Coronary heart disease risk indicators, aerobic power, and physical activity in men with spinal cord injuries

Janssen, T.W.J.; Oers, C.A.J.M.; van der Kamp, J.; ten Voorde, B.J.; van der Woude, L.H.V.; Hollander, A.P.

published in
Archives of Physical Medicine and Rehabilitation
1997

DOI (link to publisher)
10.1016/S0003-9993(97)90076-9

document version
Publisher's PDF, also known as Version of record

Coronary Heart Disease Risk Indicators, Aerobic Power, and Physical Activity in Men With Spinal Cord Injuries

Thomas W. J. Janssen, PhD, C. A. J. M. (Lidy) van Oers, MS, PT, Gerard J. van Kamp, PhD, Ben J. TenVoorde, PhD, Luc H. V. van der Woude, PhD, A. Peter Hollander, PhD

ABSTRACT. Janssen TWJ, van Oers CAJM, van Kamp GJ, TenVoorde BJ, van der Woude LHV, Hollander AP. Coronary heart disease risk indicators, aerobic power, and physical activity in men with spinal cord injuries. Arch Phys Med Rehabil 1997;78:697-705.

Objective: To compare the lipid and (apo-)lipoprotein profile and blood pressure of men with long-standing spinal cord injuries (SCI) to those of an age-matched able-bodied (AB) population, and to assess the most important determinants of this profile and blood pressure.

Design: A cross-sectional study of persons with chronic SCI residing in the community.

Setting: Tests were performed in a university research laboratory.

Subjects: Thirty-seven men (age 37.4 ± 12.0yrs) with long-standing (14.7 ± 8.6yrs) SCI ranging from level C4/5 to L5 volunteered to participate. Comparisons were made with published data from 3,498 AB men, age 20 to 59yrs, from the same country.

Main Outcome Measures: Lipid and lipoprotein profile (total cholesterol [TC], high-, low-, and very low-density lipoprotein cholesterol [HDL-C, LDL-C and VLDL-C, respectively], and triglycerides [TG]), as well as aerobic power, activity level, anthropometric variables, and blood pressure. Multiple regression analyses assessed the most important determinants of the lipid and blood pressure profile.

Results: None of the lipid variables were related to the lesion level. TC, HDL-C, and TC/HDL-C were not significantly different from the AB population. The most important determinants of TC, LDL-C, and the ratios TC/HDL and HDL-C/LDL-C were age, smoking behavior, and activity level. Aerobic power was not an important determinant of any lipid or (apo-)lipoprotein or blood pressure.

Conclusion: Men with long-standing SCI do not appear to have an essentially different coronary heart disease risk profile compared with AB persons. Modifiable risk factors such as activity level, smoking, alcohol consumption, body mass index, and adipose tissue were more important than lesion level and aerobic power in the determination of the lipid and lipoprotein profile, suggesting several potential interventions.

From the Faculty of Human Movement Sciences (Dr. Janssen, Ms. van Oers, Dr. van der Woude, Dr. Hollander) and the School of Medicine, Departments of Clinical Chemistry (Dr. van Kamp) and Medical Physics and Informatics (Dr. TenVoorde), Vrije Universiteit, Amsterdam, The Netherlands.

Submitted for publication August 19, 1996. Accepted in revised form November 26, 1996.

No commercial party having a direct or indirect interest in the subject matter of this article has or will confer a benefit upon the authors or upon any organization with which the authors are associated.

Dr. Janssen is currently affiliated with the Institute for Rehabilitation Research and Medicine, Wright State University School of Medicine, Dayton, OH.

Reprint requests to Thomas W. J. Janssen, PhD, Wright State University Institute for Rehabilitation Research and Medicine, 3171 Research Boulevard, Dayton, OH 45420.

0003-9993/97/7807-4159$3.00/0

INDIVIDUALS WITH spinal cord injuries (SCI) are at increased risk of coronary heart disease (CHD).[1] CHD is one of the most important causes of death in persons with SCI,[2] occurring at younger ages than in the able-bodied (AB) population. Some of the most important indicators of an increased risk are high serum concentrations of total cholesterol (TC) and low concentrations of high-density lipoprotein cholesterol (HDL-C).[3] HDL-C levels have been reported to be significantly lower among persons with SCI than in the AB population.[4-9] In contrast, Cardús et al[10] did not find significant differences in total cholesterol (TC), HDL-C, and low-density lipoprotein cholesterol (LDL-C) between men with SCI and age-matched AB men.

It has been suggested that the lower HDL-C levels are due to an inactive lifestyle and a concomitant reduction of cardiopulmonary fitness of persons with SCI.[7] In AB individuals an inverse relation has been reported between aerobic power, the most common measure of cardiopulmonary fitness, and TC, LDL-C, triglycerides (TG), and a direct relation between aerobic power and HDL-C, apolipoprotein A-1 (apoA-1), and HDL-C/apoA-1 ratio.[11-15]

These relationships have hardly been studied in individuals with SCI. Although direct relations between aerobic power and HDL-C[9,16] and between aerobic power and TC/HDL-C, TG, LDL-C/HDL-C, and HDL-C/apoA-1[16] have been reported in very small groups of subjects with paraplegia, the possibly interfering and maybe more important factors such as age, lesion level, time since injury (TSI), behavioral factors (smoking, alcohol consumption, activity level), genetic factors (family history of CHD), and anthropometric factors (body mass, adipose tissue) were not taken into account.

Since persons with quadriplegia have in general lower levels of daily activity (energy expenditure[17]) and aerobic power than those with paraplegia,[18] they could be expected to have an inferior CHD risk profile. However, the relation between aerobic power, activity level, and serum lipid and lipoprotein profile has not been investigated in a subject group of persons with paraplegia and persons with quadriplegia.

The purpose of this study was to describe the serum lipid and (apo-)lipoprotein profile and blood pressure among a group of men with SCI, including men with quadriplegia as well as men with paraplegia. To investigate whether these men were at increased risk of CHD, lipid, lipoprotein, and blood pressure values were compared with those obtained from AB individuals.[19] A second purpose was to assess the most important determinants of the lipid and lipoprotein profile and blood pressure among men with SCI using age, lesion level, TSI, aerobic power, behavioral factors (activity level, smoking, and drinking behavior), anthropometric factors (body mass, body mass index, and skinfolds) and family history as independent factors.

