ANNUAL REVIEW OF NURSING RESEARCH

Volume 11, 1993



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ANNUAL REVIEW OF NURSING RESEARCH

Volume 11, 1993

Joyce J. Fitzpatrick, Ph.D. Joanne S. Stevenson, Ph.D. Editors



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Preface

This eleventh volume of the Annual Review of Nursing Research (ARNR) series marks the beginning of our second decade of publication. The 1980s was an era of expansion in nursing scholarship. It is our expectation that in the coming decade we will witness an increase in both the depth and breadth of nursing research. As this occurs, the content areas under review should be more clearly and narrowly defined. Chapters in Volume 11 represent an initial effort toward this goal.

In Part I, Research on Nursing Practice, we have included a focus on patient/client symptoms. Marquis Foreman reviews research on acute confusional states; Barbara Holtzclaw describes research on shivering; Kathleen Potempa's review is focused on research of chronic fatigue; Marylin Dodd reviews research on side effects of cancer chemotherapy; and Nancy Hester describes research on pain in children.

Part II, Research on Nursing Care Delivery, includes the following chapters: Patient Care Outcomes Related to Management of Symptoms by Sue Hegyvary, and the Role of Nurse Researchers Employed in Clinical Settings by Karin Kirchhoff. The Nursing Education section, Part III, includes a chapter by Claire Andrews and Carol Davis on nurse-midwifery education. Part IV, Research on the Profession, includes a chapter by Joan Turner on AIDS-Related Knowledge, Attitudes, and Risk for Infection Among Nurses.

Part V serves as a category for chapters that do not easily fit the content theme of Part I or the categories included in the other components. In this volume, Part V includes: Family Unit Focused Research by Ann L. Whall and Carol J. Loveland-Cherry; Opiate Abuse in Pregnancy by Cathy Strachan Lindenberg and Anne B. Keith; Alcohol and Drug Abuse by Eleanor J. Sullivan and Sandra M. Handley; and Patient Falls in Health Care Institutions by Janice M. Morse.

A new Advisory Board has participated in launching the new decade. We are pleased to welcome new Board members Violet Barkauskas, Marie Cowan, Claire Fagin, Suzanne Feetham, Phyllis Giovannetti, Kathleen McCormick, Jane Norbeck, and Christine Tanner. Continuing Advisory Board members Ada Sue Hinshaw and Harriet Werley will provide continuity from the previous Board. Roma Lee Taunton, coeditor of volumes 4 through 10, now joins us as an Advisory Board member. Our Advisory Board members play a major role in setting directions for the future, as well as recommending authors, chapters, and reviewers for each volume. Joanne S. Stevenson has joined me as a coeditor for Volumes 11 through 13.

Although there are many new ARNR team members working with us to start the new decade, we would be remiss in not acknowledging the ongoing support of key staff members at Case Western Reserve University. Nikki Polis continues as a member of the editorial staff, and we have the assistance of a number of support staff members.

As a celebration of the first decade of the ARNR series, a special International State of the Science Congress was held in Washington, DC, in August 1992. Nurse leaders from around the world joined together; examplars of the significant strides in nursing research were presented in several plenary sessions. A special volume in the ARNR series, Proceedings of the State of the Science Congress, will be published in 1994. Because the Congress was cosponsored by several national and regional professional nursing organizations, the Proceedings' publication will include a broader scope of content, with particular emphasis on nursing research and its clinical applications.

We look forward to your continuing involvement in this important series. Please let us know your ideas.

JOYCE J. FITZPATRICK Senior Editor

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PART I

Research on Nursing Practice

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Chapter 1

Acute Confusion in the Elderly

Marquis D. Foreman

College of Nursing University of Illinois at Chicago

CONTENTS

Conceptual Issues in the Study of Acute Confusion Beliefs About Acute Confusion Lexicon of Acute Confusion Nature of Acute Confusion Clinical Issues in the Study of Acute Confusion Methods for Detecting Acute Confusion Etiologic Basis of Acute Confusion Epidemiology of Acute Confusion Outcomes of Acute Confusion Care of Patients Who Are Acutely Confused Lived Experience of Acute Confusion Directions for Future Research

The history of acute confusion dates back 2,500 years to the writings of Hippocrates (Lipowski, 1990), but knowledge about the condition remains incomplete. Too often acute confusion in elderly individuals has been ignored. Elderly patients have at times been perceived as uninteresting, unimportant, unworthy, and beyond help (Foreman, 1986; Francis & Kapoor, 1990; Lipowski, 1990). Historically, aging has been considered synonymous with cognitive decline; thus, the occurrence of acute confusion was thought inevitable and beyond the influence of health care professionals. Additional-

ly, the distinction was not made between acute reversible conditions and chronic states with permanent impairment. During the past 15 years, the notions and attitudes about the elderly and acute confusion have changed. As a result, there has been increasing attention to this condition within the clinical and research literature.

