# Early Detection of Protein Depletion in Alcoholic Cirrhosis: Role of Body Composition Analysis

DWI PRIJATMOKO,* BOYD J. G. STRAUSS,‡ JOHN R. LAMBERT,* WILLIAM SIEVERT,* DAN B. STROUD,‡ MARK L. WAHLQVIST,* BENJAMIN KATZ,* JOHN COLMAN§ PENELOPE JONES,§ and MELVYN G. KORMAN*

*Department of Medicine and ‡Clinical Nutrition and Metabolism Unit, Monash Medical Centre, Clayton; and §Department of Gastroenterology, Alfred Hospital, Melbourne, Australia

*Background:* Malnutrition is common in alcoholic cirrhosis. Bedside nutritional assessment techniques may be unreliable in patients with chronic liver disease. The aim of this study was to quantify changes in body composition and compare methods for measuring body composition in alcoholic cirrhosis. *Methods:* Thirty-eight men with alcoholic cirrhosis were compared with 16 age-matched healthy men. Body composition was assessed using anthropometry and bioelectrical impedance to determine fat-free mass and body fat, deuterium oxide dilution to measure total body water, in vivo neutron activation analysis to measure total body protein, and dual energy x-ray absorptiometry to measure bone mineral content and total body fat mass. *Results:* With increasing severity of cirrhosis, total body water increased, whereas total body protein decreased with a significant decrease in serum albumin levels. Total body protein levels, expressed as an index, were a more sensitive indicator of protein depletion than serum albumin levels. When patients were assessed by anthropometry and bioelectrical impedance for fat-free mass, there was no reduction compared with controls. *Conclusions:* Anthropometry and bioelectrical impedance do not accurately reflect changes in body composition associated with chronic liver disease. Quantification of body composition changes in alcoholic cirrhosis requires the use of direct methods such as in vivo neutron activation analysis, dual energy x-ray absorptiometry, or deuterium oxide dilution.

Protein energy malnutrition is common in advanced alcoholic liver disease and may have an adverse affect on morbidity and mortality.[1,2] The measurement of body protein stores, body fat, skeletal mass, and body water is an integral part of nutritional assessment in these patients. The two-compartment model of body composition divides the body into fat mass and fat-free mass (FFM). In the four-compartment model, FFM is further divided into body water (intracellular and extracellular), body protein, and skeletal mass. Changes in one or more components of body composition may be associated with an unfavorable clinical outcome.[1–3] Thus, the assessment of body composition, if precise, may provide useful information for managing patients with alcoholic liver disease, as well as a method for measuring response to nutritional supplementation.

Currently accepted methods of nutritional assessment in normal subjects may be unreliable in patients with chronic liver disease. Measurement of body mass index (BMI) by anthropometry (AN) does not accurately reflect specific changes in the composition of body compartments, particularly in the presence of gross ascites. The presence of generalized edema may prevent the accurate estimation of fat mass as measured by skinfold thickness.[4] The measurement of total body water (TBW) by bioelectrical impedance (BEI) may be affected in chronic liver disease because the conductivity of an electric current through tissue is determined by its water and electrolyte content.[5,6] Thus, the derivation of FFM and body fat by BEI may be underestimated in the presence of edema and ascites. Serum albumin levels are not a reliable estimate of visceral protein stores because hypoalbuminemia may reflect both poor dietary intake and reduced hepatic synthesis.[7]

Accurate body composition methods are now available.[8,9] We studied the body composition of patients with alcoholic cirrhosis compared with healthy control subjects. Body composition was assessed using AN and BEI for the determination of FFM and body fat, deuterium oxide dilution for the assessment of TBW, in vivo neutron activation analysis (IVNAA) for the

*Abbreviations used in this paper:* AN, anthropometry; BEI, bioelectrical impedance; BMI, body mass index; DEXA, dual energy x-ray absorptiometry; FFM, fat-free mass; IVNAA, in vivo neutron activation analysis; MC-FFM, fat-free mass calculated by multicompartment model; NI, nitrogen index; TBBM, total body bone mineral; TBN, total body nitrogen; TBP, total body protein; TBW, total body water.