Table 1: Subject Characteristics, Including BMI, SBP, DBP, and $\dot{V}O_2$ Peak, of the 4 Lesion Groups (mean ± SD)

	1: C4-C8 (n = 8)	2: T1-T5 (n = 5)	3: Ty-T10 (n = 10)	4: T11-L5 (n = 14)	Total (n = 37)	Groups Different
Age (yrs)	37.3 ± 10.0	40.6 ± 8.3	31.6 ± 6.6	40.6 ± 15.9	37.4 ± 12.0	—
Time since injury (yrs)	17.8 ± 9.3	19.0 ± 9.5	12.7 ± 8.3	12.9 ± 7.9	14.7 ± 8.6	—
Body mass (kg)	83.1 ± 15.1	84.7 ± 11.1	79.7 ± 16.7	85.6 ± 21.4	83.3 ± 17.3	—
Sum of 4 skinfolds (mm)	67.0 ± 32.2	82.5 ± 41.2	65.4 ± 31.5	77.1 ± 35.9	72.5 ± 34.7	—
BMI (kg/m^2)	23.8 ± 3.2	24.6 ± 3.3	24.3 ± 4.8	26.0 ± 6.0	24.9 ± 4.8	—
Sport participation (hours/week)	4.6 ± 4.6	1.2 ± 1.8	2.9 ± 2.7	2.7 ± 3.5	2.9 ± 3.4	—
Alcohol consumption (glasses/day)	0.7 ± 0.5	2.0 ± 2.1	1.2 ± 1.5	1.6 ± 2.3	1.4 ± 1.8	—
SBP (mmHg)	109.3 ± 15.9	127.6 ± 26.7	126.4 ± 8.8	140.4 ± 22.1	128.2 ± 18.8	1-4
DBP (mmHg)	68.8 ± 13.2	72.8 ± 20.9	77.9 ± 9.8	76.2 ± 15.7	74.6 ± 14.4	—
$\dot{V}O_2$ peak (L/min)	1.2 ± 0.4	1.4 ± 0.2	1.9 ± 0.4	2.1 ± 0.5	1.7 ± 0.6	1-3,4; 2-4
$\dot{V}O_2$ peak (mL/kg/min)	14.3 ± 4.4	17.0 ± 2.3	24.3 ± 6.1	25.6 ± 8.3	21.5 ± 7.8	1-3,4

METHODS

Subjects and experimental procedure. Thirty-seven Caucasian men (age range 19 to 71yrs, median 32) with long-standing SCI (TSI range 4 to 33yrs, median 13; 23 complete lesions) participated in this study. Four groups were formed based on the lesion level: men with quadriplegia (C4/5 through C8, n = 8), with high-level paraplegia (T1 through T5, n = 5), with mid-level paraplegia (T6 through T10, n = 10), and with low-level paraplegia (T11 through L5, n = 14). All subjects used a hand-propelled wheelchair as primary means of locomotion, lived more or less independently at home, and said that they felt healthy. Seventeen subjects reported that they used medication (predominantly antispasticity medication and medication to prevent or control urinary tract infections) on a regular basis. One subject (age 71yrs) had had a minor myocardial infarction. Eight subjects reported to have had renal problems (eg, kidney stones), and almost half (18 of 37) of all subjects had had or still had urinary tract infections. None reported being diabetic. Table 1 provides information on subject characteristics.

Subjects reported to the laboratory at noon. After having signed an informed consent statement, blood samples were collected for determination of the blood lipid profile. Subsequently, an interviewer-administered questionnaire elicited behavioral factors, family history of CHD or hypertension, medication use, and health status. Body mass and skinfolds were determined. Blood pressure was determined at least 2 hours after a light lunch, followed by a graded exercise test to assess aerobic power.

Anthropometric factors. Body mass was determined with a Berkel scale with the subjects dressed in light clothing. The body mass index (BMI) was defined as body mass divided by self-reported height squared. Subjects were classified as grade I obese when BMI exceeded 25 or as grade II obese when BMI exceeded 30. Subcutaneous adipose tissue was estimated by the sum of 4 skinfolds (Σ4SF), determined three times (and averaged) at triceps, biceps, subscapular, and suprailiac sites, with the subject sitting in his wheelchair.

Behavioral factors. Activity level was defined as the number of hours of active sport participation per week. Relatively passive sports (eg, shooting, chess, bridge) were not included. No further distinction in type or intensity of sport participation was made.

Subjects were classified according to their regular smoking behavior: nonsmoker (1), light smoker (fewer than 10 cigarettes per day [2]), heavy smoker (10 or more cigarettes per day), and/or cigar/pipe smoker (3). Alcohol consumption was defined as the average number of alcohol-containing drinks per day.

Family history. Subjects were classified on basis of family history of CHD: subjects with no history (1), or with parents (2), brother/sister (3), or parents and brother/sister (4) with CHD. Analogously, a classification based on family history of hypertension was made.

Aerobic power. To determine aerobic power all subjects performed a graded discontinuous maximal wheelchair exercise test in their daily-use wheelchair on a motor-driven treadmill. This test has been described previously.[18] A test was ended when the subject could no longer maintain his position on the belt. To determine oxygen uptake ($\dot{V}O_2$), the expired air was collected during the exercise periods and analyzed with 30-sec samples with an Oxycon OX4.[a] Aerobic power was defined as the highest 30-sec value ($\dot{V}O_2$peak) found during the test. For safety reasons no $\dot{V}O_2$peak was determined in the oldest subject (age 71yrs).

Blood lipids and (apo-)lipoproteins. Subjects had been allowed to eat a light low-fat breakfast (bread and tea only). Most subjects, however, said that they had abstained from breakfast. Blood samples were drawn from an antecubital vein while each subject was sitting in his wheelchair. TC and TG were determined using an enzymatic colorimetric test.[b] HDL subfractions HDL_2-C and HDL_3-C were determined by a dual-precipitation method using dextran sulphate and $MgCl_2$: 0.8mL of serum was mixed with 2.4mL of a saline solution (density 1.006) and centrifuged in a thick-wall polycarbonate tube using a Beckman type L8-80 ultracentrifuge.[c] Centrifugation conditions were: 20h, 25,000rpm at 20°C using a type 50.4 fixed angle rotor. After centrifugation, the top fraction (containing very low-density lipoprotein cholesterol [VLDL-C]) was separated from the bottom fraction (containing LDL and HDL). Cholesterol was determined in both fractions. LDL-C was calculated by subtracting the HDL-C determined by precipitation from the cholesterol in the bottom fraction. ApoA-1 and apolipoprotein B-1 (apoB) were determined with immunonephelometric assays and apoA-2 was determined with a turbidimetric immunoassay using microtiter plates. In one subject, the cholesterol subfractions were not determined.

Several ratios frequently mentioned as independent cardiac risk factors were calculated: TC/HDL-C, HDL-C/LDL-C, HDL-C/ apoA-1, and apoA-1/apoB. The criteria used for classification of an increased risk were: TC of ≥6.5mmol/L,[20] TC/HDL-C of >7.0,[19] HDL-C of ≤.90mmol/L, LDL-C of >4.14mmol/L,[9] TG of >2.15mmol/L, and HDL-C/LDL-C of <.22.

