# THE LEDERLE CARDIAC SYMPOSIUM: ABSTRACTS OF PAPERS

The following are summaries of papers presented at the Lederle Cardiac Symposium which was held in Johannesburg on 25-26 July 1958 under the aegis of the Southern African Cardiac Society. The chairmen of the various sessions were: Drs. B. A. Bradlow, M. Nellen, J. K. Drummond, B. van Lingen, A. F. Graham, and Prof. H. W. Snyman.

PRESENTATION—A CASE OF DIFFERENTIAL CYANOSIS IN AN ONE-YEAR-OLD WHITE BOY, DUE TO AN AORTIC SEPTAL DEFECT, BY J. M. COMBRINK, M.MED. (MED. INT.) (PRET.), *Pretoria*

*History.* Heart murmur since birth, frequent upper respiratory infections, cyanotic attacks.

*Physical Examination:* Dyspnoea, pulse rate 60, regular rhythm liver slightly enlarged. The fingers of the left hand and the toes of both feet showed clubbing with cyanosis. The fingers of the right hand were pinker in colour. Right ventricular hypertrophy. Systolic thrill at the heart base and rough systolic murmur, grade 3/6 maximal in the 2nd and 3rd left intercostal spaces. At times a diastolic murmur was audible in the same area.

*ECG.* Total A-V dissociation, right ventricular hypertrophy.

*X-ray.* Enlarged heart with increased pulmonary vascular markings.

*Oximetry.* Oxygen saturation of the right brachial artery was 78% as against 64% in the right femoral artery.

*Thoracotomy* showed a large aortic pulmonary window, which was not technically amenable to surgical closure without the pump oxygenator.

A CASE OF TETRALOGY OF FALLOT WITH SYSTEMIC HYPERTENSION IN A WOMAN AGED 39, BY W. H. DAVIS, M.MED. (MED. INT.) (PRET.), *Pretoria*

*History.* Apparently she was blue at birth. Her effort tolerance as a child and as an adult was limited and has become more defective since the birth of her child 5 years ago. She can, however, still walk up 3 flights of stairs and shop all day.

*On Examination.* Slight cyanosis, no clubbing of the fingers. Blood pressure 196/115 mm. Hg. Heaving apex between mid- and anterior axillary line. There was no parasternal pulsation. A thrill could be felt at the base. Apex grade-II systolic blowing murmur and a descrescendo early diastolic murmur. At the pulmonary area a continuous murmur. ECG showed predominantly right ventricular and right atrial enlargement with some left ventricular enlargement.

*X-ray.* Enlarged heart, especially right ventricle, with pronounced bay in the pulmonary-artery segment and a bronchial-artery circulation pattern.

*Oximetry.* At rest: 81%-76% arterial oxygenation. On effort: For 3 minutes oxygen saturation dropped to 69%. On exposure to 100% oxygen, saturation rose to 95%.

*Cardiac Catheterization.* Pulmonary-artery pressure 31/18 mm. Hg. Right ventricular pressure 180/15 mm. Right atrium mean 13 mm. Femoral artery 194/114 mm. 94% oxygen saturation. No shunts demonstrated.

*Conclusions.* Tetralogy of Fallot; severe infundibular stenosis with marked collateral bronchial flow and systemic hypertension.

*Discussion*

1. Marked collateral bronchial flow probably due to systemic hypertension and flow of blood from a high-pressure (aorta) to low-pressure circulation. (*a*) This is probably the mechanism of the continuous murmur. (*b*) This marked flow with the increased right ventricular pressure even in the presence of a severe pulmonary stenosis explains the absence of cyanosis at rest.

2. Pulmonary valvotomy alone would in a case with such hypertension result in a severe left-to-right shunt on removing the obstruction. Repair of the septum together with pulmonary valvotomy would be essential.

3. Any condition that would increase the systemic pressure without increase of the pulmonary pressure would cause an increase of the pulmonary flow and diminution in cyanosis. If a drug with such an action were available, the cross-section of the pulmonary valve could be worked out by taking arterial and venous oxygen saturation and pressure readings.

