Do Back Belts Prevent Back Injury?

To the Editor: Dr Wassell and colleagues[1] found that neither voluntary nor mandatory back belt use reduced incidence of back injury claims or low back pain. In their accompanying editorial, Drs Hadler and Carey[2] implied that there was nothing to be done prior to the onset of back pain among workers and only physical and psychological treatment afterward.

It is not yet known whether back belts give any mechanical external support. Weight lifters do use such belts, but whether they get any true support or just psychological benefit has not been determined. My observations of workers suggest that the belts are often loosely worn, like vests, and never properly buckled to give any mechanical support. Rarely are workers given any instruction about the reasons for the back belt or how to use it properly. There is also the psychological factor of looking like a weakling and undergoing teasing by fellow workers when wearing a back belt.

Many employers may fear that enforcing the mandatory wearing of back belts would disturb their relationship with employees. This would actually involve enforcing the proper wearing of back belts. Nevertheless, the use of a back belt may have some psychological value to workers by reminding them that there is a proper way to lift safely and to avoid injury. Nonetheless, there is often inadequate teaching about safe lifting, which contributes to costs borne by workers and employers.

Hadler and Carey state that "simply because a particular biomechanical challenge exacerbates back pain does not mean that such usage is the proximate or principal cause." This ignores the reality that without such biomechanical challenge the exacerbation would not have occurred. Thus, work-related injury is the proximate cause, although it may be superimposed on some preexisting weakness or mechanical deficiency in the spine. It is academic of Hadler and Carey to dismiss attempts at prevention or even physical treatment by stating "it is time to focus on the psychosocial elements of life, on and off the job, that render too much back pain so intolerable."

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1. Wassell JT, Gardner LI, Landsittel DP, Johnston JJ, Johnston JM. A prospective study of back belts for prevention of back pain and injury. *JAMA*. 2000;284:2727-2732.
2. Hadler NM, Carey TS. Back belts in the workplace. *JAMA*. 2000;284:2780-2781.

To the Editor: Dr Wassell and colleagues[1] report that back belt use was unassociated with either self-reported back pain or workers' compensation claims for back injury. This prospective cohort study included data on individual-level risk factors for back injury, which has not been measured in previous studies.[2,3]

Wassell et al failed to note, however, that the inclusion of these variables in the analysis had almost no impact on their results because these variables functioned as neither modifiers nor confounders of the relationship between back belt use and back injury. Injury history, job title, and store policy did not modify the relationship between use of back belts and back injuries. In addition, unadjusted and adjusted odds ratios for back pain were almost identical for all levels of belt-wearing frequency, demonstrating a striking absence of joint confounding. The same was true for the rate ratios for back injury compensation claims. Thus, it appears that the relationship between back belt use and back injury is subject to almost no modification or confounding by other risk factors for back injury. This observation is important for the design of future studies in this area.

The authors used multivariate Poisson and logistic regression models but fail to mention any use of the more humble, although still important, tools of stratified analyses. Conducting a full sequence of stratified analyses preparatory to the use of regression models is an essential component of any epidemiologic analysis.[4] In fact, the main analysis of the study could have been completed without recourse to any multivariate regression methods. A presentation that focused on the unadjusted rate ratios and odds ratios, accompanied by a full stratified analysis that demonstrated the absence of modification and confounding, would have yielded a greater richness of analytical detail, while still arriving at the same conclusion. Epidemiology as a whole needs to temper its zeal for regression models, which require extensive and sometimes unwarranted assumptions about the data and often add little insight beyond that gained from stratified analysis.[4]

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GUIDELINES FOR LETTERS. Letters discussing a recent *JAMA* article should be received within 4 weeks of the article's publication and should not exceed 400 words of text and 5 references. Letters reporting original research should not exceed 500 words and 6 references. All letters should include a word count. Letters must not duplicate other material published or submitted for publication. Letters will be published at the discretion of the editors as space permits and are subject to editing and abridgment. A signed statement for authorship criteria and responsibility, financial disclosure, copyright transfer, and acknowledgment is required for publication. Letters not meeting these specifications are generally not considered. Letters will not be returned unless specifically requested. Also see Instructions for Authors (January 3, 2001). Letters may be submitted by surface mail: Letters Editor, *JAMA*, 515 N State St, Chicago, IL 60610; e-mail: JAMA-letters@ama-assn.org; or fax (please also send a hard copy via surface mail): (312) 464-5824.

Letters Section Editors: Stephen J. Lurie, MD, PhD, Senior Editor; Jody W. Zylke, MD, Contributing Editor.

