

https://dspace.org/

Contributions to an understanding of community-acquired pneumonia

Feldman, Charles

2010-09-03
http://hdl.handle.net/10539/8612

Downloaded from a DSpace repository. https://dspace.org/

Quarterly Journal of Medicine, 1993; 86:669–675

A study of acute community-acquired pneumonia, including details of cardiac changes

M.A. SEEDAT, C. FELDMAN, J. SKOULARIGIS, D.A. PROMNITZ, C. SMITH and S. ZWI

From the Department of Medicine, Baragwanath and Hillbrow Hospitals and University of the Witwatersrand, Johannesburg, South Africa

Received 1 March 1993; Accepted 5 May 1993

Summary

We prospectively studied 102 patients, aged 15–50 years, with acute community-acquired lobar pneumonia without underlying cardiorespiratory illness, admitted to Baragwanath Hospital May 1990–April 1991. Demographic, clinical, microbiological and laboratory data and negative prognostic features of these patients are described. In particular, we documented electrocardiographic changes and studied their possible relevance in patients with pneumonia.

Electrocardiographic changes occurred in 32 patients (31%). The commonest changes were clockwise rotation (16%), followed by P. pulmonale (9.8%) and S1 Q3 T3 pattern (7.8%) Other changes included right axis deviation ($n=6$), right bundle branch block ($n=3$), ventricular extrasystoles ($n=2$), atrial fibrillation ($n=1$) and nodal rhythm ($n=1$). These changes returned to normal in survivors after a mean of 2 days.

The S1 Q3 T3 pattern was associated with cardiac enzyme leak (CK-MB fraction), hypoxia and a high Simplified Acute Physiology Score (SAPS). In addition, P. pulmonale, right axis deviation and clockwise rotation correlated with hypoxia and a high SAPS score. Clockwise rotation also correlated with serum (including cardiac fraction) enzymes leak (LDH and CK-MB fraction), and pulmonary artery pressure.

The overall mortality rate was 10.8%, with no association between electrocardiographic changes and mortality. The negative prognostic factors documented were hypoxia ($p<0.0001$), multilobar pulmonary consolidation ($p<0.0001$), tachycardia ($p=0.0001$), tachypnoea ($p=0.0002$), renal dysfunction ($p=0.0009$), hypotension (systolic $p<0.02$, diastolic $p<0.003$), bacteraemia ($p=0.003$), and serum (including cardiac fraction) enzymes leak: LDH ($p<0.02$), CK ($p<0.002$) and CK-MB fraction ($p=0.0002$). These factors, with the exception of renal dysfunction, also correlated with the need for intensive care unit admission.

Acute and reversible electrocardiographic changes are common in acute community-acquired lobar pneumonia. Electrocardiographic changes, especially those compatible with acute cor pulmonale and accompanied by cardiac enzyme (CK-MB fraction) leak, correlated with severity of illness but not with mortality.

Introduction

Pneumonia is an important cause of disease worldwide, and is associated with significant morbidity and mortality.[1,2,3] Despite advances in antimicrobial therapy, improved diagnostic and microbiological techniques and sophisticated respiratory support systems, pneumonia still represents a major challenge to the physician. For example, the mortality of patients with pneumonia requiring

Address correspondence to Professor C. Feldman, Department of Medicine, University of the Witwatersrand Medical School, York Road, Parktown 2193, Johannesburg, Republic of South Africa

This paper is based on the thesis submitted by MAS to the University of Witwatersrand for the degree of Master of Medicine (Internal Medicine)

© Oxford University Press 1993

admission to an intensive care unit still exceeds 50%.[4,5]

Certain negative prognostic features, when present on admission of patients to hospital, are associated with a poorer outcome.[4,5] Cardiac factors associated with a worse outcome include the presence of hypotension, need for inotropic support of the blood pressure, atrial fibrillation and/or prior treatment with digoxin.[3,5,6] In addition, one study of electrocardiographic changes in patients with pneumonia has suggested that such changes occurred more frequently in cases with moderate to severe infection, compared with mild cases of pneumonia.[7]

The present study describes the demographic, clinical and laboratory data of a large group of patients with acute community-acquired lobar pneumonia admitted to the Baragwanath Hospital in Soweto. We documented the electrocardiographic changes that occurred and their reversibility, and in particular the association of these changes with a severity of illness index, results of blood gas analysis, serum (including cardiac fraction) enzyme levels, and echocardiographic changes, to see if these changes were of any prognostic significance.

