disorders, ligament sprains, muscle strains, bursitis, and nerve entrapments. Common sites of injury are the back, shoulder, elbow, and wrist. Treatments include rest of the affected limb through work restriction and judicious use of splints; physical therapy; nonsteroidal anti-inflammatory agents; ice; corticosteroid injections; and surgery. After the symptoms have resolved, physical therapy should be directed at muscle strengthening to prepare the person to return to work. If modified duty work is not available during treatment, the employee may not be able to work. If the disorder was not triggered by a specific event or an underlying medical condition, the physician should recommend that the work be permanently modified to prevent recurrence.

Although relatively simple interventions may be called for (such as wrist supports and work breaks), the most successful programs are well coordinated and involve medical surveillance, employee training, job analysis, engineering controls, and administrative controls. The best programs have small but empowered ergonomics teams that include employees, supervisors, engineers, and health and safety personnel. Part of the success of these programs is thought to be because employees aid in evaluating the tasks and implementing solutions.

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Resurgence of Work-Hardening Programs

WORK-HARDENING programs have undergone a resurgence in popularity because of their efficacy in rehabilitating injured people when a program is appropriately organized. The essential components of work-hardening programs are physical reconditioning, aerobic conditioning, work simulation, and education. Because it is directed at injured persons, work hardening must be goal-specific and job-directed. In addition to preparing the physical capacity for a person's return to work, activities should simulate realistic occupational situations. To accomplish this, the program must be intensive, operating five days per week, four to eight hours per day. The schedule is planned in a work-like manner; it may even be appropriate to have patients "punch in" and "punch out."

Functional capacity evaluations of strength, endurance, coordination, pace, and safety are included. The program should incorporate a customized obstacle course that includes lifting, carrying, twisting, crawling, bending, stooping, and other movements peculiar to a job. Normal performance levels are established by the therapist. The client is observed and timed moving through the course, and deviations from the norm are quantified so that they can be identified. Specialized equipment may be needed for certain simulations, but simple tools, such as bricks in a box, often suffice.

Work-hardening programs are designed to treat the entire spectrum of disability preventing a return to work. This contrasts with traditional rehabilitation programs, which focus on anatomic impairment. Standard physical therapy is less expensive, less intensive, and should be started soon after the injury. Work hardening is costly, with programs costing between $5,000 and $15,000, and is more appropriate when physical deconditioning and behavioral changes are preventing job return. This is typically about four months after an injury. When a patient is referred, the diagnosis must be firmly established and no further treatment, such as surgical procedures, is anticipated.

The programs usually last about three weeks. Dropout caused by lack of motivation occurs in 10% to 15% of participants. Encouragement and support from the referring physician and the insurance company are important. Specific goals should be set, and progress measured objectively. The patient must clearly understand that this is the most appropriate treatment and possibly the last chance for a return to gainful employment.

Credibility for work-hardening programs is established. About 80% of patients previously considered permanently disabled can be returned to employment. The fundamental philosophy responsible for this success is that measure of function, rather than discussion of pain, is the basis for treating an injured worker. All components of the program must be active, and the patient must be involved. The intensity and techniques are very different from the passive approach traditionally used to treat soft-tissue injuries. This is sports medicine for working people.

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Progress in Occupational Stress—Organizational Interventions

SEVERAL FACTORS are increasingly pushing occupational stress into the limelight. Workers' compensation claims for stress grew 700% between 1979 and 1989, driven by a greater awareness of its effects by both physicians and patients. Occupational health experts and others concerned with employee health and medical expenses are beginning to recognize that the hidden costs of stress are enormous. The contribution of stress to musculoskeletal disability as well as its role in organizational losses (productivity and lost work days) has catapulted this issue into the press. Most Fortune 500 companies now have developed some form of occupational stress program.

This attention intensifies the need to develop valid measures of stress and effective ways to control it. Traditional approaches to stress management have focused on modifying a person's response to reality through meditation, relaxation

techniques, exercise, or some other activity. Some short-term subjective benefits as well as temporary objective changes in blood pressure and electromyographic activity have been reported. Benefits at the organizational level, such as increased attendance, productivity, and reduced health care costs, have not been shown, however.