0016-5085/93/$3.00

assessment of total body nitrogen (TBN), and dual-energy x-ray absorptiometry (DEXA) for the assessment of bone mineral content as well as body fat and FFM. FFM was also measured using a multicompartment model described in the equation

$$FFM = (TBN \times 6.25) + (TBW - {}^{2}H) + (TBBM \times 1.235) + (TBP \times 0.044),$$

where *TBBM* is total body bone mineral and total body protein (*TBP*) is TBN × 6.25. This model calculates the FFM as the sum of the protein (TBN), intracellular and extracellular water (TBW), and mineral compartments (TBBM) using the gold standard technique for measuring each compartment. Thus, we feel this is the closest estimation of true FFM, even though glycogen stores and trace elements are not directly measured.

## Materials and Methods

### Subjects

Thirty-eight men (aged 30–70 years) with the diagnosis of alcoholic cirrhosis based on clinical evidence and liver histology or a strong clinical suspicion of alcoholic liver disease in the absence of other causes of chronic liver disease were recruited. Those with severe intercurrent illness, steroid therapy, or diabetes mellitus were excluded because of the independent effects of such conditions on body composition. The severity of liver disease was graded using the Child–Pugh score.[10] Sixteen healthy, age-matched volunteers were recruited from hospital staff to participate in this study. None consumed more than 20 g of alcohol daily, and none of the controls had clinical evidence of hypercortisolism or abnormal renal function. Any subject with a metabolic disorder associated with muscle wasting or receiving drugs that might interfere with muscle function was excluded.

Written informed consent was obtained from all subjects before study entry. The study was approved by the Monash Medical Centre Human Research and Ethics Committee.

### Clinical Evaluation

A complete history was obtained and physical examination was performed on all subjects. A full blood examination and liver function tests were performed and prothrombin time was measured using standard automated techniques.

### Body Composition Assessment

Following an overnight fast, all subjects underwent the following assessments in the body composition laboratory.

**AN.** BMI was calculated by the formula BMI = weight/height$^2$ (kg/m$^2$). Body fat was estimated using the Durnin and Womersley equation[11] from four sites: the biceps, triceps, and subscapular and iliac skinfolds. Skinfold thickness was measured using Harpenden (Holtain Ltd., Wales, England) skinfold calipers. FFM was calculated by subtracting body fat from body weight. Waist-hip ratio was measured by assessing waist and hip circumference. Waist circumference was measured 12 cm below the xiphisternal notch on all patients. Hip circumference was measured at the level of the maximal gluteal circumference.

**BEI.** Body resistance was measured between 9 and 10 AM after an overnight fast. The assessment was performed with the patients in a supine position with their hands at their sides (not touching the body) and with their feet apart. A pair of new electrodes was placed on the left hand and foot, and resistance was obtained using a BEI analyzer (BIA-103; RJL System Inc., Detroit, MI). Body fat and FFM were calculated using the manufacturer's software (Bodycomp II, version 1.1; RJL System Inc.).

**$D_2O$ dilution.** The $D_2O$ dilution technique is a direct method for measuring TBW. A total dose of 15 g $D_2O$ was administered orally to the subject, and a minimum of 2 hours of equilibration time was required before the collection of blood for sampling. In separate experiments, we determined that equilibration of $D_2O$ occurred within 2 hours in patients with ascites (data not shown). The Fourier transform infrared technique was used to quantify plasma $D_2O$ concentration as previously described.[12] The coefficient of variation of this technique in determining $D_2O$ concentration is 1%–2% for plasma $D_2O$ concentration.

**IVNAA.** A direct measurement of TBN can be obtained with IVNAA by counting the 10.8-MeV γ rays emitted by nitrogen in the patient's body. Subjects received a radiation dose of 0.1 milliSievert from a $^{252}Cf$ neutron source. Repeated measurements on a phantom containing a urea solution had a precision of <4%.[9] TBN can be used to calculate TBP based on the equation TBP = 6.25 × TBN. Results were expressed as a nitrogen index (NI) that equals measured TBN divided by predicted TBN (for a normal sex-matched population).[9]

**DEXA.** The DEXA scanner (DPX software version 3.4; Lunar Corp., Madison, WI) uses x-ray absorption to determine total body calcium and measures total body fat and total fat-free soft tissue. A whole body scan was performed in each subject. The error associated with DEXA is between 1% and 2% for bone mineral content and 2% and 3% for body fat and FFM.[13] Bone mineral content was expressed as an index to correct for body height, such that bone mineral content index = TBBM/height (m$^2$).

