Central venous pressure (c.v.p.) has been accepted widely as a valuable index of the adequacy of blood volume replacement; an abnormally low c.v.p. in a hypotensive patient usually implies oligaeemia, whereas a raised c.v.p. suggests cardiac ("pump") failure as the more likely cause of the hypotension.

However, pulmonary oedema may occur, or persist, in spite of a normal c.v.p. This may be the result of selective left heart failure with pulmonary venous congestion. Conversely, there is sometimes a suspicion that a poor cardiac output may result from an inadequate left ventricular filling pressure although the filling pressure of the right ventricle, as shown by the c.v.p., may be normal or even increased.

The value of c.v.p. as a guide to fluid therapy in hypovolaemic shock has been studied by several groups of workers. Cohn et al. (1967) and Loeb et al. (1969) found that in shock resulting from a variety of causes, a decreased c.v.p. was an indication for the cautious transfusion of low molecular weight dextran. A favourable response to fluid administration can be recognized by an increase in arterial pressure, pulse volume and urine flow and a clinically obvious improvement in peripheral circulation. In these patients the c.v.p. will increase about 1 cm H$_2$O for each 100 ml of dextran transfused.

Unfortunately, not all patients with a decreased c.v.p. will respond favourably. Occasionally a very small infusion will produce severe pulmonary oedema. More frequently a normal or increased c.v.p. may discourage the administration of plasma expanders unless it is appreciated that the c.v.p. may remain increased in patients with raised pulmonary vascular resistance even in the face of severe hypovolaemia. Forrester et al. (1970) found no consistent relationship between c.v.p. and left heart filling pressures. In twelve patients with acute myocardial infarction Forrester et al. (1971) found that c.v.p. was of no value in assessing the presence of congestive heart failure. Three patients had pulmonary oedema in the presence of a normal c.v.p.

These difficulties in inferring left heart pressures from c.v.p. measurement have stimulated attempts to measure left heart pressures more directly. Catheterization of the left heart (either by the trans-septal or trans-aortic route) has been a routine procedure in diagnostic cardiology laboratories for many years. More recently, however, techniques have become available which are applicable to critically ill patients and which require neither experience of orthodox cardiac catheterization nor radiological control. Management of haemodynamic problems in the intensive therapy unit has been helped by continuous monitoring by these methods and it is thought that a description of this application may be useful.

Inference of left heart pressures from measurement of pulmonary artery pressure.

Forsberg (1971) has studied the relationship between pressure in the pulmonary artery, left atrium, and left ventricle in 158 patients who had undergone diagnostic cardiac catheterization at rest and concluded that:

(1) normally, at rest, the diastolic pressures in the left ventricle and the left atrium are similar;

(2) normally the pulmonary artery diastolic pres-
sure has the same value as the end-diastolic pressure in the left ventricle.

It might be concluded that, in some patients at least, useful information about left heart function might be obtained by pulmonary artery catheterization, using perhaps the miniature catheters described by Bradley (1964). However, in individual patients there may be a very large difference between the pulmonary artery diastolic pressure and the left atrial pressure.

Diseases which may produce large and unpredictable differences between pulmonary artery and left atrial pressures (i.e. increased pulmonary vascular resistance) are: pulmonary thromboembolic disease; severe mitral stenosis; chronic pulmonary heart disease (Thomas, 1972) associated with bronchitis, pneumoconiosis or sarcoidosis. In these conditions it is not possible to deduce left atrial pressures from pulmonary artery diastolic pressure or from measurement of c.v.p.; it is in fact quite possible for such patients to become fatally hypovolaemic in spite of a persistently raised c.v.p. or pulmonary artery pressure (Diamond et al., 1971).

Hypoxia and acidosis also increase the pulmonary artery systolic and diastolic pressures. In the intact anaesthetized animal and in man the hypoxic pressor response commences when arterial oxygen saturation is less than 80%. The increase in mean pressure is stated to be of the order of 4–8 mm Hg (Motley et al., 1947). Harvey and his co-workers (1967) showed that an acutely induced moderate increase in the hydrogen ion concentration of the arterial blood is associated with an increase in pulmonary artery pressure. The systolic pressure is affected most and the effect is greater when the oxyhaemoglobin saturation is less than 91%. The pressor response is independent of the left ventricular pressure and is ascribed to pulmonary vasoconstriction.