Methods

We studied prospectively 102 patients aged 15–50 years who had previously been well, with no underlying cardiac or respiratory disorder, admitted to the Baragwanath Hospital, Soweto with acute community-acquired lobar pneumonia. The study was conducted over the 12 month period between May 1990 and April 1991. The study was approved by the Committee for Research on Human Subjects of the University of the Witwatersrand.

We recorded the age and gender of the patients, initial heart and respiratory rates, blood pressure, temperature, number of affected pulmonary lobes, and Glasgow Coma scale. The severity of illness was assessed by means of the Simplified Acute Physiology Score (SAPS),[8] and the prognosis was assessed by duration of hospital stay, need for intensive care unit admission and outcome.

One set of blood cultures (both aerobic and anaerobic bottles) was drawn from each patient on admission to hospital and prior to any antimicrobial therapy. They were processed using a radiometric method in the BACTEC 460 instrument system (Towson). Deep cough sputum specimens were also obtained from each patient prior to any therapy and were Gram stained and cultured. Testing for viruses and for *Legionella pneumophila*, *Mycoplasma pneumoniae* and *Chlamydia* spp. was done only if such infections were clinically suspected. We also recorded additional laboratory data needed to calculate the SAPS index, including initial haemoglobin and haematocrit, white cell and platelet counts, serum sodium, potassium, urea, creatinine, glucose and bicarbonate, as well as the arterial blood gas profile.

Patients were clinically examined for evidence of pulmonary hypertension or right heart failure. An electrocardiogram was done for each patient by MAS on admission to hospital and daily until discharge. The same machine was always used, and the position of the electrodes was marked on the patient using an indelible pencil. An echocardiogram was performed on each patient by JS to assess ventricular function and for Doppler measurement of pulmonary artery pressures in those cases where it was technically feasible. Serum levels of lactate dehydrogenase (LDH), creatinine kinase (CPK) and the cardiac fraction of CK (CK-MB) were measured on admission and daily as necessary thereafter.

Statistical analysis of continuous variables was by means of the Student's *t* test and Mann-Whitney U test. Categorical variables were analysed by means of the χ^2 test and the Fisher's exact (2-tail) test. Survivors were compared with non-survivors to detect negative prognostic factors. The Spearman and Pearson rank correlation coefficient tests were used for assessing the correlation of echocardiographic changes (particularly pulmonary artery pressure, right ventricular diameter, and ejection fraction), and hypoxia, SAPS, and serum enzymes leak. Multivariate analysis could not be performed because of the small patient numbers in the non-survivor group, and because Doppler measurements of pulmonary artery pressure were technically possible in only 16 patients.

Results

Demographic and clinical data

Of 102 patients, 80 were male. Mean age was 32 years (range 15–50). Mean initial temperature was 38.8 °C (range 38–40), mean systolic blood pressure 113 mmHg (range 70–140), mean diastolic blood pressure 69 mmHg (range 30–90), mean heart rate 108 bpm (range 76–144), and mean respiratory rate 25 bpm (range 15–50). Ten patients were shocked on admission (systolic blood pressure <90 mmHg with poor peripheral perfusion).

Laboratory data

Mean haemoglobin was 13.6 g/dl (range 10.1–20.4), mean white cell count 14.9×10^9/l (range $1.5–35.6 \times 10^9$), and mean platelet count 247×10^9/l (range $95–579 \times 10^9$/l). Eight cases had leucopenia (WCC $<4 \times 10^9$/l) and 24 patients had a marked

leucocytosis (WCC $>20 \times 10^9$/l). Thrombocytopenia (platelets $<150 \times 10^9$/l) was noted in 13 patients.

