the morning and once in the evening. On the first occasion it contained bile, whereas on the second occasion, the vomitus contained the food he had just eaten.

Between these two 'attacks' of pain, there is no history of angina or intermittent claudication.

PAST MEDICAL HISTORY

Right inguinal herniorrhaphy 1950
Second repair 1955
No tropical illnesses (patient was in Malaya for 3 years: 57 - 60)

SYSTEM REVIEW

CVS Exercise tolerance only moderate - takes very little exercise - no ankle swelling

RS Smokes at least 40 cigarettes a day and has done so for considerable time - started cigarette smoking at the age of 5! Has had 'smoker's cough' for 28 years
occasional sputum -never blood stained - no dyspnoea

AS Appetite good - weight increasing
No haematemesis or malena - bowels regular
Has been drinking fairly heavily - has slowed up since Easter this year

GUS No frequency or dysuria
no urinary symptoms

CNS No headaches
No visual disturbances

FAMILY AND SOCIAL HISTORY

Father - died of heart disease at 63 years
Mother - died at 78 years of CVA - was a diabetic
Three siblings - no heart trouble
Wife, 2 sons and a daughter, alive and well

Has been a warrant office for 13 years
Job entirely sedentery - The patient says it involves a lot of responsibility and worries

EXAMINATION

General Looks well, but considerably overweight
not shocked or cyanosed
No lymphadenopathy
Mucous membranes well injected
Thyroid not enlarged

CVS Pulse 110/min. -regular in time and force
BP 110/80 mm Hg
JVP not raised
Apex beat in the 5th left interspace on the MCL
Heart sounds I and II closed in all areas
No ankle or sacral oedema
Peripheral pulses are all present and equal

RS Trachea central
Expansion fair and equal
Percussion shows no abnormality
Breath sounds vesicular - a few scattered vesicular rhonchi
No basal crepitations

AS Tongue clean - throat well injected
Abdomen soft, but very obese and moves on respiration
No tenderness or guarding
no hepatosplenomegaly
Hernial orifices intact
PR normal

GUS
No pain in renal angles - kidneys not palpable
Genitalis normal - no scrotal masses

CNS
cranial nerves intact
Fundus normal

\[
\begin{array}{cccccc}
L & + & + & + & + & \\
R & + & + & + & + & \\
\end{array}
\]
Reflexes

DIAGNOSIS AND TREATMENT ON ADMISSION

Myocardial Infarct

Phenobarbitone 30 mgms t.i.d.
Obesity diet - 600 cals - reduction in smoking
Anticoagulants - Phenindione - heparin

PROGRESS NOTES

No further episodes of pain in hospital
Lost nearly 10 lbs weight in 3 weeks' stay in hospital
Has not reduced number of cigarettes smoked very considerably

Discharged on 21st May on anticoagulants
Phenobarbitone 30 mgms t.i.d.
INVESTIGATIONS

Blood 4th May 1963
- HB 101%  - WBC 6,700  - ESR 56 mm/hr
- SGOT 24 units
- SHBD 480 m units/ml
- Urea 35 mgms per 100 ml

15th May 1963
- Cholesterol 331 mgm%

Prothrombin activity
9th May 1963
- Prothrombin time - patient - 22 secs
  - control - 11 secs
- Prothrombin activity - 19%

Electrocardiogram - Sinus rhythm. PR interval 0.18 sec, ventricular rate 88 per min. There is right axis deviation.
R is the major deflection in lead V1 and this is probably due to RBBB. There is a broad Q wave in lead aVL with slight ST elevation of this lead and these changes may indicate high anterolateral infarction. The record is abnormal and should be repeated including high anterior chest leads.

4.5.1963
The patient is smoking 40 cigarettes a day. He was noted to have been working in a job prone to myocardial infarction. It is therefore important to reduce this source of stress, if possible, to his lifestyle. If this is likely to be a problem, it may be necessary for a change in his expression of worries, associated with his job. An example might be something away from work, such as fishing. Further records should include V7-V9 and leads above V5 and V6. The record is suggestive of right ventricular hypertrophy.

8.5.1963
Comparison with the record of 4.5.63 shows little change. There is again marked right axis deviation and a dominant R wave in V1. The changes in aVL may be positional in character. There is no definite evidence of myocardial infarction. Further records should include V7-V9 and leads above V5 and V6. The record is suggestive of right ventricular hypertrophy.

Xrays 4.5.1963 - Chest - There is no evidence of a localised pulmonary lesion, but the quality of a portable film is not sufficient for further comment.
DISCUSSION

This patient presents with one of the commonest of symptoms in males in the fifth decade of life - namely, retrosternal pain. The clinical diagnosis of myocardial infarct was made by the General Practitioner, the Consultant who saw the patient on a domiciliary Consultation and by the hospital staff. However, the ECG evidence is not very convincing. This may be because, as indicated in the ECG report, the infarct is at a higher level than the scope of the normal chest leads. It is a pity that leads were not recorded from the third and fourth left interspace. The ESR is elevated and so is the SHBD level.

The patient showed no signs of cardiac failure or shock on admission. So the management of the case consists essentially of bed rest and adequate sedation, in a person who seems to be burdened with a lot of responsibility. Three other points need special consideration. They are anticoagulant therapy, reduction in weight and an attempt to reduce the number of cigarettes he smokes each day.

As far as anticoagulant therapy is concerned there is no doubt at all that in such a case its benefits are enormous during the stay in hospital. The patient had repeated attacks of fairly severe pain during the day, unrelated to effort. If each one of them represents an episode of thrombosis, however small the vessels involved, it is essential that such recurrences should be prevented. So the patient was started with heparin and phenindione, the latter being controlled by prothrombin activity.

The patient is manifestly overweight and the opportunity to reduce the load on the myocardium by reducing weight is taken by putting him on a 600 calorie diet.

The patient has the most incredible history of cigarette smoking. He started smoking at the age of 5 and before admission smoked no less than 40 cigarettes a day. He has had a chronic cough for 28 years. While a causal correlation between cigarette smoking and coronary heart disease has not been worked out, it is known that heavy smokers are statistically more prone to myocardial infarcts. The strong suggestion was therefore made that he reduces the number of cigarettes to at least half the number he is used to. If as is likely in this case, this addiction to heavy smoking is an expression of worries associated with his occupation, sedation and bed rest away from work should help him to achieve the aim of reducing his smoking habits to a more sensible level.

The patient's condition remained excellent during his stay in the hospital and it was felt that he could continue his convalescence at home, for about 4 weeks more before he returns to work. He was therefore discharged on phenobarbitone and anticoagulant therapy.
The management of myocardial infarct is never complete at the time the patient leaves the hospital to continue his convalescence. In persons over the age of 60-65 years, who have retired from active work, further management seldom presents great difficulties. However in a younger patient the therapeutic regime has to be something more positive than advice to be 'careful' about his heart.