In Reply: We agree with Dr Dorinson and do not want to "dismiss attempts at prevention." However, our research shows that employers and employees should not depend on back belts for prevention. Instead, prevention efforts should be based on a comprehensive workplace evaluation of work tasks and the work environment to reduce the hazards of lifting. Whatever mechanical or psychological effects that back belts may have on workers, our study shows that these effects do not result in decreased incidence of back pain or back injury claims. We are now completing a study to assess biomechanical and physiological effects of back belts.

Dorinson has observed that many workers wear belts improperly. In our study, employees had training on proper lifting techniques and proper belt wearing when first hired. Although not reported in our article, belt wearing was validated through direct observation of 227 employees in 8 stores. Back belt use was observed in 77% of participants who reported wearing belts " usually every day" based on a short observation period on a single day. However, belt tightness was not measured. The observations were made as soon as could be arranged after the interview with a median of 16 days between interview and work observation.

Dorinson suggests that back belts might have different effects for people with "some preexisting weakness or mechanical deficiency in the spine." We adjusted for a "previous back injury" in our main analysis and also evaluated 2 subgroups of employees separately (both with and without a history of previous back injury) to determine any effects of belt wearing. Back belts provided no statistically significant protection from "biomechanical challenge" for either subgroup based on our study of back pain incidence and injury claim rates.

Dr Marshall points out that the unadjusted and adjusted odds ratios and rate ratios were "almost identical" for our evaluation of belt-wearing effects on back pain and injury. Our results do not imply that future studies can dispense with individual-level risk factor data, especially if conducted in different settings or among different populations. Prior to estimating regression results, we recognized many possibilities for effect modification and confounding. Women are less likely to report wearing belts "usually every day" and are more likely to report back pain; current smokers are more likely to report wearing belts "usually every day" and also have a higher rate of back injury claims. The complicated interrelationships among these variables requires a careful regression analysis for an unbiased evaluation of the effects of belts. We did indeed conduct "a full sequence of stratified analyses" and also diagnostic evaluation of the regression models. We chose to emphasize results for the main objectives of our study and the consistency of these results in subgroup, interaction, and other ancillary statistical analyses of data, all of which demonstrate the lack of effects of back belts on back pain and injury.

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In Reply: Dr Dorinson suggests that different types of belts or ways of wearing them might prevent disabling back pain. However, there are no substantive scientific data or theoretical rationale for these claims.[1] On the other hand, the prospective study by Dr Wassell and colleagues[2] indicates that a back belt policy does not prevent disabling back pain at work even for workers who are compliant.

We do not belittle the experience of backache, which is a common and recurring part of life.[3] It is so ubiquitous across cultures, lifestyles, age groups, and employment categories that Dorinson's putative "biomechanical challenge" must be unavoidable. The degree of pain and disability is a function of the individual's ability to cope, which is related to a number of other factors.[4] Adverse aspects of the psychosocial context in which an individual experiences symptoms are far more likely to influence coping than the physical demands of life within or outside the workplace.[5]

Furthermore, although a worker's back pain may not be job-related, it will nonetheless interfere with job-related activities. If such tasks are deemed to be harmful, then the backache becomes a compensable injury. This is more likely to occur if the employer does not accommodate workers with backache as they would those with other medical illnesses. The consequences of recourse and redress indemnified by Workers' Compensation insurance all too often include iatrogenic illness and recalcitrant disability.[4] That is why we concluded that "it is time to focus on the psychosocial elements of life, on and off the job, that render too much of back pain so intolerable that it is memorable and even incapacitating."

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Loss and the Duration of Grief

To the Editor: Dr von Gunten and colleagues[1] give important advice for physician communication during end-of-life care.

My only criticism of this article is in the section on loss and grief. Most bereavement programs support families for 1 year. These authors perpetuate this notion that normal grief lasts 6 to 12 months. I believe normal grief can go on well past that first year. In fact, I believe the second year is often more difficult because the emotions can be as strong as the first year but the bereaved person feels less comfortable in talking about his or her pain and loss. In that second year fewer friends and relatives talk about the loss or allow the bereaved person to do so. In addition, during that second year bereaved persons perceive the societal message that grief should be finished after 6 to 12 months and respond by keeping their pain more to themselves. Perhaps this is why the first resolution in 1 bereaved parent's "resolutions for bereaved parents" is "I will grieve as much and for as long as I feel like grieving, and I will not let others put a time table on my grief."[2] The newer models of grief are starting to criticize and reject this 1-year time frame.[3] I believe end-of-life care advocates also need to reject this time frame and that bereavement programs should be willing to extend support beyond this 12-month period.