### Statistical Analysis

One-way analysis of variance (ANOVA) was used to assess the difference between each component of body composition among groups. When a significant difference was found by ANOVA, the Bonferroni *t* test was used to detect

**Table 1.** Case Patient and Control Patient Data

| | Control (n = 16) | Child's A (n = 17) | Child's B (n = 15) | Child's C (n = 6) |
|---|---|---|---|---|
| Age (*yr*) | 47.3 (10.9) | 58.9 (11.5) | 52.4 (10.8) | 49.8 (9.0) |
| Height (*m*) | 1.72 (0.1) | 1.70 (0.2) | 1.71 (0.1) | 1.71 (0.1) |
| Weight (*kg*) | 71.0 (9.3) | 75.0 (14.8) | 76.0 (20.9) | 76.4 (19.5) |
| BMI (*kg*) | 23.5 (2.1) | 26.9 (4.3) | 25.7 (6.8) | 26.0 (5.4) |
| GGT (*U/L*) | 21.7 (1.1) | 103.7 (25.8)* | 239.5 (28.9)* | 61.2 (28)* |
| MCV (*fL*) | 88.1 (2.8) | 97.7 (10.6)* | 101.1 (7.5)* | 96.3 (6.4)* |
| Albumin (*g/L*) | 45.1 (3) | 38.5 (6.3) | 33.5 (6.1)* | 32 (6.9)* |

NOTE. All data are expressed as mean ± SEM.
GGT, γ-glutamyl transpeptidase; MCV, mean corpuscular volume.
*$P < 0.05$.

which group differed significantly after correcting for multiple comparisons. Analyses were performed using Instat (version 1.14; Graphpad Software, San Diego, CA 1990).

## Results

Clinical and biochemical data are shown in Table 1. The cirrhotic patients weighed more than control patients, but without a significantly increased BMI. γ-Glutamyl transpeptidase values and mean corpuscular volume were significantly higher in cirrhotic subjects.

When case patients and control patients were analyzed together, serum albumin levels directly correlated with changes in the NI ($P < 0.001$; $r = 0.54$) (Figure 1) and were significantly lower in Child's B and C patients, which correlated with an increase in TBW ($P < 0.005$; $r = 0.38$) (Figure 2). When cirrhotic patients were analyzed separately, there was only a weak correlation between serum albumin levels and changes in NI ($r = 0.2$; 95% confidence interval, −0.13 to 0.48). Serum albumin levels were relatively preserved in Child's A patients (by definition), despite a significant reduction in the nitrogen index in just over half of this group (Table 2).

### Body Fat

When assessed by AN, BEI, and DEXA, Child's A ($P < 0.05$) and Child's B ($P < 0.05$) patients had a significantly higher percentage of body fat than control patients or Child's C patients. Within each group, there was no significant difference in estimating body fat among the three methods (Table 3).

### FFM and Protein Stores

The mean NI (as measured by IVNAA) of all cirrhosis groups was significantly lower ($P < 0.01$) than in healthy control patients (Figure 3). Although the mean NI of Child's A patients was within the lower range of normal, 53% were below the healthy range. Although there was no significant difference in mean NI between Child's A and B patients, Child's C patients had a significantly lower mean NI than that of either Child's A ($P < 0.01$) or B ($P < 0.05$) patients. No significant differences were found between any group

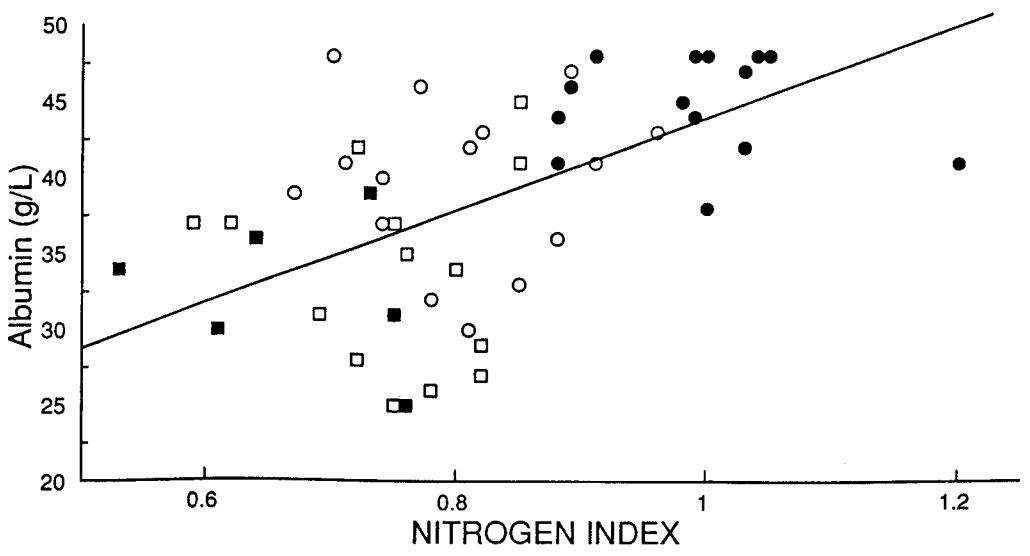


**Figure 1.** Correlation between serum albumin levels and NI in case patients and control patients ($P < 0.0001$; $r = 0.54$). ○, Child's A; □, Child's B; ■, Child's C; and ●, control patients.