Blood pressure. Systolic (SBP) and diastolic (DBP) finger arterial pressures were continuously recorded with a noninvasive blood pressure monitor[d] while subjects were sitting in their wheelchair. The Finapres is a noninvasive device capable of beat-to-beat measurement of blood pressure using an inflatable finger cuff with a built-in infrared photoplethysmograph. Subjects were instructed to hold the cuffed finger at heart level. The finger arterial pressure as measured with this device has

been shown to agree well with intraarterial blood pressure during resting conditions, showing a tendency (not significant) towards overestimation (+2 ± 11mmHg) of SBP and underestimation (−3 ± 7mmHg) of DBP.[21] Average values of 3min continuously recording were calculated. Subjects were classified as hypertensive when SBP of ≥160mmHg or DBP of ≥95mmHg was recorded.[22]

Comparison with an AB population. To compare the lipoprotein profile (TC, HDL-C, the ratio TC/HDL-C), BMI, SBP, and DBP of men with SCI with data obtained from an age- and sex-matched AB population from the same country as the subjects with SCI (n = 3,498, age 20 through 59yrs[19]), the group of subjects with SCI was divided into 4 age groups: 20 through 29 (n = 14), 30 through 39 (n = 10), 40 through 49 (n = 7), and 50 through 59 (n = 6) years; 1 subject (19yrs) was placed in group 20 through 29 and 3 subjects older than 59 (60, 61, and 71yrs) were placed in the oldest group.

Statistics. A one-way analysis of variance (ANOVA) was applied to study differences in subject characteristics, anthropometric factors, behavioral factors, blood pressure, and lipid and (apo-)lipoprotein profile among lesion groups. A Tukey post hoc test was applied to indicate which groups were different. A two-sample t test (for equal and unequal variance) compared TC, HDL-C, TC/HDL-C, SBP, DBP, and BMI of the men with SCI with those of the age-matched AB population.[19]

Pearson correlation coefficients were determined for the relations among the lipid and lipoprotein profile, blood pressure, age, TSI, aerobic power, alcohol consumption, and anthropometric factors. Spearman correlations determined the relations with the ordinal measures smoking behavior, family history, and lesion level. Multiple regression analysis was performed to estimate the most important predictors of lipid and lipoprotein fractions and blood pressure. Independent variables were age, lesion level, TSI, aerobic power, behavioral factors (activity level, smoking and drinking behavior), anthropometric factors (body mass, BMI, and skinfolds) and family history. For this purpose, each lesion level was assigned an arbitrary number ranking from 1 to 22, C4 being 1 and L5 being 22. The significance level for all tests was set at $p < .05$.

RESULTS

Relation With Lesion Level

No significant differences among lesion groups were found for age, TSI, body mass, BMI, Σ4SF, alcohol consumption, sport participation, and DBP (table 1). SBP was lower in the group with quadriplegia than in those with low-level paraplegia. Aerobic power was significantly inversely related with the level of the lesion, with the subjects with quadriplegia having significantly lower levels than those with mid- and low-level paraplegia. Twenty-three (62%) subjects were nonsmokers, 2 subjects were light smokers, and 12 subjects were heavy smokers (3 of whom were cigar smokers and 1 of whom was a pipe smoker). Table 2 displays the lipid and (apo-)lipoprotein levels for the four lesion groups. No significant differences among the lesion groups were found for any of the parameters.

Individuals at Increased Risk

Only 5 subjects (14%) had TC levels of >6.5mmol/L while 6 had HDL-C levels of <.91mmol/L. LDL-C levels were >4.14mmol/L in 7 (19%) and TG levels were >2.15mmol/L in 9 subjects. Few subjects had a TC/HDL-C ratio above 7.1 (n = 2) or a HDL-C/LDL-C ratio below .22 (n = 3).

Only 3 subjects were classified as hypertensive. In lesion group 4 (T11 through L5), 6 of 14 subjects had a SBP of >140mmHg, whereas only 2 of these 6 had a DBP of >90mmHg. None of the subjects with quadriplegia had hypertension. Ten subjects (27%) were classified as grade I obese (BMI of >25) whereas 5 subjects (14%) had grade II obesity.

Comparison With an AB Population

The percentage of nonsmokers among the subjects with SCI (23 of 37 [62%]) is very similar to that (61%) of the AB population.[19] Thirteen (35%) of our subjects did not participate in sport while 24% of the AB men between 20 and 60 years were classified as inactive (no sport participation and a sedentary job). Figure 1 compares the values for BMI, SBP, DBP, TC, TC/HDL-C, and HDL-C from the subjects with SCI with those from the age-matched AB population. BMI showed an increase with age in both populations. However, younger (younger than 30yrs) subjects with SCI had a significantly lower BMI (21.7 ± 2.8 vs. 23.3 ± 2.8) and older subjects (older than 49yrs) had a significantly higher BMI (29.6 ± 6.3 vs. 26.3 ± 3.1) than the AB subjects in the same age group. The prevalence of grade II obesity was slightly higher in our subjects (n = 5 [14%]) than in the AB men (8.1%). SBP was significantly higher in subjects with SCI younger than 40 years than in AB persons. In contrast, DBP was significantly lower in subjects with SCI older than 39 years.

TC and TC/HDL-C ratio demonstrated an increase with age in both populations and were not significantly different between the two populations. In spite of a tendency towards lower HDL-C levels in subjects with SCI in all age groups, differences between both groups were not significant. The prevalence of excess TC seems to be lower in the subjects with SCI (n = 5 [14%]) than in the AB population (19.9%).

Table 2: Blood Lipid and (Apo-)Lipoprotein Profile in the 4 Lesion Groups

	1: C4-C8	2: T1-T5	3: T6-T10	4: T11-L5	Total
TC (mmol/L)	4.75 ± 1.02	5.28 ± .81	5.34 ± 1.19	5.14 ± 1.31	5.13 ± 1.31
VLDL-C (mmol/L)	.73 ± .25	.90 ± .45	.96 ± .43	.82 ± .34	.85 ± .36
LDL-C (mmol/L)	2.95 ± 1.00	3.34 ± .68	3.35 ± .98	3.21 ± 1.29	3.22 ± 1.05
HDL-C (mmol/L)	1.03 ± .14	1.06 ± .14	1.02 ± .17	1.10 ± .23	1.06 ± .18
HDL_2-C (mmol/L)	.17 ± .10	.15 ± .09	.11 ± .09	.18 ± .17	.16 ± .13
HDL_3-C (mmol/L)	.86 ± .12	.91 ± .11	.91 ± .12	.92 ± .08	.90 ± .10
TG (mmol/L)	1.41 ± .48	1.82 ± .86	1.85 ± .85	1.56 ± .59	1.64 ± .68
HDL-C/LDL-C	.38 ± .11	.33 ± .09	.33 ± .11	.41 ± .21	.37 ± .15
TC/HDL-C	4.61 ± .93	5.07 ± 1.17	5.40 ± 1.62	4.85 ± 1.55	4.98 ± 1.40
apoA-1 (g/L)	1.26 ± .12	1.38 ± .16	1.34 ± .14	1.32 ± .17	1.32 ± .17
apoA-2 (g/L)	.37 ± .04	.39 ± .04	.41 ± .03	.40 ± .05	.39 ± .04
apoB (g/L)	1.15 ± .31	1.32 ± .27	1.40 ± .42	1.25 ± .44	1.28 ± .38
apoA-1/apoB	1.14 ± .24	1.08 ± .25	1.02 ± .29	1.21 ± .50	1.12 ± .37
HDL-C/apoA-1	31.9 ± 2.8	29.8 ± 2.6	29.4 ± 2.8	32.0 ± 3.1	30.9 ± 3.1

No significant ($p > .05$) differences were found among groups.