Here is a comparatively young person of 43, with a family of three children dependent on him, who has had two 'attacks' of pain about 4 months apart, suggestive of myocardial infarcts. We do not know if the first attack was in fact an infarct, as no ECG records were taken at that time. The fact that he was able to go to work during the following weeks suggests that it was not anything very extensive in nature, even if it were an infarct. Before considering the more controversial points about long term anticoagulant therapy and efforts to lower his cholesterol levels, there are some simple points which will make a considerable difference to the prognosis.

First of all comes the fact that he is more than 2 stones heavier than the 'ideal person' of his age and height. He seems to have been steadily gaining weight during the last few years. The sources of calories in this case are too much food and too much of alcoholic drinks. This high intake of calories achieves pernicious significance when one recalls the truth that his work is entirely sedentary and that he has taken very little exercise during the past few years. His myocardium is carrying an unnecessary load at the moment and if he can get down to about 9 stones and remain there for the rest of his days, they shall indeed be prolonged. He seems very averse to the idea of taking exercise of some kind. It needs to be impressed upon him that his body needs some regular exercise to keep it in good order.

Secondly, no efforts must be spared to make him drastically reduce his cigarette smoking. We have evidence on his body, in the shape of two scars for herniorrhaphy, that the effect of his chronic cough has not been negligible. In as much as his ECG records suggest the possibility of RV hypertrophy, it seems reasonable to postulate that he has a considerable degree of chronic bronchitis and emphysema. While it may be true that a fair degree of damage to his lungs has already been done, it may still be not too late to prevent further changes in the respiratory system.

The patient was discharged home on anticoagulants. A decision has now to be made if the therapy should be gradually tailed off, or whether there is a case here for long term anticoagulant treatment. Recently the pendulum of anticoagulant treatment in myocardial infarction has begun to swing furiously between the two extremes after a decade of relatively uncontested use in the acute phase of infarction as well as as a prophylactic agent against future episodes. The debate seems to centre
around two main points. Firstly, the argument seems to be about the long
term use of anticoagulants as a prophylactic measure in view of possible
serious side effects. Secondly it involves the criteria to be used in
selecting patients for anticoagulant therapy. Where there are no facilities
for adequate control of patients with fairly frequent estimations of the
prothrombin activity and in patients who have bleeding disorders or sources
of gastrointestinal haemorrhage, the decision is not difficult to take.
An attempt has recently been made to separate patients into good risk and
poor risk patients taking into consideration the previous history of the
illness, clinical features at the time of infarct and age and to indicate
that anticoagulants are more valuable in poor risk patients. It has been
suggested that the greatest benefit from anticoagulant therapy is obtained
in the first year after the infarct and that therefore anticoagulation on
a life-long basis is unjustifiable. The debate is far from being over.

The patient has no contraindication to anticoagulant therapy
from the previous medical history and clinical examination. He is a young
man who must be offered any method that might conceivably reduce chances
of further episodes. He is an intelligent person who understands the
dangers of prolonged anticoagulation and appreciates the need for regular
attendance at the 'Prothrombin Clinic'. He can be entrusted to steer clear
of aspirin and aspirin containing stomach powders and headache pills. From
the history it is clear that he has no anginal symptoms and is more prone
to pain at rest, which is more in favour of thrombotic phenomena. In such
a situation the patient must be given the benefit of doubt about the
efficacy of anticoagulant therapy as a prophylactic agent, even though
there is considerable debate about its value.

To enter into an even more controversial subject, one has
to consider if there is any justification for reducing his cholesterol
level in blood. Much has been written and said about the role of athero-
sclerosis and cholesterol in the causation of myocardial infarcts. Attention
has been concentrated on methods to reduce blood cholesterol by the
ingestion of unsaturated oils and the exclusion of animal fats from the
diet. Synthetic drugs of various kinds including hormones have been used
for the same purpose. The evidence brought forward to show the beneficial
effect of this 'programme' has been convincing in relation to the lowering
of cholesterol, but not to any significant reduction in the occurrence of
myocardial infarcts. It may be possible that the patient may be prepared
to follow fancy diets of low animal fats, corn oil etc... but the benefit
that may accrue is unlikely to be significant. On the other hand, as has
already been mentioned, a reduction in his weight by using a low calorie
diet will bring greater rewards.

Finally, one has to take into account the psychological
aspect of the situation. The patient is overburdened at the moment with
responsibilities. This must be remedied by relegating some of his work
and worries to others in his office. It may be necessary to continue some
form of sedation for a few weeks beyond the period of convalescence. The patient's own doctor should discuss with the patient ways and means by which the latter can be relieved of some of the psychological stress his work involves.

If in the attempt to rehabilitate this patient, we make him a cardiac invalid, by fixing his attention too much on his 'heart', more harm may perhaps be done than if we had allowed him to carry on as if this episode in his life were not of any great medical significance. In the eagerness to put him on long term therapy of any kind, this has to be borne in mind.
HISTORY OF ILLNESS

This patient was admitted to the hospital on 30th January, 1962 with a right hemiplegia and severe motor dysphasia. He had been in very good health till the afternoon of that day, when on his return from Glasgow while getting off the train at the station he was "struck down". When he came to himself he found his right side paralysed and his speech seriously affected.

The most significant finding at that time besides the neurological findings (flaccid paralysis on the R side: complete absence of deep reflexes on the same side: plantar extensor on that side and right sided homonymous hemianopia) was the raised systolic and diastolic pressures of 230/128 mm Hg and grade II retinopathy. The ECG showed evidence of LV hypertrophy and strain.

After routine investigations it was decided to put the patient on hypotensive therapy, starting with 20 mgms of guanethidine a day and gradually increasing to 50 mgms a day on discharge. His BP readings did not show any marked reduction: 220/110 mm Hg, lying and 200/110 mm Hg standing. He was followed up in the Out-patient Clinic and the dose of guanethidine increased to 60 mgms a day. When he was last seen in the OPD on 9th January 1963, his BP readings were 220/110-lying and 160/104 standing. It was felt that at that time that he should be admitted into the hospital for a complete investigation of his hypertension and for modification of treatment if indicated.

During the year since the onset of his hemiplegia, he had made a fair degree of recovery. His speech was completely recovered, except for a slight slurring. He was still not able to walk very far and was not doing any parish visits. His arm was very good but tended to go numb when it was cold. Eye sight was restricted though there was considerable improvement. There was still slight diminution of pinprick sensation on the right side.