Mean serum urea was 8.3 mmol/l (range 1.9–29.7), creatinine 112 μmol/l (range 49–356), sodium 133 mmol/l (range 119–146), and potassium 3.9 mmol/l (range 2.4–5.3). Twenty-two patients had serum sodium <130 mmol/l, which occurred in the setting of pre-renal failure in 14 cases and syndrome of inappropriate ADH secretion in 8 cases. Serum urea was >7 mmol/l in 50% of patients.

Mean pO_2 was 54.3 mmHg (range 26.2–87.2), and pCO_2 29.1 mmHg (range 28–37). Thirty-eight patients had severe hypoxia (pO_2 <50 mmHg) and 5 patients were hypercapnoeic (pCO_2 >38 mmHg). The normal values in Johannesburg (1760 metres above sea level) are 69–84 mmHg for pO_2, and 28–37mmHg for pCO_2. Seven patients had an associated metabolic acidosis.

Microbiological data

The microbiological results are shown in Table 1. These include sputum and blood culture results, and suspected pathogens based on sputum Gram stain (in the presence of negative cultures). Including all this data, *Streptococcus pneumoniae* as a pathogen was found in 56 cases, *Klebsiella pneumoniae* in 6 and *Staphylococcus aureus* in 1. Three further cases of suspected Gram-negative bacillary pneumonia were based on the sputum Gram stain. No aetiology at all was found in 36 patients by any method (35%). All serology for viruses and atypical pathogens was negative where performed.

Radiological data

Seventy-two cases (72%) had unilobar and the remainder (28%) had multilobar pulmonary consolidation.

Cardiological data

Clinical evidence of pulmonary hypertension was recorded in 7 patients. All had ECG changes; 4 had S1 Q3 T3 pattern (3 died), 2 had P. pulmonale (both died), and one had right bundle branch block (RBBB) (survived).

Electrocardiographic changes occurred in 32 patients (31%). The commonest changes were clockwise rotation (16% of all cases), P. pulmonale (9.8%), and S1 Q3 T3 pattern (7.8%). Other changes seen were right axis deviation (RAD) ($n=6$), ST-T wave changes ($n=6$), RBBB ($n=3$), ventricular extrasystoles (VES) ($n=2$), atrial fibrillation ($n=1$) and nodal rhythm ($n=1$). The mean duration of ECG changes was 2 days. All the ECG changes were reversible and all survivors had a normal ECG on discharge from hospital.

Several elevated serum enzyme levels were noted. The mean lactate dehydrogenase (LDH) was 439 U/l (range 96–1353; normal 230–462). The mean creatinine kinase (CPK) was 239 U/l (range 20–1832; normal 24–195). Among the patients with raised CPK levels, the cardiac CK-MB fraction was measured in 9 cases. It was abnormally raised in 3 cases, 2 with S1 Q3 T3 pattern and 1 with ST-T wave changes in the inferior leads.

On echocardiogram the mean left ventricular end diastolic diameter was 48.6 mm (range 40–63.8) and left ventricular end systolic diameter was 34.5 mm (range 22.8–49). Mean right ventricular diameter (RVD) was 30 mm (range 22–42). The mean LV ejection fraction was 52% (range 22–78%). Pulmonary artery pressure (PAP) could be measured in 16 cases. The mean PAP was 35 mmHg (range 21–60 mmHg) and correlated with hypoxia (both mean and absolute pO_2 <60 mmHg). Table 2 shows the correlation of hypoxia, SAPS, PAP, RVD, and serum enzymes.