Treatment with vaso-active drugs has been shown also to increase the disparity between right and left ventricular function. Cohn and his associates (1969) studied the relationship between c.v.p. and left ventricular end-diastolic pressure (l.v.e.d.p.) in patients with acute myocardial ischaemia, severe bronchopulmonary disease, or septic shock. In five out of seven patients with acute myocardial infarction isoprenaline increased the difference between l.v.e.d.p. and c.v.p. Noradrenaline and metaraminol tended to increase l.v.e.d.p. with little change in c.v.p. In the patients with bronchopulmonary disease isoprenaline caused l.v.e.d.p. to increase while c.v.p. decreased or remained constant. In contrast, in three patients with septic shock isoprenaline reduced the l.v.e.d.p. Vasoconstrictors (angiotensin, noradrenaline or metaraminol) increased l.v.e.d.p. while c.v.p. remained unchanged.

It may be concluded that, although c.v.p. or pulmonary artery end-diastolic pressure will give useful information about left heart and pulmonary venous pressures in the majority of patients, a more direct method of measurement is necessary in patients in whom there is reason to suspect that the pulmonary vascular resistance may be raised.

**Pulmonary artery “wedge” pressure.**

If a branch of the pulmonary artery is occluded the pressure recorded in the peripheral segment of the artery is left atrial pressure, transmitted from the pulmonary capillary bed. This transmitted left atrial pressure may be measured by a suitable catheter wedged into a branch of the pulmonary artery so as to occlude it totally. The wedge pressure obtained in this way (PA\(_{\text{wedge}}\)) is not significantly different from pulmonary venous or left atrial pressure (Rahimtoola et al., 1972; Fitzpatrick, Hampson and Burgess, 1972).

Bradley (1964) described miniature catheters which may be introduced into a peripheral vein and “floated” through the right heart chambers into the pulmonary artery. Although they may be used for pulmonary artery pressure measurement, mixed venous blood sampling, and for the measurement of cardiac output by either the Fick principle, dye dilution or thermal conductivity methods, they are too small to be wedged into a branch of the pulmonary artery of sufficient size to allow the measurement of PA\(_{\text{wedge}}\).

The development, in 1970, of the Swan Ganz range of catheters was an important advance. These are relatively large catheters (sizes 5FG and 7FG) and an inflatable balloon is attached near the tip. When inflated the balloon can occlude a large branch of the pulmonary artery. Although they may be used for pulmonary artery pressure measurement, mixed venous blood sampling, and for the measurement of cardiac output by either the Fick principle, dye dilution or thermal conductivity methods, they are too small to be wedged into a branch of the pulmonary artery of sufficient size to allow the measurement of PA\(_{\text{wedge}}\).

The device described by Swan et al. (1970) is a double-lumen catheter made from extruded p.v.c. (fig. 1). The major lumen is for pressure transmission and for sampling. The minor lumen is used
to inflate a delicate latex balloon fastened approximately 1 mm from the tip (fig. 2). The balloon is constructed so that when inflated it does not obstruct the major lumen, but forms a rounded end to the catheter tip (fig. 3). This appears to be a major factor in minimizing extrasystoles as the catheter traverses the heart. The balloon both helps the blood flow to carry the catheter into the pulmonary artery and facilitates wedging. When the balloon is deflated pulmonary artery pressure is transmitted.

Passage of the catheter into the pulmonary artery.