A CASE OF PERSISTENT LEFT SUPERIOR VENA CAVA DRAINING INTO THE LEFT ATRIUM AS A CAUSE OF CYANOSIS, BY W. H. DAVIS, M.MED. (MED. INT.) (PRET.), *Pretoria*

*History.* (1) Cyanosis since birth. (2) Severe cyanosis with exercise without severe dyspnoea. (3) Ordinary effort tolerance fair, but for 50 years marked dyspnoea on running. (4) Fainting attacks with severe exercise. (5) Mentally retarded.

*On Examination.* (1) Central cyanosis with clubbing of fingers and toes. (2) No cardiac enlargement; no thrill; left parasternal grade-II systolic murmur, soft mid-diastolic murmur, and a soft second pulmonary sound.

*Cardiac catheterization* from the left arm showed oxygen saturation values the same as for the femoral artery. The pressure curves were left ventricular and aortic. The catheter actually passed from the left cephalic vein into a persistent left superior vena cava, left atrium, left ventricle and aorta. Catheterization from the right arm showed normal findings for right atrium, ventricle and pulmonary arteries as far as pressures and oxygen saturation were concerned.

*At operation* the finding was a persistent left superior vena cava draining into the left atrium with a very large hemi-azygos vein.

*Discussion*

1. The embryology of a persistent left superior vena cava and related venous anomalies were discussed. The importance of a vein connecting the two superior venae cavae to make the condition amenable to surgery was emphasized.

2. Pressure curves were discussed.

AMOEBIC PERICARDITIS, BY J. KELMAN DRUMMOND, M.R.C.P. (EDIN.), AND N. MCE. LAMONT, M.D., F.R.F.P.S., *Durban*

A series of 8 cases was reported out of a study of 250 cases of hepatic amoebiasis and 1 additional case.

Reference to world literature showed a low rate of diagnosis before death (ante-mortem diagnosis in only 4 cases), and included one survey (2 out of 47 cases). Of the 8 cases of the present series the diagnosis was missed in 1.

It was stressed that the features of amoebic pericarditis were closely linked with those of the liver abscess, its invariable precursor, and some of the problems of diagnosis of liver abscess were mentioned.

There were 3 distinct phases in the pathogenesis, viz. (1) Pericarditis with 'sympathetic' effusion, (2) intrapericardial rupture of the liver abscess, and (3) the phase of occlusion, with thickening of the pericardium.

Of the 8 cases, 2 were arrested in phase 1, and recovered; 1 died in phase 2 in state of shock; 4 died in phase 3, after showing initial improvement, death taking place even months after admission from progressive cardiac tamponade; the outcome of the last case (also in phase 3) was left in some doubt.

*Prognosis and Therapy*

Good drainage, surgical if necessary, of any abscess of the left lobe of the liver (which will suffice for phase 1) is imperative.

Inadequate drainage of the pericardium is likely to lead to constrictive pericarditis (in phase 2).

Adequate drainage in phase 2 is provided by an indwelling polythene tube in the *posterior pericardium.*

Simple aspiration is probably inadequate if later possible constrictive pericarditis is to be avoided.

The polythene tube may be introduced *via* the liver and the diaphragm.

The value of irrigation with either emetine or antibiotics is not established. The pus is sterile, but secondary infection must be watched for.

Amoebic pericarditis is a grave illness with severe toxaemia, shock and tamponade, but alert diagnosis and thorough drainage should give good results.

HYPOTHERMIA—ITS APPLICATION TO CARDIOVASCULAR SURGERY, BY DENNIS N. FULLER, F.R.C.S., *Johannesburg*

The effect of lowering body temperatures is to diminish metabolism. Temperatures of 28-29°C are desirable. In this series the lowest temperature recorded was 27·0°C. At this range of temperature the brain can tolerate 8-10 minutes cessation of circulation without damage. Probably longer periods would be safe, particularly if the carotid circulation were perfused with a low flow (as little as 15-30 c.c. per kg.) of arterialized venous blood. It is known from cardiac ischaemia in open heart surgery with a pump oxygenator that the myocardium at rest will tolerate an hour or more of complete cessation of the coronary flow.

