Mechanisms of Injury and Death Proximal to Restraint Use

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Restraints have been subsumed under the rubric of “interventions” in the nursing literature. However, alarming reports of lethal consequences proximal to their use raise the issue to a life and death matter that requires immediate attention from professionals. Little is known about the mechanisms of death and other adverse consequences of restraint use. This article describes these mechanisms and also considers some hypothesized factors and adverse effects associated with their use.

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Restraints are among the most coercive interventions available to psychiatric staff members. The modern practice of this procedure has a long history in psychiatric settings. Yet despite the remarkable progress in our understanding of the brain and human behavior, historical studies suggest that rates of restraint use in psychiatric institutional settings remain unchanged during the past centuries (Esther, 1997).

Physical restraint, as used in acute psychiatric settings, is a security measure designed to protect patients or staff. The term “restraint” refers to physical restriction of movement and is most commonly used to restrain limbs on a bed (“four-point” or “five-point” restraint). The term is also used to refer to restricting patients to a chair or to limit the movement of their arms or legs (“ambulatory restraint”).

Restraints are labeled “interventions” in the nursing literature. However, the extent to which restraints are therapeutic interventions is questionable. Their efficacy as therapeutic measures has not been empirically shown in any outcome studies to date (Singh, Singh, Davis, Latham, & Ayers, 1999). Alarming reports of lethal consequences proximal to the use of restraints requires immediate attention from professionals.

The practice of restraints puts patients and staff at great risk for injury and even death. Consequences related to the use of restraints can be both traumatic and lethal. Moreover, even when they do not result in injury and death, the practice of restraints and seclusion are not benign. Although the incidence and prevalence of deaths and injury are not collected (U.S. General Accounting Office, September 1999), the 1998 Hartford Courant investigation suggested that within the past 10 years 142 reported deaths were connected to the use of physical restraint in mental health settings (Weiss, 1998). Those who died were disproportionately young children. The newspaper exposé, as well as an exposé of conditions in some Charter facilities conducted by the Columbia Broadcasting System, prompted Congress to request an investigation by the U.S. General Accounting Office (September 1999). This investigation confirmed the risks inherent in the use of mechanical restraints. There has been little discussion regarding the sequelae associated with restraints and seclusion. Mental health professionals have not written or spoken up about this important topic, which has led to questions regarding the extent of the professional knowledge with respect to the restraints’ adverse effects. This article describes some of what is known about death in restraints and also considers some hypothesized factors and adverse effects as-
associated with their use. Because the use of restraint with adults has been widely discussed in the geriatric literature, children are the primary focus of this discussion.

CHILDREN AT SPECIAL RISK FOR TRAUMA

While caring for adult patients can be trying at times, children in psychiatric facilities are exceedingly vexing and difficult. They are wounded and mistrustful, and their main mode of coping is to lash out in attack. This can be frightening to inadequately prepared staff, and they can construe a verbal assault by a child in a personal way, especially if staff members do not have a ready arsenal of understanding, verbal skills, and self-awareness. These staff can lose all boundaries between their issues and the child’s issues. This is the essence of negative countertransference reactions, and its effects result in fear and paralysis in staff members who then tend to resort to survival and control tactics. This begins the aggression coercion cycle described by Goren, Singh, and Best (1993) in which staff members and patients provoke each other in a game of one-upmanship. Staff members who seek to control a frightening or uncertain situation engage in a power play. This places the child in a “no-win situation” and the potential for a take-down, restraint, and possible abuse and injury escalate.

Yet it is extremely important to understand the effects of restraints on children who are still in the process of development. Placing a child in restraints by way of a temporary therapeutic hold or in leather 5-point restraints strapped to a bed for hours is indisputably a highly aversive experience. It is also not a rational option given what we know about children who exhibit aggressive tendencies and disruptive behavior disorders.