Table 1 Results of sputum Gram stain, sputum culture and blood culture in 102 patients with acute community-acquired pneumonia

Aetiology	Sputum		Blood culture	Total positives ($n=66$)
	Gram stain	Culture		
Streptococcus pneumoniae	19*	29	13**	56
Klebsiella pneumoniae	–	4	3***	6
Gram-negatives	3****	–	–	3
Staphylococcus aureus	–	1	–	1

*Suspected pathogen, based on predominant presence in the Gram stain of Gram-positive diplococci in significant numbers in the absence of positive sputum or blood cultures.
**Five of these cases were also sputum culture positive.
***One of these cases was also sputum culture positive.
****Suspected Gram-negative pathogen, based on the presence in the Gram stain of Gram-negative bacilli in significant numbers in the absence of positive sputum or blood cultures.

Table 2 Correlation of hypoxia, SAPS, right ventricular diameter, pulmonary artery pressure, ejection fraction and serum enzymes

	SAPS	RVD (mm)	PAP (mmHg)	EF (%)
Mean arterial pO_2 (mmHg)*	$p=0.0001$	NS	$p<0.02$	NS
Arterial pO_2 <60 mmHg**	$p=0.0001$	NS	$p<0.04$	NS
	LDH (U/l)	CK (U/l)	CK-MB (U/l)	CKMB%
SAPS***	$p<0.03$	$p=0.0001$	$p=0.0001$	$p<0.002$

NS, not significant; SAPS, simplified acute physiology score; RVD, right ventricular diameter; PAP, pulmonary artery pressure; EF, ejection fraction; LDH, lactate dehydrogenase; CK, creatinine kinase; CK-MB, creatinine kinase MB fraction; CK-MB%, creatinine kinase-MB fraction expressed as a percentage of total creatinine kinase.
*Pearson rank coefficient test.
**Spearman rank coefficient test.
***Pearson/Spearman rank coefficient test.

The S1 Q3 T3 pattern was associated with cardiac enzyme (CK-MB fraction) leak ($p=0.001$), hypoxia ($p=0.004$) and a high SAPS ($p<0.006$). In addition P. pulmonale, RAD and clockwise rotation were associated with hypoxia ($p<0.004$, $p<0.03$ and $p=0.002$, respectively) and a high SAPS ($p<0.02$, $p=0.003$ and $p=0.008$, respectively).

Clockwise rotation was associated with serum enzymes (including cardiac fraction) leak: LDH ($p<0.04$) and CK-MB ($p<0.02$), and pulmonary arterial hypertension ($p=0.005$). Although there was no association between other electrocardiographic changes (S1 Q3 T3, P. pulmonale and right axis deviation) and pulmonary arterial hypertension, all the patients with these electrocardiographic changes and in whom PAP could be measured, had evidence of significant pulmonary arterial hypertension (systolic pulmonary artery pressure >30 mmHg).

There was no association between ST segment-T wave changes (consistent with myocardial ischaemia) and serum enzymes (including cardiac fraction CK-MB) leak, blood gas profiles, echocardiographic parameters and SAPS.

Prognosis and mortality

The mean SAPS for the entire group was 6 (range 0–21), being 5 for survivors and 15 for non-survivors. SAPS correlated with need for ICU admission and mortality ($p<0.0001$), presence of hypoxia ($p<0.0001$) and serum enzyme leaks ($p<0.03$) (Table 2).

Eleven cases were admitted to the ICU (10.8%), with a mortality of 55%, while 91 cases were not admitted to the ICU (89.2%) with a mortality of 5.5% ($p=0.0007$). Significant differences in clinical and laboratory data of patients admitted to ICU and those not admitted to the unit, are shown in Table 3.

Eleven patients died (mortality 10.8%), of whom six had been admitted to the ICU. Significant clinical and laboratory data differences between survivors and non-survivors are shown in Table 4. In addition the parameters shown, bacteraemia ($p<0.003$) and multilobar pulmonary consolidation ($p<0.0001$) were predictors of a worse prognosis.

The mortality rate for patients with and without ECG changes was not significantly different (15% vs. 8.4%), nor was there a correlation with the need for ICU admission.

