Table 3—*Patient prevalence at baseline and hazard ratios for coronary heart disease, stroke, or both in Japanese study subjects grouped by metabolic syndrome status*

	Prevalence at baseline		Hazard ratios for CHD		Hazard ratios for stroke		Hazard ratios for CHD and/or stroke	
	Men	Women	Men	Women	Men	Women	Men	Women
Criteria of individual components								
1a. BMI >30 or WHR >0.90 (men) or >0.85 (women)	39.4	37.5	1.3 (0.7–2.5)	1.2 (0.5–3.0)	1.3 (0.7–2.6)	1.1 (0.5–2.3)	1.4 (0.8–2.2)	1.2 (0.6–2.1)
1b. Waist circumference ≥85cm (men) or ≥90 cm (women)	36.7	9.6	1.7 (0.9–3.0)	1.0 (0.2–4.4)	0.90 (0.4–1.9)	1.1 (0.3–3.7)	1.3 (0.8–2.1)	1.1 (0.4–2.8)
2a. SBP ≥140 or DBP ≥90 mmHg	38.9	38.9	0.8 (0.4–1.6)	1.0 (0.4–2.6)	2.1 (1.1–4.3)	2.4 (1.1–5.5)	1.3 (0.8–2.1)	1.8 (1.0–3.2)
2b. SBP ≥130 or DBP ≥85 mmHg	60.7	62.2	0.9 (0.5–1.6)	0.9 (0.4–2.2)	1.4 (0.7–2.9)	1.8 (0.7–4.5)	1.1 (0.6–1.7)	1.2 (0.7–2.4)
3. Triglycerides ≥150 mg/dl	24.8	21.0	2.9 (1.6–5.3)	1.7 (0.6–4.4)	1.1 (0.5–2.4)	0.7 (0.2–1.9)	2.0 (1.2–3.2)	1.1 (0.5–2.2)
4. HDL cholesterol ≤40 mg/dl	19.3	36.3	1.8 (0.9–3.5)	1.5 (0.6–3.6)	1.0 (0.4–2.5)	1.3 (0.6–2.9)	1.6 (0.9–2.6)	1.3 (0.7–2.4)
5. Triglycerides ≥150 mg/dl or HDL cholesterol <35 mg/dl	28.5	27.0	2.8 (1.6–5.2)	1.8 (0.7–4.5)	0.9 (0.4–1.9)	1.6 (0.7–3.5)	1.8 (1.1–2.9)	1.6 (0.9–2.9)
6. Urinary albumin excretion >30 μg/g creatinine	51.2	57.7	1.2 (0.6–2.3)	2.9 (0.9–8.7)	1.8 (0.9–3.8)	1.1 (0.5–2.4)	1.4 (0.9–2.3)	1.6 (0.8–3.0)
7. LDL cholesterol ≥120 mg/dl	45.1	65.2	2.1 (1.1–3.9)	1.2 (0.5–3.2)	0.9 (0.5–1.8)	0.6 (0.3–1.3)	1.4 (0.9–2.3)	0.8 (0.4–1.4)
8. Current smoker	43.9	8.7	1.4 (0.7–2.5)	0.6 (0.1–4.3)	0.9 (0.4–1.8)	2.5 (0.8–7.3)	1.2 (0.7–1.9)	1.6 (0.6–4.1)
9. Alcohol intake >3 drinks/day*	12.4	0.2	0.7 (0.3–2.1)	0.0 (0.0–0.0)	1.0 (0.4–2.8)	0.0 (0.0–0.0)	0.9 (0.4–1.8)	0.0 (0.0–0.0)
Number of components comprising WHO-MetS other than diabetes (i.e., among 1a, 2a, 5, and 6)								
0	18.6	16.4	1.00	1.00	1.00	1.00	1.00	1.00
≥1 (vs. <1)	81.5	83.6	1.7 (0.7–4.5)	3.9 (0.5–28.4)	1.0 (0.4–2.5)	2.3 (0.5–9.7)	1.2 (0.7–2.4)	2.8 (0.9–9.0)
≥2 (vs. <2; i.e., WHO-MetS)	51.2	52.5	1.3 (0.7–2.4)	2.8 (1.0–7.9)	2.0 (0.9–4.1)	3.7 (1.4–9.9)	1.6 (1.0–2.6)	3.2 (1.6–6.5)
≥3 (vs. <3)	21.8	20.7	1.8 (0.9–3.5)	1.3 (0.5–3.7)	2.1 (1.0–4.4)	1.1 (0.4–2.7)	1.9 (1.2–3.2)	1.2 (0.6–2.4)
Number of components comprising NCEP-MetS other than diabetes (i.e. among 1b, 2b, 3, and 4)								
0	20.1	21.6	1.00	1.00	1.00	1.00	1.00	1.00
≥1 (vs. <1)	79.9	78.4	1.9 (0.7–4.9)	1.6 (0.4–5.6)	1.0 (0.4–2.2)	6.4 (0.9–46.7)	1.3 (0.7–2.4)	2.7 (0.9–7.7)
≥2 (vs. <2; i.e., NCEP-MetS)	45.0	38.0	1.9 (1.0–3.6)	1.7 (0.7–4.0)	1.4 (0.7–2.8)	1.3 (0.6–2.8)	1.8 (1.1–2.8)	1.4 (0.8–2.5)
≥3 (vs. <3)	14.5	11.5	2.5 (1.3–4.9)	0.9 (0.2–3.7)	0.9 (0.3–2.4)	0.3 (0.0–2.2)	1.8 (1.0–3.2)	0.5 (0.2–1.7)