Children who are placed in restraints most often suffer from some type of disruptive behavior disorder. Disruptive behavior disorders refer to a core set of behaviors and symptoms represented by the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994) categories that include attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder (CD). CD is a sufficiently disruptive condition that it accounts for up to 50% of consultations in psychiatric clinics (Fergusson, Horwood, & Lynskey, 1994; Steiner, 1997). CD is typified by a “repetitive and persistent pattern of behavior in which the basic rights of others and major age appropriate societal norms or rules are violated” (American Psychiatric Association, 1994, p. 85). Although debate as to the genesis of CD continues, scholars have found that children with this disorder share remarkably similar risk factors (Kazdin, 1993). One of these is a history of trauma or physical abuse or neglect, another is low verbal intelligence quotient and difficulties with receptive language (Moffitt, 1993; Oftnow-Lewis, 1992; Richters & Cicchetti, 1993; Wolff, Waber, Bauermeister, Cohen, & Ferber, 1982).

The interaction of traumatic exposure (abuse) to single or recurring violence within an environment of chronic danger is a relatively recent field of study (Cicchetti, 1989; Garbarino, Kostelnk, & Dubrow, 1991). However, scholars have developed some remarkable hypotheses and have conducted compelling research about what happens to children who experience chronic abuse, and those exposed to violence.

Researchers in this area (Perry & Pollard, 1998; Perry, Pollard, Blakley, Baker, & Vigilante, 1995; van der Kolk & Greenberg, 1987) posit that a child’s acute responses to threat can develop in a pattern based on their individual traumatic experiences. This response can be one of hyperarousal or dissociation. The hyperarousal response is an alarm reaction that begins to activate the sympathetic nervous system, resulting in stimulation of the hypothalamic pituitary axis. The dissociative response involves a variety of mechanisms in which the child disengages from the world attending to stimuli in their own internal world—a kind of defeat reaction from an unavoidable and painful situation. The mechanism behind this latter response is not well understood but it seems to begin with central nervous system activation, yet what follows is a marked increase in vagal tone with lowering of blood pressure and heart rate, despite increasing levels of circulating epinephrine. This response seems to be mediated by naturally occurring opioids (van der Kolk & Greenberg, 1987; Pynoos, 1993). Females and younger children are more apt to react along the dissociative continuum to severe stress than males and older children (Perry & Pollard, 1998).

Increasing evidence suggests that in children who experience chronic abuse, the adrenal system goes awry, causing an imbalance of the steroids in the brain (Perry, 1997; Perry & Pollard, 1998; Schwartz & Perry, 1994). Researchers believe that
excess cortisol leads to damage in the hippocampus, causing memory lapses, anxiety, and an inability to control emotional outbursts. Cortisol can also alter brain centers that regulate attention and affect a child’s capacity to attend to meaningful stimuli.

Abused children show a variety of disturbances in physiology, thinking, and behavior. Similar to children who have experienced any other traumatic stress, many have elevated resting heart rates, temperature, and blood pressure (Ontnow-Lewis, 1992; Pynoos, 1993). If they respond along the dissociative continuum, they may have lower heart rates and blood pressures (Perry & Pollard, 1998; Perry et al., 1995). Verbal deficits are one of the neuro-psychologic sources of abused children’s difficult behavior. In addition, several studies indicate that this misbehavior may be associated with deficiencies in the brain’s self-control functions, commonly referred to as “executive functions” (Moffitt, 1993; Moffitt & Henry, 1991; Newman, 1987). These functions include sustaining attention and concentration; abstract reasoning and concept formation; formulating goals; anticipating and planning; programming and initiating purposive sequences of behavior; self-monitoring and self-awareness; inhibiting unsuccessful, inappropriate, or impulsive behaviors; and interrupting ongoing behavior patterns to shift to a more adaptive alternative behavior.

After repeated exposure to trauma, the same systems initially activated in the brain by the original trauma will be reactivated when the child is merely exposed to reminders of the traumatic event. For example, these children may react physiologically to a playful scream heard on a playground in the same way they may have reacted to a scream heard during a violent altercation at home. These systems may also be reactivated in response to a child’s thoughts or dreams about the traumatic event. They may even begin reactivation when they are warned by an adult: “If you don’t do this, I will…” (Perry & Pollard, 1998, p. 8). Such stimulus reminders may generalize to other situations and lead to the repeated activation of the stress-response apparatus, although the child may be temporally and geographically distanced from any apparent threat. The helpless and frightened child begins to view the world through a filter (or template) of the original trauma and will respond accordingly (Cicchetti, 1995; Perry et al., 1995).

