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Bouter, L.M.; Knipschild, P.G.

published in

Physician and Sportsmedicine
1989

document version

Publisher's PDF, also known as Version of record

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citation for published version (APA)

Bouter, L. M., & Knipschild, P. G. (1989). Causes and Prevention of Injury in Downhill Skiing. *Physician and Sportsmedicine*, 17(11), 81.

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Causes and Prevention of Injury in Downhill Skiing

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In brief: This literature review summarizes the current state of knowledge regarding the causes of injury in downhill skiing and discusses the incidence, distribution, risk factors, and prevention of injuries. The incidence of serious injury ranges from two to four per 1,000 ski days. About 55% of all downhill ski injuries involve the lower extremity; many of these are equipment-related. Potential risk factors fall into categories including personal characteristics, skill level, physical condition, behavior, equipment, and environment. Beginners are at higher risk, but taking lessons probably lowers the risk to some extent. Properly adjusted ski bindings undoubtedly can further lower the incidence of lower extremity injury. Similarly, improvements in the design of boots, bindings, and poles can probably make skiing safer.

Downhill skiing has become enormously popular over the last few decades. Today there are at least 25 to 30 million active skiers around the world.¹ According to one estimate, the total number may be as high as 200 million.² Ever since studies on this subject were first published (around the beginning of this century),³ a sub-
continued

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◀ *Children (especially between the ages of 11 and 13) and adults over 40 have a higher risk of injury than do other age-groups. Instruction probably lowers the risk somewhat.*

ski injuries continued

Table 1. Potential Risk Factors Involved in Downhill Ski Injuries

Personal Characteristics	Behavior
Age	Fear
Gender	Risk taking
Height	Alcohol consumption
Weight	Nutrition
Education	
Skill Level	Equipment
Self-rated ability	Bindings
Experience	Boots
Lessons	Poles
	Brakes
Physical Condition	Environment
Fitness level	Snow quality
Sports participation	Visibility
Ski gymnastics	Weather conditions
Warm-up	Difficulty of ski run
Sleep	Time of day
Menstruation	Duration of exposure

Table 2. Incidence of Downhill Ski Injuries*

Type of Injury	No. of Injuries (per 1,000 Ski Days)
Upper body	1.6
Lower extremity/ equipment related	1.5
Knee	0.9
Other than knee	0.6
Lower extremity/ non-equipment related	0.3
Total	3.4

*From Johnson et al.⁹

stantial risk of injury has been documented. Since the 1943 report on injury statistics by Moritz,⁴ information on ski injuries has been published regularly. However, it is difficult to compare various sets of statistics because of differences in the definition and registration of injuries.⁵ Furthermore, underreporting by certain groups of skiers (eg, men) and of specific injuries (eg, thumb) is a problem.⁶ Incidence is usually expressed as the number of injuries that occur per 1,000 ski days. To calculate this, knowing which population has incurred the injuries is essential,^{5,7} which might be based on ski-lift ticket sales⁸ or on responses from a random

sample of skiers⁹ or a whole cohort that is at risk.¹⁰

Prevention of ski injuries requires an understanding of their causes, which means determining the quantitative contribution of the most important risk factors. This requires investigating the occurrence of ski injuries in relation to risk factors.⁵ Haddon et al¹¹ in 1962 were probably the first to conduct research in which injured and uninjured skiers were compared.

Potential risk factors can be divided into several main categories: personal characteristics, skill level, physical condition, behavior, equipment, and environment (table 1). These main categories can then be subdivided; for example, within the environment category it is useful to distinguish snow quality, visibility, weather conditions, difficulty of the ski run, time of day, and duration of exposure. Many risk factors are interrelated, which makes it difficult to study the contribution of a particular risk factor to injury incidence. Unconcerned by this complexity, some so-called experts offer recommendations for injury prevention that often are based on a mixture of plausibility, case reports, and prejudice. Their claims are frequently "proved" by quasiempirical reasoning based on injury statistics.⁵

Injuries

Incidence. Estimates of injury incidence range from 1 to 10 per 1,000 ski days.^{6,9,12} Most researchers agree that the incidence of serious injuries (those that require extensive medical treatment) lies in the range of two to four per 1,000 ski days.¹³⁻¹⁵ This measure can also be considered the mean risk percentage for one skier during a vacation that includes 10 days of skiing. This figure is probably much lower than it was a few decades ago.¹³⁻¹⁵ It is generally agreed that the development of modern ski equipment—especially the release binding—has contributed substantially to this decline.^{9,16} The improved design and maintenance of ski runs may also have contributed to this trend.¹⁶

Anatomic Site. The location of ski injuries has also changed over the years.⁶ Between 1960 and 1980 the proportion of lower extremity injuries declined substantially (from 80% to 55%), especially of the foot and ankle (from 45% to 10%).⁶ Similarly, the proportion of tibial fractures declined (from 25% to 15%), while knee injuries