He was admitted to the hospital on 17th April 1963 for reassessment. His main complaint at that time was postural hypotension on getting up in the morning.
EXAMINATION

General
Cheerful person, walks with a slight limp - slight slurring of speech - no cyanosis - good colour
No lymphadenopathy - no thyroid enlargement - no clubbing
? Slightly overweight

CVS
Pulse 72 per minute - regular in time and force
BP lying 190/130 standing 160/110
JVP not raised
Apex Beat in the 5th left intercostal space on MCL
Heart sounds - splitting of both heart sounds
accentuated second sound at aortic area
No ankle or sacral oedema
Peripheral pulses present - carotids equal, no stenosis

RS
Trachea central
Expansion good and equal
Breath sounds vesicular in all areas - no accompaniments

AS
Tongue moist and clean
No masses palpable in the abdomen
Hernial orifices intact

GUS
Kidneys not palpable
No pain in renal angles
No scrotal enlargements

CNS
All cranial nerves intact, except for Right temporal lower quadrantinopia - fundus grade II
No diminution of sensation on right side of face
Right hemiparesis - diminished pinprick on right side
other sensations normal
very slight increase in tone
No loss of muscle power in the arm - slight
weakness in the R leg

Reflexes

<table>
<thead>
<tr>
<th>B</th>
<th>T</th>
<th>S</th>
<th>K</th>
<th>A</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>L</td>
</tr>
</tbody>
</table>

Reflexes

| R | - | - | - | - | ↑ |

DIAGNOSIS
Residual neurological deficits of right hemiplegia
Right hemianaesthesia
Right temporal lower quadrant loss of visual field
Hypertension
INVESTIGATIONS IN 1962

**JANUARY - MARCH**

**Blood**
- Hb 107%
- WBC 18,900
- ESR 20 mm/hr

- **BUN** - 17 mg/dL
- **Prothrombin activity** 55%
- **Serum bilirubin** - 0.4 mg/dL
- **SGOT** - less than 20 units
- **Alkaline Phosphate** - 13 K-A units
- **Zn turbidity** - 1 unit
- **Thymol Turbidity** - 1 unit

**Urine**
- trace albumin, otherwise NAD
- MSU - few epithelial cells - no growth

**Lumbar**
- Puncture Pressure 230 mm Hg of CSF - rises to 270 Quickendenst
- Free rise and fall

**CSF**
- **Protein** - 57 mg/dL
- **Glucose** - 55 mg/dL
- **Chloride** - 720 mg/dL
- **Cell count** - 100/cu.mm on 1.2.1962
- 8/cumm on 15.2.1962
- Wasserman negative
- Gold test - 0000011110 on 1.2.1962
- 0000000000 on 15.2.1962

**ECG**
- **Left Ventricular hypertrophy and strain**

**Xray**
- Chest - lung fields clear
- skull - normal
- IVP - normal

**EEG**
- essentially normal - some reduction in activity amplitude of waves on left side

**IN APRIL 1963**

**Blood**
- Hb 95%
- WBC 11,300
- ESR 5 mm/hr
- N 60%
- L 32%
- M 6%
- E 2%

**Biochemistry**
- Sodium 137 mEq/L
- Potassium 4.7 mEq/L
- Chloride 97 mEq/L
- HCO3 22.4 mEq/L
- BUN 40 mg/dL
**Urine** 20.4.1963
Volume 1300 ml/24 hrs
Urinary creatinine: 120mgms
Concentration: 92 mgms/1
Serum Creat. concen: 1.2 mgm/1
Creatinine Clearance: 77mls/min.

22.4.1963
Creatinine clearance: 82mls/min

24.4.1963
Creatinine clearance: 75mls/min

**X-rays** 19.4.1963
The C/T ratio equals 155/300 - some LV enlargement - aorta is unfolded
lung fields are clear

23.4.1963
Heart screening - There is calcification of the left coronary artery affecting the descending
and circumflex branches - plaques of calcium are also present in the right coronary artery
The penetrated view of the chest shows an enlarged heart and an unfolded aorta - The lung
fields are clear

**Radio-hippuran Renogram**
26.4.1963
The tracing is not entirely satisfactory from the technical point of view, but it shows
evidence of good function in each kidney and there does not appear to be any significant
disparity in function between the two organs

**BLOOD PRESSURE PICTURE WHEN IN HOSPITAL - ON METHYL DOPA 250 mgm 2 - 4x a day**
DISCUSSION

This patient who is a Roman Catholic priest was first admitted into the hospital with neurological signs suggestive of a cerebrovascular accident. The sudden onset might have suggested an embolic phenomenon, but there was no identifiable source in the heart or in the carotid vessels. The permanence of the neurological phenomena even a year after the episode militates against the diagnosis of hypertensive encephalopathy. The most probable diagnosis therefore is of a cerebral haemorrhage secondary to hypertension. The transient rise of cells in the cerebro-spinal fluid, in the absence of xanthochromia suggests this diagnosis.

At that time he was put on hypotensive therapy, which has not been very successful in reducing his diastolic pressure. Hence it was felt that it was justifiable to assess his hypertension more fully than was done previously and to investigate his response to Methyl Dopa.

This patient had always been in very good health, till the cerebral vascular accident, at which time it was discovered that he had a moderate degree of hypertension. The uncovering of the existence of hypertension after such an episode is not an unusual event in the practice of medicine. It seemed very unlikely to be a hypertension secondary to some other cause, but it was felt that in as much as the patient was only 48, it would be worth investigating him. However, the fact that he has presented with a serious complication of hypertension suggests that the condition is well established and that even if it were secondary in nature, considerable amount of irreversible damage has been done.

In the history or clinical examination, there is nothing to incriminate endocrine abnormalities or coarctation of the aorta. The only likelihood is therefore limited to that of renal disease. The clearance tests, blood analyses and renogram gave no indication of renal abnormality. In a younger person, it may have been justifiable to push ahead with aortogram and divided renal function studies. In this case they are hardly justifiable in view of the clinical picture.

During his second admission to the hospital, it was decided to change his hypotensive drug from Guanethidine to Methyl Dopa. It was started at 250 mgms twice a day and gradually increased to 250 mgms four times a day. At the time of discharge it was very doubtful, if Guanethidine was any less effective. However, the one advantage of Methyl Dopa in this particular case seems to be that the patient who complained of severe postural hypotension before did not do so now.

It is very tempting to ask if a 15 month period on Guanethidine has done the patient any good. It must be admitted that there has been no deterioration in the creatinine clearance. The retinopathy has stayed at Grade II, the level at which it was a year ago. His neurological deficits
not been made good to any significant extent. There has been a slight deterioration in his cardiac status. While there was no sign of cardiac enlargement during the first admission, it is present during the second admission. Though there is no history of angina of effort, on screening of the heart, considerable calcification is present in the coronary arteries.