This template or filter is a result of the high levels of adrenergic and cortisone derivative substances that remain after the original trauma and are responsible for permanently altering the neurosynaptic junctions in a way that fixates memories and impulse response. Perry and colleagues (1995) refer to this event as an acute state that eventually becomes a permanent trait. In essence, without intervention, the children develop a disarming of the down-regulating circuit that would return them to more normal function.

HYPOTHEZIZED CUMULATIVE AND ADDITIVE TRAUMA

Although the study of life events is not as advanced in child psychiatry as in adult psychiatry, research has shown that noxious life events have a cumulative or additive effect on children. The effects of these events may be delayed, resulting in a lag of approximately 4 months (Goodyer, Kolvin, & Gatzanis, 1987).

As mentioned previously, research shows that traumatized children may suffer from language deficits, both receptive and expressive, and cognitive impairments that include distorting the intent of others. These contribute to a “present-oriented” cognitive style and limit their responses in threatening or ambiguous social situations. Because of their cognitive handicaps, these children are at a significant disadvantage in high stress situations in which demands are put on them to make “appropriate” decisions and remain rational (Dodge, Price, Bachorowski, & Newman, 1990).

These children may become aggressive and threatening and are subject to being “taken down” by staff and placed in restraints. A “take down” is a highly emotional and volatile situation that is highly stressful to both staff members and patients. Although it is not designed to be a punishment, it is often perceived this way by the children who experience it (Mohr, Mahon, & Noone, 1998). When a child exhibits threatening behavior, a team of staff members converge on the child intending to subdue and restrain him and/or administer medication. It follows that in such a situation the predominant emotions would be an escalation of galvanizing anger or paralyzing fear. Faced with the threat of being taken down by 5 or more persons larger than him or her, the child’s body will initiate a cascade of physiologic reactions. He or she experiences a dramatic increase in the brain regions
that are involved in the hyperarousal response induced by the threat (locus coeruleus and ventral tegmental nucleus activity). These regions play a critical role in regulating the stress response, arousal, vigilance, affect, attention, sleep, and the startle response. In addition, a corresponding increase in the release of catecholamines occurs in response to the threat. Catecholamines surge, causing tachycardia and hypertension while readying the muscles for action. At the same time corticosteroids are released in response to the perceived danger. The perceptual field narrows and the ability to process information decreases. This is especially critical in a child who already has difficulties in verbal receptive skills.

This situation repeats the abuse cycle. The act of restraint may constitute an event that appears to the child much like an instance of abuse. Given the child’s diminished judgement, caused by their mental illness and as a result of the high intensity stress of the “take down” situation, and given their impaired verbal competence, actions that lead to restraint could be as damaging to the child from a physiologic standpoint as any other noxious life event. It becomes one more layer of trauma.

Hypothesized Adverse Health Effects From Stress Studies

Patients report that being placed in restraints is a traumatic and stressful experience even when that stress is purely “psychologic” and does not involve physical injury (Gallop, McCay, Guha, & Khan, 1999; Mohr et al., 1998; Norris & Kennedy, 1992). In recent years researchers have produced an impressive amount of literature showing the effect of psychologic stress on the down-regulation of various aspects of the immune system. The mechanism for this is caused by the disruption of the communication pathways that link the nervous, endocrine, and immune systems under psychologic stress (Ader, Cohen, & Felten, 1995; Glaser, Rabin, Chesney, Cohen, & Natelson, 1999). Studies have shown that persons reporting high levels of psychologic stress also have a higher incidence and greater severity of illness (Cohen, Doyle, & Skoner, 1999).

Because of the obvious ethical constraints of studying the effects of experimentally induced immobilization stress, studies are conducted with animals. Scientists worldwide have been conducting research on the effects of immobilization on the physiologic functioning of rats. Researchers in Japan sought a link between the immune response and immobilization stress and found that neutrophil functioning was negatively affected as a result of the immobilization. Moreover, they found that immobilization affected blood cells and serum elements and damaged the host’s defense and physiologic functioning (Kuriyama, Oishi, Kakazu, & Machida, 1998).