continued

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INDICATIONS AND USAGE. ZESTRIL is indicated for the treatment of hypertension. It may be used alone as initial therapy or concomitantly with other classes of antihypertensive agents. In using ZESTRIL, consideration should be given to the fact that another angiotensin converting enzyme inhibitor, captopril, has caused agranulocytosis, particularly in patients with renal impairment or collagen vascular disease, and that available data are insufficient to show that ZESTRIL does not have a similar risk. (See WARNINGS.) **CONTRAINDICATIONS.** ZESTRIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an angiotensin converting enzyme inhibitor. **WARNINGS. Angioedema:** Angioedema of the face, extremities, lips, tongue, glottis and/or larynx has been reported in patients treated with angiotensin converting enzyme inhibitors, including ZESTRIL. In such cases, ZESTRIL should be promptly discontinued, and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis or larynx, likely to cause airway obstruction, appropriate therapy, eg, subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL) should be promptly administered. (See ADVERSE REACTIONS.) Hypotension:** Excessive hypotension was rarely seen in uncomplicated hypertensive patients but is a possible consequence of use with ZESTRIL in salt/volume-depleted persons, such as those treated vigorously with diuretics or patients on dialysis. (See PRECAUTIONS, Drug Interactions and ADVERSE REACTIONS.) In patients with severe congestive heart failure, with or without associated renal insufficiency, excessive hypotension has been observed and may be associated with oliguria and/or progressive azotemia, and rarely with acute renal failure and/or death. Because of the potential fall in blood pressure in these patients, therapy should be started under very close medical supervision. Such patients should be followed closely for the first two weeks of treatment and whenever the dose of ZESTRIL and/or diuretic is increased. Similar considerations apply to patients with ischemic heart or cerebrovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses which usually can be given without difficulty once the blood pressure has increased after volume expansion. **Neutropenia/Agranulocytosis:** Another angiotensin converting enzyme inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment especially if they also have a collagen vascular disease. Available data from clinical trials of ZESTRIL are insufficient to show that ZESTRIL does not cause agranulocytosis at similar rates. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered. **PRECAUTIONS. General. Impaired Renal Function:** As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe congestive heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with angiotensin converting enzyme inhibitors, including ZESTRIL, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death. In hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine may occur. Experience with another angiotensin converting enzyme inhibitor suggests that these increases are usually reversible upon discontinuation of ZESTRIL and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy. Some hypertensive patients with no apparent pre-existing renal vascular disease have developed increases in blood urea nitrogen and serum creatinine, usually minor and transient, especially when ZESTRIL has been given concomitantly with a diuretic. This is more likely to occur in patients with pre-existing renal impairment. Dosage reduction of ZESTRIL and/or discontinuation of the diuretic may be required. **Evaluation of the hypertensive patient should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.) Hyperkalemia:** In clinical trials hyperkalemia (serum potassium greater than 5.7 mEq/L) occurred in approximately 2.2% of hypertensive patients and 4.0% of patients with congestive heart failure. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in approximately 0.1% of hypertensive patients. Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with ZESTRIL. (See Drug Interactions.) **Surgery/Anesthesia:** In patients undergoing major surgery or during anesthesia with agents that produce hypotension, ZESTRIL may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion. **Information for Patients. Angioedema:** Angioedema, including laryngeal edema, may occur especially following the first dose of ZESTRIL. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician. **Symptomatic Hypotension:** Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patient should be told to discontinue the drug until they have consulted with the prescribing physician. All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with their physician. **Hyperkalemia:** Patients should be told not to use salt substitutes containing potassium without consulting their physician. **Neutropenia:** Patients should be told to report promptly any indication of infection (eg, sore throat, fever) which may be a sign of neutropenia. **NOTE:** As with many other drugs, certain advice to patients being treated with ZESTRIL is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects. **DRUG INTERACTIONS. Hypotension — Patients on Diuretic Therapy:** Patients on diuretics and especially those in whom diuretic therapy was recently instituted, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with ZESTRIL. The possibility of hypotensive effects with ZESTRIL can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with ZESTRIL. If it is necessary to continue the diuretic, initiate therapy with ZESTRIL at a dose of 5 mg daily, and provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS, and DOSAGE AND ADMINISTRATION.) When a diuretic is added to the therapy of a patient receiving ZESTRIL, an additional antihypertensive effect is usually observed. Studies with ACE inhibitors in combination with diuretics indicate that the dose of the ACE inhibitor can be reduced when it is given with a diuretic. (See DOSAGE AND ADMINISTRATION.) **Indomethacin:** In a study in 36 patients with mild to moderate hypertension where the antihypertensive effects of ZESTRIL alone were compared to ZESTRIL given concomitantly with indomethacin, the use of indomethacin was associated with a reduced effect, although the difference between the two regimens was not significant. **Other Agents:** ZESTRIL has been used concomitantly with nitrates and/or digoxin without evidence of clinically significant adverse interactions. No clinically important pharmacokinetic interactions occurred when ZESTRIL was used concomitantly with propranolol or hydrochlorothiazide. The presence of food in the stomach does not alter the bioavailability of ZESTRIL. **Agents Increasing Serum Potassium:** ZESTRIL attenuates potassium loss caused by thiazide-type diuretics. Use of ZESTRIL with potassium-sparing diuretics (eg, spironolactone, triamterene or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. **Lithium:** Lithium toxicity has been reported in patients receiving lithium with drugs which cause elimination of sodium, including ACE inhibitors. Lithium toxicity was usually reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if ZESTRIL is administered concomitantly with lithium. **Carcinogenesis, Mutagenesis, Impairment of Fertility.** There was no evidence of a tumorigenic effect when lisinopril was administered for 105 weeks to male and female rats at doses up to 90 mg/kg/day (about 56 times* the maximum recommended daily human dose) or when lisinopril was administered for 92 weeks to (male and female) mice at doses up to 135 mg/kg/day (about 84 times*) the maximum recommended daily human dose). *Based on patient weight of 50 kg. Lisinopril was not mutagenic in the Ames microbial mutagen test with or without metabolic activation. It was also negative in a forward mutation assay using Chinese hamster lung cells. Lisinopril did not produce single strand DNA breaks in an in vitro alkaline elution rat hepatocyte assay. In addition, lisinopril did not produce increases in chromosomal aberrations in an in vitro test in Chinese hamster ovary cells or in an in vivo study in mouse bone marrow. There were no adverse effects on reproductive performance in male and female rats treated with up to 300 mg/kg/day of lisinopril. **Pregnancy. Pregnancy Category C:** Lisinopril was not teratogenic in mice treated on days 6-15 of gestation with up to 1,000 mg/kg/day (625 times the maximum recommended human dose). There was an increase in fetal resorptions at doses down to 100 mg/kg; at doses of 1,000 mg/kg this was prevented by saline supplementation. There was no fetotoxicity or teratogenicity in rats treated with up to 300 mg/kg/day (188 times the maximum recommended dose) of lisinopril at days 6-17 of gestation. In rats receiving lisinopril from day 15 of gestation through day 21 postpartum, there was an increased incidence in pup deaths on days 2-7 postpartum and a lower average body weight of pups on day 21 postpartum. The increase in pup deaths and decrease in pup weight did not occur with maternal saline supplementation. Lisinopril, at doses up to 1 mg/kg/day, was not teratogenic when given throughout the organogenic period in saline supplemented rabbits. Saline supplementation (physiologic saline in place of tap water) was used to eliminate maternotoxic effects and enable evaluation of the teratogenic potential at the highest possible dosage level. The rabbit has been shown to be extremely sensitive to angiotensin converting enzyme inhibitors (captopril and enalapril) with maternal and fetotoxic effects apparent at or below the recommended therapeutic dosage levels in man. Fetotoxicity was demonstrated in rabbits by an increased incidence of fetal resorptions at an oral dose of lisinopril of 1 mg/kg/day and by an increased incidence of incomplete ossification at the lowest dose tested (0.1 mg/kg/day). A single intravenous dose of 15 mg/kg of lisinopril administered to pregnant rabbits on gestation days 16, 21 or 26 resulted in 88% to 100% fetal death. By whole body autoradiography, radioactivity was found in the placenta following administration of labeled lisinopril to pregnant rats, but none was found in the fetuses. **Human Experience:** There are no adequate and well-controlled studies of lisinopril in pregnant women. However, data are available that show drugs of this class cross the human placenta. Because the risk of fetal toxicity with the use the ACE inhibitors has not been clearly defined (see below), lisinopril should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality. When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed in utero to ACE inhibitors should be closely observed for hypotension, oliguria and hyperkalemia. If oliguria occurs, attention should be directed toward