Correlations Among Lipid Profile and Blood Pressure

TC was found to be directly related ($p < .01$) with LDL-C, apoB, and TC/HDL-C, and significantly inversely related with HDL-C/LDL-C (table 3). HDL-C was significantly inversely related with VLDL-C, TG, and directly related ($p < .05$) with apoA-1 and apoA-2. ApoB was significantly related with LDL-C, TC/HDL-C, and HDL-C/LDL-C. The ratio apoA-1/apoB reflected the ratios TC/HDL-C and HDL-C/LDL-C. VLDL-C was significantly related to TG. SBP and DBP were only moderately related ($r = 0.3$) to apoA-2.

Relation Between Lipid and Lipoprotein Profile, Blood Pressure, and Selected Factors

TC, LDL-C, and apoB were significantly and directly related to age, TSI, Σ4SF, body mass, BMI, and smoking behavior, and significantly inversely related to sport participation and $\dot{V}O_2$peak (mL/kg/min) (table 4). HDL-C and HDL_2-C were directly related to alcohol consumption and inversely related to BMI, body mass, and Σ4SF. HDL_3-C, in contrast, was not significantly related to any parameter. VLDL-C and TG were directly related ($p < .05$) to Σ4SF, BMI, body mass, age, and TSI, and significantly inversely related to alcohol consumption. The TC/HDL-C ratio was significantly directly related to Σ4SF, body mass, age, TSI, and smoking behavior, and inversely ($p < .05$) related to alcohol consumption. The HDL-C/LDL-C ratio was significantly directly related to the sport participation and inversely related to Σ4SF, body mass, BMI, age, and smoking behavior. ApoA-1 was only significantly related to alcohol consumption, whereas apoA-2 was inversely related ($p < .05$) to active sport participation. SBP was most strongly related to lesion level: subjects with lower lesions had higher SBP levels. Both SBP and DBP were positively related to family history of hypertension.

In the group of subjects with SCI, absolute levels of aerobic power ($\dot{V}O_2$peak) were not significantly related to any of the lipids or (apo-)lipoproteins (table 4). Some within-group correlations were only significant in group T11 through L5 ($n = 14$): $\dot{V}O_2$peak was significantly inversely related to TC ($r = -.62$), LDL-C ($r = -.60$), apoB ($r = -.57$), and TC/HDL-C ($r = -.56$). Age was notably more strongly related to these parameters in the T11 through L5 group than was aerobic power (TC: $r = .77$; LDL-C: $r = .75$; apoB: $r = .78$; TC/HDL-C: $r = .77$).

Prediction of Lipid and (Apo-)Lipoprotein Profile and Blood Pressure

With the current set of independent parameters (age, lesion level, TSI, hours of sport participation, body mass, BMI, Σ4SF, smoking and drinking behavior, family history, and aerobic power), 12% to 71% of the variance in lipid, (apo-)lipoprotein, and blood pressure parameters could be accounted for after multiple regression analysis (table 5). Because of missing data the number of subjects used for these equations varied between 33 and 35.

Age was the most important predictor of TC, LDL-C, apoB, and of the ratios HDL-C/LDL-C, TC/HDL-C, and apoA-1/apoB. Smoking behavior explained an additional part of the variance in TC, LDL-C, apoB, TC/HDL-C, and HDL-C/LDL-C. Nonsmokers had on average a TC .42mmol/L lower and a

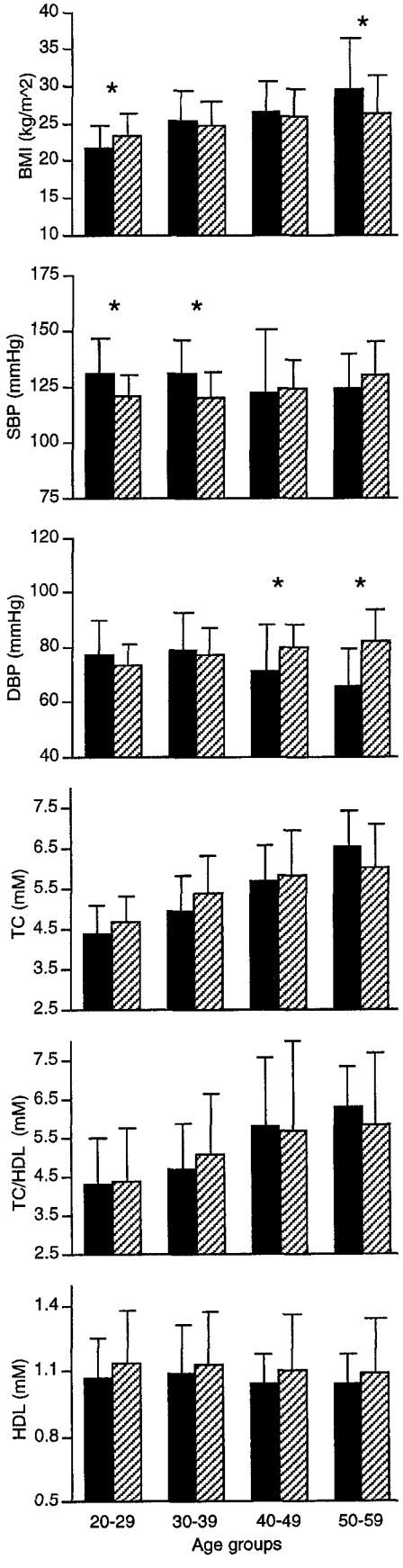

Fig 1. Comparison of values for BMI, SBP, DBP, TC, HDL-C, and TC/HDL-C ratio in subjects with SCI (■) to values derived from an age-matched AB male population (▨)[19] (*significant [$p < .05$] differences).

Table 3: Correlations Among Blood Lipid and (Apo-)Lipoproteins, and Blood Pressure

	TC	HDL	HDL_2	HDL_3	LDL	VLDL	TC/HDL	HDL/LDL	TG	apoA-1	apoA-2	apoB	apoA1/apoB	HDL/apoA-1	SBP
HDL-C	−.03														
HDL_2-C	−.15	.85*													
HDL_3-C	.13	.75*	.28†												
LDL-C	.97*	.00	−.09	.12											
VLDL-C	.36†	−.64*	−.66*	−.34†	.17										
TC/HDL-C	.80*	−.59*	−.58*	−.34†	.74*	.71*									
HDL-C/LDL-C	−.80*	.41*	.46*	.18	−.82*	−.37†	−.84*								
TG	.35†	−.67*	−.71*	−.32†	.21	.87*	.70*	−.45*							
apoA-1	.18	.84*	.61*	.75*	.16	−.35†	−.33†	.20	−.35†						
apoA-2	.03	.59*	.40*	.58*	.05	−.44*	−.30†	.12	−.27	.54*					
apoB	.95*	−.23	−.32†	−.01	.92*	.48*	.89*	−.85*	.48*	.07	−.04				
apoA-1/apoB	−.78*	.52*	.54*	.27	−.77*	−.51*	−.90*	.97*	−.56*	.29†	.19	−.87*			
HDL-C/apoA-1	−.27	.75*	.74*	.43*	−.18	−.76*	−.67*	.48*	−.79*	.28†	.35†	−.48*	.58*		
SBP	−.14	.02	−.03	.08	−.13	−.12	−.16	.08	.03	.01	.32†	−.07	.05	.06	
DBP	−.14	−.05	−.04	−.03	−.14	−.04	−.10	.04	.10	.06	.31†	−.01	−.02	−.12	.77*

* $p < .01$.
† $p < .05$.