The catheter is passed through a peripheral vein which may be entered using a Seldinger technique or by a "cut down". If an antecubital vein is used it must be medial. The catheter is flushed through and connected to a pressure transducer via manometer connecting tubing. The tip is inserted into the vein carefully to avoid damaging the balloon. When the veins at the shoulder are reached the balloon is half-inflated; the blood flow carries the catheter to the right atrium, which is recognized by a characteristic pressure wave form. The balloon is then inflated fully (1.5 ml for size 7FG) and the catheter may be advanced rapidly through the right ventricle and into a branch of the pulmonary artery where it wedges.
The procedure is usually straightforward and the only skills required are the ability to set up and calibrate a transducer with a pressure wave form display and to insert a catheter into a vein. As the catheter enters the great veins, variation in the pressure with respiration will be seen. As the right atrium is entered pulsations in time with the heart rate are superimposed on the respiratory pattern. The transition to the ventricular pattern is dramatic. The small amplitude atrial waves are replaced by tall waves (fig. 4). The systolic pressure in the right ventricle is usually about one-sixth of the systemic arterial systolic pressure; the diastolic level is close to zero. As the pulmonary artery is entered the most marked change is a shift in diastolic pressure to a higher value than that seen in the ventricle. Wedging of the catheter causes left atrial pressure to be transmitted: this resembles the right atrial pattern although the magnitude may be different (fig. 5). Once the catheter has been positioned, wedge pressure may be measured subsequently without the transducer. The mean pulmonary artery pressure may be measured with an ordinary central venous pressure manometer set connected via a three-way tap between the catheter and the transducer. After the manometer level has stopped falling and the mean pressure has been noted, the balloon is slowly inflated. When enough air has been injected (and in any case not more than 1.5 ml) to occlude the artery, the manometer level will suddenly fall further to the mean wedge pressure. In this way a nurse can measure wedge pressure as frequently as is necessary. This simpler arrangement is more acceptable to nursing colleagues.

Complications and precautions.

These are described in detail in the literature which accompanies each catheter, as is the catheterization procedure. If the technique described here is adhered to the incidence of minor complications will be small and major complications should be avoided.

Arrhythmias. Premature ventricular contractions, usually single, are seen occasionally but have not been a problem. Nevertheless continuous e.g. monitoring is advised.

Difficulty in positioning the catheter. Swan et al. (1970) have described their experience of 100 attempted catheterizations. The pulmonary artery was reached in 92% of patients and the wedge position in 80%. Of the first twenty patients catheterized by the author, difficulty was experienced in only two. Both had pulmonary hypertension with a large right ventricle, and the catheter tended to slip back from the pulmonary artery into the ventricle. Size 7F catheters, which have pre-formed curved tips, were used and it was possible to position both catheters with the help of an image intensifier to guide the tip to a more distal branch of the pulmonary artery.

Movement of the catheter. For a short time after it has been positioned, the catheter tends to continue to pass distally as the slack loop in the right ventricle is “taken up”. This may cause wedging even with the balloon deflated. The catheter should be withdrawn until the pulmonary artery pressure wave form reappears.

Balloon rupture. Swan et al. (1970) described a 10% incidence of balloon rupture but they were using the catheters more than once. The release of 1.5 ml of air into the pulmonary circulation appears to be harmless. The balloon or inflation tube has failed in two patients catheterized by the author. In one case inspection of the central venous and pulmonary wedge pressure traces showed them to be closely related and the patient was then successfully managed by reference to c.v.p. alone.

Perforation of the pulmonary artery. This has been described in one patient (Chun and Ellestad,
However, the prescribed technique was not followed: under radiographic control the deflated catheter was passed into a peripheral branch of this small vessel. The complication is unlikely if the balloon is inflated as soon as a large calibre vein is entered.

**Knotting of the catheter.** Intracardiac knotting of both conventional cardiac catheters (Johansson, Malmstrom and Uggla, 1954) and a flow directed catheter (Lipp, O'Donoghue and Resnekov, 1971) have been described. In the one reported case in which a knot formed in a Swan Ganz catheter, premature ventricular beats were observed although the pressure tracing showed the catheter tip to be in the atrium. This implies that the catheter had looped into the ventricle and back to the atrium. It was possible to remove the knotted catheter, with the balloon deflated, through the original venepuncture without anaesthesia. The manufacturers recommend that if the catheter (which has 10-cm markings) has not entered the ventricle after 50 cm it should be withdrawn to the 30-cm mark and reinserted with the balloon partially inflated.

None of the described complications has caused serious morbidity, and correct placement of the catheter is possible in a higher proportion of cases than has been reported for the placement of c.v.p. catheters (Johnston and Clark, 1972).

**INDICATIONS FOR PULMONARY ARTERY CATHETERIZATION**

**Pulmonary oedema.**

Probably the most clearly established use of the Swan Ganz catheter is in the control of pulmonary venous pressure to prevent pulmonary oedema. Sarim et al. (1970) showed that pulmonary oedema following cardiac surgery could be prevented by maintaining left atrial pressure below 10–12 mm Hg. Patients with a diseased left ventricle are at risk whenever fluid replacement or intravenous therapy is necessary. Usually the volume of fluid to be infused may be estimated by careful observation of fluid loss, or by measurement of c.v.p. In some cases, however, this is inadequate to prevent pulmonary oedema. Measurement of wedge pressure in such patients will determine whether or not the pulmonary venous pressure has increased above the safe limit suggested above, and enable the infusion to be adjusted to a rate acceptable to the failing heart.