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support of blood pressure and renal perfusion with the administration of fluids and pressores as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension or the underlying prematurity. Another ACE inhibitor, enalapril, has been removed from the neonatal circulation by peritoneal dialysis and theoretically may be removed by exchange transfusion, although there is no experience with the latter procedure. There is no experience with either of these procedures for removing lisinopril or other ACE inhibitors from the neonatal circulation. **Nursing Mothers:** Milk of lactating rats contains radioactivity following administration of ¹⁴C lisinopril. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZESTRIL is given to a nursing mother. **Pediatric Use:** Safety and effectiveness in children have not been established. **ADVERSE REACTIONS.** ZESTRIL has been found to be generally well tolerated in controlled clinical trials involving 2003 patients and subjects. The most frequent clinical adverse experiences in controlled trials with ZESTRIL were dizziness (6.3%), headache (5.3%), fatigue (3.3%), diarrhea (3.2%), upper respiratory symptoms (3.0%), and cough (2.9%), all of which were more frequent than in placebo-treated patients. For the most part, adverse experiences were mild and transient in nature. Discontinuation of therapy was required in 6.0% of patients. In clinical trials, the overall frequency of adverse experiences could not be related to total daily dosage within the recommended therapeutic dosage range. For adverse experiences which occurred in more than 1% of patients and subjects treated with ZESTRIL or ZESTRIL plus hydrochlorothiazide in controlled clinical trials, comparative incidence data are listed in the table below.

Percent of Patients in Controlled Studies			
	ZESTRIL (n = 2003†) Incidence (discontinuation)	ZESTRIL/Hydrochlorothiazide (n = 644) Incidence (discontinuation)	Placebo (n = 207) Incidence
Dizziness	6.3 (0.6)	9.0 (0.9)	1.9
Headache	5.3 (0.2)	4.3 (0.5)	1.9
Fatigue	3.3 (0.2)	3.9 (0.5)	1.0
Diarrhea	3.2 (0.3)	2.6 (0.3)	2.4
Upper Respiratory Symptoms	3.0 (0.0)	4.5 (0.0)	0.0
Cough	2.9 (0.4)	4.5 (0.8)	1.0
Nausea	2.3 (0.3)	2.5 (0.2)	2.4
Hypotension	1.8 (0.8)	1.6 (0.5)	0.5
Rash	1.5 (0.4)	1.6 (0.2)	0.5
Orthostatic Effects	1.4 (0.0)	3.4 (0.2)	1.0
Asthenia	1.3 (0.4)	2.0 (0.2)	1.0
Chest Pain	1.3 (0.1)	1.2 (0.2)	1.4
Vomiting	1.3 (0.2)	1.4 (0.0)	0.5
Dyspnea	1.1 (0.0)	0.5 (0.2)	1.4
Dyspepsia	1.0 (0.0)	1.9 (0.0)	0.0
Paresthesia	0.8 (0.0)	2.0 (0.2)	0.0
Impotence	0.7 (0.2)	1.6 (0.3)	0.0
Muscle Cramps	0.6 (0.0)	2.8 (0.6)	0.5
Back Pain	0.5 (0.0)	1.1 (0.0)	1.4
Nasal Congestion	0.3 (0.0)	1.2 (0.0)	0.0
Decreased Libido	0.2 (0.1)	1.2 (0.0)	0.0
Vertigo	0.1 (0.0)	1.1 (0.2)	0.0

†Includes 420 patients treated for congestive heart failure who were receiving concomitant digitalis and/or diuretic therapy.