LDL-C .37mmol/L lower than light smokers (after adjusting for age and activity level). The hours of weekly sport participation accounted for an extra 4% to 6% of the variance in TC and LDL-C. One hour more sport participation was associated with a TC .05mmol/L lower and a LDL-C .07mmol/L lower.

Only a small part of the variance in VLDL-C (R^2 = .16), TG (R^2 = .24), and the ratio HDL-C/apoA-1 (R^2 = .16) was explained with Σ4SF being the most important predictor of these parameters, with no other variables adding significantly to the explanation of the variance. Alcohol consumption accounted for 12% of the variance in HDL-C: the consumption of 1 drink per day was associated with a HDL-C .035mmol/L higher. BMI together with alcohol consumption accounted for 26% of the variance in HDL_2-C.

The hours of weekly sport participation explained 15% of the variance in apoA-2: every additional hour was associated with a .005g/L reduction in apoA-2. Age was also an independent predictor: 1 year older was related to a .001g/L decrease in apoA-2.

Lesion level was the most important determinant of SBP, explaining 35% of the variance. After adjusting for lesion level, family history of hypertension explained an additional 9%. Not lesion level, but family history was entered first into the equation to predict DBP, explaining 13% of the variance. Age appeared to have an independent effect on both SBP and DBP: an increase of 1 year was associated with a SBP and DBP 0.5mmHg lower. The amount of sport participation added significantly to the explanation in variance of DBP: 1 hour more was associated with a 1.4mmHg reduction.

DISCUSSION

The mean TC level of 5.1 ± 1.3mmol/L found in the present study agrees with other studies on men with chronic SCI.[9,16,23,24] In spite of a tendency toward a lower TC among those with quadriplegia, differences among lesion groups were not significant, which is in accordance with other studies.[9,10,23,24] In agreement with other studies,[5,10] TC was not higher than in AB persons. On the contrary, TC tends to be or is significantly lower among persons with SCI.[4,6,7,9,23] In only 5 of the 37 subjects did TC exceed 6.5mmol/L. Hence, persons with SCI do not seem to be at higher risk for CHD than AB when only TC is considered.

In agreement with other studies,[4,6,9,23] HDL-C values tended to be lower than reported in AB persons,[19] although the differences were not significant. Six subjects (17%) had a HDL-C level of <.91mmol/L, which is an independent risk factor of CHD. In older men with SCI (mean age 48), an even higher percentage of 37% was reported.[9] Based on risk estimates from the Framingham Study,[25] the mean HDL-C level of 1.06mmol/L represents an excess risk of CHD of approximately 18%. Hence, the tendency toward lower HDL-C values in persons with SCI suggests a slightly higher CHD risk.

The tendency toward lower HDL-C levels may be related to

Table 4: Correlations of Lipid and (Apo-)Lipoprotein Profile and Blood Pressure to Subject Characteristics, Behavioral Factors, Anthropometric and Family History of CHD, and Hypertension (Family BP)

	TC	HDL	HDL_2	HDL_3	LDL	VLDL	TC/HDL	HDL/LDL	TG	apoA-1	apoA-2	apoB	apoA1/apoB	HDL/apoA-1	SBP	DBP
Age	.68*	−.13	−.17	−.02	.58*	.30†	.58*	−.56*	.28†	.03	−.16	.64*	−.57*	−.26	−.12	−.27
Lesion level	.08	.12	.04	.21	.06	.04	.00	.02	−.00	.12	.24	.09	.01	.10	.57*	.20
TSI	.55*	−.16	−.14	−.06	.58*	.31†	.57*	−.56*	.31†	.06	−.11	.61*	−.55*	−.35†	−.25	−.05
Body mass	.49*	−.25	−.34†	−.03	.49*	.30†	.54*	−.51*	.43*	−.10	−.13	.48*	−.47*	−.32†	−.05	−.11
Sum 4 skinfolds	.45*	−.28	−.33†	−.09	.43*	.38†	.50*	−.44*	.48*	−.07	−.20	.45*	−.44*	−.40*	.03	−.02
BMI	.47*	−.29†	−.39*	−.04	.46*	.32†	.53*	−.47*	.47*	−.11	−.13	.50*	−.48*	−.38†	−.02	−.06
Sport participation	−.33†	−.14	−.18	−.03	−.40*	.20	−.17	.28†	.06	−.12	−.40*	−.28†	.25	−.11	−.20	−.25
Smoking	.51*	.05	−.09	.08	.52*	.21	.41†	−.45*	.26	.22	−.20	.51*	−.43*	−.15	−.27	−.09
Alcohol use	−.22	.36†	.38†	.18	−.19	−.35†	−.38†	.22	−.31†	.30†	.18	−.25	.29†	.28†	.11	.10
Family CHD	.23	.00	.07	.00	.21	.11	.11	−.14	.06	.12	.28	.13	−.02	−.00	.26	.00
Family BP	.08	−.20	−.18	−.09	.08	.14	.15	−.14	.26	−.18	−.03	.20	−.25	−.05	.28*	.29†
$\dot{V}O_2$ (l/min)	−.15	.09	−.02	.19	−.21	.07	−.11	.21	.09	.06	.23	−.15	.23	.06	.47*	.23
$\dot{V}O_2$ (mL/kg/min)	−.39*	.16	.12	.14	−.44*	−.06	−.35†	.45*	−.10	.08	.24	−.37†	.42*	.17	.46*	.29†

* $p < .01$.
† $p < .05$.