Data are percent or hazard ratios (95% CIs) and are grouped according to individual and combined cardiovascular risk factors mostly comprising the metabolic syndrome as defined by the World Health Organization or the National Cholesterol Education Program. *Equivalent to 38 g ethanol/day. DBP, diastolic blood pressure; SBP, systolic blood pressure; WHR, waist-to-hip ratio.

tion being a significant predictor for stroke, whereas 130/85 mmHg in the NCEP definition is not.

The strengths of our study were that *1*) it is the first prospective study to determine the predictive value of MetS on CVD in Asian subjects, *2*) the two most widely used definitions of MetS were applied to the same cohort for the evaluation of their clinical usefulness, and *3*) the follow-up was mainly carried out in university or large general hospitals, which facilitated the reliable assessment of follow-up data and event diagnosis/records. Nevertheless, we acknowledge that the study had certain limitations: *1*) Our study subjects were hospital-based patients with diabetes of a relatively long duration; therefore, we cannot make inferences beyond a similar group. *2*) We analyzed both intervention (lifestyle modification through diabetes self-management care) and control (continuance of conventional care) groups of the JDCS together, although mild intervention produced only limited differences in glycemic control (0.1–0.2% in HbA_{1c}) as well as a lack of significant differences in known classical cardiovascular risk factors, as previously reported (38). *3*) We did not consider medication use in the diagnosis of MetS in this study. *4*) Mortality was not analyzed because we did not have sufficient occurrences at this stage of the study.

In conclusion, we found a high prevalence of MetS among diabetic patients with no history of CVD. For Japanese female patients with type 2 diabetes, WHO-MetS but not NCEP-MetS was predictive for CVD. In male patients, although both WHO-MetS and NCEP-MetS were somewhat predictive for CVD, hyperlipidemia or hypertension had equivalent or higher HRs for CVD and seemed to be sufficient for the prediction of CVD. We suggest that the commonly used definitions of MetS, at least in their present forms, have limited clinical usefulness for Asian diabetic patients and may need some ethnic group–specific modifications for global use.

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APPENDIX

The Japan Diabetes Complications Study (JDCS) Group

Primary investigator: Nobuhio Yamada (University of Tsukuba)

Chief of Assessment Committee: Yasuo Akanuma (Institute for Adult Diseases Asahi Life Foundation)