Recent investigations have explored ways to assess the sources of stress in the environment and to modify these. Research based on organizational interventions has yielded some encouraging results. New survey questionnaires, some of which are commercially available, can help determine the personal and organizational sources of stress; the data can be aggregated to provide an organizational stress profile that, in turn, can serve as the basis for planning interventions. One program resulted in reductions in all types of workers' compensation claims from 3.08 to 0.63 claims per month, with the average cost per claim dropping from $7,329 to $384. A key factor in the success of these programs is the commitment of managers to implement changes based on survey results.

Several recent studies have focused on job decision latitude (JDL) as a powerful predictor of workplace stress and future morbidity. Defined as an employee's ability to exercise decision-making authority at work and to choose the skills used on the job, JDL has been widely studied. In this model, work stress is the result of the interaction between JDL and job demands. Other factors being equal, a person with a highly demanding job but little control over its implementation is likely to be under more stress. Some of the other factors that are considered are lack of personal scheduling freedom and few opportunities for new learning on the job.

Low JDL has proved in several studies to be strongly associated with cardiovascular disease. A relationship between low JDL and cardiovascular disease in employed Swedish men was shown in one study. Another study reported a 22-year prospective study of pulp mill employees that showed higher mortality in people with low JDL. An interventional study showed that redesigning a work unit in order to increase JDL resulted in improved employee health as well as productivity.

Limited by their cost, complexity, and risk, organizational stress interventions are still relatively poorly studied and understood. They now represent the leading edge of occupational stress research and appear to hold promise for improved control of this problem.

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Mercury

MERCURY IS AN old occupational hazard that remains a problem in dental offices, health care institutions, and some homes.

Elemental mercury is a heavy metal that interferes with the sulfhydryl groups of proteins, particularly active sites of enzymes. The major manifestations of these chemical reactions are nephrotoxicity, primarily proteinuria and tubular necrosis, and neurotoxicity, which can be profound with high exposure. Cardinal symptoms of mercury toxicity include tremors, oral and gingival inflammation, ataxia, excessive and uncontrollable salivation, anorexia and weight loss, labile affect and irritability (erethism), pathologic shyness and avoidance of people, and acrodynia (erythema and painful desquamative dermatitis of the hands and feet). The deterioration of handwriting over time is often taken as one indication of subclinical effect. Persons with congenital mercury toxicity will have severe mental retardation and motor abnormalities, including disturbances in swallowing. Congenital organic mercury poisoning was perhaps most poignantly illustrated by the severe outbreak in Minamata, Japan, in the 1950s.

Organic mercury compounds are more rapidly absorbed than elemental mercury, especially by ingestion, and also are more toxic. These compounds are products of metabolism, accumulated in fish in mercury-contaminated rivers and estuaries, for example, and have been used as fungicides, particularly methylmercury on seeds. Organic mercury compounds are metabolized to mercury in the body, with the same toxicity as metallic mercury. Organomercurials were once used extensively as medications in the preantibiotic era and were responsible for many cases of toxicity. Organic mercury compounds are also used as preservatives in some latex paints and have also been associated with excessive exposure to mercury and clinical acrodynia.

Overt mercury toxicity has been reported in heavily contaminated dental offices, particularly where there has been an ineffective cleanup of spills. Most dental personnel are now aware of the hazard and often request blood mercury determinations as screening tests during physical examinations. There is also a debate over whether the minute amounts of mercury that vaporize from dental amalgams in patients may be toxic; the best evidence to date suggests that this is not the case. In health care institutions, mercury remains a part of many instruments. In the home, mercury exposure has been associated with spilling small quantities kept in the home for hobbies, as a by-product of efforts at home smelting, and as a constituent of interior latex paint.

Mercury vaporizes at room temperature. Inhalation can be substantial, especially if the mercury is heated at the source. A recent severe case of home exposure involved a quantity of mercury inadvertently spilled into a space heater that became a source of exposure for many months.

Mercury levels in blood or urine do not correlate with symptoms, probably because toxicity is intracellular and much circulating mercury is bound with protein. Cases of mercury poisoning, however, will show elevated levels. Relatively low levels of exposure, with urine or blood levels just outside the range of a reference population, have not been associated with demonstrable abnormalities; levels may vary by laboratory, with 24-hour yields from urine ranging from 0 to 50 nmol per liter. Hair analysis is not reliable for clinical diagnosis.

Treatment of serious mercury intoxication should be left to physicians with experience in clinical toxicology. Chela-