Where massive transfusion is necessary in patients with heart failure or raised pulmonary vascular resistance (for example, in the treatment of haematemesis in a patient with coronary artery disease or chronic bronchitis) monitoring of wedge pressure may be the only reliable way of assessing the adequacy of blood volume replacement. The hypovolaemia which is often associated with acute myocardial infarction (Loeb et al., 1969) may also be treated by transfusion regulated by reference to the PA$_{wedge}$ pressure. In this condition c.v.p. has been shown to be an unreliable index of the need for transfusion. A low wedge pressure indicates hypovolaemia and implies that cardiac output may be improved by plasma volume expansion. As in the surgical patient, pulmonary oedema may be avoided by adjusting the rate of transfusion so that a wedge pressure of 10 mm Hg is not exceeded.

It should be recognized, however, that not all pulmonary oedema is the result of pulmonary venous congestion (Karliner, 1972). Uraemic patients have excessively permeable pulmonary capillaries and develop pulmonary oedema without abnormal vascular pressures (Gibson, 1966). Oedema may occur also in lungs damaged by pneumonia, parenchymatous trauma, inhalation of chlorine or the ingestion of paraquat. Of the patients described in table I, in two out of three cases of pulmonary oedema resulting from the aspiration of stomach contents the PA$_{wedge}$ was grossly increased. The author has investigated also a patient suffering from pulmonary oedema accompanied by sodium retention associated with long-term intermittent positive pressure ventilation (Gett, Sherwood Jones and Shepherd, 1971). It was found that the pulmonary oedema was not prevented by maintaining the wedge pressure at the normal value. In these non-cardiogenic forms of pulmonary oedema, PA$_{wedge}$ pressure monitoring will only help to exclude pulmonary venous hypertension as a causative or aggravating factor.

PA$_{wedge}$ pressure has been measured in 14 patients with pulmonary oedema treated by the author (table I). In 5 patients pulmonary venous pressure was less than 10 mm Hg. The pulmonary oedema in these patients is assumed to be partly the result of abnormal pulmonary capillary permeability. In 7 of the 9 patients with an increased pulmonary venous pressure, the c.v.p. was 5 cm H$_2$O or less (table I) and was therefore of no help in the prevention of circulatory overloading.

**Assessment of the adequacy of left ventricular filling.**

PA$_{wedge}$ pressure is related closely in certain circumstances to the left ventricular filling pressure.
### TABLE I. Patients with pulmonary oedema investigated by measurement of pulmonary artery wedge pressure.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Diagnosis and comments</th>
<th>c.v.p. (cm H$_2$O)</th>
<th>PA$_{wedge}$ (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pneumonia and septal infarct (c.v.p. and PA$_{wedge}$ continued to coincide approximately)</td>
<td>19</td>
<td>17</td>
</tr>
<tr>
<td>2</td>
<td>Pneumonia, coronary insufficiency (as l.v.f. improved PA$_{wedge}$ decreased to 2 mm Hg and circulation was improved by dextran)</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>Pulmonary oedema, possibly the result of inhalation of gastric contents</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>Inhaled gastric contents</td>
<td>5</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>Uraemia. Pulmonary oedema at least partly the result of pulmonary venous hypertension</td>
<td>23</td>
<td>22</td>
</tr>
<tr>
<td>6</td>
<td>Staphylococcal pneumonia</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>7</td>
<td>Ischaemic heart disease; mitral valve disease</td>
<td>-2</td>
<td>18</td>
</tr>
<tr>
<td>8</td>
<td>Peritonitis (also chronic bronchitis and hypertension)</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>9</td>
<td>Multiple injuries, chronic bronchitis</td>
<td>5</td>
<td>18</td>
</tr>
</tbody>
</table>