Committee members: Keita Ato, Masaaki Eto, Hiroshi Ito (Asahikawa Medical College); Azuma Kanatsuka, Naotake Hashimoto, Yasushi Saito, Kazuo Takahashi, Kazuo Yagi (Chiba University); Tadami Takekoshi, Takanobu Wakasugi (Fukui Prefectural Hospital); Shigetake Toyooka (Fukui Red Cross Hospital); Yukihiro Bando (Fukui Saiseikai Hospital); Tsugihiko Nakai, Koji Oida, Jinya Suzuki (Fukui University); Yasuaki Fukumoto, Seiichi Sumi (Garatia Hostiptal); Genshi Egusa, Rumi Fujikawa, Masamichi Okubo, Kiminori Yamane (Hiroshima University); Takao Koike, Narihito Yoshioka (Hokkaido University); Motonobu Anai, Ritsuko Honda, Masatoshi Kikuchi (Institute for Adult Diseases Asahi Life Foundation); Shun Ishibashi (Jichi Medical School); Masanobu Kawakami, Kazuyuki Namai (Jichi Medical School Omiya Medical Center); Takashi Sasaki, Masami Nemoto (Jikei University); Ryuzo Kawamori, Yasushi Tanaka (Juntendo University); Toshihiko Ishida (Kagawa University); Izumi Takei (Keio University); Yoshikuni Fujita, Keiji Tanaka, Yoshihiro Yajima (Kitazato University); Hideki Kishikawa, Tetsushi Toyonaga (Kumamoto University); Shingo Komichi, Zenji Makita, Kyohei Nonaka, Kentaro Yamada (Kurume University); Naoto Nakamura, Koji Nakano (Kyoto Prefectural University of Medicine); Toyoshi Iguchi, Hajime Nawata (Kyushu University); Yasuhisa Matsushima (Matsudo City Hospital); Hideo Takahashi (Minami Akatsuka Clinic); Hiroyuki Toyoshima (Minoh City Hospital); Shoichi Akazawa, Eiji Kawasaki, Shigenobu Nagataki (Nagasaki University); Nigishi Hotta, Jiro Nakamura (Nagoya University); Kentaro Doi, Yu Harano, Yasunao Yoshimasa (National Cardiovascular Center); Yoichi Hayashi (Nihon University); Shinichi Oikawa (Nippon Medical School); Ryuzo Abe, Hiroaki Seino, Daishiro Yamada (Ohta-Nishinouchi Hospital); Mitsuru Hoshi, Takao Watarai (Osaka Koseinenkin Hospital); Masatoshi Imaizumi, Ryohei Todo (Osaka National Hospital); Keisuke Kosugi, Yasuhisa Shimizu, Yutaka Umayahara (Osaka Police Hospital); Junichiro Miyagawa, Mitsuyoshi Namba, Kaoru Takemura, Yoshimitsu Yamasaki (Osaka University); Kazuhiro Hosokawa, Kempei Matsuoka (Saiseikai Central Hospital); Junko Nakano, Hirotaka Umezu (Saiseikai Fukushima General Hospital); Akihiko Hoshino, Toshihiko Nishiyama, Tetsushi Nogami (Saisekai Kumamoto Hospital); Hideo Nunome (Saiseikai Mito Hospital); Shigehiro Katayama, Atsuhito Togashi (Saitama Medical College); Kenichi Yamada (Sakura National Hospital); Atsunori Kashiwagi, Yoshihiko Nishio (Shiga University of Medical Science); Yukio Yoshimura (Shikoku University); Tatsuhide Inoue (Shizuoka General Hospital); Masafumi Kitaoka (Showa General Hospital); Toshio Kitada, Akio Shirai, Ryoichiro Watanabe (Takeda General Hospital); Takaichi Miyagawa (Tama Minami Clinic); Yoshikazu Sakamoto, Osamu Mokuta, Ryo Okazaki (Teikyo Universiy Ichihara Hospital); Kazuma Takahashi (Tohoku University); Koji Shirai, Hiroshi Miyashita (Toho University Sakura Hospital); Akira Tanaka (Tokyo Medical and Dental University); Yoshiaki Fujita (Tokyo Metropolitan Institute of Gerontology); Hideki Ito (Tama-Hokubu Medical Center) Reiko Kawahara, Yasue Omori, Asako Sato (Tokyo Women's Medical University); Toshio Murase, Mitsuhiko Noda, Masato Odawara (Toranomon Hospital); Masashi Kobayashi, Masaharu Urakaze (Toyama Medical and Pharmaceutical University); Hitomi Fujii, Satoshi Iimuro, Takashi Kadowaki, Sachiko Mizuno, Yasuo Ohashi, Junichi Osuga, Yasuyoshi Ouchi, Akane Takahashi (University of Tokyo); Hirohito Sone, Kamejiro Yamashita (University of Tsukuba); Ryo Kawasaki, Hidetoshi Yamashita (Yamagata University); Hisahiko Sekihara, Yasumichi Mori (Yokohama City University); Tetsuo Nishikawa (Yokohama Rosai Hospital); Hiroto Furuta, Kishio Nanjo (Wakayama Medical University).

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variable. In the largest Japanese series of patients with left ventricular apical ballooning, 90 percent of the patients had ST-segment elevation.[2] This finding is in contrast to the less frequent observation of ST-segment elevation in our experience and that reported by others.[3,4]

We thank Dr. Kadhiravan for highlighting the clinical features of scorpion envenomation. Many of the cardiac abnormalities seen with this catecholamine-mediated disorder are similar to those observed with stress cardiomyopathy. The observation that prazosin may reduce the risk of pulmonary edema and death after a scorpion sting suggests that adrenergic blockade may indeed have a role in the management of stress cardiomyopathy.

Ilan S. Wittstein, M.D.
Hunter C. Champion, M.D., Ph.D.
Johns Hopkins University School of Medicine
Baltimore, MD 21287
iwittste@jhmi.edu

1. Bybee KA, Prasad A, Barsness GW, et al. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. Am J Cardiol 2004;94:343-6.
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Vascular Risk Factors and Diabetic Neuropathy

TO THE EDITOR: In the article by Tesfaye et al. (Jan. 27 issue)[1] on the European Diabetes (EURODIAB) Prospective Complications Study, which looked at modifiable risk factors for diabetic neuropathy, the first line of the abstract states, "Other than glycemic control, there are no treatments for diabetic neuropathy." Although there have been no prospective, randomized trials to date, there is a growing literature regarding the role of nerve decompression in carefully selected patients with diabetic distal, large-fiber, symmetric polyneuropathy.

A recent analysis of 50 patients with diabetes who underwent decompression of the tibial and peroneal nerves in one leg and not the other showed that no ulcers or amputations occurred in the leg that had been operated on, whereas in 15 patients, there were 12 ulcers and three amputations in the unoperated leg, with an average follow-up of 4.5 years ($P<0.001$).[2] A recent review that included our series of 25 patients showed that "in properly selected patients, surgical releases can decrease pain and improve sensation."[3]

Gedge D. Rosson, M.D.
A. Lee Dellon, M.D.
Johns Hopkins University School of Medicine
Baltimore, MD 21287
gedge@jhmi.edu

1. Tesfaye S, Chaturvedi N, Eaton SEM, et al. Vascular risk factors and diabetic neuropathy. N Engl J Med 2005;352:341-50.
2. Aszmann O, Tassler PL, Dellon AL. Changing the natural history of diabetic neuropathy: incidence of ulcer/amputation in the contralateral limb of patients with a unilateral nerve decompression procedure. Ann Plast Surg 2004;53:517-22.
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TO THE EDITOR: Tesfaye et al. report that the incidence of diabetic neuropathy is associated with a history of cardiovascular disease at baseline and with cardiovascular risk factors. Additional information might further elucidate their important findings.