Clinical adverse experiences occurring in 0.3% to 1.0% of patients in the controlled trials and rarer, serious, possibly drug related events reported in uncontrolled studies or marketing experience included: **BODY AS A WHOLE:** Chest discomfort, fever, flushing. **CARDIOVASCULAR:** Angina pectoris, orthostatic hypotension, rhythm disturbances, tachycardia, peripheral edema, palpitation. **DIGESTIVE:** Abdominal pain, anorexia, constipation, flatulence. **METABOLISM:** Gout. **MUSCULOSKELETAL:** Joint pain, shoulder pain. **NERVOUS SYSTEM/PSYCHIATRIC:** Depression, somnolence, insomnia, stroke. **RESPIRATORY SYSTEM:** Bronchitis, sinusitis, pharyngeal pain. **UROGENITAL:** Oliguria, progressive azotemia, acute renal failure. **OTHER:** Blurred vision, pruritus, urinary tract infection, vasculitis of the legs. **ANGIOEDEMA:** Angioedema has been reported in patients receiving ZESTRIL (0.1%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis and/or larynx occurs, treatment with ZESTRIL should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.) **HYPOTENSION:** In hypertensive patients, hypotension occurred in 1.2% and syncope occurred in 0.1% of patients. Hypotension or syncope was a cause of discontinuation of therapy in 0.5% of hypertensive patients. (See WARNINGS.) In patients with congestive heart failure, hypotension occurred in 5.0% and syncope occurred in 1.0% of patients. These adverse experiences were causes for discontinuation of therapy in 1.3% of these patients. **Clinical Laboratory Test Findings. Serum Electrolytes:** Hyperkalemia. (See PRECAUTIONS.) **Creatinine, Blood Urea Nitrogen:** Minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 2.0% of patients with essential hypertension treated with ZESTRIL alone. Increases were more common in patients receiving concomitant diuretics and in patients with renal artery stenosis. (See PRECAUTIONS.) Reversible minor increases in blood urea nitrogen and serum creatinine were observed in approximately 9.1% of patients with congestive heart failure on concomitant diuretic therapy. Frequently, these abnormalities resolved when the dosage of the diuretic was decreased. **Hemoglobin and Hematocrit:** Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.4 g% and 1.3 vol%, respectively) occurred frequently in patients treated with ZESTRIL but were rarely of clinical importance in patients without some other cause of anemia. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia. **Other (Causal Relationship Unknown):** Rarely, elevations of liver enzymes and/or serum bilirubin have occurred. Overall, 2.0% of patients discontinued therapy due to laboratory adverse experiences, principally elevations in blood urea nitrogen (0.6%), serum creatinine (0.5%), and serum potassium (0.4%). **OVERDOSAGE.** The oral LD₅₀ of lisinopril is greater than 20 g/kg in mice and rats. The most likely manifestation of overdosage would be hypotension, for which the usual treatment would be intravenous infusion of normal saline solution. Lisinopril can be removed by hemodialysis. **DOSAGE AND ADMINISTRATION. Initial Therapy:** In patients with uncomplicated essential hypertension not on diuretic therapy, the recommended initial dose is 10 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 20-40 mg per day administered in a single daily dose. The antihypertensive effect may diminish toward the end of the dosing interval regardless of the administered dose, but most commonly with a dose of 10 mg daily. This can be evaluated by measuring blood pressure just prior to dosing to determine whether satisfactory control is being maintained for 24 hours. If it is not, an increase in dose should be considered. Doses up to 80 mg have been used but do not appear to give greater effect. If blood pressure is not controlled with ZESTRIL alone, a low dose of a diuretic may be added. Hydrochlorothiazide, 12.5 mg has been shown to provide an additive effect. After the addition of a diuretic, it may be possible to reduce the dose of ZESTRIL. **Diuretic Treated Patients:** In hypertensive patients who are currently being treated with a diuretic, symptomatic hypotension may occur occasionally following the initial dose of ZESTRIL. The diuretic should be discontinued, if possible, for two to three days before beginning therapy with ZESTRIL to reduce the likelihood of hypotension. (See WARNINGS.) The dosage of ZESTRIL should be adjusted according to blood pressure response. If the patient's blood pressure is not controlled with ZESTRIL alone, diuretic therapy may be resumed as described above. If the diuretic cannot be discontinued, an initial dose of 5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, Drug Interactions.) Concomitant administration of ZESTRIL with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium. (See PRECAUTIONS.) **Use in Elderly:** In general, blood pressure response and adverse experiences were similar in younger and older patients given similar doses of ZESTRIL. Pharmacokinetic studies, however, indicate that maximum blood levels and area under the plasma concentration time curve (AUC) are doubled in older patients so that dosage adjustments should be made with particular caution. **Dosage Adjustment in Renal Impairment:** The usual dose of ZESTRIL (10 mg) is recommended for patients with creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≥ 10 mL/min ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 5 mg once daily. For patients with creatinine clearance < 10 mL/min (usually on hemodialysis) the recommended initial dose is 2.5 mg. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Renal Status	Creatinine Clearance mL/min	Initial Dose mg/day
Normal Renal Function to Mild Impairment	> 30	10
Moderate to Severe Impairment	≥ 10 to 30	5
Dialysis Patients	< 10	2.5†

†Dosage or dosing interval should be adjusted depending on the blood pressure response.
Stuart Pharmaceuticals, A business unit of ICI Americas Inc., Wilmington, DE 19897 USA Rev J 4/89

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ski injuries continued

remained approximately the same (20%). Thus it seems that modern ski equipment protects the tibia more effectively than it does the knee.⁶ As a consequence of the decline in lower extremity injuries, the proportion of upper extremity injuries increased (from 10% to 25%), as did head and torso injuries (from 10% to 20%).⁶

Johnson et al^{9,17} introduced the concept of lower extremity, equipment-related (LEER) injury, "... in which the mechanism is consistent with the ski acting as a lever to bend or twist the leg."^{9(p107)} These authors classified 80% of lower extremity injuries as equipment-related. Between 1972 and 1978 the incidence of LEER injuries decreased by 43%, compared with a decrease of only 25% for upper body injuries. The incidence of tibial fracture and ankle sprain declined substantially—each by more than 70%.

Some researchers^{6,15} suggest that the ulnar collateral ligament of the metacarpophalangeal joint of the thumb is the most frequent injury site (as high as 20% of all ski injuries). It is assumed that this injury is severely underreported in most studies because it does not immobilize the skier and thus often goes undocumented. Table 2 lists the incidence of several types of ski injuries based on observations made at a ski area in Vermont.⁹ Hauser and Gläser⁶ reported similar figures based on a sample of more than 200,000 ski days.