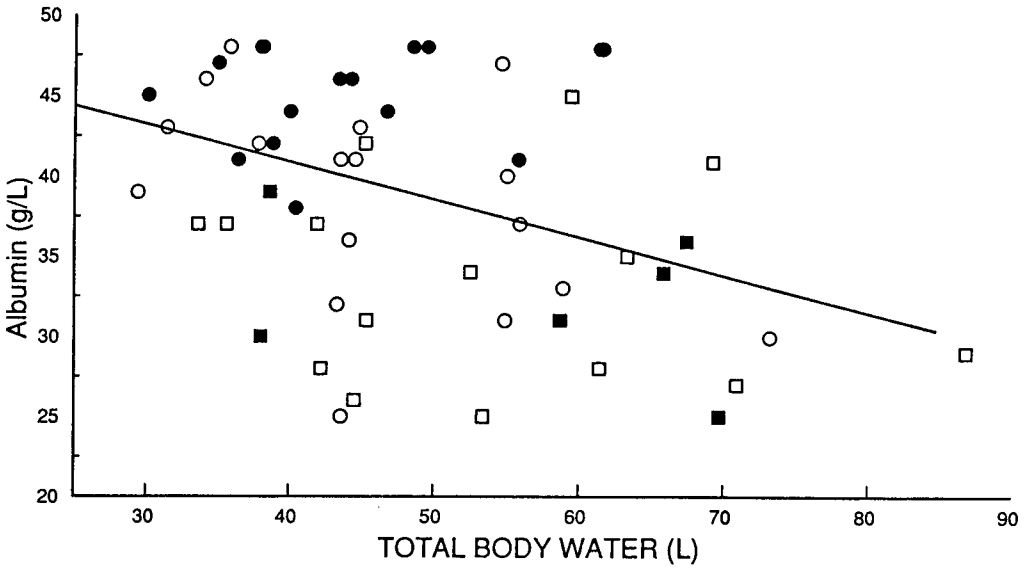


**Figure 2.** Correlation between serum albumin levels and TBW in case patients and control patients ($P < 0.005$; $r = 0.38$). ○, Child's A; □, Child's B; ■, Child's C; and ●, Control patients.

when FFM was estimated by AN, BEI, or DEXA (Table 4). However, when compared with FFM calculated by the multicompartment model (MC-FFM), AN, BEI, and DEXA each underestimated FFM in Child's B patients compared with the multicompartment model. Only BEI underestimated FFM in the Child's C patients, which may reflect the small number of patients in this category. The hydration of the MC-FFM was significantly increased in all cirrhotic patients (Table 5).

To determine if the protein depletion reflected by the NI was apparent by more widely available methods, we compared body protein as a percentage of FFM measured by AN, BEI, and DEXA (Table 6). All methods were able to discriminate between control patients and Child's groups, but only the multicompartment method and AN discriminated between Child's A groups and more advanced liver disease.

### TBW

When measured by BEI, there was no significant difference in TBW between control patients and all cirrhosis groups (Table 7). Between the control patients and Child's A patients, the mean TBW estimated by BEI and $D_2O$ dilution was not significantly different. However, in Child's B and C patients, the mean TBW estimated by BEI was significantly lower than that estimated by the $D_2O$ dilution technique, which showed that TBW was significantly increased in Child's B and C patients compared with control patients.

Fat distribution is commonly expressed as the waist-hip ratio. There was a significant correlation between an increase in waist-hip ratio and an increase in TBW ($r = 0.54$; $P < 0.001$).

### Bone Mineral Content

The total bone mineral content index for control patients and cirrhosis groups as assessed by DEXA is shown in Table 8. The bone mineral content index in cirrhosis groups was not significantly different from control patients.