Table 5: Significant Multiple Regression Analyses to Predict Lipid and (Apo-)Lipoprotein Profile and Systolic and Diastolic Blood Pressure

	Regression Coefficient (+ Intercept)	Variable	N	Significance	Multiple R^2
TC =	+.063 ± .011	Age	35	.000	.52
	+.422 ± .110	Smoking		.026	.64
	−.054 ± .033	Hours Sport		.045	.68
	+2.294 ± .462			.000	
VLDL-C =	+.004 ± .002	Skinfolds	34	.020	.16
	+.541 ± .136			.000	
LDL-C =	+.055 ± .009	Age	34	.000	.51
	+.367 ± .110	Smoking		.002	.65
	−.070 ± .029	Hours Sport		.021	.71
	+.829 ± .399			.046	
HDL-C =	+.035 ± .017	Alcohol Consumption	34	.045	.12
	+1.017 ± .039			.000	
HDL_2-C =	−.009 ± .004	BMI	34	.028	.16
	+.022 ± .011	Alcohol Consumption		.048	.26
	+.361 ± .105			.002	
HDL-C/LDL-C =	−.007 ± .002	Age	34	.001	.36
	−.045 ± .020	Smoking		.030	.45
	+.679 ± .066			.000	
TC/HDL-C =	+.062 ± .017	Age	34	.001	.36
	+.444 ± .195	Smoking		.030	.45
	−.211 ± .099	Alcohol Consumption		.041	.52
	+2.294 ± .706			.003	
TG =	+.010 ± .003	Skinfolds	35	.003	.24
	+.911 ± .244			.001	
apoA-2 =	−.005 ± .002	Hours Sport	35	.005	.15
	−.001 ± .001	Age		.021	.29
	+.478 ± .022			.000	
apoB	+.020 ± .004	Age	35	.000	.46
	+.152 ± .046	Smoking		.003	.60
	+.280 ± .154			.078	
apoA-1/apoB	−.017 ± .004	Age	35	.001	.35
	−.115 ± .052	Smoking		.033	.44
	+1.932 ± .172			.000	
HDL-C/apoA-1	$-3.6*10^{-4} \pm 1.4*10^{-4}$	Skinfolds	35	.019	.16
	+.335 ± 0.011			.000	
SBP	+2.028 ± .531	Lesion level	33	.001	.35
	+6.537 ± 2.612	Fam. Hypertension		.018	.44
	−.500 ± .244	Age		.049	.51
	+115.207 ± 10.617			.000	
DBP	+5.367 ± 1.863	Fam. Hypertension	33	.007	.13
	−.547 ± .183	Age		.006	.29
	−1.407 ± .654	Hours Sport		.040	.38
	+91.759 ± 7.965			.000	

the higher TG (1.6mmol/L) compared to values (1.1 to 1.4mmol/L) in AB persons.[6,7,26,27] TG was inversely associated with HDL-C levels ($r = -.67$), which agrees with studies among AB persons[28-30] and among men with SCI.[9,24] Plasma lipoprotein lipase, a lipolytic enzyme, hydrolyzing TG into lipoproteins, plays an important role in the metabolism of lipoproteins and has been shown to increase through exercise.[31] Lower activity levels in men with SCI may therefore result in a higher TG and a lower HDL-C. In addition, the lower HDL-C and higher TG levels may also be the result of an elevated plasma insulin level caused by a lower insulin sensitivity.[9]

Several studies stress that TC/HDL-C is a more important risk indicator of CHD than TC or HDL-C levels alone.[3,32] Because of the tendency toward lower levels of both TC and HDL-C in our subjects, TC/HDL-C was not significantly different from that in the AB population.[19] Moreover, HDL-C/LDL-C, suggested to be a better CHD risk indicator than TC or HDL-C in persons with SCI,[9] was below .22 in only 3 subjects, indicating that the lower HDL-C also corresponds with a lower LDL-C. Indeed, the mean LDL-C in the present study (3.2mmol/L) was lower than reported for AB men (4.1mmol/L,[26]). The average HDL-C/LDL-C of .37 ± .15 in the present study is higher than the .31 reported[24] in a group of middle-aged (mean age 49.5) men with SCI but consistent with other studies using relatively young men with long-standing SCI,[16] and only slightly lower than reported (0.4 to 0.5) among AB men.[14,33] Hence, the lower HDL-C coincides to a certain extent with lower levels of TC and LDL-C, resulting in an equal or moderately higher CHD risk among persons with SCI.

TC, LDL-C, apoB, and the ratios TC/HDL-C and HDL-C/LDL-C were highly intercorrelated (table 3), with age as the most important determinant of these parameters in our group of subjects. Smoking behavior added significantly to the explanation in variance with the smokers having a less favorable

CHD risk profile, which has also been observed in the AB population. Weekly sport participation was associated with lower TC levels, predominantly due to lower LDL-C levels. Hooker and Wells[34] showed that an increased activity level (an 8-week moderate-intensity training regimen) can actually reduce TC and LDL-C in men with SCI. Hence, it seems that modifiable risk indicators determine to an important extent TC and LDL-C, and consequently HDL-C/LDL-C and TC/HDL-C, suggesting a possible way of intervention.

The most important predictor of HDL-C in the present study was alcohol consumption. The positive relation has also been found in AB persons.[3,27,31] In contrast to studies with AB persons,[27,30,31] variables such as BMI, age, smoking, aerobic power, and activity level did not add significantly to the explanation of the variance. BMI, however, showed a significant but inverse relation with HDL-C and was the most important determinant of HDL_2-C. The decrease of .009mmol/L in HDL_2-C with one unit decrease in BMI is similar to values reported in AB men.[27,31] Hence, if the proposed relations are causal, weight reduction and moderate alcohol consumption may improve HDL-C levels in men with SCI. Moreover, reducing the amount of adipose tissue may result in a decrease in TG levels and a concomitant increase in HDL-C levels.

In the present study, the amount of sport participation was not related to HDL-C. However, an experimental study by Hooker and Wells[34] showed a significant increase in HDL-C (from 1.0 to 1.2mmol/L) in five subjects with SCI after a moderate-intensity training regimen. In contrast, a low-intensity training group in the same study displayed a tendency towards a decrease (from 1.3 to 1.1mmol/L). Since the intensity of sport participation was not considered in our study, differences in intensity may have influenced the relations studied. Additional experimental research with larger subject groups is indispensable to verify whether HDL-C levels can be augmented through exercise in these individuals, and if so, which mode of exercise and level of intensity provide the best results.

Mean values for apoA-1 (1.32 ± .17g/L) were in agreement with those of men with long-standing SCI[16] and only slightly lower than reported (1.4 to 1.5g/L) in AB men.[4,26,35] Analogous to apoA-1 levels, apoA-2 showed no significant differences among the four lesion groups and levels were similar to values (0.3 to 0.4g/L) reported among AB.[3,28] ApoB, the structural protein of VLDL-C and LDL-C, has been directly associated with an increased risk of CHD.[28] Mean apoB (1.28 ± .38g/L) was considerably higher than values (0.9 to 1.1g/L) reported in AB men[3,26,28] and similar to or even higher than reported (1.1 to 1.4g/L) among men with CHD.[3,28,35] In this respect, individuals with SCI seem to have a higher risk of CHD than AB. However, more prospective research is necessary to confirm the proposed causal relation between apoB and CHD in persons with SCI.