Circumstances. A number of studies mention self-reported direct causes of accidents that led to injury. It is difficult to compare these studies because they use different categories. They attribute from 11% to 20% of injuries to a collision—6% to 16% with an object and 3% to 8% with another skier.^{6,14,16} Collision injuries usually are relatively serious. It is not surprising that falls (due to several causes) are blamed for most injuries (70% to 90%).^{6,16} Problems with ski lifts (especially T-bar lifts) account for 2% to 9% of injuries.¹⁶ Most accidents that result in injury (90% to 95%) happen on the ski runs, probably because that is where most skiing is done.^{5,6} About 15% of these skiers reported going very fast just before the accident; about 55% were going moderately fast, 25% were skiing slowly, and 5% were standing still.⁵ The fatality rate is less than one per million ski days; most cases involve a high-speed collision with a tree.^{14,16,18}

Most injuries occur on weekends,⁶ although Monday is also mentioned as a hazardous day.¹⁹ Danger associated with the third day (usually

Monday) of skiing is not supported by the data.^{6,19} Some researchers conclude that accidents occur relatively less frequently during the morning.¹⁹ Others hypothesize that accident risk remains constant throughout the day.^{5,6,12} On average, injured skiers are on the slopes for about 3 hours before getting hurt.^{5,19}

Risk Factors

Personal Characteristics. There are proportionately more children (especially between ages 11 and 13) and adults over 40 among injured skiers, indicating a higher risk for these age-groups.^{8,12} The fact that children are less skilled seems to be at least partly responsible for this finding. Injured skiers under age 15 have a high proportion of lower extremity injuries, especially tibial fracture. According to some studies,^{8,20} this indicates that children use poor quality equipment and/or do not adjust their bindings properly.

Some authors^{12,16} report that the injury risk is higher for females than males; others^{3,10} report the same risk for both sexes. Furthermore, females are more likely than males to report their injuries to the local facilities.⁶ Injury to the ligaments of the knee occurs relatively often among females, indicating differences in anatomy and/or in efficacy of binding adjustment^{12,16} between males and females.

Differences in height and weight between injured and noninjured skiers have not been found or have been confounded by age.^{6,11} Some authors mention that more highly educated skiers have a lower risk.^{5,19}

Skill Level. Researchers agree that skiers who consider themselves beginners have a risk about twice that of more advanced skiers.^{5,12,14} This also applies to skiing experience (number of seasons),⁵ which correlates highly to self-rated ability. However, a preventive effect from experience still seems to remain when skill level is adjusted for.⁵

It seems reasonable to expect that formal instruction will increase skill level and thus lower injury risk. However, an overall effect is lacking,⁵ and even an increased risk for those who take ski lessons has been reported.²¹ The latter finding is probably due to the absence of adjustment for differences in ability and/or duration of exposure.^{5,14} One study⁵ adjusted for these factors in a multivariate analysis and reported a preventive effect from lessons, but only during

continued

The introduction of modern equipment in the 1970s contributed to the decline in ski injuries.

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INDICATIONS AND USAGE. Doxycycline is indicated in infections caused by the following microorganisms:

- Rickettsiae (Rocky Mountain spotted fever, typhus fever and the typhus group, Q fever, rickettsialpox and tick fevers).
- Mycoplasma pneumoniae* (PPLO, Eaton's agent).
- Agents of psittacosis and ornithosis.
- Agents of lymphogranuloma venereum and granuloma inguinale.
- The spirochetal agent of relapsing fever (*Borrelia recurrentis*).

The following gram-negative microorganisms:

- Haemophilus ducreyi* (chancroid)
- Yersinia pestis* (formerly *Pasteurella pestis*)
- Francisella tularensis* (formerly *Pasteurella tularensis*)
- Bartonella bacilliformis*
- Bacteroides* species
- Vibrio cholerae* (formerly *Vibrio comma*)
- Campylobacter fetus* (formerly *Vibrio fetus*)
- Brucella* species (in conjunction with streptomycin)

Because many strains of the following groups of microorganisms have been shown to be resistant to tetracyclines, culture and susceptibility testing are recommended.

Doxycycline is indicated for treatment of infections caused by the following gram-negative microorganisms, when bacteriological testing indicates appropriate susceptibility to the drug:

- Escherichia coli*
- Enterobacter aerogenes* (formerly *Aerobacter aerogenes*)
- Shigella* species
- Mima* species and *Herellea* species
- Haemophilus influenzae* (respiratory infections)
- Klebsiella* species (respiratory and urinary infections)

Doxycycline is indicated for treatment of infections caused by the following gram-positive microorganisms when bacteriological testing indicates appropriate susceptibility to the drug:

Streptococcus species: Up to 44 percent of strains of *Streptococcus pyogenes* and 74 percent of *Streptococcus faecalis* have been found to be resistant to tetracycline drugs. Therefore, tetracyclines should not be used for streptococcal disease unless the organism has been demonstrated to be susceptible.

For upper respiratory infections due to group A beta-hemolytic streptococci, penicillin is the usual drug of choice, including prophylaxis of rheumatic fever.

Diplococcus pneumoniae, *Staphylococcus aureus*, (respiratory, skin and soft-tissue infections). Tetracyclines are not the drug of choice in the treatment of any type of staphylococcal infection.

When penicillin is contraindicated, doxycycline is an alternative drug in the treatment of infections due to:

- Treponema pallidum* and *Treponema pertenue* (syphilis and yaws)
- Listeria monocytogenes*
- Clostridium* species
- Bacillus anthracis*
- Fusobacterium fusiforme* (Vincent's infection)
- Actinomyces* species

In acute intestinal amebiasis doxycycline may be a useful adjunct to amebicides.

In severe acne doxycycline may be useful adjunctive therapy.

Doxycycline is indicated in the treatment of trachoma, although the infectious agent is not always eliminated, as judged by immunofluorescence.