There is no doubt that the patient is severely handicapped by the effects of the cerebral haemorrhage and by the side effects of hypotensive therapy. His inability to walk considerable distances has prevented him from undertaking his Parish visits. The postural hypotension caused by Guanethidine seemed to cause considerable distress. In such a situation, it is very difficult to decide whether the patient's comfort or the control of hypertension should be the goal of therapeutic efforts. It seems very unlikely that physiotherapy would restore any more function to his right leg. Therefore it may be necessary for the patient to accept the fact that he shall have to give up any hope of being able to visit all his parishioners in their homes.

It seems to be difficult to ascribe to the drugs the credit for maintaining the status quo, especially because they have not consistently or substantially reduced the blood pressure. The fact that he feels faint on standing, while the pressure is 160/110 mm Hg suggests that his cerebral flow is not very adequate at that systolic pressure.

One would have liked to think that on hypotensive agents, this patient may be able to live longer, protected from the ill effects of hypertension on his CNS and kidneys. However, the effect of hypertension on his heart may present a less optimistic prognosis. Though there is still no history of myocardial ischemia, the X-ray and ECG findings suggest that an episode of myocardial infarct or left ventricular failure cannot be far away. Reduction of diastolic pressure much further would be necessary to prevent this outcome. While higher doses of methyl dopa or guanethidine may achieve this, the price to be paid in side-effects may be far too great. One can only hope that this patient has a reasonably benign form of essential hypertension and that his myocardium may stand the strain for some few years by hypertrophy.
COMPLAINTS AND THEIR DURATION

1. "Flu" about a month ago

2. Lack of sleep due to bouts of cough during the last few days before admission

HISTORY OF PRESENT ILLNESS

This patient had an attack of "flu" about 4 weeks before admission. It started as an infection of the upper respiratory tract, but later went into his chest. He then developed pain all over his body. After a few days he felt better and thought he had "shaken it off". However he developed an irritating cough and felt he had "something to get rid of down in the front of my chest". He stopped smoking at this time in the hope that it would help clear up the cough. His sputum became yellowish at this time and he went to see his doctor who put him on a 3 day course of oral penicillin and cough mixture. The cough eased up during the day, but continued at night and prevented his getting adequate sleep. He would wake up at night with a bout of cough, become breathless after that and feel he had to sit up to make himself comfortable. His doctor gave him some new tablets, but his cough at night continued to interfere with his sleep.

PAST MEDICAL HISTORY

In 1956 while working for long hours each day, the patient noticed that he felt asleep at work. He was very easily exhausted. He was referred by his GP to a Consultant Cardiologist at that time. No special treatment was given at that time except rest in bed for three months. He went back to work and has been carrying on without difficulty since.

In 1958 while on holiday, he woke up one morning and discovered that he was blind in his right eye. The loss of vision did not improve with time. He was seen in the Eye Department and the condition in the right eye was diagnosed as due to 'heart damage'.

He had an attack of 'virus pneumonia' about 2 years ago. He felt febrile and complained of rigors and headaches and malaise. He was in bed for a week at that time.

He has not had rheumatic fever or scarlet fever. He has had the exanthemata of childhood and jaundice.
SYSTEM REVIEW

CVS feels a heaviness in the praecordium on overwork, but can walk as fast as anyone else. No paroxysmal nocturnal dyspnoea - no ankle swelling. Exercise tolerance good.

RS used to smoke about 20 cigarettes a day - stopped a month ago has had a slight cough for a year or so. not much sputum before recent episode - no chest pain no haemoptysis

AS Appetite good till a month ago - has lost 10 lbs during the last month - bowels regular no dyspepsia - no malena

GUS No nocturia, dysuria or other urinary symptoms

CNS no headaches - loss of vision, mainly central, in the right eye for about 5 years - no dizziness

SOCIAL HISTORY

Laundry manager - works hard and very successful in business Likes to go on holidays and get out into the open air

FAMILY HISTORY

Father - died of heart disease Mother - died of old age Wife - has had ileostomy for ulcerative colitis 2 children - one boy and one girl - both alive and well

EXAMINATION

General - Looks well, but slightly apprehensive No cyanosis or jaundice No lymphadenopathy - mucous membranes well injected No thyroid enlargement No clubbing or splinter haemorrhages

CVS Pulse 90 per min. regular in time and force - peak not sustained BP 130/85 mm Hg. JVP not raised Apex Beat - in the SIXTH left intercostal space on the Anterior axillary line Diffuse and heaving in character
Heart Sounds

Aortic area
Neither heard distinctly. They are replaced by two murmurs giving a see-saw effect. Grade IV harsh grating systolic ejection murmur radiating into neck and apex - also heard all over the praecordium and anterior chest wall. Marked systolic thrill with patient sitting forward and holding breath in expiration.

Pul. area
Faint first and second heart sounds along with the aortic murmurs in systole and diastole.

Tri. area
Only the aortic murmurs are audible, the diastolic more marked than the systolic.

Apex area
Faint first sound - systolic murmur not radiating into axilla and harsh - most likely to be propagated aortic systolic second sound not heard - propagated diastolic murmur from the aortic area. No thrills.

Slight ankle and sacral oedema - femoral pulses not delayed peripheral pulses present.

Trachea central
Expansion fairly good and equal anteriorly - slightly diminished posteriorly on the right side.

Percussion revealed no abnormality.

Breath sounds - vesicular in all areas - diminished in the middle zone on the right posteriorly - basal crepitations on both sides.

Vocal resonance - diminished in posterior right mid-zone.

Tongue clean - fauces clear.

Liver enlarged 2 fingerbreadths below costal margins - not tender not nodular - no splenomegaly.

Hernial orifices intact.

Kidneys not palpable - no tenderness in renal angle.

No scrotal swellings.

All cranial nerves intact, except for central scotoma in the right eye.

Fundoscopy - right disc very much paler than left arteries and veins much smaller on the right.
DIAGNOSIS AND TREATMENT ON ADMISSION

Left Ventricular failure secondary to Aortic stenosis and incompetence
Pneumonic process in the right lung

Digoxin 0.5 stat, then 0.25 mgm q.i.d.
Neonaclex 5 mgms 3days a week
KCl 3 Gms on alternate days

PROGRESS NOTES

Patient has responded well to treatment
Blood cultures excluded possibility of SBE

11th May '63 Seen by consultant cardiologist
Opinion: Aortic Stenosis - dominant
Aortic incompetence
Grade IV on admission
Recommend:
1. phenobarbitone
2. full digitalisation
3. increase diuretics - either 10mgm neonaclex
or 2cc mersalyl 3x week
4. repeat xray when failure fully controlled
5. reconsideration thereafter for aortography
and LV catheterisation as assessment for
possible but equivocal aortic surgery