Of the cardiological parameters measured, only serum enzymes leak (including the cardiac CK-MB fraction) was predictive of poor outcome (LDH, $p=0.02$; CPK, $p=0.002$; MB percentage of the total CK [CK-MB%], $p=0.02$). Raised cardiac enzyme (CK-MB fraction) was associated with the need for intensive care unit admission. Echocardiographic parameters measured and electrocardiographic changes did not predict poor outcome or the need for intensive care unit admission.

Discussion

This study of patients with community-acquired pneumonia documents a number of important negative prognostic factors, and factors predicting the need for admission of patients to an ICU similar to those shown in several previous studies.[4–6] In addition, there are a number of significant changes in cardiac parameters not previously noted.

The present study suggests that ECG changes in patients with acute community-acquired pneumonia are not uncommon, occurring in 31% of cases. The commonest abnormality was clockwise rotation, followed by P. pulmonale and S1 Q3 T3 pattern. Other changes noted included RAD, ST-T wave changes, RBBB, VES, atrial fibrillation and idionodal rhythm. All these changes returned to normal (within a mean

Table 3 Significant differences found in clinical and laboratory data of patients with acute community-acquired pneumonia admitted ($n=11$) and not admitted ($n=91$) to an intensive care unit

Clinical data	Not admitted ($n=91$)		Admitted ($n=11$)		p
	Mean ± SD	Range	Mean ± SD	Range	
Temperature (°C)	38.7 ± 0.6	38.0–40.0	39.4 ± 0.6	38.0–40.0	<0.002
Heart rate (bpm)	107 ± 12	76–144	118 ± 10	105–140	=0.005
Breath rate (per min)	24 ± 7	15–44	39 ± 8	25–50	=0.0001
Systolic BP	114 ± 14	80–140	100 ± 15	70–125	=0.003
Diastolic BP	71 ± 10	30–90	59 ± 10	40–80	=0.0004
SAPS	5 ± 4	0–21	13 ± 3	7–14	<0.0001
Laboratory data					
Serum Na (mmol/l)	133 ± 6.0	119–146	130 ± 5	124–141	<0.02
Arterial pO_2 (mmHg)	56.2 ± 11.6	26.2–87.2	37.9 ± 6.9	30.0–53.3	<0.0001
LDH (U/l)	420 ± 158	96–933	618 ± 338	358–1353	<0.003
CK (U/l)	207 ± 283	20–1832	530 ± 430	64–1080	<0.03
CK-MB (U/l)	2 ± 8	0–55	11 ± 19	0–52	<0.02
CKMB%	0.4 ± 2	0–23	1 ± 2.5	0–8	<0.02

Abbreviations as for Table 2.

Table 4 Significant differences in clinical and laboratory data of survivors ($n=91$) and non-survivors ($n=11$) among patients with acute community-acquired pneumonia

Clinical data	Survivors ($n=91$)		Non-survivors ($n=11$)		p
	Mean ± SD	Range	Mean ± SD	Range	
Age (years)	32 ± 10	15–55	35 ± 12	20–55	NS
Temperature (°C)	38.8 ± 0.7	38.8–40.0	39.3 ± 0.5	38.6–40.0	<0.02
Heart rate (bpm)	106 ± 11	76–130	124 ± 13	110–144	=0.0001
Breath rate (per min)	24 ± 7	15–50	36 ± 8	19–46	=0.0002
Systolic BP (mmHg)	114 ± 13	80–140	99 ± 22	70–140	<0.02
Diasystolic BP (mmHg)	71 ± 9	40–90	57 ± 17	30–85	=0.003
SAPS	5 ± 3.5	0–16	15 ± 3	10–21	<0.0001
Laboratory data					
Serum urea (mmol/l)	7.7 ± 4.2	2.0–5.2	14.1 ± 6.9	1.9–28.9	=0.0009
Serum creatinine (μmol/l)	70.1 ± 8.4	62–144	140.6 ± 17.8	65–356	=0.04
Arterial pO_2 (mmHg)	56.4 ± 11.3	26.5–87.2	36.4 ± 7.3	26.2–48.4	<0.0001
LDH (U/l)	425 ± 169	96–1135	573 ± 307	134–1353	<0.02
CK (U/l)	189 ± 232	20–1590	692 ± 542	60–1832	<0.002
CKMB (U/l)	1.4 ± 6.9	0–49.0	15.6 ± 22.9	0–55	=0.0002
CKMB%	0.4 ± 2.6	0–23	0.9 ± 1.3	0–23	0.0005

Abbreviations as Table 2.

of 2 days), and ECGs were normal in all survivors on discharge from hospital. In general, ECG changes were associated with greater severity of illness (particularly as recorded by SAPS), but did not correlate with outcome. This finding may have been due to the relatively low mortality rate.