One graph shown illustrates that the oxygen requirements of a hypothermized (27°C) arrested heart are 3% of a normal beating heart. In an other graph the effect on the glycogen, creatine phosphate and adenosine triphosphate contents of the myocardium are illustrated under conditions of (*a*) hypothermia, (*b*) potassium arrest, and (*c*) citrate arrest.

21 cases were operated on in Johannesburg under hypothermia (2 at Baragwanath Hospital, 12 at the Transvaal Memorial Hospital for Children, and 7 at the Florence Nightingale Nursing Home). The particulars were as follows:

| *Diagnosis* | *No. of Cases* | *Deaths* |
|---|---|---|
| Pulmonary valvular stenosis | 4 | 0 |
| Isolated right ventricular infundibular stenosis | 1 | 0 |
| Tetralogy of Fallot (infundibular stenosis) | 3 | 2 |
| Congenital aortic valvular stenosis | 3 | 2 |
| Congenital tricuspid atresia | 3 | 1 |
| Hypertensive patent ductus arteriosus | 2 | 0 |
| Truncus arteriosus | 1 | 1 |
| Traumatic aortic aneurysm | 1 | 0 |
| Atrial septal defect | 2 | 0 |

The ages ranged from 4 months to 48 years. There were 6 deaths in the series of 20 cases. All deaths could be adequately

accounted for by technical or myocardial reasons. In none could the method of hypothermia be incriminated.

THE ASSESSMENT OF DYSPNOEA IN CARDIAC DISEASE, BY R. KAMENER, M.B., B.CH. (RAND) AND S. ZWI, M.B., B.CH. (RAND), *Johannesburg*

A cardio-respiratory unit has to investigate dyspnoea—to verify its presence, seek its cause, determine the necessity for treatment, or assess changes in its severity after treatment. Until recently, dyspnoea was regarded as the consequence of a reduction in vital capacity or cardiac output, anoxia, hypercapnia, altered pH, or over-activation of the Hering-Breuer reflex. These, however, may be accompaniments, not causes, for they are rarely detectable before dyspnoea is advanced.

To ventilate the lungs, the respiratory muscles do 'work' which can be measured. The work depends on the volume ventilated, the force to stretch the elastic lung (compliance), and the force to draw air into the lung (non-elastic resistance). In cardiopulmonary diseases, at least 1 of these 3 factors is excessive, and the resultant increased respiratory work causes fatigue of the respiratory muscles. This muscular fatigue is probably the basis of the symptom of dyspnoea. Two practical examples illustrate this approach to dyspnoea:

1. In mitral stenosis, disability is sometimes difficult to assess. In the early stages, measurement of compliance or cardiac catheterization usually produces equivocal results. We have found it more helpful to subject patients in the early stages to standardized grades of moderate exercise and measure the volume of air ventilated. When this is excessive, dyspnoea is present. The procedure is safe and simple, and appears to be reliable.

2. In normal subjects or patients with cardiopulmonary disease in which the heart is not under stress, exercise (and hyperventilation) lowers the non-elastic resistance of the lung. However, when the heart is 'decompensated' (as may be inferred from cardiac catheterization or other studies), the opposite occurs, i.e. on exercise non-elastic resistance rises above its resting level. This appears applicable to what is commonly regarded as right heart failure as well as left heart failure.

DIRECT LEFT AURICULAR PRESSURES IN MITRAL DISEASE, BY P. E. MARCHAND, M.CH., M.D. (RAND), F.R.C.S., *Johannesburg*

Left atrial pressures were measured by the bronchoscopic route as described by Allison and Linden (*Circulation*, 1953, **7**, 669). There have been no serious complications.

Normal left and right atrial traces are similar, though the left V wave is generally larger. In mitral stenosis the A wave is usually dominant and the diastolic gradient across the valve may reach 40 mm. Hg. Hypotension due to anaesthesia or cardiac irritation lowers the gradient. For this reason it is advisable to administer a small dose of a short-acting hypertensive drug immediately before pressures are measured. This also accentuates the incompetent pattern (Crawshaw *et al. Brit. J. Surg.*, 1954, **92**, 1).