Mean BMI and Σ4SF were similar to those reported among sedentary persons with SCI, but considerably higher than among wheelchair athletes.[7] The considerable number of subjects with grade I (n = 10 [27%]) or grade II obesity (n = 5) together with the large Σ4SF connotes that a major part of our subjects had excessive adipose tissue. Although the mean BMI (24.9 ± 4.8) was not essentially different from values reported among AB men,[19,36] the average Σ4SF was considerably larger than in AB populations.[37] It seems that men with SCI, especially those at advanced age, are more likely to develop excessive adipose tissue, probably as a result of the (forced) sedentary lifestyle and the concomitant lower daily energy expenditure,[17] a decreased basal (and/or resting) metabolism,[17] and a higher than recommended fat consumption.[38] Nutritional intervention and an increase in regular activity seem to be indispensable to prevent persons with SCI from becoming obese, especially when the increasing longevity of individuals with SCI[2] is considered.

SBP appeared to be inversely related to the lesion level, which is consistent with other studies.[39,40] None of the subjects with quadriplegia could be classified as hypertensive, whereas only three subjects with paraplegia had hypertension. The low resting blood pressure may be the result of a decreased sympathetic nervous system activity below the lesion. In agreement with the results from an Australian study,[23] SBP and DBP were considerably lower than reported among AB (age 47 ± 6yr) men.[36] Comparison with the age-matched AB population,[19] DBP was significantly lower in older subjects with SCI while SBP was somewhat higher among the younger subjects. Hence, in comparison with the AB population, individuals with SCI, especially those with high- and mid-level lesions, seem to have an equal or even lower blood pressure profile.

However, blood pressure may increase dramatically during periods of autonomic dysreflexia,[41] during functional electrical stimulation,[42] or during activities with a large isometric component (eg, transfers), which is very common in daily life of persons with SCI. This elevated blood pressure is not only a risk factor for CHD, but for cerebrovascular accidents as well. In addition, chronic renal failure, a common problem among individuals with SCI, may increase SBP significantly in persons with SCI.[6]

The most important determinants of SBP were lesion level, family history of hypertension, and age, together accounting for 51% of the variance in SBP, suggesting that SBP in this group is for a major part determined by nonmodifiable factors. In contrast, every additional hour of sport participation was associated with a 1.4mmHg reduction in DBP after statistically adjusting for family history and age, suggesting a potential for intervention.

Absolute aerobic power was not significantly related to any of the lipids or (apo-)lipoproteins in this group of subjects. This is in contrast to significant relations found in AB persons between aerobic power and HDL-C[14] and HDL-C/LDL-C,[14,33] and also in contrast to the relations between aerobic power and HDL-C, TC/HDL-C, TG, VLDL-C, LDL-C/HDL-C, apoB/ApoA-1, and HDL-C/apoA-1 found in men with SCI.[9,16] The latter studies,[9,16] however, did not take age of the subjects into account, and since age has been shown to be an important determinant of the major part of the lipoprotein levels, the relations found may have been affected by (or even the result of) the variance in age in their subjects. Regression analyses revealed that in persons with SCI, activity level is more important than aerobic power in predicting lipoprotein levels, suggesting that those with a low aerobic power as a result of a high lesion are not predisposed to have a higher CHD risk per se, but may improve their risk profile through regular exercise.

The limited number and the rather young mean age of our subjects hinders generalization of results to the entire population of men with SCI. Moreover, all subjects were volunteers, which may have resulted in a younger, healthier, and more active subject group not representative of the whole population. The only criteria for exclusion, however, were the inability to propel a wheelchair manually and an age below 16yrs, which resulted in a rather heterogeneous subject group with regard to age, lesion level, sport participation, and body mass.

In conclusion, men with SCI do not seem to have an essentially different CHD risk profile in comparison to AB persons. Although HDL-C tends to be slightly lower than in AB men, TC and LDL-C are correspondingly lower, resulting in similar HDL-C/LDL-C and TC/HDL-C ratios. The higher apoB levels

than reported among AB persons suggest a moderately higher CHD risk in men with SCI. In contrast, the essentially lower DBP values, especially found in persons with quadriplegia, may indicate a lower CHD risk than in AB persons. However, men with SCI, especially older individuals, seem to have a higher risk of developing obesity (with concomitant changes in TG, VLDL-C, and HDL-C levels).

In addition, the present study shows that the lipid and (apo) lipoprotein profile is not related to the lesion level nor to the absolute aerobic power in a heterogeneous group of men with long-standing SCI. More important predictors are age, activity level, smoking behavior, alcohol consumption, and adipose tissue (as estimated by BMI or skinfolds), indicating that modifiable risk factors determine, as in the AB population, CHD risk indicators to an important extent, leaving a number of possibilities for intervention. However, experimental and prospective analyses of CHD in relation to the risk factors studied remain necessary to establish the causality of the relations found and whether they also apply to individuals with SCI.

Acknowledgments: The authors gratefully acknowledge the involvement of CMH van de Heuvel, CP Kesselaar, EEP Rozendaal, and EM Willemsen in the data collection. Thanks are due to the subjects for their participation.