## Discussion

Protein calorie malnutrition in alcoholic liver disease is common. Mendenhall et al. documented

**Table 2.** Serum Albumin Concentrations in Patients With Normal (>0.8) and Low (<0.8) NI

| | Serum albumin concentration (g/L) | |
|---|---|---|
| | NI > 0.8 | NI < 0.8 |
| Child's A | 39.38 (5.4) (n = 8) | 37.67 (6.9) (n = 9) |
| Child's B | 35.2 (6.88) (n = 5) | 32.6 (5.4) (n = 10) |
| Child's C | NA (n = 0) | 32.0 (4.5) (n = 6) |

NOTE. All data are expressed as mean ± SEM.

**Table 3.** Comparison of Three Methods to Determine Percentage of Body Fat

| | Control | Child's A | Child's B | Child's C | P |
|---|---|---|---|---|---|
| AN | 20.1 (6.2) | 24.9 (6.0) | 22.1 (6.6) | 16.9 (6.2) | <0.05 |
| BEI | 19.0 (4.5) | 22.7 (4.2) | 26.1 (8.7) | 23.5 (4.7) | <0.02 |
| DEXA | 21.0 (5.8) | 26.2 (7.2) | 27.1 (6.9) | 21.0 (5.8) | <0.05 |
| P | NS | NS | NS | NS | |

NOTE. All data are expressed as mean percentage ± SEM.

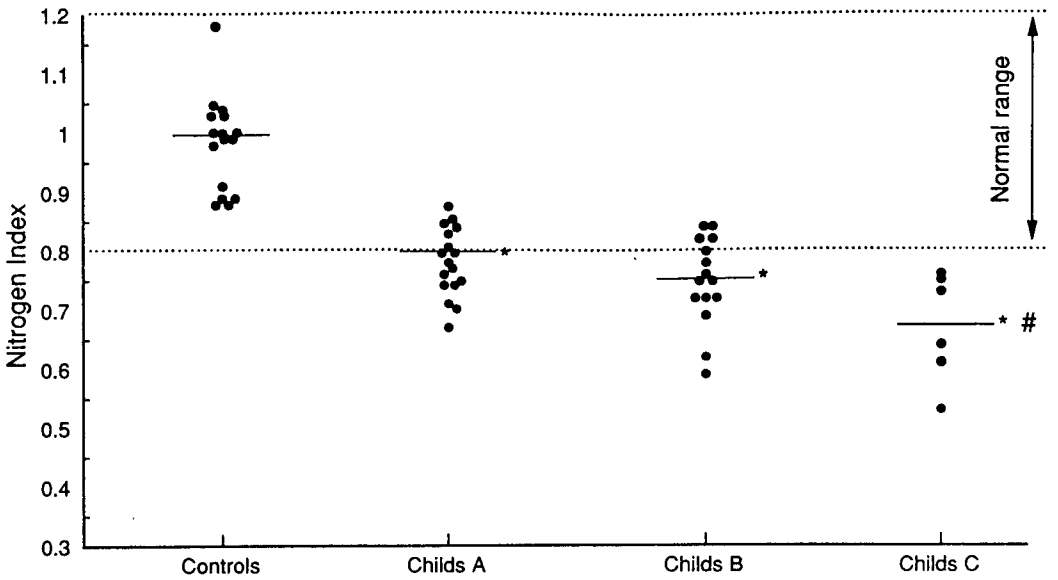


**Figure 3.** The mean NI (measured TBN divided by predicted TBN) was significantly higher in the control group compared with all patient groups (see text for further discussion). *$P < 0.001$ compared with control patients; #$P < 0.05$ compared with Child's A.

protein calorie malnutrition in all patients with alcoholic hepatitis compared with 62% of alcoholic patients without liver disease.[1] A similarly high incidence of protein calorie malnutrition in cirrhosis has been confirmed by other investigators.[2] More importantly, severe protein calorie malnutrition is associated with increasingly severe liver disease and, in one study, a 76% 12-month mortality.[3] Several trials of nutritional supplementation in cirrhosis (with or without alcoholic hepatitis or hepatic encephalopathy) have been reported with variable outcomes.[14–17] Such variability reflects patient selection but may also indicate variation in the methods used for nutritional assessment, which is an integral part of such studies.