In auricular fibrillation the X descent disappears and the C and V waves merge to form a plateau curve.

It is too soon to formulate fixed criteria for mitral incompetence. Provided the left ventricular pressure is high the following two appearances, associated with a high diastolic gradient, suggest incompetence, viz.: (1) Dominant V wave, starting early in ventricular systole, with a steep Y descent which does not flatten at its base; and (2) a plateau curve with dominant V peak in the presence of normal rhythm.

These appearances were illustrated by 8 of our 63 pre- and post-operative pressure traces where mitral incompetence of known extent had been produced.

AORTIC EJECTION SOUND, BY MAURICE NELLEN, M.D. (CAPE TOWN), M.R.C.P. (EDIN.), M.R.C.P. (LOND.), LOUIS VOGELPOEL, M.R.C.P. (LOND.), AND VELVA SCHRIRE, M.R.C.P. (LOND.), *Cardiac Clinic, Groote Schuur Hospital, Cape Town*

A study has been made of 64 cases in which the aortic ejection sound was heard. The cases consisted of acquired aortic stenosis, aortic incompetence, aortic atheroma, hypertension, the congenital defects of severe tetralogy of Fallot, aortic coarctation, aortic stenosis, and 13 cases of pulmonary atresia.

The sound occurs soon after the first heart sound, is high-pitched like a click, and is loudest in the 3rd and 4th intercostal spaces but well heard at the mitral area and best at the aortic area.

The early aortic ejection sound may be the loudest component of the first sound and may be the only component of this sound audible at the base of the heart.

The sound must be differentiated from the innocent mid- or late-systolic click, the pulmonary ejection sound, splitting of the first sound, and an auricular sound.

When the first sound is split and the aortic ejection is also present, triplication of the first sound can be recorded.

Indirect carotid tracings show that the rise of pressure in the carotid artery precedes the ejection sound; thus it must be associated with ejection and not with opening of the aortic valves. In 3 cases of aortic stenosis calcification of the valves did not abolish the added sound.

Inhalation of amyl nitrate may make the sound inaudible or shortens its distance from the first heart sound, whereas it increases the audibility of a pulmonary ejection sound and its distance from the first sound.

THE ELECTROCARDIOGRAM IN BERI-BERI HEART DISEASE, BY V. SCHRIRE, M.R.C.P. (LOND.) AND J. GANT, M.R.C.P. (EDIN.) *Cardiac Clinic, Groote Schuur Hospital, Cape Town*

Fifty cases of beri-beri heart disease were studied electrocardiographically during 58 episodes of heart failure.

The most striking finding was a normal ECG when the heart failure was at its worst—33 cases. In the remainder, leads over the right ventricle were abnormal in 10 cases and over the left ventricle or both ventricles in 15.

Transient changes were a characteristic and diagnostic feature of the disease. Often fleeting and variable, changing from day to day and, on occasion, from ventricle to ventricle, they required daily ECG for their detection. They were frequently restricted to 1 or 2 praecordial leads. The right ventricular leads alone were affected 26 times. Changes in the left ventricular surface leads occurred 13 times and both ventricles were affected in 11. In occasional cases one ventricle was involved during one episode and the other ventricle during a recurrent attack. In 8 cases the tracings remained normal throughout.

The changes often preceded the diuresis but were maximal during the period of recovery from heart failure. Minor alterations also occurred and hypokalaemic patterns were an occasional complication during diuresis. Ultimate return of the ECG to normal was the rule where the period of observation was adequate (81%).

CLINICAL AND PHYSIOLOGICAL ASPECTS OF OEDEMA, BY B. SENIOR, M.R.C.P. (LOND.), M.R.C.P. (EDIN.), *Johannesburg*

In health, conservation of sodium and of water is largely mediated by secretion of aldosterone and of antidiuretic hormone. The stimulus to the secretion of aldosterone is a decrease in the volume of the intravascular component. A relatively greater osmolar concentration in the extracellular component than in the cells prompts secretion of antidiuretic hormone.