About 35 percent of the patients who qualified for follow-up either did not reach follow-up or were not assessed for neuropathy. Although the authors compared baseline data between patients who were assessed and those who were not, data on two of the major risk factors they identified (i.e., cardiovascular disease and smoking) were not included in that comparison. This raises the question of whether those data were collected only retrospectively, at the time of assessment for neuropathy, with a possibility of recall bias. If that is not the case, it is important to know whether there was differential loss to follow-up with regard to these factors, which could potentially compromise validity. Additional information on the role of variables known to be associated both with vascular risk factors and glycemic control and with the risk of diabetic complications — specifically, socioeconomic status,[1,2] ethnic group,[3] and the presence or absence of depression[4] — would be instructive.

Raz Gross, M.D., M.P.H.
Columbia University Medical Center
New York, NY 10032
rg547@columbia.edu

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TO THE EDITOR: Tesfaye et al. report that, apart from glycemic control, the incidence of neuropathy in a European cohort of patients with type 1 diabetes was associated with potentially modifiable cardiovascular risk factors, including a raised triglyceride level, a high body-mass index, smoking, and hypertension. We analyzed the database of subjects with type 2 diabetes who had participated in our Japan Diabetes Complications Study[1-3] for factors that were associated with neuropathy, defined as either the lack of an ankle tendon reflex or the development of abnormal sensation. During the seven-year study, neuropathy developed in 332 of 1618 patients. Odds ratios were calculated with the use of most of the variables included in the study by Tesfaye et al., and we similarly controlled for the glycosylated hemoglobin level and the duration of diabetes. Our data indicate that only obesity was a risk factor for neuropathy in our Japanese subjects (Table 1). These results support the notion that risk factors for diabetic neuropathy vary depending on the ethnic group of patients and on the type of diabetes.

Hirohito Sone, M.D., Ph.D.
University of Tsukuba
Tsukuba 305-8575, Japan
Sachiko Mizuno, Ph.D.
University of Tokyo
Tokyo 113-0033, Japan
Nobuhiro Yamada, M.D., Ph.D.
University of Tsukuba
Tsukuba 305-8575, Japan
jdcstudy@mc.tsukuba.ac.jp

Table 1. Risk Factors for Neuropathy after Adjustment for Glycosylated Hemoglobin and Duration of Diabetes in 1618 Japanese Patients with Type 2 Diabetes.*

Variable	Unit Increase	Odds Ratio (95% CI)	P Value
Body-mass index	1	1.10 (1.00–1.21)	0.051
Waist-to-hip ratio	0.1	1.65 (1.14–2.39)	0.008
Systolic blood pressure	10 mm Hg	1.01 (0.84–1.21)	0.93
Diastolic blood pressure	10 mm Hg	1.11 (0.82–1.51)	0.50
Low-density lipoprotein cholesterol	10 mg/dl	0.99 (0.90–1.09)	0.82
High-density lipoprotein cholesterol	10 mg/dl	0.95 (0.79–1.14)	0.57
Triglycerides	10 mg/dl	1.02 (0.98–1.05)	0.37
Lp(a) lipoprotein	1 mg/dl†	0.86 (0.63–1.19)	0.36
Urinary albumin excretion	10 mg/g of creatinine†	0.99 (0.76–1.29)	0.95
Fasting plasma insulin	5 μU/ml‡	1.10 (0.88–1.38)	0.40
Insulin use		1.47 (0.75–2.88)	0.26
Smoking at baseline		1.31 (0.69–2.48)	0.41
Excessive alcohol intake at baseline§		1.43 (0.51–4.03)	0.50

* The patients included 877 men and 741 women (mean [±SD] age, 58.4±7.4 years). Each risk factor was evaluated individually by discrete-type Cox regression. CI denotes confidence interval.
† Values were log-transformed.
‡ Patients receiving insulin therapy were excluded from this analysis.
§ Excessive intake was defined as more than 3 drinks (approximately 38 g of ethanol) per day.

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2. Sone H, Ito I, Ohashi Y, Akanuma Y, Yamada N. Obesity and type 2 diabetes in Japanese patients. Lancet 2003;361:85.
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THE AUTHORS REPLY: Dr. Gross comments on the completeness of information and follow-up. None of the data in the EURODIAB Prospective Complications Study were collected retrospectively. Recall bias is therefore not an issue. When we compare the presence of cardiovascular disease and smoking at baseline between patients with and those without a full neuropathy assessment at follow-up, we find no significant difference in the prevalence of cardiovascular disease at baseline. There were 6 percent more smokers among those who did not have a neuropathy assessment at follow-up. A possible consequence of the association between patients who were lost to follow-up and a slightly higher cardiovascular risk profile might be that our results underestimate the true incidence of neuropathy. However, loss of patients to follow-up is unlikely to bias observed associations between risk factors and disease, because a situation in which a poor cardiovascular risk profile reduces the risk of neuropathy in those lost to follow-up and increases the risk in those returning for follow-up is unlikely. All study

centers were instructed not to include subjects from ethnic minority groups, since this would have generated small but extremely heterogeneous subgroups. The population of the EURODIAB Prospective Complications Study can thus be regarded as white European. Because of the varying situations in the 15 countries from which participants were recruited, it was difficult to assess socioeconomic status. Information was gathered concerning higher education; however, adjustment for this variable did not affect our findings. Information on depression was not collected, although we think that depression may be more a consequence of rather than a cause of diabetic complications.