Inclusion conjunctivitis may be treated with oral doxycycline alone, or with a combination of topical agents.

Doxycycline is indicated for the treatment of uncomplicated urethral, endocervical or rectal infections in adults caused by *Chlamydia trachomatis*.

Doxycycline is indicated for the treatment of nongonococcal urethritis caused by *Chlamydia trachomatis* and *Ureaplasma urealyticum* and for the treatment of acute epididymo-orchitis caused by *Chlamydia trachomatis*.

Doxycycline is indicated for the treatment of uncomplicated gonococcal infections in adults (except for anorectal infections in men), the gonococcal arthritis-dermatitis syndrome and acute epididymo-orchitis caused by *N. gonorrhoeae*.

CONTRAINDICATIONS. The drug is contraindicated in persons who have shown hypersensitivity to any of the tetracyclines.

WARNINGS. THE USE OF DRUGS OF THE TETRACYCLINE CLASS DURING TOOTH DEVELOPMENT (LAST HALF OF PREGNANCY, INFANCY AND CHILDHOOD TO THE AGE OF 8 YEARS) MAY CAUSE PERMANENT DISCOLORATION OF THE TEETH (YELLOW-GRAY-BROWN). This adverse reaction is more common during long term use of the drugs but has been observed following repeated short term courses. Enamel hypoplasia has also been reported. TETRACYCLINE DRUGS, THEREFORE, SHOULD NOT BE USED IN THIS AGE GROUP UNLESS OTHER DRUGS ARE NOT LIKELY TO BE EFFECTIVE OR ARE CONTRAINDICATED.

Results of animal studies indicate that tetracyclines cross the placenta, are found in fetal tissues and can have toxic effects on the developing fetus (often related to retardation of skeletal development). Evidence of embryotoxicity has been noted in animals treated early in pregnancy. If any tetracycline is used during pregnancy or if the patient becomes pregnant while taking these drugs, the patient should be apprised of potential hazard to the fetus.

As with other tetracyclines, doxycycline forms a stable calcium complex in any bone-forming tissue. A decrease in the fibula growth rate has been observed in prematures given oral tetracycline in doses of 25 mg/kg every six hours. This reaction was shown to be reversible when the drug was discontinued.

Photosensitivity manifested by an exaggerated sunburn reaction has been observed in some individuals taking tetracyclines. Patients apt to be exposed to direct sunlight or ultraviolet light should be advised that this reaction can occur with tetracycline drugs, and treatment should be discontinued at the first evidence of skin erythema.

The antianabolic action of the tetracyclines may cause an increase in BUN. Studies to date indicate that this does not occur with the use of doxycycline in patients with impaired renal function.

PRECAUTIONS. As with other antibiotic preparations, use of this drug may result in overgrowth of nonsusceptible organisms, including fungi. If superinfection occurs, the antibiotic should be discontinued and appropriate therapy instituted.

All infections due to group A beta-hemolytic streptococci should be treated for at least 10 days.

Laboratory tests: In venereal disease when coexistent syphilis is suspected, dark-field examination should be done before treatment is started and the blood serology repeated monthly for at least 4 months.

In long term therapy, periodic laboratory evaluation of organ systems, including hematopoietic, renal and hepatic studies should be performed.

Drug interactions: Because tetracyclines have been shown to depress plasma prothrombin activity, patients who are on anticoagulant therapy may require downward adjustment of their anticoagulant dosage.

Since bacteriostatic drugs may interfere with the bactericidal action of penicillin, it is advisable to avoid giving tetracyclines in conjunction with penicillin.

For concomitant therapy with antacids or iron-containing preparations and food see "Dosage and Administration" section.

Carcinogenesis, mutagenesis, impairment of fertility: Long term studies are currently being conducted to determine whether tetracyclines have carcinogenic potential. Animal studies conducted in rats and mice have not provided conclusive evidence that tetracyclines may be carcinogenic or that they impair fertility. In two mammalian cell assays (L51784 mouse lymphoma and Chinese hamster lung cells *in vitro*) positive responses for mutagenicity occurred at concentrations of 60 and 10 mcg/mL respectively. In humans no association between tetracyclines and these effects have been made.

Pregnancy: Pregnancy Category D (See Warnings section).

Nursing mothers: Tetracyclines are present in the milk of lactating women who are taking a drug in this class. Because of the potential for serious adverse reactions in nursing infants from the tetracyclines, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the importance of the drug to the mother (see Warnings section).

Pediatric use: See Warnings and Dosage and Administration sections.

ADVERSE REACTIONS. Due to oral doxycycline's virtually complete absorption, side effects to the lower bowel, particularly diarrhea, have been infrequent. The following adverse reactions have been observed in patients receiving tetracyclines:

Gastrointestinal: Anorexia, nausea, vomiting, diarrhea, glossitis, dysphagia, enterocolitis, and inflammatory lesions (with monilial overgrowth) in the anogenital region. These reactions have been caused by both the oral and parenteral administration of tetracyclines. Rare instances of esophagitis and esophageal ulcerations have been reported in patients receiving capsule and tablet forms of drugs in the tetracycline class. Most of these patients took medications immediately before going to bed. (See Dosage and Administration).

Skin: Maculopapular and erythematous rashes. Exfoliative dermatitis has been reported but is uncommon. Photosensitivity is discussed above (see Warnings).

Renal toxicity: Rise in BUN has been reported and is apparently dose related. (See Warnings).

Hypersensitivity reactions: Urticaria, angioneurotic edema, anaphylaxis, anaphylactoid purpura, pericarditis, and exacerbation of systemic lupus erythematosus.

Bulging fontanels in infants and benign intracranial hypertension in adults have been reported in individuals receiving tetracyclines. These conditions disappeared when the drug was discontinued.

Blood: Hemolytic anemia, thrombocytopenia, neutropenia, and eosinophilia have been reported with tetracyclines.