Apart from menstrual variations or excessive sweating or abuse of alcohol, control is so adjusted that fluctuation in the quantities of these substances within the body is minimal. The oedema of cardiac failure results from a discrepancy between pump input and output. Secretion of aldosterone increases and tubular reabsorption of sodium becomes greater. Oedema becomes manifest.

In long-standing cardiac oedema potassium depletion of the cells may occur through the use of antidiuretics or as a result of continued secretion of aldosterone. The decrease in cellular osmolar concentration consequent on potassium loss may provoke a secretion of antidiuretic hormone.

A paradox yet to be explained is the fact that in primary hyperaldosteronism excessive sodium reabsorption is not associated with the presence of oedema.

Treatment should be directed at restoring the effective action of the pump. Where this is not achieved various agents are employed to produce a loss of body sodium. These agents and their actions are further discussed.

LONG-TERM ANTICOAGULANT THERAPY IN CORONARY THROMBOSIS, BY M. M. SUZMAN, M.D. (DURH.), F.R.C.P. (LOND.), *Johannesburg*

Observation was kept on 1,157 survivors of acute myocardial infarction. They were drawn from unselected hospital admissions and private practice, and had recieved anticoagulant therapy

during the acute phase of the presenting attack. They were followed till death or to the end of each year of survival for periods ranging from 1 to 10 years till the end of the present study.

Anticoagulant therapy was continued on a long-term basis in 353 patients for a total duration of 1,093 patient-years and a mean duration of 37·1 months; and of these 57 died—a total fatality rate of 16%.

To serve as a control group for comparison, 687 patients who received anticoagulants only during the acute phase of the infarction were observed for a total period of 2,084 patient-years and a mean duration of 36·4 months; and of these 329 died—a total fatality rate of 48%.

In 117 patients who discontinued their long-term anticoagulant therapy there were 47 subsequent deaths—a fatality rate of 40%. The duration of treatment in these cases did not influence the subsequent mortality.

During the first 5-year period annual fatality rates were significantly lower in the patients maintained on anticoagulants. It is concluded that long-term anticoagulant therapy improves the prognosis in survivors of acute myocardial infarction.

### Systemic Embolism—Evidence at Mitral Valvotomy, by J. C. van der Spuy, Dip. Surg. (Rand), *Pretoria*

Out of a total of 58 patients operated upon for mitral stenosis, 20 patients either had presented pre-operative evidence of systemic embolism or were found at operation to have thrombus formation in the left atrium or its appendage. Of the 20 patients, 4 had a left-sided and 2 a right-sided hemiplegia, 1 a verbal aphasia and 1 a saddle embolus at the aortic bifurcation. In the absence of subacute bacterial endocarditis and of valve calcification, a thrombus arising in the left atrium was taken to be the cause of the embolism even when no thrombus could be found at operation. In such cases it was assumed that the only thrombus present had been dislodged and swept into the sytemic circulation.

These 20 cases could be divided into 2 very distinct groups, viz. (A) 10 which showed marked enlargement of the left auricle, and (B) 10 which had a small or shrivelled left auricle.

Of the 10 in group A, 8 developed systemic emboli, in 4 of whom relatively small, dark-red, single or multiple thrombi were present in the periphery of the auricle, and in 4 no thrombosis was found; 7 showed sinus rhythm and 1 atrial fibrillation.

Of the 10 in group B, in none was there evidence of systemic embolization. In 2 the auricle contained whitish, organized thrombus only. In the remaining 8 the whole of the left atrium was lined with a very thick layer of thrombus having a soft muddy inner surface and an organized outer layer. In addition, 4 of these patients had one or more large loose thrombi which escaped from the left atrium on releasing the auricular clamp. In 9 atrial fibrillation was present and in 1 sinus rhythm.

In all the 20 patients there was a tightly stenosed valve and pulmonary hypertension.

*Conclusions.* In a patient with mitral stenosis, pulmonary hypertension and a very large auricle, one or more somewhat small thrombi tend to form in the periphery of the left auricle with marked tendency towards embolization. Organization of the thrombus, and consequent shrinkage of the auricle, markedly reduces the tendency towards embolization, even in the presence of massive thrombus formation in the left atrium.