We are aware of surgical decompression of peripheral nerves as a possible treatment for diabetic distal symmetric polyneuropathy. However, the review cited in the letter by Drs. Rosson and Dellon concludes that "the role that this surgery can play remains controversial."[1] Thus, this promising form of treatment will need to be confirmed in a randomized controlled trial involving several centers.

Solomon Tesfaye, M.D.

Royal Hallamshire Hospital
Sheffield S10 2JF, United Kingdom
solomon.tesfaye@sth.nhs.uk

Daniel R. Witte, Ph.D.
John H. Fuller, M.A.

Royal Free and University College Medical School
London WC1E 6BT, United Kingdom

1. Biddinger KR, Amend KJ. The role of surgical decompression for diabetic neuropathy. Foot Ankle Clin 2004;9:239-54.

Health Care in the 21st Century

TO THE EDITOR: In his Shattuck Lecture on health care in the 21st century (Jan. 20 issue),[1] William Frist takes pride in the "tough but wise dec isions" by America's leaders that "unleashed the creative power of the competitively driven marketplace," promising to bring "lower costs, higher quality, greater efficiency, and better access to care" by 2015. Frist's confidence seems strangely misplaced, since it is precisely this marketplace model that has brought us our currently escalating health care costs, shrinking access to care, and mounting dissatisfaction on the part of patients and the medical profession alike. We in the United States are unique in the world in regarding health care as a commodity, and as a consequence, we spend more for health care than any other country does. At the same time, the United States ranks 37th in quality of service, according to the World Health Organization, and 27th in rates of infant mortality, and our life expectancy is shorter than that in several European countries that spend far less on health care. This is hardly a record that merits such praise and confidence in our system. Rather, it is one that should call into question the industrial model that, Frist maintains, holds such promise for our future health care.

Victor Gurewich, M.D.

Beth Israel Deaconess Medical Center
Boston, MA 02215
vgurewic@bidmc.harvard.edu

1. Frist WH. Shattuck Lecture health care in the 21st century. N Engl J Med 2005;352:267-72.

TO THE EDITOR: Dr. Frist's vision of health care in the 21st century is imaginative but risky. His reliance on "consumer-driven health care" threatens to exacerbate the inequalities and inefficiencies in U.S. health care. His call for "affordable health coverage for all Americans" does not equal affordable health care for all Americans. High-deductible insurance with health savings accounts is less expensive than traditional coverage, but the financial barriers with this approach discourage primary and preventive care.[1] If his fictitious patient, Mr. Rogers, had a low or moderate income, he might question, while having chest pain, whether he could afford "nanocath" laboratory services.

Instead, everyone should have an inviting medical home[2,3] where they can get the care they need. We should make high-quality information more available to patients, but the burden of reducing costs should be focused on policy makers, hospitals, and clinicians through a realignment of care incentives. We encourage Congress to follow the recent recommendations of the Institute of Medicine by "taking action to achieve universal health insurance . . . with enactment by 2010."[4]

Jeffrey Huebner, M.D.

University of Washington
Seattle, WA 98105
eino@u.washington.edu

Kenneth Frisof, M.D.

Case Western Reserve University
Cleveland, OH 44106

Appropriate body-mass index for Asians

Sir—Vivien Choo (July 20, p 235)[1] reports WHO's recommendation for a narrower body-mass index (BMI) range (18·5–23·0 kg/m²) for Asians, based on data from body-fat percentage and BMI from ten Asian countries. We have completed a study of cardiovascular risk factors in a community of Indian migrants living in the UK (n=242) and their contemporaries in rural India (305). Using standardised methods in age-matched and sex-matched cohorts, we found that the rates of obesity were different between the two populations.

BMI was greater in Indian migrants (26·3 kg/m² [95% CI 25·8–26·9]) than their rural contemporaries (21·1 kg/m² [20·6–21·5]). Only 24% of Indian men and 21% of Indian women living in the UK were within the suggested Asian BMI range. Migrant men aged 25–44 years were also significantly taller than their rural counterparts ($p<0·05$). Hence, migration is an important consideration that needs to be taken into account before an Asian BMI range is accepted. Furthermore, we do not think it appropriate to pool people from different Asian countries under a single Asian category. India alone is home to people of Indo-Aryan, Dravidian, and Mongolian origin,[2] and anthropometric features are likely to differ between them.