Furthermore, scientists in France showed the effects of immobilization stress on sleep rebound in rats (Marinesco, Bonnet, & Cesplugio, 1999). Levels of reparative sleep following episodes of stress were significantly more impaired after longer periods of immobilization. Some of these changes are caused by endocrine systems. It is well-known that aldosterone and cortisol suppress the immune system, whereas melatonin appears to enhance it. Melatonin, a product of the pineal gland, helps to regulate the circadian rhythm and sleep-wake cycle. Although little is known about the specific functions of sleep, the literature suggests that it is important for the proper functioning of the host defense system (Benca & Quintas, 1997). As a result, researchers suggest that the deleterious effect on sleep precipitated by stress events could be responsible for the development of pathologies in humans.

Studies in this area are in their infancy and caution should be used when extrapolating from animal studies. However, these studies suggest that being placed in restraints and immobilized is not benign, and that restraints may not only be subjectively noxious but also physically harmful.

RESTRAINTS AND THEIR HYPOTHOSIZED ROLE IN DEATH

There is little consensus in the scientific literature concerning the causes of death proximal to the use of restraints. The psychiatric literature pays scant attention to this issue. The forensic and emergency research contain a number of studies on the subject, and the Joint Commission on the Accreditation of Health Care Organizations (JCAHO) has studied reports of restraint-related deaths. JCAHO officials reviewed 20 restraint-related deaths voluntarily reported under their sentinel event reporting procedure. In 40% of the cases the cause of death was asphyxiation, while strangulation, cardiac arrest, or fire caused the remainder (JCAHO, 1998). JCAHO determined that asphyxiation was
related to putting excessive weight on the back of the patient in a prone position, placing a towel or sheet over the patient’s head to protect against spitting or biting, or obstructing the airway when pulling the patient’s arms across the neck area. Among the deaths reported by the Hartford Courant as cases in which restraint or seclusion was a factor, the causes included asphyxia, cardiac complications, drug overdoses or interactions, blunt trauma, strangulation or choking, fire/smoke inhalation, and aspiration (Weiss, 1998).

Restraint Asphyxia
The most common cause of death, asphyxiation, is termed “restraint asphyxia” (Milliken, 1998; Reay, 1998). To date, studies conducted on this issue have varied as to population, but the most common reports concern adults who often have been in police custody and frequently involve the victim’s use of alcohol or some other illegal substance (Pollanen, Chiasson, Cairns, & Young, 1998). Admittedly the aforementioned presents a confounding factor in applying this research to the field of psychiatry. However, it could be posited that many patients receiving psychiatric care are under the influence of psychotropic medications, some which depress central nervous system activity.

Investigating the mechanism of unexpected deaths of otherwise healthy persons in restraint, Reay, Howard, Fligner, and Ward (1988) found that 9 of 10 subjects in their study experienced a prolonged recovery phase from exercise when they were restrained in a prone position. The mechanism for this might include restriction of thoracic respiratory movements, airway compromise, or the release of catecholamines during the physical exertion. Interestingly, Reay (1998) stated that the findings to support a diagnosis of restraint asphyxia can be meager to nonexistent, with no typical pathologic findings on autopsy. The only sources for a determination of restraint asphyxia are the historical events surrounding the physical struggle.

Positional Asphyxia
Positional asphyxia occurs when the body’s position interferes with respiration. Respiration in humans depends on the exchange of gases in the lungs, the patency of the airway, and the pump action of the muscles and bony thorax that are responsible for ventilating the lungs. This coordinated activity is mediated by the nervous system.

In the forensic literature, death from positional asphyxia was found to occur when individuals were placed in a position that did not allow adequate breathing, most often a prone position. This may have involved a restrictive or confining position, a simple flexion of the head onto the chest, a partial or complete external airway obstruction, or neck compression. Persons who later died were not able to disengage themselves from the physical or mechanical restraint causing the fatal position (Bell, Rao, Wetli, & Rodriguez, 1992; Howard & Reay, 1997; O’Halloran & Lewman, 1994).

Reay, Fligner, Stilwell, and Arnold (1992) reported that positional asphyxia occurred in 3 cases of individuals who were in transport by law enforcement personnel. These individuals were being transported in a prone position, and on autopsy they were found to have died as a result of respiratory compromise. All 3 persons investigated by Reay’s Los Angeles Medical Examiners Unit died as a result mechanical interference with respirations. Although fatal positional asphyxia has been documented in adults, it has not been in children. However, it could be argued that a child’s small size alone would be a significant factor in their increased susceptibility to death from this mechanism.