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This letter was shown to Dr von Gunten and colleagues, who think Dr Davis makes excellent points.—ED.

RESEARCH LETTER

Rapid Loss of Insulin Secretion in a Patient With Fulminant Type 1 Diabetes Mellitus and Carbamazepine Hypersensitivity Syndrome

To the Editor: Type 1 diabetes mellitus (DM) is caused by severe insulin deficiency that typically results from autoimmune destruction of pancreatic β-cells, although in some cases (classified as "idiopathic") there is no evidence of autoimmunity.[1] Recently, a novel etiology of the disease has been described as nonautoimmune fulminant type 1 DM,[2] which involves abrupt onset of insulin-deficient hyperglycemia, high serum pancreatic enzyme concentrations, and neither diabetes-related autoantibodies nor evidence of insulitis in pancreatic biopsy specimens. We report a case of rapid-onset type 1 DM with diabetic ketoacidosis (DKA), for which the clinical course was precisely documented. Interestingly, the onset of diabetes coincided with development of hemolytic anemia due to carbamazepine-induced cold agglutinin disease.

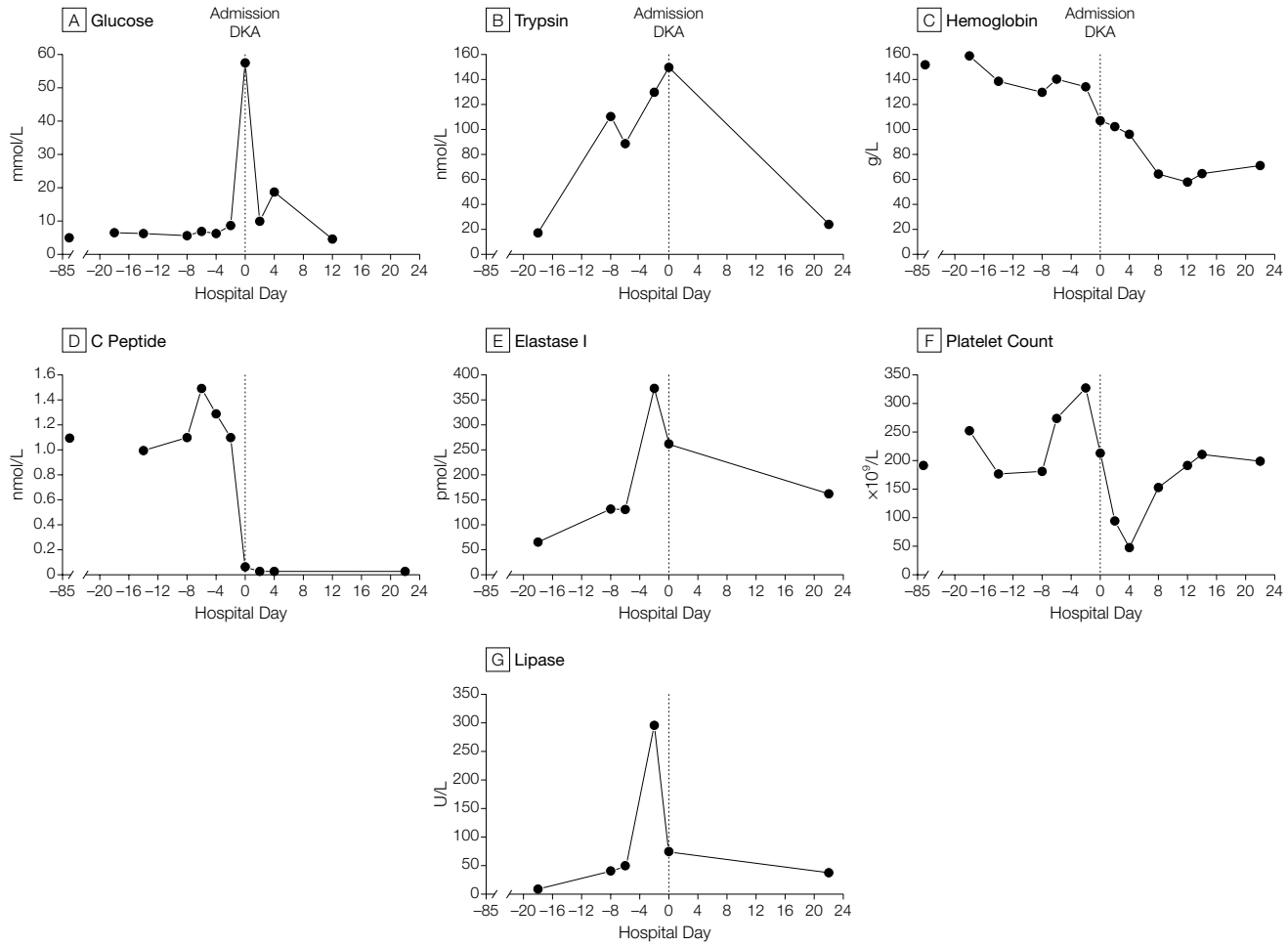
Report of a Case. A 77-year-old woman without history of alcohol use developed DKA after 2 weeks of inpatient treatment for allergic skin reactions. She had been treated with carbamazepine (200 mg/d) for 1 month for postherpetic neuralgia before developing generalized skin rash. Carbamazepine was discontinued at that time and prednisone, 20 mg per day, was administered. The patient had neither hyperglycemic symptoms nor abdominal pain until the day of discharge when she began to feel thirsty and complained of epigastric discomfort. The next morning she was found comatose and was transferred to our hospital, where she was found to be markedly dehydrated with systolic blood pressure of 80 mm Hg, diastolic blood pressure of 60 mm Hg, and body temperature of 92.1°F (33.4°C). Laboratory data showed severe hyperglycemic ketoacidosis (plasma glucose, 58.0 mmol/L [1045.0 mg/dL]; 3-hydroxyburytic acid, 7170 µmol/L [74.6 mg/dL]; arterial pH, 6.91; bicarbonate, 2.6 mmol/L), but normal glycosylated hemoglobin (5.9% of total hemoglobin) and glycated albumin (14.9% of total albumin) levels. The results of a pancreatic ultrasound were unremarkable. After recovery from coma with standard treatment for DKA, she required multiple insulin injections to control her blood glucose levels. She then developed anemia and thrombocytopenia, both of which spontaneously improved (FIGURE). Diagnosis of hemolytic anemia due to carbamazepine-induced cold agglutinin disease[3] was made on the basis of undetectable haptoglobin, autoagglutination on her blood film, an elevated cold agglutinin titer (1:128), and positive results on both lymphocyte stimulation test and patch testing using carbamazepine.