In all the above, pulmonary oedema was associated with an increased pulmonary venous pressure, but only in patients 1 and 5 was the c.v.p. increased significantly.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Diagnosis and comments</th>
<th>c.v.p. (cm H$_2$O)</th>
<th>PA$_{wedge}$ (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Primary pulmonary hyperventilation syndrome; pneumonia</td>
<td>-2</td>
<td>9</td>
</tr>
<tr>
<td>11</td>
<td>Peritonitis (chronic bronchitis). Pulmonary oedema possibly the result of infection. Patient rehydrated cautiously and recovered</td>
<td>-7</td>
<td>6</td>
</tr>
<tr>
<td>12</td>
<td>Uraemia and pulmonary oedema. Oedema persisted although PA$_{wedge}$ maintained below 10 mm Hg</td>
<td>0-5</td>
<td>10</td>
</tr>
<tr>
<td>13</td>
<td>Head injury. Inhalation of gastric contents. C.v.p. misleadingly high. Improved by transfusion</td>
<td>16-24</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>Chest injury. Persistent pulmonary oedema necessitated IPPV for 4 weeks. Improved after negative sodium balance established</td>
<td>20±</td>
<td>8</td>
</tr>
</tbody>
</table>

In patients 10-14 pulmonary oedema was present without a significantly increased pulmonary venous pressure. In two patients (13 and 14) the very high c.v.p. might have suggested pulmonary venous hypertension.

Note: All pressures measured using the sternal angle as the reference point during expiration.

l.v.f. = left ventricular failure.

However, when the ventricle is failing, this relationship is disturbed by the booster pump effect of the left atrium (Rahimtoola et al., 1972; Mitchell et al., 1962). Atrial contraction boosts the failing ventricle by causing an acute increase in ventricular diastolic pressure just before ventricular systole. This pressure (l.v.e.d.p. "post-a") is the true filling pressure of the ventricle, and in cardiac failure it may be significantly different from the mean left atrial pressure and therefore from PA$_{wedge}$.

Moreover, it is not possible to define a range of filling pressure which can be considered normal; at any given filling pressure the energy or ejectile force produced by the ventricle will depend on its condition at that time, its exposure to sympathetic stimulation, and the amount of stretch associated with that filling pressure. The amount of "stretch", in the sense of lengthening of ventricular muscle fibres, which is associated with any particular filling pressure is dependent upon the "compliance" of the ventricle (Levine, 1972). In a non-compliant ventricle, the inflow of a normal stroke volume during diastole will increase the end diastolic pressure to a high level. In constrictive disease of the pericardium or endocardium, or in infiltrative disease of the myocardium, uncompliant ventricles may be associated with high filling pressures even in the absence of a defect in myocardial contractility. Change in compliance is also seen in patients with acute myocardial ischaemia, although in these cases changes in contractility may predominate.

In deciding whether a particular level of PA$_{wedge}$ pressure is associated with inadequate left ventricular filling or, perhaps, with ventricular failure, it is necessary to observe the effect of a change in PA$_{wedge}$ on cardiac output, the latter either measured directly or inferred from clinical observation. If it is suspected that the filling pressure of the left ventricle may be inadequate, the effect of raising the PA$_{wedge}$ to 10 mm Hg by transfusion may be observed. If the PA$_{wedge}$ is found to be initially greater than 15 mm Hg, the use of inotropic agents and diuretics is indicated. As ventricular function improves and PA$_{wedge}$ falls, hypovolaemia may be revealed and further improvement achieved by cautious expansion of the intravascular space (as in patient 2, table I).

The author has investigated 9 patients in whom the distinction between left heart failure and hypovolaemia has been difficult (table II). Four were grossly oligoametic in spite of a normal or increased c.v.p. One patient who was vasodilated, hypotensive...
TABLE II. Pulmonary artery wedge pressure measurement to assess the adequacy of left ventricular filling pressure.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Diagnosis and comments</th>
<th>Initial pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>c.v.p. (cm H₂O)</td>
</tr>
<tr>
<td>15</td>
<td>Gram-negative sepsicaemia. Improved by further transfusion</td>
<td>22</td>
</tr>
<tr>
<td>16</td>
<td>Haematemesis (chronic bronchitis)</td>
<td>11</td>
</tr>
<tr>
<td>17</td>
<td>Peritonitis. No definite history of pulmonary disease. Improved by transfusion</td>
<td>5</td>
</tr>
<tr>
<td>18</td>
<td>Gram-negative sepsicaemia (PA wedge pressure reduced and anuria recovered when adrenaline infusion commenced)</td>
<td>8</td>
</tr>
<tr>
<td>19</td>
<td>Perforated gastric ulcer (chronic bronchitis)</td>
<td>4-7</td>
</tr>
<tr>
<td>20</td>
<td>Pneumonia and pre-renal uraemia (chronic bronchitis)</td>
<td>7</td>
</tr>
<tr>
<td>21</td>
<td>Septicaemia; anuria. Filling pressure apparently adequate. Did not improve with transfusion</td>
<td>10</td>
</tr>
<tr>
<td>22</td>
<td>Prolonged postoperative bleeding</td>
<td>9</td>
</tr>
<tr>
<td>23</td>
<td>Pneumonia. Because of chronic bronchitis oligaeemia suspected in spite of normal c.v.p. PA wedge indicated adequate left ventricular filling and transfusion produced no improvement</td>
<td>1</td>
</tr>
</tbody>
</table>