Death by Aspiration
The JCAHO (1998) posits that while restraining patients in the prone position may predispose them to suffocation, restraining patients in a supine position may predispose them to aspiration. Aspiration occurs when persons have decreased levels of consciousness, either as a result of their illness or secondary to medications. Patients may aspirate either through vomiting or regurgitation of gastric contents. Supine positions, during which patients are rendered immobile in conjunction with decreased or altered levels of consciousness, interfere with their ability to protect their airway. Death occurs as a result of asphyxia, acute pulmonary edema, or pneumonitis (Plantadosi, 1996).

Asphyxia Secondary to Neck Compression
Deaths resulting from neck compressions were investigated by Reay and Holloway (1982). Neck holds are a commonly used law enforcement technique taught to some law enforcement personnel as
a means of subduing suspects resisting arrest or to control combative or difficult to manage prisoners. One kind of neck hold is intended to impede the flow of blood in the carotid arteries. Although there is compensating collateral circulation from vertebral arteries through the Circle of Willis, occlusion of the blood flow in carotids can produce brain ischemia or carotid sinus stimulation, which causes bradycardia and hypotension. In extreme cases it can result in cardiac arrest (Reay & Eisele, 1982; Reay & Holloway, 1982).

In the second type of neck hold the intent is the occlusion of the airway itself by forearm compression. This kind of hold can collapse the trachea and, given enough pressure, can cause tracheal fractures. Given that the combative individual is trying to free himself, his struggle strengthens the force around his neck. The individual then becomes aware that they cannot breathe, which leads to further agitation and increasing demand for oxygen and intensifying the force even further. Death can result from cardiac arrest secondary to hypoxia (Reay & Eisele, 1982; Reay & Holloway, 1982).

**Blunt Trauma to the Chest**

Blunt trauma to the chest may occur during the restraint process. Myocardial concussion (commotio cordis) resulting from this trauma has been reported to cause sudden death, presumably by cardiac arrhythmia secondary to myocardial injury. Although rare, deaths have been primarily reported in children, probably because of thinness of their chest wall. Again the diagnosis is not made by autopsy, in that no morphologic changes are seen in the myocardium, but rather by history of the event surrounding the trauma (Boglioli, Taff, & Harleman, 1998).

**Catecholamine Rush**

In situations where children become increasingly agitated, followed by intense struggle with staff members, taken down to the ground, carried to a quiet room, and secured with restraints a massive release of adrenal catecholamines occurs. This response, mediated by the adrenal medulla, induces an output of epinephrine and norepinephrine that sensitizes the heart and produces rhythm disturbances (Lown, DeSilva, & Lenson, 1977). Neural and psychologic factors have been implicated as risk factors for ventricular arrhythmias and sudden death. For example, behavioral arousal and psychologic stress have been shown to induce malignant cardiac rhythm disturbances (Johnson, Pinto, Kirby, & Lown, 1992; Kirby, Pinto, Hottinger, Johnson, & Lown, 1991).

Neural integration of body functions takes place through a complex system of feedback loops when information from inside and outside the organism is catalogued by the brain. These pathways play a major role in causing sudden death in persons who find themselves in perilous situations. Moreover, the situations need not be perilous to precipitate cardiac arrhythmias. Events that do not compare in anxiety intensity to being restrained, such as making a speech in public, have been found to have profound effects on cardiac vagal control (Coumel, Rosengarten, Leclercq, & Attuel, 1982; Grossman, Watkins, Wilhelm, Manolakis, & Lown, 1996).

More than 2 decades ago Lown and colleagues (Lown et al., 1977) identified psychic stress as a mediating factor for advanced cardiac arrhythmias, and emotional extremes have been suggested in the literature as a triggering mechanism for sudden cardiac death (Engel, 1978). In a classic study Lown and colleagues (1977) systematically explored the relationship between psychologic stress that consisted of mental arithmetic, reading from colored cards, and recounting emotionally charged experiences. Such testing induced a significant increase in ventricular premature beat frequency in 11 of the total 19 patients. One patient experienced paroxysms of ventricular tachycardia.