J K Patel, E A Hughes, M I Mackness, A Vyas, J K Cruickshank

*University Department of Medicine (JP, MM) and Clinical Epidemiology Unit (AV, JC), University of Manchester, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK; Sandwell and West Birmingham Hospitals NHS Trust, Birmingham, UK (EH)
(e-mail: jeeteshpatel@SWBH.nhs.uk)

1 Choo V. WHO reassesses appropriate body-mass index for Asian populations. *Lancet* 2002; **360:** 235.
2 Padmavati S. Epidemiology of cardiovascular disease in India II: ischemic heart disease. *Circulation* 1962; **25:** 711–17.

Obesity and type 2 diabetes in Japanese patients

Sir—The incidence and morbidity of type 2 diabetes mellitus is known to be higher in obese individuals, and is especially high in ethnic groups with high body-mass indices (BMI), such as Pacific Islanders and Pima Indians. However, few studies have compared the BMIs of type 2 diabetic patients from different ethnic groups, and this scarcity perhaps reflects the difficulties in obtaining large matched-sample cohorts from different ethnic groups.

Comparison of data from two prospective studies (table) reveals a striking difference in the average BMI of type 2 diabetic patients from two different ethnic populations—white individuals from the UK Prospective Diabetes Study (UKPDS)[1] and Japanese patients from the Japan Diabetes Complication Study (JDCS).[2] Both groups were similar in terms of numbers of patients, age, glycohaemoglobin A_{1c} concentration, and disease duration. However, the BMI of white diabetic patients was much higher than that of the Japanese patients. Moreover, whereas the BMI of white diabetic patients was higher than that reported for non-diabetics of the same ethnic origin,[3] the BMI of Japanese diabetic patients was normal compared with that of the Japanese non-diabetic population.[4] The average BMI of the white UKPDS patients continued to increase during the 10 years after diagnosis, and although we do not have retrospective BMI data from the time of diagnosis in the JDCS cohort, there has been no significant increase in the average BMI over the 6 years of the study.

The origin of this large difference in BMI is unknown, but it might reflect differences in insulin secretion and sensitivity between the two ethnic groups. Unfortunately, this hypothesis is difficult to prove because plasma insulin concentrations were measured only at baseline in the UKPDS, and are not comparable with the subsequent insulin measurements taken in the JDCS. In general, diabetic patients who are obese have greater insulin secretion and lower insulin sensitivity (ie, insulin resistance) than non-obese diabetics, and the white population is regarded as more obese and insulin resistant than the east Asian population.[5]

Notwithstanding the lack of comparative insulin data, the higher systolic blood pressure and triglyceride concentrations in the UKPDS patients compared with those in the JDCS patients (table) could imply that they have insulin resistance syndrome, which is characterised by the accumulation of multiple cardiovascular risk factors. The lower fasting plasma glucose concentrations in the face of higher glycohaemoglobin concentrations in the UKPDS patients could suggest a more severe postprandial hyperglycaemia than that in the JDCS patients; postprandial hyperglycaemia is also known to be an independent risk factor for cardiovascular disease. This association might partly explain the higher incidence of cardiovascular complications in white diabetics than in east Asian diabetics.

	UKPDS (n=2015)	JDCS (n=2205)
Age (years)	62	59
Diabetes duration (years)	9	11
Blood pressure (mm Hg)	140/80	132/77
Fasting plasma glucose (mmol/L)	8·14	8·75
Glycohaemoglobin A_{1c} (%)	7·9	7·7
Total cholesterol (mmol/L)	5·3	5·2
Triglycerides (mmol/L)	1·53	1·40
BMI (kg/m²)	29·4	23·1
Mean BMI of whole population	24·1	22·7

Comparison of mean baseline characteristics from the Japan Diabetes Complication Study (JDCS) and averaged year-9 data for white individuals from the UK Prospective Diabetes Study (UKPDS)

*Hirohito Sone, Hideki Ito, Yasuo Ohashi, Yasuo Akanuma, *Nobuhiro Yamada, for the Japan Diabetes Complication Study Group*

Department of Internal Medicine, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan
(e-mail: jdcstudy@md.tsukuba.ac.jp)

1 Davis TM, Cull CA, Holman RR. Relationship between ethnicity and glycemic control, lipid profiles, and blood pressure during the first 9 years of type 2 diabetes: UK Prospective Diabetes Study (UKPDS 55). *Diabetes Care* 2001; **24:** 1167–74.
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Reversible ocular myasthenia gravis or mitochondrial myopathy from statins?

Sir—Ever-increasing millions of people are being treated to lower their cholesterol concentration. In a Correspondence letter, Parmar and colleagues (Aug 31, p 717)[1] report a patient who developed "ocular myasthenia" of 2 years' duration, associated with proximal limb-muscle weakness, induced by treatment with each of three statins and by a fibrate. To determine the mechanism of this

showing areas at risk of drought or rainfall does not include that country.

A further point I would like to make is that the Review included only articles that involved human infectious diseases. Having worked in an emergency team in a community destroyed by the floods provoked by El Niño, I know that the most severe and immediate problems associated with the phenomenon, as with any other disasters, are the issues related to mental health and reconstruction.