In this group of nine patients, four (patients 15, 17, 19 and 20) were seriously oligaeemic in spite of normal or high c.v.p. One had gross left heart failure with only a slightly raised c.v.p. (patient 18).

Measurement of cardiac output.

The pulmonary artery catheter may be used in the Fick technique to withdraw a true “mixed venous” blood sample, and in the dye dilution method the dye may be injected directly into the pulmonary artery. A special catheter has been devised for use in the thermal dilution method for measuring cardiac output. A bolus of cold dextrose solution may be injected into the right ventricle and the degree of dilution of its “coolth” by the pulmonary artery blood flow may be measured by a thermistor carried near the tip of a specially constructed Swan Ganz catheter. Many advantages are claimed for this method, which does not require arterial catheterization, and is said to be more accurate than the dye dilution methods in low cardiac output states (Forrester et al., 1972). The principal disadvantage is the price of the disposable catheter which is approximately £35.

Pulmonary angiography.

To facilitate fast injection of contrast medium into the pulmonary artery a catheter is available with side holes proximal to the balloon. It is, of course, not possible to measure wedge pressure with this catheter. Injection of contrast medium through the standard size 7FG catheter is possible although the relatively slow injection speed produces poor definition when using an image intensifier. Nevertheless, it has been possible to localize a pulmonary embolus in the left pulmonary artery in two patients investigated by the author using the standard catheter. Treatment with streptokinase injected into the site of the obstruction by continuous infusion through the catheter was followed by resolution, which was confirmed by further angiography on the two subsequent days.

CONCLUSION

Swan Ganz catheters are expensive (£12.50 each for size 7FG), and must obviously be used with discrimination. However, in the management of complicated haemodynamic problems, measurement of the wedge pressure may provide valuable information which is difficult to obtain otherwise.

REFERENCES

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Intracardiac knotting of the catheter in heart catheteri-


Johnston, A. O. B., and Clark, R. G. (1972). Malposition-

Karlins, J. S. (1972). Noncardiogenic forms of pulmon-


Extrasystoles and Allied Arrhythmias (2nd edn). By David

Scherf and Adolf Schott. Published by William


1041. Price £16.

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grams used to illustrate the text could have been im-

proved by a more ruthless replacement policy on the part

of the authors. Equally the dependence on recordings

from dogs seems misplaced when even the most bizarre

combinations of arrhythmias are readily found in any

department of cardiac surgery.

The technical aspects are almost entirely neglected in

this book. No description of relevant recording apparatus

is provided and although the cellular electrophysiological

changes associated with cardiac activity are discussed no

account of the composition of a normal e.g. complex is

furnished. Similarly the reader seeking information about

the relative value of scalar and vector electrocardiograms

must look elsewhere; indeed little guidance is given even

on the choice of leads. Furthermore, since extrasystoles

and arrhythmias are among the easiest cardiac variables

to be detected and analysed electronically some reference

to analogue and digital computing methods for handling

the data might have been included particularly as an

extensive literature on the subject has built up over the

past 10 years.

Those sections of particular interest to anaesthetists are

disappointing. The material is presented almost in cata-

logue form without any reasoned exposition and with

little attempt to interpret the evidence; in particular too

many opinions are offered as facts. The references in

these sections appear to have been chosen indiscriminately

and many regarded by anaesthetists as definitive in their

fields are absent whereas other relatively minor contribu-

tions are given prominence.

Overall the bibliography is extensive and for those

departments with a substantial library budget this could be

a useful reference book.

J. P. Payne