When given over prolonged periods, tetracyclines have been reported to produce brown-black microscopic discoloration of thyroid glands. No abnormalities of thyroid function are known to occur.

DOSAGE AND ADMINISTRATION. THE USUAL DOSAGE AND FREQUENCY OF ADMINISTRATION OF DOXYCYCLINE DIFFERS FROM THAT OF THE OTHER TETRACYCLINES. EXCEEDING THE RECOMMENDED DOSAGE MAY RESULT IN AN INCREASED INCIDENCE OF SIDE EFFECTS.

Adults: The usual dose of oral doxycycline is 200 mg on the first day of treatment (administered 100 mg every 12 hours) followed by a maintenance dose of 100 mg/day. The maintenance dose may be administered as a single dose or as 50 mg every 12 hours. In the management of more severe infections (particularly chronic infections of the urinary tract), 100 mg every 12 hours is recommended.

For children above eight years of age: The recommended dosage schedule for children weighing 100 pounds or less is 2 mg/lb of body weight divided into two doses on the first day of treatment, followed by 1 mg/lb of body weight given as a single daily dose or divided into two doses on subsequent days. For more severe infections up to 2 mg/lb of body weight may be used. For children over 100 pounds, the usual adult dose should be used.

Uncomplicated gonococcal infections in adults (except anorectal infections in men): 100 mg, by mouth, twice-a-day for 7 days. As an alternate single visit dose, administer 300 mg stat followed in one hour by a second 300 mg dose. The dose may be administered with food, including milk or carbonated beverage, as required.

Acute epididymo-orchitis caused by *N. gonorrhoeae*: 100 mg, by mouth, twice-a-day for at least 10 days.

Primary and secondary syphilis: 300 mg a day in divided doses for at least 10 days.

Uncomplicated urethral, endocervical, or rectal infection in adults caused by *Chlamydia trachomatis*: 100 mg by mouth, twice-a-day for at least 7 days.

Nongonococcal urethritis caused by *C. trachomatis* and *U. urealyticum*: 100 mg, by mouth, twice-a-day for at least 7 days.

Acute epididymo-orchitis caused by *C. trachomatis*: 100 mg, by mouth, twice-a-day for at least 10 days.

The therapeutic antibacterial serum activity will usually persist for 24 hours following recommended dosage.

When used in streptococcal infections, therapy should be continued for 10 days.

Administration of adequate amounts of fluid along with capsule and tablet forms of drugs in the tetracycline class is recommended to wash down the drugs and reduce the risk of esophageal irritation and ulceration (see Adverse Reactions).

If gastric irritation occurs, it is recommended that doxycycline be given with food or milk. The absorption of doxycycline is not markedly influenced by simultaneous ingestion of food or milk.

Concomitant therapy: Antacids containing aluminum, calcium or magnesium, sodium bicarbonate, and iron-containing preparations should not be given to patients taking oral tetracyclines.

Studies to date have indicated that administration of doxycycline at the usual recommended doses does not lead to excessive accumulation of the antibiotic in patients with renal impairment.

Caution — Federal law prohibits dispensing without prescription.

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ski injuries continued

the first and second seasons of skiing.

Physical Condition. It has been suggested that good physical condition can prevent exhaustion and thus lower accident risk.²² Despite the physiologic plausibility of this hypothesis, empirical evidence is lacking. A recent study⁵ of Dutch skiers indicates that a self-reported good physical condition was a risk factor. This is consistent with the higher prevalence among injured skiers of participation in other sports. Furthermore, we know of no published empirical evidence that supports the recommendation to prepare for a ski vacation by taking a course in ski gymnastics or by taking lessons on an artificial ski run.⁵ The same is true for the assumed preventive effect of warm-up exercises.

Skiers seem to get plenty of sleep.¹⁹ One study found that injured skiers sleep better and longer than uninjured skiers,⁵ and another reported no differences between these two groups.¹¹ A possible association between accident risk and menses has been suggested¹⁹ but could not be confirmed.⁵

Behavior. Skiers who report that before a vacation they feel somewhat afraid of having a skiing accident seem to have a lower injury risk.⁵ Recklessness and risk-taking behavior are often mentioned as risk factors in popular literature on downhill skiing. The personality trait referred to as "sensation seeking" appears to be relatively high among downhill skiers, especially on the subscale that indicates a desire to engage in hazardous activities. But contrary to what might be expected, injured skiers seem to have a lower score than uninjured skiers when tested for this trait.⁵

Several authors list alcohol consumption as an important risk factor in downhill skiing.^{19,22,23} Two mechanisms are mentioned in this respect. First, alcohol affects reaction time, accuracy of movements, and perception of risk.¹⁹ Stanley²³ supposes that alcohol consumption amplifies the effect of hypoxia at high altitudes. Second, Brouns et al²² point out that alcohol often lowers carbohydrate intake, especially immediately after skiing, when glycogen resynthesis is stimulated most strongly. Consequently, the skier will experience glycogen depletion sooner the next day, increasing injury risk because of exhaustion and diminished control of movement.

Little data are available concerning alcohol consumption among downhill skiers. Only 2%

of a group of injured skiers were found to have an alcohol permillage of more than 0.8 at the time of injury.^{19,24} Hauser and Gläser⁶ found only one skier with alcohol permillage of 0.7 among 39 randomly selected uninjured male skiers. Thus alcohol does not appear to be an important risk factor. According to self-reported drinking behavior, alcohol even seems to have a protective effect, but this finding might be due to selective underreporting by injured skiers.⁵

Glycogen depletion in the muscle fibers is evident at the end of a day of skiing.²² The resulting fatigue is considered an important risk factor.²⁵ To prevent an increasing glycogen depletion throughout the vacation, skiers are advised to consume frequent, small meals that are rich in carbohydrate.²² Empirical evidence of a relationship between nutrition and injury risk among skiers is lacking.