References

1. Yekutiel M, Brooks ME, Ohry A, Yarom J, Carel R. The prevalence of hypertension, ischaemic heart disease and diabetes in traumatic spinal cord injured patients and amputees. Paraplegia 1989;27:58-62.
2. Geisler WO, Jousse AT, Wynne-Jones M, Breithaupt D. Survival in traumatic spinal cord injury. Paraplegia 1983;21:364-73.
3. Stampfer MJ, Sacks FM, Salvini S, Willett WC, Hennekens CH. A prospective study of cholesterol, apolipoproteins, and the risk of myocardial infarction. N Engl J Med 1991;325:373-81.
4. Nikkilä EA, Kuusi T, Myllynen P. High density lipoprotein and apolipoprotein A-1 during physical inactivity. Atherosclerosis 1980; 37:457-62.
5. Heldenberg D, Rubinstein A, Levtov O, Werbin B, Tamir I. Serum lipids and lipoprotein concentrations in young quadriplegic patients. Atherosclerosis 1981;39:163-7.
6. Vaziri ND, Gordon S, Nikakhtar B. Lipid abnormalities in chronic renal failure associated with spinal cord injury. Paraplegia 1982; 20:183-9.
7. Brenes G, Dearwater S, Shapera R, LaPorte RE, Collins E. High density lipoprotein cholesterol concentrations in physically active and sedentary spinal cord injured patients. Arch Phys Med Rehabil 1986;67:445-50.
8. Dearwater SR, LaPorte RE, Robertson RJ, Brenes G, Adams LL, Becker D. Activity in the spinal cord-injured patient: an epidemiologic analysis of metabolic parameters. Med Sci Sports Exerc 1986; 18:541-4.
9. Bauman WA, Spungen AM, Zhong Y-G, Rothstein JL, Petry C, Gordon SK. Depressed serum high density lipoprotein cholesterol levels in veterans with spinal cord injury. Paraplegia 1992;30:697-703.
10. Cardús D, Ribas-Cardús F, McTaggart WG. Lipid profiles in spinal cord injury. Paraplegia 1992;30:775-82.
11. Berg A, Keut J, Ringwald R, Deus B, Wybitul K. Physical performance and serum cholesterol fractions in healthy young men. Clin Chim Acta 1980;106:325-30.
12. Schnabel A, Kindermann W. Effect of maximal oxygen uptake and different forms of physical training on serum lipoproteins. Eur J Appl Physiol 1982;48:263-77.
13. Haskell WL. Exercise induced changes in plasma lipids and lipoproteins. Prev Med 1984;13:23-36.
14. Sallis JF, Patterson TL, Buono MJ, Nader PR. Relation of cardiovascular fitness and physical activity to cardiovascular disease risk factors in children and adults. Am J Epidemiol 1988;127:933-41.
15. Abbott RF, Levy D, Kannel WB. Cardiovascular risk factors and graded treadmill exercise endurance in healthy adults: The Framingham Offspring Study. Am J Cardiol 1989;63:342-6.
16. Bostom AG, Toner MM, McArdle WD, Montelione T, Brown CD, Stein RA. Lipid and lipoprotein profiles relate to peak aerobic power in spinal cord injured men. Med Sci Sports Exerc 1991;23:409-14.
17. Mollinger LA, Spurr GB, El Ghatit AZ, Barboriak JJ, Rooney CB, Davidoff DD, et al. Daily energy expenditure and basal metabolic rates of patients with spinal cord injury. Arch Phys Med Rehabil 1985;66:420-6.
18. Janssen TWJ, Van Oers CAJM, Hollander AP, Veeger HEJ, Van der Woude LHV. Isometric strength, sprint power, and aerobic power in individuals with a spinal cord injury. Med Sci Sports Exerc 1993;25:863-70.
19. Kromhout D, Obermann-De Boer GL, Blokstra A, Verschuren WMM. Report 528901003 (in Dutch). The Netherlands: National Institute of Public Health and Environmental Protection, September 1990.
20. Erkelens DW. Cholesterol consensus in the Netherlands. Eur J Clin Nutr 1989;43:89-96.
21. Imholz BPM, Settels JJ, Van den Meiracker AH, Wesseling KH, Wieling W. Non-invasive beat to beat finger blood pressure measurement during orthostatic stress compared to intraarterial pressure. Cardiovasc Res 1990;24:214-21.
22. World Health Organization. Arterial hypertension. Technical Report Series No. 628. Geneva: World Health Organization, 1978.
23. Krum H, Howes LG, Brown DJ, Ungar G, Moore P, McNeil JJ, et al. Risk factors for cardiovascular disease in chronic spinal cord injury patients. Paraplegia 1992;30:381-8.
24. Maki KC, Briones ER, Langbein WE, Inman-Felton A, Nemchausky B, Welch M, et al. Associations between serum lipids and indicators of adiposity in men with spinal cord injury. Paraplegia 1995;33:102-9.
25. Gordon T, Castelli WP, Hjortland MC, Kannel WB. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. Am J Med 1977;62:707-13.
26. Boomsma DI, Kaptein A, Kempen HJM, Gevers Leuven JA, Princen HMG. Lipoprotein(a): relation to other risk factors and genetic heritability. Results from a Dutch parent-twin study. Atherosclerosis 1993;99:23-33.
27. Lakka TA, Salonen JT. Physical activity and serum lipids: a cross-sectional population study in Eastern Finnish men. Am J Epidemiol 1992;136:806-18.
28. Kukita H, Hamada M, Hiwada K, Kokubu T. Clinical significance of measurements of serum apolipoprotein A-I, A-II and B in hypertriglyceridemic male patients with and without coronary artery disease. Atherosclerosis 1985;55:143-9.
29. Laakso M, Pyörälä K, Voutilainen E, Marniemi J. Plasma insulin and serum lipids and lipoproteins in middle-aged non-insulin dependent diabetic and non-diabetic subjects. Am J Epidemiol 1987;125: 611-21.
30. Hughes K, Leong WP, Sothy SP, Lun KC, Yeo PPB. Relationships between cigarette smoking, blood pressure and serum lipids in the Singapore general population. Int J Epidemiol 1993;22:637-43.
31. Patsch W, Sharrett AR, Sorlie PD, Davis CE, Brown SA. The relation of high density lipoprotein cholesterol and its subfractions to apolipoprotein A-1 and fasting triglycerides: the role of environmental factors. Am J Epidemiol 1992;136:546-57.
32. Castelli WP. Epidemiology of coronary heart disease. Am J Med 1984;76 Suppl 2A:4-12.
33. Sedgwick AW, Thomas DW, Davies M, Baghurst K, Rouse I. Cross-sectional and longitudinal relationships between physical fitness and risk factors for coronary heart disease in men and women: "The Adelaide 1000." J Clin Epidemiol 1989;42:189-200.
34. Hooker SP, Wells CL. Effects of low- and moderate-intensity training in spinal cord-injured persons. Med Sci Sports Exerc 1989;21: 18-22.
35. Pujia A, Gnasso A, Cortese C, Angotti E, Colonna A, Mattioli PL. Early extracoronary heart disease risk factors in a sample of civil servants in Southern Italy. Atherosclerosis 1993;102:1-7.
36. Bovens AM, Van Baak MA, Vrencken JG, Wijnen JA, Saris WH, Verstappen FT. Physical activity, fitness, and selected risk factors

for CHD in active men and women. Med Sci Sports Exerc 1993; 25:572-6.
37. Wallace ES, White JA, Downie A, Dalzell G, Doran D. Influence of exercise adherence level on modifiable coronary heart disease risk factors and functional-fitness levels in middle-aged men. Br J Sports Med 1993;27:101-6.
38. Levine AM, Nash MS, Green BA, Shea JD, Aronica MJ. An examination of dietary intakes and nutritional status of chronic healthy spinal cord injured individuals. Paraplegia 1992;30: 880-9.
39. Kessler KM, Pina I, Green B, Burnett B, Laighold M, Bilsker M, et al. Cardiovascular findings in quadriplegic and paraplegic patients and in normal subjects. Am J Cardiol 1986;58:525-30.
40. Imai K, Kadowaki T, Aizawa Y, Fukutomi K. Morbidity rates of complications in persons with spinal cord injury according to the site of injury and with special reference to hypertension. Paraplegia 1994;32:246-52.
41. Naftchi NE, Demeny M, Lowman EW, Tuckman J. Hypertensive crises in quadriplegic patients. Changes in cardiac output, blood volume, serum dopamine-β-hydroxylase activity, and arterial prostaglandin PGE_2. Circulation 1978;57:336-41.
42. Ashley EA, Laskin JJ, Olenik LM, Burnham R, Steadward RD, Cumming DC, et al. Evidence of autonomic dysreflexia during functional electrical stimulation in individuals with spinal cord injuries. Paraplegia 1993;31:593-605.

Suppliers

a. Jaeger/Tonnies, Nikkelstraat 2, 4823 AB Breda, The Netherlands.
b. CHOD-PAP; Boehringer Mannheim GmbH, Sandhoferstrasse 116, D-68305, Mannheim, Germany.
c. Beckman Instruments, 200 South Kraemer Blvd, Brea, CA 92621.
d. Finapres BP2300; Ohmeda, 355 Inverness Drive South, Englewood, CO 80112-5810.