Our findings highlight the limitations that must be kept in mind when using simple bedside methods such as AN and BEI for predicting body composition and nutritional status. The assumptions underlying these methods are based on healthy subjects[18,19] in whom the ratio between FFM and TBW (intracellular and extracellular fluid) is constant. The use of skinfold thickness to estimate body fat is based on the assumptions that the thickness of the subcutaneous adipose tissue is proportional to total body fat and that the site of measurement is representative of the average thickness of subcutaneous fat. Although these assumptions are valid for the reference group studied, variations can occur in different patient groups and among different operators.[6]

We estimated body fat using AN, BEI, and DEXA. Our findings indicate a higher percentage of fat mass in this group of Child's A and B patients than in control patients (Table 3). Using anthropometric methods, there was a significant reduction in body fat in Child's C patients compared with all other groups. The cause for the increased fat mass in earlier stages of alcoholic liver disease is unclear. Six of the patients were heavy drinkers at the time of study (as judged by blood ethanol levels); therefore, increased total energy intake may have been a factor in the observed increase in fat mass. In addition, there is evidence to suggest that endogenous protein is a preferred fuel in alcoholic cirrhosis, allowing preservation of fat mass.[20]

In cirrhosis, muscle wasting may be masked by an

**Table 4.** Comparison of Percentage of FFM Determined by MC-FFM, AN, BEI, and DEXA

| | MC-FFM | AN-FFM | BEI-FFM | DEXA-FFM |
|---|---|---|---|---|
| Control | 83 (1.3) | 79.2 (1.3)[a] | 80.7 (1.1) | 83.2 (1.5) |
| Child's A | 80 (1.7) | 75.1 (1.4)[a] | 77.3 (1.0) | 77.5 (1.7) |
| Child's B | 86 (2.0) | 78 (1.5)[b] | 73.9 (2.2)[b] | 76.8 (1.8)[b] |
| Child's C | 89 (2.4) | 83.2 (2.0) | 76.5 (1.8)[b] | 83.7 (1.7) |

NOTE. All data are expressed as mean percentage ± SEM.
[a]$P < 0.05$ compared with MC-FFM in that category.
[b]$P < 0.001$ compared with MC-FFM in that category.

**Table 5.** FFM and Fractional Hydration of Control Patients and Cirrhotic Patients Determined by the MC-FFM and Compared With BEI

| | Control | Child's A | Child's B | Child's C |
|---|---|---|---|---|
| MC-FFM[a] | 83 (1.3) | 80 (1.7) | 86 (2) | 89 (2.4) |
| Hydration[b] | 72.9 (0.7) | 77.7 (0.8) | 80.4 (0.7) | 82.6 (1.2) |
| BEI | 73.2 (0.4) | 73.0 (0.02) | 73.0 (0.02) | 73.0 (0.04) |

NOTE. Values are expressed as mean percentage ± SEM.
[a]$P$ = NS between any group.
[b]Control patients were significantly less hydrated than all Child's groups ($P < 0.0001$), and Child's A patients were less hydrated than Child's C patients ($P < 0.05$).

**Table 6.** TBP Expressed as a Mean Percentage (±SEM) of the FFM

| | Control | Child's A | Child's B | Child's C |
|---|---|---|---|---|
| FFM | 20 (2) | 16 (2) | 13 (2)[a] | 11 (2)[a] |
| AN-FFM | 21 (1) | 17 (2) | 15 (2)[b] | 12 (2)[b] |
| BEI-FFM | 21 (2) | 16 (3)[c] | 16 (2)[c] | 13 (3)[c] |
| DEXA-FFM | 20 (1) | 16 (2) | 15 (2) | 12 (2)[d] |

NOTE. By any method, Child's groups differed significantly from control patients ($P < 0.001$).
[a]Child's B and C had a significantly lower body protein fraction than Child's A ($P < 0.01$).
[b]Child's B and C had a significantly lower body protein fraction than Child's A ($P < 0.05$).
[c]There was no statistically significant difference among the Child's groups in fractional body protein.
[d]Child's C had a significantly lower body protein fraction than Child's A ($P < 0.001$) and Child's B ($P < 0.05$).

increase in body weight caused by a substantial increase in TBW from ascites or peripheral edema. Measurement of FFM by AN, BEI, or DEXA estimates both body water and body protein compartments and thus may fail to show a significant reduction in body protein. We found no significant difference in FFM when measured by these three methods (Table 4). We then examined the body protein fraction of the FFM as determined by the different methods as a discriminator for subtle changes in protein mass. All methods identified patients with advanced protein depletion, but only FFM determined by the multicompartment model and AN showed a difference between Child's A compared with Child's B and C patients. The ability to directly measure body nitrogen provided the most precise discrimination. When body nitrogen was measured by IVNAA and an NI was derived, there was a significant and progressive reduction in body protein (Figure 3). All cirrhosis groups had lower body nitrogen than control patients, with a progressive decrease from Child's A to Child's C. More importantly, nine of the Child's A patients were below the normal limit for NI, and six of these patients had a normal serum albumin concentration (Table 2). This suggests that generalized muscle wasting occurs early in cirrhotic patients. Although a correlation exists between serum albumin levels (used in determining Child's classification) and the

