TABLE: Patient data

Parameter	Before fast Mean value ± SD	After fast Mean value ± SD	t*	P
Chloride level (mmol/L)	105 ± 2.0	103 ± 3.5	2.29	0.02-0.01
Bicarbonate level (mmol/L)	27 ± 3.2	21 ± 2.5	5.30	<0.001
Bilirubin level (μmol/L)	13 ± 6.5	17 ± 6.0	2.81	0.02-0.01
Uric acid level (mmol/L)	0.34 ± 0.068	0.59 ± 0.14	8.67	<0.001
Creatinine level (mg/L)	0.09 ± 0.011	0.10 ± 0.018	4.28	<0.001
Glucose level (mmol/L)	4.4 ± 0.73	3.5 ± 0.69	3.15	0.01-0.005
Cholesterol level (mmol/L)	5.0 ± 0.94	5.6 ± 0.96	2.45	0.05-0.025
Triglyceride level (g/L)	1.08 ± 0.65	1.26 ± 0.38	1.35	0.3-0.2
Lactate dehydrogenase level (U/L)	251 ± 74	207 ± 54	2.42	0.05-0.025
Iron concentration (μmol/L)	19 ± 5.1	16 ± 4.4	2.06	0.1-0.05
Red cell count ($\times 10^{12}$/L)	4.6 ± 0.54	4.8 ± 0.57	1.41	0.2-0.1
Haemoglobin concentration (g/dL)	13.9 ± 1.2	14.6 ± 1.6	2.29	0.05-0.025
Haematocrit	0.40 ± 0.040	0.42 ± 0.043	1.60	0.2-0.1

*Paired *t*-test with 13 df.

congestive cardiac failure increased and progressed to death. That digoxin and all the patient's other medications were ceased at the beginning of the fast, no doubt contributed to the cardiac failure. In contrast to this, for example, Duncan et al. treated nearly 900 patients by periods of fasting that varied from 10–14 days without a single death or the occurrence of a significant complication.[8]

The purpose of this letter is to report the changes in haematological and biochemical parameters during four to five days of water-fasting.

Fourteen consecutive patients who were being investigated in hospital for suspected food-intolerance reactions, underwent four to five days of fasting; only deionized pure water was ingested in unlimited amounts. Haematological and biochemical measurements were obtained on the morning of the first day, that is, after an overnight fast — the way in which "fasting blood levels" are normally carried out. These measurements were repeated on either the fourth or fifth day of the fast, immediately before the fast was terminated by the commencement of food-testing. The age range of the patients was 12–55 years (average, 34 years); there were nine men and five women. The weight loss ranged from 4.35 to 1.15 kg (average, 2.73 kg). The following levels were measured: sodium; potassium; chloride; bicarbonate; bilirubin; alanine transaminase; gamma glutamyl transferase; alkaline phosphatase; total protein; albumin; urea; creatinine; uric acid; glucose; cholesterol; triglycerides; aspartate transaminase; lactate dehydrogenase; calcium; and iron. The full blood count included the haemoglobin concentration, red cell characteristics and the sedimentation rate, as well as the white cell count and differential count.

The Table lists the 10 biochemical and three haematological measures which were the only ones that appeared to show any change of possible significance.

The slight increases in red blood cell counts, haemoglobin concentrations and haematocrit values in 11 patients is suggestive of a mild degree of haemoconcentration, perhaps reflecting mild dehydration. The drop in bicarbonate levels from a mean of 27 to 21 mmol/L ($P<0.001$) could be explained on the basis of ketosis. Creatinine levels showed a slight significant rise and would parallel the changes in haematocrit values. It is not surprising that the glucose levels fell, although not markedly, which shows the efficiency of gluconeogenesis in maintaining the blood glucose level. The rises in cholesterol and triglyceride levels would reflect the mobilization of fat stores. The small falls in lactate dehydrogenase levels, the slight falls in serum iron levels and the small rises in bilirubin and chloride levels were felt not to be significant. The most marked changes were in the uric acid levels which rose from a mean of 0.34 to 0.59 mmol/L ($P<0.001$).

Although all 14 patients experienced some degree of withdrawal symptoms (for example, headache, nausea), no one was affected severely by the fast and all patients completed the desired length of fast (between four to five days) during which time normal sedentary activities were maintained.