21st May '63 Patient discharged - to come back to hospital in 6 weeks
Digoxin 0.25 mgm b.i.d.
bendrofluozide 10 mgm 3x week
KCl 3 Gm on the other days of the week.
### INVESTIGATIONS

<table>
<thead>
<tr>
<th>Blood</th>
<th>2.5.1963</th>
<th>Hb 92%</th>
<th>WBC 11,500</th>
<th>ESR 29mm/hr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>M 78%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>L 19%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>M 2%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>E 1%</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Urine</th>
<th>2.5.1963</th>
<th>Colour</th>
<th>amber</th>
<th>S.G.</th>
<th>1020</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sugar</td>
<td>ve</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Protein</td>
<td>ve</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acetone</td>
<td>ve</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bile</td>
<td>ve</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Micro</td>
<td>No RBC seen</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Biochemistry</th>
<th>2.5.1963</th>
<th>Sodium 138 mEq/l</th>
<th>Bicarbonate 25.3 mEq/l</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Potassium 4.3 mEq/l</td>
<td>BU 30mEq%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chloride 102 mEq/l</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bacteriology</th>
<th>3.5.1963</th>
<th>Blood culture</th>
<th>No growth</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4.5.1963</td>
<td>Sputum</td>
<td>Commensals and a small number of pneumococci</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>No alcohol or acid fast bacilli</td>
</tr>
<tr>
<td></td>
<td>8.5.1963</td>
<td>Blood culture</td>
<td>No growth</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wasserman and Kahn</td>
<td>negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>negative</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>X-rays</th>
<th>1.5.1963</th>
<th>Chest X-ray</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Marked cardiac enlargement - CT ratio 195/340</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulmonary vascular congestion and pulmonary oedema</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Septal lines in costophrenic angle</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ossification in Right Midzone is heavier than and more patchy than opacities elsewhere - may be due to infarction or infection superimposed on pulmonary oedema</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Posterior segment of upper lobe and the anterior segment to some extent seem to be involved</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.5.1963</td>
<td>Heart screening</td>
</tr>
<tr>
<td></td>
<td>Very gross calcification in the aortic valves - no mitral calcification - LV considerably enlarged</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RV and both atria appear normal</td>
<td></td>
</tr>
</tbody>
</table>
Electrocardiograms

3.5.1963 Sinus rhythm 88 per minute PR 0.2 sec
Left axis deviation - 50°- occasional ventricular extrasystoles - P wave in V\(_1\) is biphasic - in aVL the R measures 15mm S wave in V\(_2\) exceeds 22 mm, the nadir is not reached - in leads I, aVL & V\(_5\) and V\(_6\) there is symmetrical inversion of T waves V\(_5\) is still transitional V\(_7\) and V\(_8\) should be recorded
Appearances are those of LV hypertrophy and LA hypertrophy

X-rays

13.5.1963
The lung fields are clearing but there is evidence of either unresolved infarction infection in the right upper zone The cardiac outline indicated left ventricular enlargement consistent with an aortic valvular lesion

21.5.1963
There is marked cardiac enlargement, the enlargement being mainly left ventricular. The ascending aorta is slightly prominent. The LA is slightly enlarged. Congestive changes are present in the lung fields. The area of increased density involving the right upper zone is still present, but the previous films are not available for comparison.
DISCUSSION

This patient's history suggests how easily respiratory illnesses precipitate cardiac failure in those who have previous heart disease. The fact that cough is a symptom of both respiratory disease as well as left sided cardiac failure makes diagnosis difficult unless this particular predisposition is borne in mind, and a very careful examination undertaken. The transition is shown by a slight change in the nature of the symptom after treatment with antibiotic and 'cough mixtures'. The cough cleared up by day, but continued at night, waking the patient from sleep and giving him a period of breathlessness.

To somebody who knows the patient's past history, the connection is obvious. The episode in 1956 when the patient was seen by a consultant cardiologist does not seem like a typical story of rheumatic fever. There is no history of an earlier episode in childhood. His attack of 'virus pneumonia' suggests that he is predisposed to chest infection.

The diagnosis, from the history and examination, seems to be rheumatic heart disease with the involvement of the aortic valve causing stenosis and incompetence, without involvement of any of the other valves of the heart. That there is no previous history of rheumatic fever is not surprising, because in 40% of cases of mitral stenosis, there is no rheumatic history. However, involvement of aortic valve without involvement of mitral valve is uncommon. It would have been nice to be able to fit in his visual defect in some manner to the cardiac lesion. The sudden onset suggests an embolic phenomenon, but there is no fibrillation of the heart. On examining his visual fields it is clear that the defect is in the centre of the field, suggesting that it is the macular area that is involved. The disc on the affected side looks a little paler, but there is no obvious abnormality of the blood vessels, except that the arterial branches look slightly smaller than on the nonaffected side. The macular area of the retina itself shows no visible change. There has been no progression in the field of defect. The only possible way of explaining these findings seems to be the selecting obstruction of the arterial supply to the macular fibres or the macular part of the retina.

When the patient was first seen, he looked ill, but it was only the clinical examination and X-ray investigations that showed the extent of cardiac failure. There was gross cardiac enlargement and considerable dyspnoea on the slightest exertion. The fact that the right middle lobe was consolidated by some infective process or had undergone infarction made his extensive dyspnoea explicable. How the patient managed to carry on his work as a laundry manager with such disability is surprising.

In view of the presence of infection with valvular disease, it was considered wise to exclude any possibility of subacute bacterial endocarditis by blood culture. The results were negative. The Wasserman and Kahn tests were done, as syphilis classically gives isolated lesions of the aortic valve more commonly than rheumatic endocarditis. These tests also
were negative.

The patient was treated with bed rest, digitalisation and diuretic therapy. For his chest condition which had not yet shown complete radiological resolution he was put on benzyl penicillin a mega-unit a day. In a few days his cough disappeared and he was able to have undisturbed sleep at night. He began to appreciate the fact that though he thought he was reasonably well before admission, after a week in hospital he was feeling much better than he had for many weeks. He was a little anxious about his business affairs, but his very capable wife showed herself a very competent manageress. This enabled him to continue his rest and treatment in the hospital for another two weeks without business worries.

A consultant cardiologist saw the patient. In view of the fact that the patient was a young man of 43, it was thought advisable to consider the possibility of surgical correction of the aortic valvular lesion and to assess the patient from this angle. However, it was felt that in view of the considerable calcification of the aortic valves seen on screening of the heart, surgery is not indicated. The electrocardiograph showed gross enlargement and hypertrophy of the left Ventricle. The left atrium was also hypertrophied. The Cardiologist was very doubtful if surgery was indicated in this case.

Three weeks after admission, the patient was discharged from the hospital, showing no signs of left ventricular failure. His chest showed no clinical evidence of a continuing inflammatory process. He was advised to continue on his digitalis and diuretic therapy.