Equipment. As stated above, studies suggest an important preventive effect from the introduction of modern equipment in the 1970s—especially in the design of the ski binding. The question is whether binding adjustment is still an important risk factor. Bindings seem to release less frequently in accidents that result in LEER injuries than those resulting in non-LEER injuries.^{5,15} However, it is doubtful that even an optimally adjusted ski binding will always release in time to prevent a LEER injury. The knee is not sufficiently protected by the current binding design.^{3,5,26} Studies indicate that binding adjustment generally is more adequate among controls than injured skiers (particularly those with LEER injuries).^{6,17,20} While the bindings of both controls and skiers with a non-LEER injury are adjusted an average of 50% above the recommended setting, the average deviation is 85% for skiers with a knee sprain and 150% for those with a tibial fracture.⁶ More recently Hauser demonstrated with a randomized controlled trial²⁷ that optimal adjustment can substantially lower the risk for LEER injuries.

Ski boots and bindings form a functional unit, and it is recognized that the properties of the boot can dramatically affect release performance of the binding.²⁶ Early models of the modern plastic ski boot (late 1960s and early 1970s) were associated with a sudden increase in isolated fractures of the fibula at the level of the boot top.³ The stiffness of the boot was modified, but there still seems to be room for further

Properties of the ski boot can dramatically affect release performance of the binding.

continued

Photo: David Madison © 1989

Snow quality contributes to the incidence of ski injuries. Skiers on icy or hard-packed snow are more likely to injure the upper extremity; skiers on wet snow or powder, the lower extremity.

improvement in ski boot design.²⁶ No data are available on the risks associated with the types of ski boots currently available, although some authors²⁴ warn that binding function may be impaired when the boot sole is made of thermoplastic rubber—a material that is still used in children's boots.

Falling with the hand on the ski pole handle is an important cause of thumb injury.^{6,27} To avoid risking this injury, skiers are advised either not to use ski-pole straps or to use grips of a different design.²⁷ To prevent lacerations caused by the edges of the ski, Tapper¹⁶ warns against the use of safety straps around the ankle and advises instead the use of a ski brake (called a "ski stopper").

Environment. Snow quality is often investigated as a risk factor. Most researchers agree that icy or hard-packed snow leads to a higher proportion of upper extremity and head injuries. Wet snow and powder lead to lower ex-

tremity injuries; particularly, tibial fractures and knee lesions are relatively frequent.⁶ There is some doubt concerning the effect of snow quality on overall injury incidence,^{12,19} but the risk is substantially elevated when icy spots are present.⁵

Jaffin¹² found that about 20% of skiers reported problems with visibility. However, no clear relation to injury risk was established. The risk may be greater when the sky is cloudless, even when taking into account the fact that ski runs are more crowded on clear days.^{5,19} Some authors^{28,29} attribute the elevated risk during good weather to a biological or psychological influence of high atmospheric pressure.

Discussion

Despite the problems of selective underreporting, a fair amount of information is available regarding the overall incidence of ski injuries and the frequency of specific injuries. The systems currently used to document injuries seem reliable enough to detect major trends. Also, there is consensus as to the circumstances of accidents that result in injury. Some of these circumstances seem to call for relatively straightforward preventive measures—eg, removing objects from ski runs, modifying the design of T-bar lifts, creating ski runs that make it impossible to ski at high speeds. Generally, though, the situation is more complex, and although there is agreement with respect to some generally accepted risk factors, uncertainty remains about others.

Both case-control and cohort studies have been conducted to increase our knowledge about the preventable causes of ski injuries. The results of these investigations usually were presented in terms of crude frequencies of the factor at issue, without reports of appropriate measures of association (eg, odds ratios). Although most researchers agree that risk factors are probably interrelated to some extent, stratified analysis is rare, and an adequate multivariate analysis is almost never performed. Another problem is the fact that many of these studies were based on information collected by questionnaire—a method that is open to imprecision and bias. Future studies should try to avoid these weaknesses and focus more specifically on risk factors that are open to manipulation. Because of the inherent uncertainty of nonexperimental (associational) studies, it is always

continued

HER HIP FRACTURE WAS 50 YEARS IN THE MAKING.

A lifetime of skeletal health may depend on adequate bone development in adolescence.^{1,2} But from ages 11 through 18 when the calcium RDA increases to 1,200 mg/day,³ many youngsters get considerably less calcium than they need.^{2,4} Therefore, they may not achieve the normal adolescent contribution of 45% to peak adult bone mass.²

A 60% reduction in hip fracture incidence has been noted in a recent 14-year prospective study of 531 women and 426 men (ages 50–79) among those in the top third of *lifetime* dietary calcium intake (primarily from dairy).⁵ This is strong evidence that adequate consumption of dairy during adolescence and after can help build and maintain a “bone bank” sufficient to help reduce the risk of geriatric hip fractures.⁵

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ski injuries continued

advisable to try a preventive measure (eg, education) in a well-designed randomized trial before introducing it on a large scale. Unfortunately, only one such trial has been performed.²⁷

Conclusion

Despite the limitations, some conclusions can be drawn from the existing data with acceptable confidence: Beginners are at higher risk, but taking ski lessons can probably lower this risk to some extent. Empirical evidence is lacking with respect to the possible risk-reducing effect of being in good physical condition. Further clarification will depend on studies that measure condition directly (prospectively), or even better, studies that actively manipulate physical condition among a group of randomly chosen skiers. Sound knowledge of behavior factors also is virtually absent. Alcohol is prob-

ably not an important risk factor, but nutrition might be; this area has not been thoroughly investigated. Risk-taking behavior seems to play a role, but its impact is uncertain, and effective manipulation of this factor is questionable.

There is little doubt that adequate adjustment of the ski binding can lower the incidence of LEER injury. Moreover, it is plausible that improved design of ski boots and bindings can reduce knee injuries and thus make skiing a safer sport (especially for women and children). The current design of ski poles seems to contribute to the occurrence of thumb injuries. Little is known yet about environmental risk factors. **PSM**

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