Mechanisms of ECG changes in patients with pneumonia may be multifactorial and may include hypoxia, electrolyte changes, adrenergic stimulation, and direct cardiac involvement.[9–11] In an attempt to detect possible cardiac muscle injury we measured serum enzymes (LDH and CPK) as well as the cardiac MB fraction (noting its percentage of the total CPK) while acknowledging that some of the raised serum enzyme levels may have been due to non-cardiac skeletal muscle affects. In summary, the changes that we detected in this study included features of acute cor pulmonale, (associated with hypoxia, cardiac enzyme leak [CK-MB fraction] and increased RV diameter), and ST-T waves changes (compatible with myocardial ischaemia), which were not associated with serum enzyme leak, blood gas profiles or echocardiographic changes.

In the present study, there was no association between ST segment-T wave changes and respiratory rate, severity of illness, mortality and blood gas

profiles. Although the complete data have not been shown, it was noted that patients with ST segment-T wave changes had significantly lower mean serum potassium concentrations on admission compared with patients in the group with no electrocardiographic changes. However, with the exception of one patient, in whom a serum potassium of 3.4 mmol/l was recorded, all patients with ST-segment and T wave changes had normal serum potassium. Further, there was no association between ST segment-T wave changes and serum enzymes leak, thus excluding myocardial injury from whatever cause as a possible explanation for the ECG changes compatible with myocardial ischaemia.

The phenomenon of electrocardiographic changes compatible with myocardial ischaemia in patients with pneumonia remains unexplained. Electrocardiographic changes consistent with myocardial ischaemia can occur with angiographically normal coronary arteries and have been documented in patients with severe acute asthmatic attacks[9–11] and during hyperventilation.[12,13]

Electrocardiographic abnormalities consistent with acute cor pulmonale (including S1 Q3 T3 pattern, P. pulmonale, clockwise rotation and right axis deviation) seen in this study appear to reflect right ventricular strain secondary to acute pulmonary hypertension.[9–11]

Patients with electrocardiographic changes compatible with acute cor pulmonale had greater degree of hypoxaemia than those without electrocardiographic changes. Furthermore, patients with P. pulmonale and clockwise rotation had a greater right ventricular mean diameter than those with normal electrocardiograms. A correlation between systolic pulmonary artery pressure and ECG changes was documented with clockwise rotation only. Despite the absence of a statistical association between systolic pulmonary artery pressure and other electrocardiographic changes reflecting right heart embarrassment, systolic pulmonary arterial hypertension was documented in all the patients with P. pulmonale and S1 Q3 T3 pattern.

Moreover, there was an association between hypoxia (both mean arterial pO_2 and arterial pO_2 <60 mmHg) and systolic pulmonary artery pressure but not right ventricular diameter. This is indirect evidence that the electrocardiographic changes compatible with acute cor pulmonale reflect hypoxia-induced acute vasoconstrictive pulmonary hypertension.