A raised serum uric acid level is well recognized to be part of fasting, and although levels as high as 1 mmol/L have been reported,[9] no case of clinical gout has occurred. Ketosis affects the renal clearance of uric acid; increased ketosis is associated with a reduction in the excretion of uric acid and vice versa. The highest uric acid levels were all associated with gross ketonuria and the lower values with lesser degrees of ketonuria, as were the changes in bicarbonate levels.

Short-term fasting is well tolerated; the biochemical changes do not cause clinical symptoms and reflect the metabolic changes that are associated with the fast.

David R. Collison, FRACP
1&2/35 Albert Avenue
Chatswood, NSW 2067
Craig Eagle
106 Bannockburn Road
Pymble, NSW 2073

1. Bloom WL. Fasting as an introduction to the treatment of obesity. *Metabolism* 1959; 8: 214-220.
2. Harrison MT, Harden RMcG. The long-term value of fasting in the treatment of obesity. *Lancet* 1966; 2: 1340-1342.
3. Thompson TJ, Runcie J, Miller V. Treatment of obesity by total fasting for up to 249 days. *Lancet* 1966; 2: 992-996.
4. Collison DR. Total fasting for up to 249 days. *Lancet* 1967; 1: 112.
5. Mackarness R. Not all in the mind. London: Pan Books, 1983.
6. Spencer IOB. Death during therapeutic starvation for obesity. *Lancet* 1968; 1: 1288-1290.
7. Kahan A. Death during therapeutic starvation. *Lancet* 1968; 1: 1378-1379.
8. Duncan GG, Dunca TG, Schless GL, Cristofori FC. Contraindications and therapeutic results of fasting in obese patients. *Ann NY Acad Sci* 1965; 131: 632-636.
9. Lawlor T. Death during therapeutic starvation. *Lancet* 1968; 2: 44.

Vitamin C and congestive cardiac failure

To the Editor: A 45-year-old farmer was documented as having intramedullary lymphoma in March 1978. He was managed with chemotherapeutic regimens which did not include anthracycline drugs. He responded well initially but remained pancytopenic. His red cell transfusion requirements increased markedly in late 1981; a total of 150 units had been transfused by July 1983. By this time he had heavily pigmented skin, hepatomegaly and testicular atrophy. His serum iron level was 38 μmol/L with 88%

saturated iron-binding capacity. His serum ferritin level was 6300 μg/L.

In spite of frequent monitoring in association with his transfusions, no cardiomegaly was detected until June 1983, when he first required diuretic therapy with each transfusion because of mild pulmonary oedema. Over subsequent weeks he developed a persistent tachycardia with increasing cardiomegaly and overt cardiac failure.

In September 1983 he presented in gross biventricular cardiac failure with a tachycardia of 140 bpm and a central venous pressure of 20 cm of water. The apex beat was in the sixth intercostal space in the anterior axillary line; the only abnormality on auscultation was a loud third heart sound. Bilateral basal crepitations were present to the mid-zone of the lung. The liver span was 20 cm with no splenomegaly. Moderate ankle and sacral oedema was present. An electrocardiogram showed a sinus tachycardia with frequent atrial and ventricular ectopic beats, right bundle-branch block, widespread T-wave inversion and a normal PR interval.

Diagnoses of myocardial ischaemia, thyroid dysfunction, endocarditis and viral myocarditis were considered and excluded. His cardiac failure was controlled with the use of frusemide and prazosin. Two days later he developed atrial fibrillation which was treated with digoxin.

However, it became apparent that the patient had been taking 4 g of vitamin C a day since May 1983 under the misconception that this might "improve his blood". It was felt probable that vitamin C had precipitated acute cardiac decompensation in the presence of transfusional iron overload. The intravenous administration of desferrioxamine was commenced with measurements of urinary iron excretion and was changed two weeks later to a subcutaneous pump infusion of 1 g of desferrioxamine over 10 hours each night indefinitely.

No additional therapy was given to treat the cardiac failure. Serial two-dimensional echocardiograms over the ensuing 30 months demonstrated the gradual return of his ejection fraction towards a normal value (Figure). His exercise tolerance improved to the extent that he could play 18 holes of golf regularly, and clinical examination of the cardiovascular system remained normal until his death of advanced lymphoma in November 1986.