In as much as the left ventricle is capable of considerable hypertrophy of its musculature, it is expected that it will be able to stand the strain imposed on it by valvular incompetence or stenosis for much longer time than the left atrium in a case of mitral stenosis. On the other hand, when the left ventricle fails, it is of much graver significance than the failure of the left atrium. It was obvious from the history of the patient that what precipitated failure in this case was the pulmonary infection. The fact that the failure responded very adequately to therapy and effective treatment of the infection is a good sign. It must be emphasised here that the treatment of pulmonary infection in a patient with cardiac lesions with oral penicillin for the very short time of three days is a very poor therapeutic regime. If he had been put on sufficiently high and effective doses of an antibiotic at the very beginning of his respiratory illness, he might have not gone into failure at this time. Subacute bacterial endocarditis was a very real possibility in this case, and so no infection should be allowed to smoulder on for long periods without sufficient treatment. Any further damage to his aortic valves, would throw him into further failure, from which he may not recover as well as he has done this time.
It seems essential that every effort must be made to prevent him going into cardiac failure again. The first step in the further management of the patient is therefore adequate follow up at the Cardiac clinic of the hospital where he must be seen at three monthly intervals. In the meanwhile he should continue with his digitalis and diuretics.

It is absolutely necessary to prevent further infection of his lungs. So every upper respiratory illness must be taken more seriously than in the past and treated from the very start with an antibiotic to prevent secondary infection of the lungs. The patient gave up smoking during the last episode of "flu", and he should be encouraged to continue to do so.

He is very successful in his business and has been working fairly long hours without adequate rest. His recent stay in the hospital has shown him that in his wife he has a very competent business partner, and that a considerable amount of the burden he carries can be relegated to his wife and junior staff. His illness in 1956, when he was off work for three months was brought on to a great extent by over-work. Even during the more recent episode, had he not continued at work when he was not feeling too well, he might have preserved a little more of his myocardial capacity. In view of these events, it is worthwhile impressing on the patient that he MUST not work beyond the abilities of his heart and shorten his life unnecessarily. It is hoped that his wife, who is a qualified nurse will be able to put his across to him without alarming him unduly.

The prognosis in this does not seem very hopeful. On admission he was in Grade IV cardiac disability. It has been possible with digoxin and diuretics to bring him back to Grade II. It is possible that he may go into frank failure again, even without any considerable addition to the load the left ventricular myocardium is bearing. It seems worthwhile, investigating at greater length the possibilities of cardiac surgery by aortography and LV catheterisation, so that should he become grossly incapacitated, he may be offered the services of cardiac surgeons. This formidable procedure carries a very high mortality still, so that one hesitates to recommend it till at a later stage.
DAVID WATT
1 Tobago Place
EDINBURGH

Date of Birth 14.1.1936
Age 27 years
Occupation Asst. Storekeeper

DATE OF ADMISSION 10.4.1963

COMPLAINTS AND THEIR DURATION

1. "Gastric flu" four weeks before admission for 2 weeks
2. Pain across the stomach for 2 weeks

HISTORY OF PRESENT ILLNESS

This patient who was well till four weeks before admission began to feel tired and weak at that time. It was accompanied by a sense of lethargy, loss of interest in work and an uncomfortable feeling in his stomach. He did not have any nausea or vomiting or any bowel symptoms at that time. However, he noticed that when the pain came on, he felt sweaty and clammy. There was no radiation of this pain. Occasionally a gnawing pain started in the lower abdomen and radiated to the small of his back. The illness was diagnosed as "gastric flu".

He returned to his work after two weeks. He was well for three or four days after which he noticed while walking home from work that he had a dull pain in his epigastrium. The pain did not radiate nor was it relieved by rest. However, food seemed to relieve the pain. A few days later he was woken from sleep by the pain, but it disappeared as soon as he sat up in bed. His symptoms of lethargy and weakness also returned. He vomited on two occasions and the vomitus was dark brown and watery.

During the three days prior to admission, the patient felt breathless on slight exertion and had repeated attacks of epigastric pain for which his own doctor gave him a mixture and some pills.

A few hours before admission, the patient felt severe pain in the epigastrium accompanied by sweats. The pain radiated to the neck. He did not feel faint or breathless. His doctor was called, but the pain disappeared by the time he arrived. He was sent into the hospital by ambulance.

PAST MEDICAL HISTORY

Herniorrhaphy on the right side at age 1
Operation on the left upper eyelid forptosis
Recurrent attacks of sore throat 5 years ago when working in paper mill
SYSTEM REVIEW

CVS: Good exercise tolerance till recently
No chest pain related to effort
No ankle oedema - feeling of tiredness for over a month

RS: Smokes about 5 cigarettes a day
Has had an irritating cough with occasional white sputum
No chest pain or haemoptysis

AS: Appetite normally good
Bowels regular - no loss of weight recently
Alcohol - 5 pints of beer at weekends

GUS: No nocturia, dysuria or frequency

CNS: No headache, visual disturbances
No giddiness or vertigo
Slight weakness in lower limbs recently

FAMILY HISTORY

Father alive and well
Mother has had rheumatic fever - breathless on exertion - ankle oedema
Wife alive and well
No children
Other members of family have had congenital ptosis of eyelid

EXAMINATION ON ADMISSION

GENERAL Pale, cyanosed and dyspnoeic
Skin moist and sweaty
No lymphadenopathy - thyroid not enlarged
No finger clubbing - no pyrexia

CVS Pulse 200/min: irregular in time and force
BP 70/40 mm Hg - JVP raised 2½"
Apex Beat - heaving in the 5th left interspace on MCL
Heart Sounds: Loud split first sound
  Grade 2 systolic murmur
  Single second sound at apex
No sacral or ankle oedema
Peripheral pulses
  Femoral + +
  Popliteal + +
  Post.tibial + +
  Dorsalis Pedis + +

RS Dyspnoeic and cyanosed - trachea central
Expansion good and equal on both sides
Percussion does not disclose any abnormality
Breath sounds vesicular - no accompaniments

AS
Tongue a little dry - breath smells of acetone
Abdomen soft and moves with respiration
No hepatic or splenic enlargement

GUS
Kidneys not palpable - testes normal
Hernial orifices intact

CNS
Cranial nerves intact
Fundus normal

Reflexes
\[
\begin{array}{cccccc}
L & + & + & + & + & \downarrow \\
R & + & + & + & + & \downarrow \\
\end{array}
\]

DIAGNOSIS AND TREATMENT ON ADMISSION

Acute left and right ventricular failure with atrial fibrillation

Digitalised with 1.0 mgm Digoxin intravenously
0.5 mgm Digoxin intramuscularly
Mersalyl 2 ml at 6.00 a.m. the following morning