### The By-pass Operation for Peripheral Arterial Obstruction, by F. A. K. van Wyk, M.Med. (Chir.) (Pret.), *Pretoria*,

The most important cause of peripheral arterial obstruction is atherosclerosis. The occlusion is often localized in large vessels and may be suitable for an arterial graft.

Pre-operative investigations should include an arteriogram. The most suitable cases are those occurring in young subjects where the occlusion has occurred in a short segment of a major vessel such as the aorta and iliac arteries. The peripheral arterial tree must be patent to ensure satisfactory results.

The operation consists of the insertion of an arterial homograft to act as a by-pass of the obstruction. The affected segment of artery is not excised. Anastomosis of end of graft to side of host artery is made above and below the occlusion.

Indications for operation are intermittent claudication, rest pain, and impending and early gangrene. The best results are obtained in intermittent claudication, although in early cases of gangrene healing has taken place without amputation.

### Pulmonary Stenosis with Intact Ventricular Septum and Fallot's Tetralogy—Pre- and Post-operative Assessment of Severity, by Auscultation, by L. Vogelpoel, M.R.C.P. (Lond.), and V. Schrire, M.R.C.P. (Lond.)

*Cardiac Clinic, Groote Schuur Hospital, Cape Town*

1. A clinical and phonocardiographic study was made on 45 cases of pulmonary stenosis (valvular in 36 and infundibular in 9) with intact ventricular septum and on 52 cases of Fallot's tetralogy. Increasing severity of stenosis has an opposite effect on the duration of the systolic murmur in the two conditions, owing to the different dynamic situations present.

2. With an intact ventricular septum, the severer the stenosis the greater the duration of the murmur and the wider the splitting of the second sound. Auscultatory and phonocardiographic criteria were defined for grading the severity of the stenosis.

3. In Fallot's tetralogy, the severer the stenosis the shorter, earlier and softer the murmur becomes. The length of the systolic murmur was shown to provide an accurate means of assessing the severity of the stenosis.

4. Auscultation was found to be an excellent bedside method of predicting the surgical result. When severe pulmonary stenosis is converted into mild stenosis by a successful valvotomy or resection, the auscultatory features of severe stenosis are changed to those of mild stenosis. In Fallot's tetralogy a successful pulmonary or infundibular resection will convert a severe case into a mild one with corresponding change in the auscultatory findings. Less successful operations fail to produce the auscultatory features of mild stenosis.

### Atrial Septal Defect in Infancy, by Joan M. Wagner, M.R.C.P. (Lond.), *Johannesburg*

The physical findings in 4 patients dying of atrial septal defect are described, in the hope that in the future such cases, which are difficult to diagnose in infancy, may be submitted for early surgery.

The ages at the time of death were 3 months (in 2), 4½ months and 2½ years. In each case autopsy showed pure atrial septal defect of 1·5 cm. diameter or more.

All 4 were markedly under-weight; 2 had repeated chest infections. All were severely dyspnoeic; 2 had collapsing pulses confirmed by blood pressure readings. The pulmonary second sound was split in all but markedly in only 1. In all, systolic murmurs were heard—in 1 a grade-III murmur at all areas, in 2 a grade-II at all areas, in 1 a grade III in the third left interspace. Diastolic murmurs were heard in 3, in 1 a pulmonary-incompetence murmur, in 1 a mid-diastolic and in 1 a scratchy early-diastolic murmur. The jugular pulsations were not helpful. Electrocardiograph showed a large right auricle and right ventricle in all, but none showed bundle heart block. X-ray showed cardiomegaly in all, with large right ventricle and full lung fields but no hilar dance.

### Experiences with the Artificial Circulation in the Dog, by V. H. Wilson, M.D. (Camb.), M.R.C.P. (Edin.), *Johannesburg*

Dr. Wilson reported experience with the Lillehei pump and 3 bubble oxygenators in 36 dogs. The surgeons and anaesthetists had been supported by their own theatre staff, physicians, a clinical pathologist, a morbid anatomist, and a technician, working with facilities which offered satisfactory conditions for animal survival.