In this study a definite association between PAP and electrocardiographic changes was not established. In a previous study of patients with severe asthma and ECG changes of acute cor pulmonale, even cardiac catheterization failed to demonstrate elevated right heart pressures.[14] In the present study, systolic PAP could be measured only in 16 patients. No measurement could be made in the others due to a technical difficulty encountered with the Doppler measurement of PAP. Systolic PAP was measured non-invasively using Doppler flow velocity by measuring peak regurgitant velocity in those patients with tricuspid incompetence. Doppler echocardiography was used to measure systolic PAP because as many as 80% of normal subjects have Doppler evidence of tricuspid regurgitation,[15,16] thus evaluation of tricuspid flow should provide a high yield. In the present study, however, the absence of tricuspid regurgitation in the majority of patients precluded measurements of systolic PAP, as noted by Feigenbaum.[17] In fact, a pulmonary artery mean pressure of 20 mmHg or more is necessary before one can expect any change in pulmonary and tricuspid valve motion.[17]

We found changes of cor pulmonale associated with cardiac enzyme (CK-MB fraction) leaks. This implies myocardial cell ischaemia or injury. The exact mechanism of this myocardial injury is not understood. It may be due to the direct toxic effect of the organism, mediator damage as part of the immune-mediated injury, or the effect of hypoxia. Hypoxia has a direct myocardial depressant effect whereby cardiac output decreases with severe hypoxia.[18,19] Indirectly, myocardial ischaemia occurs secondary to hypoxia induced vasoconstrictive pulmonary hypertension and increased right ventricular afterload.[20,21]

None of the patients in this study had impaired left ventricular function (assessed by echocardiographic parameters), thus global myocardial injury or depression secondary to hypoxia,[18,19] alcohol,[22] myocardial depressant factor release during sepsis [23–25] or myocarditis are unlikely mechanisms.

It is theoretically possible that some of the ECG changes of acute cor pulmonale may have been due to pulmonary thromboembolic disease. Invasive exclusion of embolism was not routinely performed in these cases on clinical grounds.

Hypotension and the frequent use of inotropes to support the cardiovascular system, well-documented negative prognostic factors in patients with pneumonia, may be due in part to right-side myocardial injury resulting in right ventricular dysfunction and decreased left ventricular filling and compliance. This assumption is strongly supported by the association between (a) electrocardiographic changes compatible with acute cor pulmonale and cardiac enzymes leak and (b) the association between cardiac enzymes leak and both severity of illness and mortality.

These observations raise the possibility that death in pneumonia may, in some cases, be due to acute cor pulmonale, secondary to hypoxia-induced pul-

monary vasoconstrictive or direct hypoxia-induced myocardial injury. Indeed, it has been shown that the occurrence of pulmonary hypertension in patients with respiratory failure and accompanied by bacterial sepsis is associated with a poor outcome [20,26–28]. In this study no association between pulmonary arterial hypertension and mortality was established, probably due to the small number of patients who died and the small number of patients in whom it was technically feasible to measure pulmonary artery pressures.