RESPONSE TO TREATMENT

\[
\begin{array}{ll}
10.4.'63 & 11.4.'63 \\
\hline
12.00 & 1.00 & 2.00 & 3.00 & 4.00 & 5.00 & 6.00 \\
60 & 80 & 100 & 120 & 140 & 160 & 180 & 200 \\
\end{array}
\]
### INVESTIGATIONS

#### Blood

<table>
<thead>
<tr>
<th>Date</th>
<th>Hb</th>
<th>WBC</th>
<th>ESR</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.4.1963</td>
<td>112%</td>
<td>11,300</td>
<td>1 mm/hr</td>
</tr>
<tr>
<td>13.4.1963</td>
<td>126%</td>
<td>7,700</td>
<td>1 mm/hr</td>
</tr>
<tr>
<td>17.4.1963</td>
<td>109%</td>
<td>8,400</td>
<td>1 mm/hr</td>
</tr>
</tbody>
</table>

- PCV 55%
- MCHC 34
- Reticulocytes less than 1%
- slight anisocytosis
- normochromia

#### Urine

<table>
<thead>
<tr>
<th>Date</th>
<th>Colour</th>
<th>S.G.</th>
<th>Sugar</th>
<th>Protein</th>
<th>Acetone</th>
<th>Micro</th>
<th>Micro</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.4.1963</td>
<td>Amber</td>
<td>1020</td>
<td>- ve</td>
<td>- ve</td>
<td>trace</td>
<td>- ve</td>
<td>- ve</td>
</tr>
</tbody>
</table>

#### Biochemistry

<table>
<thead>
<tr>
<th>Date</th>
<th>Sodium</th>
<th>Potassium</th>
<th>Bicarbonate</th>
<th>Total Protein</th>
<th>Albumin</th>
<th>Globulin</th>
<th>Globulin</th>
<th>Globulin</th>
<th>Globulin</th>
<th>PBI</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.4.1963</td>
<td>136 mg/dl</td>
<td>4.2 mEq/l</td>
<td>22.6 mEq/l</td>
<td>5.7 Gms</td>
<td>3.1 Gms</td>
<td>0.2 Gm</td>
<td>0.6 Gm</td>
<td>0.8 Gm</td>
<td>1.0 Gm</td>
<td>4.4 ug%</td>
</tr>
</tbody>
</table>

#### Prothrombin Time

- Patient's serum: 19 secs
- Control: 11 secs
- Prothrombin activity: 22%

#### Bacteriology

- 11.4.1963: Blood Culture No growth
- 12.4.1963: Blood Culture No growth
- 15.4.1963: ASO titre 200 Todd units

#### X-rays

- 12.4.1963: Heart Screening
- Well marked auricular fibrillation
- LA enlarged
- Congestive changes in lung fields
- Appearance suggestive of mitral disease
18.4.1963  Barium swallow and meal
Marked atrial fibrillation, LA slightly enlarged
No intrinsic lesion in the oesophagus seen
No abnormality in stomach or duodenum

22.4.1963  Chest X-ray
Heart Transverse diameter within normal limits
Aorta small - Left Pulmonary artery segment is prominent
Little enlargement of left atrium
Lung fields clear - lung markings in upper zone more
prominent than in lower zone
consistent with mitral disease

Electrocardiogram  
15.4.1963  Atrial fibrillation, ventricular rate about 100/min
Right axis deviation - no ECG evidence of RV hypertrophy
Marked clockwise rotation of heart and general form
suggests presence of LV hypertrophy and strain, but
additional leads V₇ and V₈ will be necessary to confirm
this.
Record unusual for a man of this age
19.4.1963  Atrial fibrillation still persists - frequent ventricular
extrasystoles - aQRS approximately + 100° - T wave
inversion from V₃ - V₇ - appearances are not typical of
LV strain - no evidence of preponderant hypertrophy of
either ventricle
Further records must be taken

TREATMENT
10.4.1963  Digoxin 1.0 mgm IV injection
0.5 mgm IM injection
then 0.25mgm q.i.d. orally  Na amytal 200 mgm nocte
11.4.1963  Mersalyl 2ml IM injection
19.4.1963  Bendrofluozide 5.0 mgm 3 days a week
KCl 3 Gms on alternate days
22.4.1963  Phenindione 200 mgm
23.4.1963  "" "" 100 mgm
24.4.1963  "" "" 50 mgm
25.4.1963  "" "" 25 mgm  Quinidine 200 mgm stat
26.4.1963  "" "" 50 mgm  Quinidine 400 mgm 6 hrly
three doses given

PROGRESS NOTES
11.4.1963  Patient improved - No raised JVP - still fibrillating
rate approximately 100/min.

19.4.1963  Seen by Consultant cardiologist
"RV lift at LSE - loud split 1st sound
variable SM₁-₂ - Normal 2nd heart sound at base
apex - No MDM or OS No ELM
"RV failure - strangely acute onset for RV failure due to mitral stenosis -
" recommend Diuretics and Quinidine under anticoagulant cover

26.4.1963 "unlikely to be rheumatic mitral stenosis - Atrial fibrillation not well controlled
"Recommend: exclude thyrotoxicosis
restore normal rhythm
if all proves negative and atrial fibrillation persists
 discharge on digitalis regime - to attend at cardiac Outpatient - review prior to angiography

27.4.1963 5.20 a.m. Patient collapsed after "calling out and looking dreadful"
Doctor attended in five minutes, but patient was pulseless
External and Internal Cardiac Massage, intubation and Mouth to mouth respiration attempted without success
Massage and Inflation stopped at 6.15 p.m.

POST-MORTEM REPORT (RELEVANT POSITIVE FINDINGS)

CVS The heart was moderately enlarged - RA and RV little dilated
myocardium normal - LA moderately dilated: no hypertrophy
Slit like patent Foramen Ovale
LV definitely dilated - hypertrophy, slight to moderate degree
varying in different parts of the chamber - LV myocardium
showed no obvious lesion apart from pallor
Valves - dilated
Coronary arteries - a few very small patches of intimal
thickening - myocardium patchily congested
Aorta - thoracic and abdominal aorta were slightly narrow
for age of patient - no atheroma

LIVER Moderately enlarged - obviously congested
On section one or two small round nodules, yellow in colour,
scattered throughout the substance of the liver - largest
0.75 cm in diameter - ? deposits of an abnormal tissue
DISCUSSION

This is the strange and tragic story of a young man of 27 who developed an acute illness from which he seemed to have recovered considerably, but died suddenly due to cardiac arrest which could not be reversed inspite of external and internal cardiac massage.