The haematology is of special interest. Dr. Greig had established that a prolonged clotting time was associated with fibrin deposition if the artificial surface was unsuitable. Dr. Greig had also demonstrated that some protamine samples were inactive and that others, though active, might be anticoagulant in action if used in the wrong concentration. Heparin dosage according to weight produced a very variable effect upon the blood in both human and dog. He had found empirically that 1 mg. per kg. of dog weight provided satisfactory conditions for by-pass work.

### An Assessment of the Exercise Capacity of Cardiac Patients, by C. H. Wyndham, M.R.C.P. (Lond.), and J. S. Ward, *Johannesburg*

Heart rate, oxygen consumption and minute ventilation volume were meassured at 3 grades of work on 4 trained men, 5 untrained men, and 10 ambulatory patients with rheumatic heart disease. Ventilation volume per minute at different levels of oxygen consumption were also measured on 24 young men to set up standards

against which to judge abnormality at the Johannesburg altitude of 6,000 feet above sea level.

Of the 10 cardiac patients, 8 showed ventilation volumes per minute at the 3 levels of oxygen consumption which were essentially the same as in normal subjects. Therefore, ventilatory function, as in normal subjects, did not appear to be the factor limiting the maximum possible level of oxygen consumption during severe exercise. The maximum level of oxygen consumption was obtained by extrapolation to 190 beats per minute of the heart rates obtained at 3 submaximal grades of work when heart rates are plotted as a function of oxygen consumption.

The other 2 cardiac patients showed an abnormal pattern of ventilatory response to exercise. Even at mild effort they had a larger minute volume than normal, and this departed further from normal as the level of exercise was raised. The possible implications of this observation is discussed and the primary cause is considered to be an inability to increase the cardiac output in a normal manner in relation to exercise.

The ventilatory responses to exercise, therefore, serve to distinguish 2 clear categories of patients with rheumatic heart disease. The method of assessing the maximum possible level of oxygen consumption of patients in these 2 categories is discussed.

It is suggested that assessment of the individual cardiac patient's maximum level of oxygen consumption can be employed as an objective index in choosing a safe level of work in employment, in assessing the influence of therapy or surgery, and in following the course of the disease with time.

CONTINUED MORBIDITY AFTER MITRAL VALVOTOMY, BY M. M. ZION, M.R.C.P. (LOND.) AND J. L. BRAUDO, M.R.C.P. (EDIN.), *Johannesburg*

Of 300 patients submitted to mitral valvotomy 98 were followed over a period of 1-5 years; 42 (43%) had good, 16 (16%) fair, and 40 (41%) poor results.

The problems presented by patients could be classified as follows:

1. Post-commissurotomy syndrome—readily controlled by steroids.

2. Inadequate splitting of the mitral valve at the first operation.

3. Late re-stenosis of the mitral valve, which occurred in 2 young women following acute rheumatic fever after the initial valvotomy (the importance of prophylactic penicillin was stressed).

4. Persistent right heart failure or tricuspid incompetence, which occurred in 8 cases despite a satisfactory initial valvoltomy They all show an increased exercise tolerance despite an increased heart size. Three factors may be responsible—persistence of increased pulmonary arterial resistance, irreversible myocardial damage, and organic tricuspid disease.

5. Combined mitral stenosis and mitral incompetence.

6. Other causes of poor results despite adequate post-operative size of the mitral valve:

(*a*) Associated valvular defects considered insignificant before operation.

(*b*) Presence of systemic hypertension or ischaemic heart disease.

(*c*) Persistent rheumatic fever.

(*d*) Chronic bronchial infection.

(*e*) Cardiac arrhythmias.

(*f*) Persistent pulmonary hypertension.

(*g*) Subacute bacterial endocarditis.

(*h*) A small group in whom failure to improve cannot be explained.

## OTHER PAPERS

The following papers were also read:

Diphtheritic Myocarditis, by A. L. Jackson, M.R.C.P. (Lond.), *Johannesburg*.

The Ballistogram in Mitral Valve Disease, by B. van Lingen, M.D. (Rand.), *Johannesburg*.