**Table 7.** TBW as a Mean Percentage (±SEM) of Body Weight

| | Control | Child's A | Child's B | Child's C |
|---|---|---|---|---|
| $D_2O$ | 60.5 (1.1) | 62.2 (1.8) | 67.4 (2.0) | 73.7 (2.0)[a] |
| BEI | 59.0 (0.9) | 56.5 (1.7) | 53.9 (1.6) | 55.9 (1.3)[b] |

[a]$P < 0.0001$ (ANOVA).
[b]$P$ = NS (ANOVA).

**Table 8.** Bone Mineral Content Index

| | |
|---|---|
| Controls | 0.99 (0.03) |
| Child's A | 0.99 (0.04) |
| Child's B | 0.99 (0.05) |
| Child's C | 1.09 (0.15) |

NOTE. Results are expressed as mean percentage (±SEM). Bone mineral content index was calculated as bone mineral content ($kg/m^2$) divided, by height ($m$). There were no significant differences among the groups.

NI when case patients and control patients are examined together, the correlation is relatively weak when analyzing the cirrhotic patients separately because changes in serum albumin levels may reflect changes in TBW. IVNAA provides a sensitive measure of changes in TBP, which more accurately reflects the extent of protein depletion in Child's A patients. The ability to detect significant changes in TBP stores early in the course of alcoholic liver disease provides an opportunity to assess therapeutic intervention before the onset of irreversible liver damage.

TBW was measured by deuterated water dilution and BEI. An underlying assumption of the BEI method is that the FFM contains 73% water. In our patients with cirrhosis, BEI significantly underestimated TBW, whereas $D_2O$ dilution showed a significant increase in TBW in Child's B and C patients. An increase in TBW in patients with alcoholic liver disease may not reflect an increase in FFM but rather reflects an increase in extracellular water, which was not measured in this study. Similarly, the waist-hip ratio has been used to define abdominal obesity and has been shown to be an independent indicator of cardiovascular mortality. The abdominal circumference in cirrhotic patients may reflect an enlarged liver, the accumulation of ascites, or a combination of both. Similarly, muscle wasting in the hips may alter this ratio. In cirrhotic patients, we observed a significant correlation between TBW and waist-hip ratio; thus, the waist-hip ratio is not accurate in predicting fat distribution in patients with alcoholic liver disease.

This study failed to support findings by other investigators[21–24] showing a reduced bone mineral content in cirrhotic patients. The increased sunlight exposure of the Australian population, the social status of the subjects studied, the techniques utilized, or the type of alcohol consumed may explain this difference.[25] The DEXA method measures bone mineral content, but it does not provide histological interpretation. Thus, we cannot comment on changes in bone architecture.

In summary, we have shown that in men with alcoholic cirrhosis, body composition is altered with ad-

vancing severity of liver disease. IVNAA and $D_2O$ dilution techniques reflect the anticipated clinical changes in cirrhosis, whereas AN and BEI may not accurately reflect changes in body composition. A significant decrease in NI found early in the course of alcoholic liver disease may provide useful prognostic information but, more importantly, may indicate the need for early and aggressive nutritional supplementation.