The early symptoms of his illness which started about a month before admission were not recognised as indications of cardiac failure because of their bizarre nature. The diagnosis of "gastric flu" was merely an admission of the inability to piece together the data available into a cogent diagnosis. At the time of admission into the hospital, the diagnosis of acute cardiac failure caused at least partly by the rapid ventricular rate of 200 was all too obvious. Immediate intravenous digitalisation reduced the heart rate considerably and improved the blood pressure. In less than 24 hours, the most prominent sign of cardiac failure on admission, the markedly raised Jugular venous pressure was back to normal. It was evidence par excellence of the effectiveness of digitalis therapy in cardiac failure associated with atrial fibrillation.

Though the condition of the patient improved very much during the few days following admission, from the point of view of therapy, the situation was unsatisfactory because an aetiological diagnosis of the atrial fibrillation was not available. On clinical grounds the commoner causes of atrial fibrillation could not be accepted as aetiological factors in this case. There was no history of rheumatic fever - nor did the auscultatory findings show that valvular involvement suggestive of rheumatic mitral disease was present. As the consultant cardiologist observed, it was a curiously acute history for rheumatic valvular disease to be the cause of atrial fibrillation or failure. Thyrotoxicosis was excluded on the basis of the history and clinical examination. In a young person who was in good health till recently, the likelihood of thyrotoxicosis producing heart failure without other signs and symptoms of the disease was only a very remote possibility. Ischaemic heart disease it was felt could hardly be present in a young man of 27 and therefore could not account for the fibrillation. So on clinical grounds alone one was forced to consider the existence of some rare cause of atrial fibrillation in this patient.

Ancilliary aids showed that this clinical judgement was a sound one. Though X-ray screening of the heart suggested the possibility of mitral disease, the ECG evidence for right ventricular hypertrophy was negative. On the other hand ECG suggested left ventricular strain. Serum Protein bound Iodine was 4.4ug% and excluded the possibility of thyrotoxicosis.

Meanwhile, the patient was making satisfactory progress. There was no further attack of epigastric pain, discomfort or dyspnoea. This suggested that the epigastric pain of which the patient was complaining during the weeks before admission was very likely to have been a result of portal congestion. Though atrial fibrillation was still present, the heart rate was only about 80 per minute. On the recommendation of the cardiologist, diuretic therapy with Bendrofluozide was instituted, with adequate response.
In view of the fact that no definite aetiological factor could be found and that the fibrillation was possibly of very recent onset, it was considered worthwhile to attempt to establish normal rhythm by quinidine therapy under anticoagulant therapy. The patient was allowed up at this time because it was not considered that nursing in bed was necessary in the face of dramatic improvement in the clinical condition of the patient.

Anticoagulant therapy was started on 22nd April and had reduced the prothrombin activity to 22% by 24th April. A test dose of 200 mgms of Quinidine was given on the 25th April without any signs of idiosyncrasy to the drug following the administration. Three doses of Quinidine, each of 400 mgms, were given on 26th April.

When the patient was seen by the Consultant Cardiologist on that day, it was observed that the fibrillation had not been well controlled. It was however suggested that the patient might be discharged on a digitalis regime if no cause could be found for the fibrillation and if it persisted, with an appointment to attend the cardiology out-patient clinic. Unfortunately the patient collapsed and died on the 27th April at 5.20 a.m. Efforts at resuscitation were of no avail.

This sudden death of the patient while maintaining steady clinical improved deepened the mystery about the diagnosis. It was hoped that the post-mortem examination might give a satisfactory explanation for the sudden death as well as offer clues to the cause of the acute onset of cardiac failure and atrial fibrillation. However gross examination of the internal organs has not helped to solve the puzzle.

The negative findings of the post-mortem exclude once and for all ischaemic heart disease, rheumatic valvular lesions and thyrotoxicosis as the cause of fibrillation and acute failure of the heart. The dilatation and hypertrophy of the LV, noted at post-mortem suggest the failure of that chamber as the source of the generalised failure of the heart. In the absence of aortic valvular lesions, hypertensive disease and myocardial infarct, the suggestion that the patient was suffering from an acute myocarditis or a rare cardiomyopathy - considered earlier in the management of the patient - deserves to be given serious thought. However, histological studies of the myocardium and the yellowish nodules found in the liver are essential before such a diagnosis can be confirmed. From the clinical picture the possibility of a rheumaticcarditis may be ruled out.

The nagging suspicion that this sudden death may be a "quinidine death" must arise in the minds of at least some of those who attended the patient. Even if only rarely, quinidine is credited with the ability to produce sudden deaths. However, it must be recalled that the test dose did not reveal any idiosyncrasy to the drug. The total amount administered, namely, 1.4Gms, is however not a high enough dose to have caused toxic symptoms due to overdosage.
The third possibility seems to be that the death was due to over-digitalisation. The ECG on 19th April had shown that there were multiple extrasystoles - a feature not present in the ECG of 15th April. While it may be possible to attribute this to the underlying disease process, it may with greater justification be considered to be a toxic symptom of digitalis. Though the absence of other toxic signs may throw considerable doubt on this suggestion, the presence of multiple ventricular extrasystoles should have been an indication for stopping digitalis or reducing dosage.

Whatever the diagnosis of the acute cardiac failure, atrial fibrillation and the cardiac arrest, a few comments may be made about the management of the case.

It does not seem that the seriousness of the situation was adequately appreciated. It seemed very much on the cards from the clinical picture and the many investigations undertaken that the patient had some primary cardiomyopathy or myocarditis. In view of this, the patient ought to have been nursed for much longer time in bed, in spite of the clinical improvement shown. It is true that the low erythrocyte sedimentation rate of 1mm per hour seems to rule out any occult inflammatory process in the body. However the ESR, it is universally recognised, is a poor criterion for excluding a disease process. The fact that the atrial fibrillation, the most important and prominent sign in this case was difficult to control should have suggested the continued existence of some serious pathological condition in the myocardium.

Secondly, the question must be raised whether it was advisable to have undertaken Quinidine therapy in the presence of serious myocardial disease as shown by the acute onset of symptoms in a young man and marked failure. Would it have been wiser (in retrospect) to have delayed treatment with quinidine until after the possibilities of treating the primary condition, say with steroids, was considered?

Thirdly, the inadequate use of the ECG must be commented upon. On 19th April, the reader of the ECG had drawn attention to the presence of multiple ventricular extrasystoles and the inversion of T waves in leads V3 - V7 and indicated the advisability of further records. Even if it be argued that there was no special need to take serial ECGs to exclude digitalis poisoning, it would seem to have been definitely indicated before the start of quinidine therapy.

It is easy to be wise after the event. If as is a possibility, to be confirmed by histological studies, the patient had involvement of the myocardium in sarcoidosis, the sudden death is not likely to be due to quinidine, but a "diagnostic death" - for often in infiltration of the myocardium by sarcoid, sudden death is not uncommon.