J Jaime Miranda

International Health and Medical Education Centre, University College London, London N19 5LW, UK
(e-mail: j.miranda@ucl.ac.uk)

1 Kovats RS, Bouman MJ, Hajat S, Worrall E, Haines A. El Niño and health. *Lancet* 2003; **362:** 1481–89.

The hidden patient

Sir—I wonder whether general medicine might take a leaf out of the psychiatric model of care in its approach to the wellbeing of carers—the "hidden patients" (Nov 15, p 1682).[1] As a matter of course, when assessing a patient with a significant disease, the carer of the patient is automatically offered a carer's assessment. The result is that services are extended not only to the patient but also to the patient's social unit as a whole. Additionally, with the care programme approach, it is not infrequently the case that the carer is care programmed.

The benefits of these procedures are numerous and include issues such as illness education, medication concordance improvements, explanation of prognostic expectations, and various other psychosocial and practical inputs. Although it involves a lot of work, the payoff more than rewards the effort. Carers become virtual members of the multidisciplinary team, and their contribution to care plans is valued and respected. Relapses are spotted earlier and admissions become less frequent as carers become more adept at managing problems and knowing whom, where, and when to call for assistance. Further advantages are seen in carers' involvement in governmental and non-governmental bodies, and their provision of a voice for those who have difficulty in making themselves heard. The tragedy of the hidden patient can be avoided.

Andrew Al-Adwani

Department of Psychiatry, Scunthorpe General Hospital, Scunthorpe DN15 7BH, UK
(e-mail: andrew.al-adwani@dsh.nhs.uk)

1 Hill J. The hidden patient. *Lancet* 2003; **362:** 1682.

Energy intake and obesity in Japanese patients with type 2 diabetes

Sir—Obesity is known to be one of the most important risk factors for the development and deterioration of type 2 diabetes. Nevertheless, we have previously revealed a discrepancy in body-mass indices (BMI) between white and Japanese patients with type 2 diabetes (about 29 kg/m^2 in white patients from the UK Prospective Diabetes Study [UKPDS] *vs* 23 kg/m^2 in Japanese patients from the Japan Diabetes Complications Study [JDCS]) whose other characteristics were very similar.[1] Moreover, by contrast with white patients with type 2 diabetes who have a higher BMI than does the white population as a whole (about 24 kg/m^2), the BMI of Japanese patients is similar to that of the general Japanese population, indicating that Japanese patients with type 2 diabetes are not obese, at least on average.[1]

We recently completed the baseline nutrition analysis of our JDCS patients. Comparing our results with those of the UKPDS,[2] we were surprised to find that the mean daily energy intake of both cohorts was almost the same despite the large differences in BMI and bodyweight (table). In other words, the UKPDS patients developed obesity with a relatively lower energy intake than the JDCS patients, considering the mean height difference between the groups. Accordingly, the daily energy intake per unit of bodyweight was 22% lower in the UKPDS patients than in the JDCS patients. Moreover, 19% of male (27% of female) patients in our cohort overate, taking more than 35 kcal per ideal bodyweight daily. Of those patients, 20% of men (29% of women) had a BMI greater than 25 kg/m^2, which is regarded as being overweight. Accordingly, only 3·8% of male and 7·8% of female patients in the JDCS study population had obesity associated with actual overeating.

This finding runs contrary to the conventional wisdom that the major pathophysiological background to type 2 diabetes is insulin resistance and obesity associated, at least to some extent, with excessive energy intake.

Obesity is known to have preceded and triggered the explosive increase in diabetes among Pacific Islanders and Pima Indians.[3] In Japan, however, despite the lack of a major increase in mean BMI and a decrease in mean total dietary intake since the 1970s, the prevalence of type 2 diabetes is now very high: a sixth of the adult population in Japan had known or strongly suspected diabetes in the most recent national survey. This proportion is much higher than in the European population,[3] and the prevalence is still increasing. The results of recent large-scale epidemiological surveys also suggested that a disturbance of insulin secretion rather than insulin resistance was strongly associated with the development of the disease in Japanese and Chinese patients, unlike in the European population.[4]

Risk factors other than obesity and insulin resistance seem to affect the development of type 2 diabetes in Japanese people. Additionally, even relatively mild obesity could have a major effect on the pathogenesis of diabetes in the Japanese population.[5]

*Hirohito Sone, Yukio Yoshimura, Hideki Ito, Yasuo Ohashi, *Nobuhiro Yamada, for the Japan Diabetes Complications Study Group*

Department of Internal Medicine, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, 305-8575 Ibaraki, Japan
(e-mail: jdcstudy@md.tsukuba.ac.jp)

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	JDCS	UKPDS
Patients (men/women)	1076 (576/500)	108 (61/47)
Age (years)	59·4 (7·4)	55·1 (7·7)
Bodyweight (kg)	58·6 (10·1)	78·2 (12·2)
Body-mass index (kg/m^2)	23·1 (3·0)	27·9 (4·3)
Glycohaemoglobin A1C (%)	7·7 (1·4)	7·1 (1·5)
Total energy intake (kcal/day)	1580 (398)	1650 (424)
Men	1778 (428)	1797 (63)*
Women	1598 (390)	1439 (44)*
Total energy intake per kg weight (kcal/kg daily, mean)	27·0	21·1

Values are mean (SD) unless otherwise indicated. *SE.