## References


1. Mendenhall CL, Anderson S, Weesner RE, Goldberg SJ, Crolic KA. Protein-calorie malnutrition associated with alcoholic hepatitis. Am J Med 1984;76:211–222.
2. Abad A, Cabre E, Gonzales-Huix F, Dolz C, Xiox X, Gussull MA. Influence of the nutritional status in the prognosis and clinical outcome of hospitalized patients with liver cirrhosis. Preliminary report. J Clin Nutr Gastroenterol 1987;2:63–68.
3. Mendenhall CL, Tosch T, Weesner RE, Garcia-Pont P, Goldberg SJ, Kiernan T, Seeff LB, Sorrell M, Tamburro C, Zetterman R, Chedid A, Chen T, Rabin L. VA cooperative study on alcoholic hepatitis II: Prognostic significance of protein-calorie malnutrition. Am J Clin Nutr 1986;43:213–218.
4. Heymsfield SB, Casper K. Anthropometry of the adult hospitalized patient. JPEN 1987;11:368–418.
5. Deurenberg P, Weststrate JA, Paymans I, van der Kooy K. Factors affecting bioelectrical impedance measurements in humans. Eur J Clin Nutr 1988;42:1017–1022.
6. Lukaski HC. Methods for the assessment of human body composition: traditional and new. Am J Clin Nutr 1987;46:537–556.
7. Morgan MY, Levine JA. Nutritional management of patients with liver disease. J Clin Nutr Gastroenterol 1986;1:368–414.
8. Rayner HC, Stroud DB, Salamon KM, Strauss BJG, Thomson NM, Atkins RC. Anthropometry underestimates body protein depletion in haemodialysis patients. Nephron 1991;59:33–40.
9. Stroud DB, Borovnicar DJ, Lambert JR, McNeill KG, Mark SJ, Rassool RP, Rayner HC, Strauss BJG, Tai EH, Thomson NM, Wahlqvist ML, Watson BA, Wright CM. Clinical studies of total body nitrogen in an Australian hospital. Basic Life Sci 1990;55:177–182.
10. Pugh RNH, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg 1973;60:646–649.
11. Durnin JVGA, Womersley J. Body fat assessment from total body density and its estimation from skinfold thicknesses: measurements on 481 men and women aged from 16 to 72 years. Br J Nutr 1974;32:77–97.
12. Blagojevic N, Allen BS, Gaskin KJ, Baur LA. Determination of total body water by fourier transform infrared analysis. Australas Phys Eng Sci Med 1990;13:110–116.
13. Mazess RB, Bardin HS, Bisek JP, Hanson J. Dual energy x-ray absorptiometry for total body and regional bone-mineral and soft tissue composition. Am J Clin Nutr 1990;51:1106–1112.
14. Mendenhall C, Bongiovanni G, Goldberg S, Miller B, Moore J, Rouster S, Schneider D, Tamburro C, Toch T, Weesner R. VA cooperative study on alcoholic hepatitis III: changes in protein-calorie malnutrition associated with 30 days of hospitalization with and without enteral nutrition therapy. JPEN 1985;9:590–596.
15. Cabre E, Gonzalez-Huix F, Abad-LaCruz A, Esteve M, Acero D, Fernandez-Banare SF, Xiol X, Gassull MA. Effect of total enteral nutrition on the short-term outcome of severely malnourished cirrhotics. Gastroenterology 1990;98:715–720.
16. Horst D, Grace ND, Conn HO, Schiff E, Schenker S, Viteri A, Law D, Atterbury CE. Comparison of dietary protein with an oral, branched chain-enriched amino acid supplement in chronic portal-systemic encephalopathy. Hepatology 1984;4:279–287.
17. Calvey H, Davis M, Williams R. Controlled trial of nutritional supplementation, with and without branched chain amino acid enrichment, in treatment of acute alcoholic hepatitis. J Hepatol 1985;1:141–151.
18. Linscheer WG. Malabsorption in cirrhosis. Am J Clin Nutr 1970;232:488–492.
19. Deurenberg P. Weststrate J, van der Kooy K. Body composition changes assessed by bioelectrical impedance. Am J Clin Nutr 1989;49:401–403.
20. Swart GR, Van den Berg IWO, Wattimena JLD, Rieveld T, Van Vuure JK, Frenkel M. Elevated protein requirements in cirrhosis of the liver is investigated by whole body protein turnover studies. Clin Sci 1988;75:101–107.
21. Bikle DD, Genant HK, Cann C, Recker RR, Halloran BP, Strewler GJ. Bone disease in alcohol abuse. Ann Intern Med 1985;103:42–48.
22. Seeman E, Melton LJ III, O'Fallon WM, Riggs BL. Risk factor for spinal osteoporosis for men. Am J Med 1983;75:977–983.
23. Schapira D. Alcohol abuse and osteoporosis. Semin Arthritis Rheum 1990;19:371–376.
24. Diamond T, Stiel D, Lunzer M, Wilkinson M, Posen S. Ethanol reduces bone formation and may cause osteoporosis. Am J Med 1989;86:282–288.
25. McDonald JT, Margen S. Wine versus ethanol in human nutrition. I. Nitrogen and calorie balance. Am J Clin Nutr 1976;29:1093–1103.


Received June 9, 1992. Accepted July 20, 1993.

Address requests for reprints to: William Sievert, M.D., Department of Medicine, Monash Medical Centre, 246 Clayton Road, Clayton 3168, Australia.

Dwi Prijatmoko was supported by a fellowship from the International Development Program of Australian Universities and Colleges.