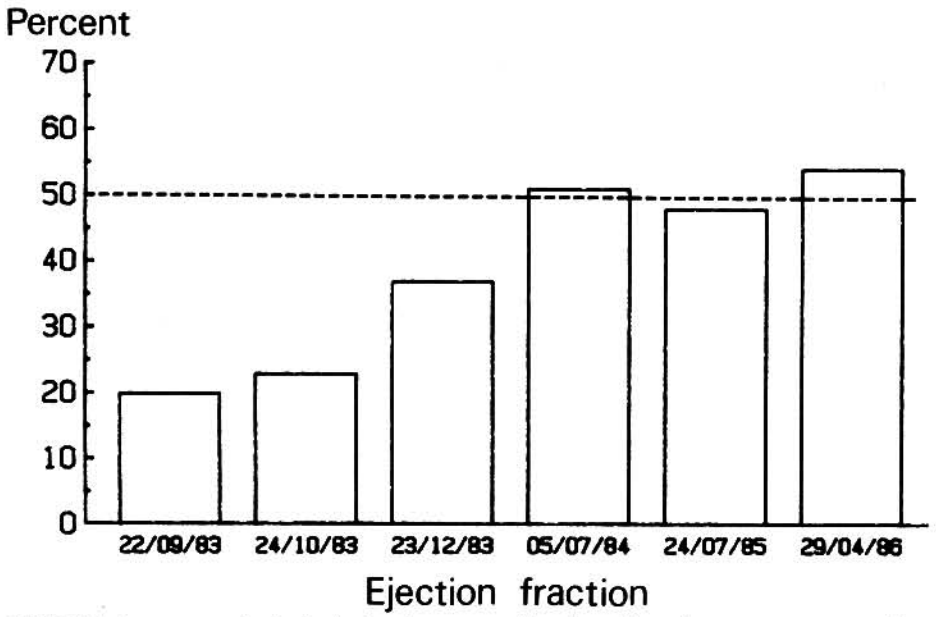

FIGURE: The improvement in ejection fraction that was noted by echocardiographic measurements over three years with desferrioxamine therapy.

This sequence of events has been described in patients with thalassaemia major,[1,2] and in a patient with congenital pyruvate kinase deficiency in association with the HLA haplotype for idiopathic haemachromatosis.[3,4] However, the possibility that transfusions, which are given in the treatment of neoplastic disease, can affect the marrow and may contribute to morbidity and mortality by the production of systemic iron overload should always be considered. Therapeutic strategies that utilize early treatment, or prophylactic therapy with desferrioxamine may improve considerably the quality of life in these individuals.

As such patients are more likely to seek non-orthodox sources of therapy, they should be warned of the potentially lethal effects of vitamin C.

Richard G. Pestell, MB BS
Registrar in Endocrinology
St Vincent's Hospital
Victoria Avenue, Fitzroy, VIC 3065
Andrew L. Barr, FRACP
Hematologist
Royal Perth Hospital
Wellington Street, Perth, WA 6000
Glen Brand, MB BS
Registrar
Royal Perth Rehabilitation Hospital
Selby Street, Shenton Park, WA 6008

1. Nienhuis AW, Griffith P, Strawczyniski H, et al. Evaluation of cardiac function in patients with thalassemia major. *Ann NY Acad Sci* 1980; 344: 382-396.
2. Grisaru D, Goldfarb AW, Gotsman MS, et al. Desferoxamine improves left ventricular function in beta thalassemia. *Arch Intern Med* 1986; 146: 2344-2349.
3. Rowbotham B, Roeser HP. Iron overload associated with congenital pyruvate kinase deficiency and high dose ascorbic acid ingestion. *Aust NZ J Med* 1984; 14: 667-669.
4. Bett JHN, Wilkinson RK, Boyle CM. Iron overload associated with congenital pyruvate kinase deficiency and high dose ascorbic acid ingestion. *Aust NZ J Med* 1985; 15: 270.

The Italians revisited

To the Editor: Minc [1] and Pasquarelli [2] have observed that Italian migrants often attributed their ailments to the "air".