Total energy intake and other characteristics of patients with type 2 diabetes from Japan Diabetes Complications Study (JDCS) and UK Prospective Diabetes Study (UKPDS)

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Underestimation of allergies in elderly patients

Sir—The incidence of immediate (type 1) allergy is increasing worldwide. We did a MEDLINE search using the terms "epidemiology and allergy" and found many references covering children, fewer in adults, and hardly any in elderly people.

Our findings show that there is a lack of research on the incidence and prevalence of allergic sensitisation in the non-paediatric population. However, the few studies that have been done suggest that allergies in elderly people are not rare. In the 1980s, Wüthrich and colleagues[1] did a study in an unselected Swiss population, which showed that more than 4% still had atopic diseases after their 60th birthday. We have described a case of newly developed allergic conjunctivitis in a woman of 70 years,[2] and Huss and colleagues[3] found 74·7% positive test results to a type 1 allergy skin test battery in 80 elderly patients.

Because of changing world demographics, it is estimated that the elderly population will increase by 75% between 2010 and 2030.[4] Hence, an ever-growing proportion of allergy patients will be from this population, and it will become increasingly important not to miss the not-so-rare differential diagnosis of allergy, especially in patients with rather mild symptoms such as conjunctivitis or rhinitis.

Stefan Wöhrl, George Stingl

Division of Immunology, Allergy and Infectious Diseases, Department of Dermatology, University of Vienna Medical School, A-1090 Vienna, Austria
(e-mail: stefan.woehrl@akh-wien.ac.at)

1 Wüthrich B, Schnyder UW, Henauer SA, Heller A. Häufigkeit der Pollinosis in der Schweiz: Ergebnisse einer repräsentativen demoskopischen Umfrage unter Berücksichtigung anderer allergischer Erkrankungen. *Schweiz Med Wochenschr* 1986; **116:** 909–17.
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Lyme borreliosis

Sir—In their review on Lyme borreliosis (Nov 15, p 1639),[1] Gerold Stanek and Franc Strle do not mention the organism's association with cutaneous B-cell lymphomas (CBCLs). Myself[2] and others[3] have detected the presence of *Borrelia burgdorferi* DNA within skin lesions of various types of CBCL in endemic and non-endemic regions, proving that this micro-organism can be involved in the pathogenesis of malignant lymphomas of the skin. These studies revealed that about 20% of cases of CBCL harboured borrelial DNA. There might be some regional differences, however, since two studies on patients from the USA that used immunohistochemical and molecular techniques gave negative results.

The link between disorders associated with *B burgdorferi* and CBCLs was suggested more than 50 years ago on the basis of clinical findings (although at that time, of course, *B burgdorferi* was not yet known about), and similar observations have been made more recently on clinical or serological bases.[4] This association is conceptually and clinically similar to that seen in B-cell lymphomas of the stomach (mucosa-associated lymphoid tissue [MALT] lymphomas), a distinct proportion of which are associated with infection by *Helicobacter pylori*. In fact, in a manner similar to that observed in the MALT lymphomas of the stomach, complete regression of CBCL lesions has been seen after effective antibiotic treatment for *B burgdorferi*. In regions endemic for *B burgdorferi*, antibiotics should be considered among the first-line treatments for patients with low-grade CBCL.

Finally, I and others have shown that in patients with B-cell chronic lymphocytic leukaemia (B-CLL), *B burgdorferi* can trigger the development of specific infiltrates at sites that are typical for borrelial lymphocytoma (eg, the nipples and scrotum).[5] In the past, lesions of B-CLL arising at these sites were well known, and were described as "leukaemia lymphatica mamillae" in old textbooks. Development of specific infiltrates of B-CLL at sites of antigenic stimulation is well known, and post-herpetic specific skin manifestations are a typical example of it. The onset of leukaemic infiltrates at sites typical for lymphocytoma, and the detection of borrelial DNA within these skin lesions, confirms the hypothesis that many so-called specific cutaneous manifestations of B-CLL are in fact antigen-driven, and that neoplastic cells in patients with B-CLL are capable of responding to antigenic stimulation.

Borrelia spp are the cause of several cutaneous and extracutaneous disorders with protean clinical features, ranging from mild manifestations such as erythema migrans to potentially life-threatening disorders such as complete heart block. CBCLs should be included among the most important aspects of Lyme borreliosis, since they represent one of its most serious manifestations.

Lorenzo Cerroni

Department of Dermatology, University of Graz, Auenbruggerplatz 8, A-8036 Graz, Austria
(e-mail: lorenzo.cerroni@uni-graz.at)

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Sonography-guided injection of botulinum toxin in children with cerebral palsy

Sir—Botulinum toxins are used to treat spastic muscles and drooling in children with cerebral palsy. For anatomically correct application to the salivary glands, sonography is the standard procedure.[1] However, palpation of anatomical landmarks is assumed to be sufficiently accurate for precise injection into spastic muscles.

Chin and colleagues[2] have shown that needle placement by palpation in