Analysis of conserved sera obtained during the patient's first hospitalization (Figure) revealed a rapid loss of C peptide leading to DKA within 2 days. There was a transient elevation of pancreatic enzyme levels, which preceded the loss of insulin secretion. The results of tests for islet cell and glutamic acid decarboxylase antibodies were both negative. Viral antibody titers examined in paired samples were low for the viruses that have been implicated in type 1 DM (Epstein-Barr virus, coxsackievirus B4, and cytomegalovirus). Human herpesvirus 6 (HHV-6) DNA was detected by polymerase chain reaction in the serum a week before the onset of DKA, and disappeared 1 month later, while HHV-6 antibodies were detected at both time points. At 6 months of follow-up, the patient's serum C peptide remained undetectable with no response to an intravenous glucagon load. She was found to have a type 1 DM–resistant human leukocyte antigen haplotype (DRB1*1501,DQA*10102,DQB1*0602/DRB1*0803,DQA1*0103,DQB1*0601).[4]

Comment. We have documented the onset of fulminant type 1 DM in a patient who showed a rapid loss of C peptide during the 2 days prior to onset of DKA. The time course of the pancreatic enzyme elevations suggested that the pancreatic injury led to complete β-cell destruction within several days. It is not clear whether reactivation of HHV-6, which can contribute to the development of drug-induced hypersensitivity syndrome,[5] could cause pancreatic injury leading to type 1 DM. Only 1 case report is available of pancreatitis associated with HHV-6 infection.[6] Other viruses might be involved, especially with the use of corticosteroids.

Another unique feature of this case is the simultaneous onset of type 1 DM and cold agglutinin disease, suggesting a causal

Figure. Clinical Course of the Changes in the Parameters for Type 1 Diabetes Mellitus, Hemolytic Anemia, Thrombocytopenia, and Pancreatic Enzyme Elevations in a Patient With Fulminant Type 1 Diabetes Mellitus and Carbamazepine Hypersensitivity Syndrome



DKA indicates diabetic ketoacidosis.

link or a common pathogenic mechanism underlying these 2 disorders. It is possible that an interaction between reactivation of HHV-6 and carbamazepine exposure caused a drug hypersensitivity reaction, somehow resulting in both the fulminant-onset DM and hemolytic anemia.

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