References

1. Bradshaw D, Botha H, Joubert G, Pretorius JP, Van Wyk R, Yach D. *Review of South African mortality (1984).* MRC Technical Report No 1, SA Medical Research Council, Parow, 1987.
2. MacFarlane JT. Acute infection of lower respiratory tract. In Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford Textbook of Medicine,* Vol. 15, 2nd edn. New York, Oxford Medical Publications, 1987:91–100.
3. British Thoracic Society. Community-acquired pneumonia in adults in British Hospital in 1982–1983. A survey of aetiology, mortality, prognostic factors and outcome. *Q J Med* 1987; **62**:195–220.
4. Hook EW, Horton CA, Schaberg DR. Failure of intensive care units support to influence mortality from pneumococcal bacteremia. *JAMA* 1983; **249**:1055–7.
5. Feldman C, Kallenbach JM, Levy H, Reinach SG, Hurwitz MD, Thornburn JR, Koornhof HJ. Community-acquired pneumonia of diverse aetiology: prognostic features in patients admitted to an intensive care unit and a 'severity of illness' score. *Intensive Care Med* 1989; **15**:302–7.
6. Van Eeden SF, Coetzee AR, Joubert JR. Community-acquired pneumonia—factors influencing intensive care admission. *S Afr Med J* 1988; **73**:77–81.
7. Trushinskii ZK, Struik RI, Kransnopolskaia SP. Prognostic significance of electrocardiographic changes in acute pneumonia. *Ter Arkh* 1984; **56**:74–8.
8. LeGall JR, Loirat P, Alperovitch A, *et al.* A simplified acute physiology score for ICU patients. *Crit Care Med* 1984; **12**:975–7.
9. Rebuck AS, Read J. Assessment and management of severe asthma. *Am J Med* 1971; **51**:788–98.
10. Siegler D. Reversible electrocardiographic changes in severe acute asthma. *Thorax* 1977; **32**:328–32.
11. Efthimiou J, Hassan AB, Ormerod O, Benson MK. Reversible T wave abnormality in severe acute asthma: an electrocardiographic sign of severity. *Respiratory Medicine* 1991; **85**:195–202.
12. Biberamn L, Sarma RN, Surawicz B, Lexington K. T wave abnormalities hyperventilation and isoproterenol infusion. *Am Heart J* 1971; **81**:166–74.
13. Lary D, Goldschlager W. Electrocardiographic changes during hyperventilation resembling myocardial ischaemia in patients with normal angiogram. *Am Heart J* 1974; **87**:383–90.
14. Gunstone RF. Right heart pressures in bronchial asthma. *Thorax* 1971; **87**:383–90.
15. Pearlman AS. The use of Doppler in the evaluation of cardiac disorders and function. In: Hurst JW, Schlant RL, Rackley CF, Sonneblick EH, Wengner NK, eds. *The Heart,* 7th edn. New York, McGraw-Hill, 1990; 2039–63.
16. Liebson PR. Echocardiography. In: Parrillo JE, ed. *Current Therapy in Critical Care Medicine,* 2nd edn. Philadelphia, BC Decker, 1991:35–45.
17. Feigenbaum H. Haemodynamic information derived from echocardiography. In: *Echocardiography,* 4th edn. Philadelphia, Lea & Febiger, 1986:188–229.
18. Downing SE, Talner NS, Gardner TH. Influences of hypoxaemia and acidemia on left ventricular function. *Am J Physiol* 1966; **210**:1327–1334.
19. Ng ML, Levy MN, De Geest H, Zieske H. Effects of myocardial hypoxia on left ventricular performance. *Am J Physiol* 1966; **211**:43–50.
20. Sibbald WJ, Driedger AA. Right ventricular function in acute disease state: Pathophysiologic considerations. *Crit Care Med* 1983; **11**:339–45.
21. Wiedeman HP, Matthay RA. Management of acute and chronic cor pulmonale. In: Scharf SM, Cassidy SS, eds. *Heart-Lung Interaction in Health and Disease.* Marcel Dekker Inc, 1989:915–81.
22. Wynne J, Braunwald E. The cardiomyopathies and myocarditidies. In: Braunwald E, ed. *Heart Disease. A Textbook of Cardiovascular Medicine,* 3rd edn. Philadelphia, WB Saunders, 1988:1410–69.
23. Brunet F, Dhainaut JF, Devaux JY, Huygebaert MF, Villemant D, Monsallier JF. Right ventricular performance in patients with acute respiratory failure. *Intensive Care Med* 1988; **14**:474–7.
24. Cunnion RE, Parrillo JE. Myocardial dysfunction in sepsis. Recent insights. *Chest* 1989; **95**:941–945.
25. Parker MM, McCarthy KE, Ognibene FP, Parrillo JE. Right ventricular dysfunction and dilation similar to left ventricular changes, characterize the cardiac depression of septic shock in humans. *Chest* 1990; **97**:126–31.
26. Sibbald WJ, Paterson NA, Holliday RL, Anderson RA, Lobb TR, Duff VH. Pulmonary hypertension in sepsis: measurement by the pulmonary arterial diastolic pulmonary wedge pressure gradient and the influence of passive and active factors. *Chest* 1978; **73**:583–91.
27. Zapol WM, Snider MT. Pulmonary hypertension in severe acute respiratory failure. *N Engl J Med* 1977; **296**:476–80.
28. Sibbald WJ, Prewitt RM. Right ventricular function (editorial). *Crit Care Med* 1983; **11**:321–2.