**Psychotropic Medications, Muscarinic Receptor Antagonists, and Hyperpyrexia**

Sudden cardiac death caused by an abrupt change in clinical status of the patient is frequently caused by the onset of a lethal cardiac arrhythmia (Braunwald, 1997). A syndrome that has been linked to psychotropic medication is prolonged Q-T interval, which is a functional abnormality thought to be associated with neurogenic influences that cause lethal arrhythmias (Schwartz, Periti, & Malliani, 1975.) The prolonged Q-T syndrome may be hereditary or acquired (Braunwald, 1997). Hereditary forms appear to be related to sympathetic nervous system imbalance (Schwartz et al., 1975). Among the many causes of the acquired form is the presence of a psychotropic drug idiosyncracies. Lithium carbonate may prolong the Q-T interval and has been reported to be associated with an increased incidence of sudden death (Ly-
man, Williams, Dinwoodie, & Schocken, 1984). Because certain psychotropic medications can cause Q-T prolongation, the stress of being placed in restraints in conjunction with the effects of these medications may be deadly.

Another example of potential complications caused by psychopharmacotherapeutics is the use of psychotropic agents with anticholinergic properties. Compounds with anticholinergic properties act by inhibiting the action of acetylcholine at autonomic effector sites innervated by postganglionic cholinergic nerves, and at presynaptic and postsynaptic receptors of neurons (Hyman & Nestler, 1993). Antihistamine agents, phenothiazines, tricyclic antidepressants, and some anti-Parkinsonian medications also possess this muscarinic receptor blocking activity (Hyman, Arana, & Rosenbaum, 1995). Children are more susceptible to the adverse effects of anticholinergic drugs. These range from dryness of skin and mucous membranes, mild tachycardia, constipation, urinary hesitancy to more severe signs, such as urinary retention, agitation, motor restlessness, confusion, myoclonus, delirium, fever, and seizures (Watemberg, Roth, Alehan, & Epstein, 1999).

One of the systemic effects of these medications is the attenuation of normal body cooling mechanisms. Children with extreme agitation who are struggling with staff and against restraint have decreased ability to discharge or release the heat generated by this increased activity. Body temperature may increase and a serious and potentially life-threatening hyperpyrexia may result.

The Possible Role of Rhabdomyolysis

There is no specific evidence that rhabdomyolysis occurs in situations of restraint, but this possibility has not been investigated either. However, a review of the literature yields data that might lead to future investigations. Rhabdomyolysis is a disorder that occurs both as a primary condition secondary to hereditary metabolic or structural abnormalities of the skeletal muscle cell, or as a complication of various disease states. However, the majority of the cases occur in otherwise healthy persons as a result of strenuous exertion, infections, intoxications, deficiency states, prolonged stasis, or trauma (Knochel, 1993; Manchip & Hurel, 1995). Rhabdomyolysis (lysis of skeletal muscle cells) is a potentially lethal condition that has a broad spectrum of clinical and biochemical findings. Current evidence suggests that some noxious factor injures the plasma membrane of the cell, which interferes with its metabolism and disrupts the integrity of the skeletal muscle cells. Common features of this condition are myalgia, pigmenturia, and increased serum creatinine kinase. Rhabdomyolysis is also associated with severe metabolic disturbances and involvement of other organ systems. Major complications are acute renal failure and death (Poels & Gabriels, 1993).

Some reports and studies on rhabdomyolysis might shed light on some of the unexplained deaths that have been associated with the use of restraints. Manchip and Hurel (1995) reported a case of rhabdomyolysis in a man medicated with lorazepam who was suffering from an acute manic episode, which they attributed to excessive exertion and dehydration. Physicians in Germany reported a case of rhabdomyolysis in a 19-year-old healthy man following his completion a 15-km march. This young man developed acute respiratory failure, renal failure, and consumption coagulopathy. He required 8 days of intensive care treatment (Derstappen, Mathias, & Losse, 1995). Likewise the condition was reported in 5 otherwise healthy sailors and marines who had begun a vigorous exercise program 1 to 3 days before the development of symptoms (Brown, Elliott, & Sray, 1994). Rhabdomyolysis was also reported in a 21-year-old man who performed more than 100 deep knee bends (Frucht, 1994).

Hyponatremia and benzodiazepines, the use of chlorpromazine, and full-sheet restraint were reported as contributing factors in the development of rhabdomyolysis (Fernandez-Real, Ricard-Engel, Camafort-Babkowski, 1994; Jermain & Crismon, 1992). Finally, Mercieca and Brown (1984) reported a case of rhabdomyolysis in an otherwise healthy patient who was experiencing a psychotic reaction secondary to the use of a hallucinogen and had been restrained in a straitjacket.

Nursing Implications

Clearly further research must be conducted on this important issue. But the information that we gleaned from the literature suggests that nurses are obliged to recognize that restraining patients is not a benign event from a physiologic standpoint. It suggests that well-educated psychiatric nurses need to take back psychiatric nursing practice. By this the authors mean that a prerequisite of caring
for vulnerable patients under psychiatric care is to be with those patients. Patients deserve knowledgeable caretakers with the ability to recognize a potentially escalating situation, intervene in that situation, and monitor patients for adverse effects related to the situation and the intervention.

Yet as far back as 1973, Rosenhan found that mental health aides spent an average of 11.3% of their time outside of the nursing station and that nurses and physicians spent even less time interacting with patients. In a more recent study Aragon and Holmes (1990) observed that conversations between adult psychiatric patients and staff occurred most often when patients stood immediately outside of the nursing station. They also found that staff members were engaged in direct care and engagement with patients an average of 27.55% and in indirect care, such as charting, an average of 72.45%. These 2 studies suggest that staff members may have insufficient contact with patients to make a meaningful assessment or even to observe the initial signs of impending violent behavior. They also suggest that the least educated staff members have the most patient contact.

The review of the literature also suggests that while waiting for further research on this issue, the psychiatric nursing community is obliged to identify a common approach on which to base practice with potentially violent patients. The National Association of State Hospital Program Directors (NASHPD, October 1999) has developed such an approach that is based on a public health model. Although an in-depth discussion is beyond the scope of this article, in short, such a model addresses (1) primary prevention (preventing and reducing the need for seclusion and restraint); (2) secondary prevention (early intervention, using the least restrictive methods possible); and (3) tertiary prevention (intervention to reverse or prevent negative consequences). The model also suggests that feedback from each stage should be used to inform and improve subsequent actions, and will always lead to the selection and use of the least possible restriction consistent with the purpose of the intervention.

Based on suggestions in the literature, nurses practicing in settings with high seclusion and restraint use would also benefit their patients, their fellow staff members, and themselves by obtaining mandatory consultation with a behavior modification specialist. Donat (1998) found that such consultation could help reduce seclusion and restraint use by more than 60%.

Finally, moving beyond the subject of restraints per se, this review of the literature points out that “mental states” have their representation in brain neuronal functions. It also underscores the remarkable plasticity of the brain and other parts of the nervous system at all levels of organization. It points out that each environmental influence results in brain changes and that the days of the “mind” as something disconnected from body are long gone. Advances in the understanding of the brain-body connection and the effects of the environment on the brain are exciting new knowledge. This knowledge comes with a responsibility and mandate for psychiatric nursing to learn and incorporate it into both education and practice.

The psychiatric nursing community is encouraged to base their practice, their textbooks, and their educational programs on these current and relevant research findings. McCabe (2000) called on the specialty to identify their core content and divest itself of outdated and empirically unsupported practices. As she points out, nursing textbooks that still contain theories and unsupported practices in empirical study do not enhance the credibility or the future of the specialty.

CONCLUSION

The purpose of this article was to bring together an extensive literature review to present an overview of some hypothesized and established complications of restraint use. These range from injury to immune systems and patient trauma to death. These authors conclude that given the lack of discussion of this issue in nursing texts and journals, the extant literature concerning the use of restraints and seclusion does not currently represent a body of knowledge on which clinicians can continue to use this security measure uncritically. Clearly research is needed that will provide clinicians with data that will effect a reduction in these coercive measures.

Finally, to the extent that caring for human beings and protecting them from harm is part of the ethical codes of many health care professionals, there are ethical and moral elements to the issues of restraints. Consistent with the principle of beneficence and the obligation to do no harm, professionals are obliged to minimize any unintended adverse consequences of any intervention. In view
of evidence of restraint’s deleterious effects, their continued use in circumstances where less restrictive or intrusive alternatives are reasonably available to accomplish the legitimate goal of child safety or the safety of others violates the principles of beneficence and nonmaleficence (Moss & La Puma, 1991; Strumpf & Evans, 1991).

REFERENCES