Clinical anecdotes about acutely confused patients and debates about nomenclature proliferate in the literature. The research about acute confusion is primarily descriptive in nature; experimental investigations about the causes and treatment of acute confusional states are few. The purposes of this review are to (a) critically review the research on acute confusion in the elderly; (b) summarize and integrate this literature, (c) highlight unresolved issues in the study of acute confusion, (d) identify gaps in the knowledge of this condition, (e) specify the implications of this knowledge for nursing practice, and (f) recommend future directions for research.

Literature for this review was identified using multiple techniques: (a) ancestry (tracking citations from publications), (b) computerized abstracting services, (c) hand bibliographic searches of Dissertation Abstracts and Masters Abstracts, and (d) written and telephone communication with investigators (internationally) known to have studied or be studying within this substantive area. The substantive area was defined as encompassing any of the following terms: acute brain failure, acute brain syndrome, acute cerebral insufficiency, acute mental status change, acute organic psychosis, acute organic reaction, acute organic syndrome, agitated confusional state, altered mental status, cerebral insufficiency syndrome, delirium, dystergastic reaction, exogenous psychoses, intensive care unit delirium, intensive care unit psychosis, metabolic encephalopathy, organic brain syndrome, postcardiotomy delirium, postcardiotomy psychosis, pseudosenility, reversible cognitive dysfunction, reversible dementia, reversible toxic psychosis, subacute befuddlement, sundown syndrome, toxic confusional state, toxic delirious reaction, toxic encephalopathy, toxic-metabolic encephalopathy, toxic psychosis, or transient cognitive impairment.

Criteria for inclusion in this review stipulated that the source had to be (a) research or data based, (b) written in English, and (c) conducted using adult samples. Most samples, however, were of older adults (\geq 65 years of age). Because impaired cognition attributable to alcohol ingestion or intoxication is by definition different from the phenomenon of interest (delirium vs. delirium tremens) this literature was excluded from review. Both published and unpublished works were reviewed. The literature resulting from these searches and sampling strategies is reviewed subsequently. To facilitate this review, the literature about acute confusion was divided into two foci: conceptual and clinical. Topics related to each focus are discussed.

CONCEPTUAL ISSUES IN THE STUDY OF ACUTE CONFUSION

Conceptual issues in the study of acute confusion pose serious obstacles to integrating the body of information into a comprehensive body of knowledge. The main conceptual issues include (a) beliefs about acute confusion, (b) the lexicon, and (c) the nature of acute confusional states.

Beliefs About Acute Confusion

Beliefs are foundational to the study of any phenomenon. Beliefs influence the perception of a phenomenon, and, consequently, determine what are considered legitimate and significant areas of study relative to that phenomenon. Beliefs also can facilitate or obscure the understanding of a phenomenon. Historically, the beliefs about acute confusional states in the elderly have served to delay understanding.

Although it has been demonstrated that cognitive decline is not an inevitable concomitant of aging (Rowe & Khan, 1987), cognitive decline remains an expectation of aging (Brady, 1987), as acutely confused behavior fits the stereotype many individuals have of the elderly. Consequently, it is only recently that cognitive decline generally and acute confusion specifically have been considered legitimate areas of study (Foreman, 1986).

Beliefs, as reflected by perceptions of acute confusion, were examined by Wolanin (1973), who reviewed descriptions of the behavior of acutely confused patients found in hospital records of older patients. Wolanin found that descriptions of acutely confused behavior varied by discipline. Physicians used terms such as "poor historian," "forgetful," "poor memory," "cannot understand directions," and "incoherent" to describe acutely confused patients. Wolanin interpreted these behaviors as indicators of impaired intellectual functioning or indicators of cognitive inaccessibility—a characteristic of these patients interfering with the physician's instrumental function of diagnosis. Conversely, nurses noted behaviors such as "uncooperative," "combative," "hostile," "difficult to manage," "belligerent," and "agitated" to describe patients, many of whom may have been acutely confused. The problem with the chart review method is that it is not possible to know if these patients were or were not acutely confused.

Conversely, the attitudes of the professionals in using the language noted in the chart reviews seemed concerned primarily with their compromised ability to function as a professional rather than with the patients. Others have noted this professional ethnocentrism as well (Ludwick & Scott, 1991; Ludwick, Scott, & O'Toole, 1991; Morgan, 1985).

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Another pervasive belief is that acute confusion equates with agitation, hyperactivity, and uncooperativeness (Lipowski, 1983b). One consequence of this association is that hypoactive and cooperative confused patients escape detection, treatment, and study. Yet, by definition, it is agreed that the behavioral manifestations of acute confusion vary between the extremes of hypoactivity and hyperactivity (American Psychiatric Association, 1987; Foreman, 1991; Liposwki, 1983a). Recent evidence suggests that these behavioral manifestations of acute confusion are a function of the etiologic agent(s) and not the acute confusional state per se (Neelon, Champagne, & Moore, 1989). It seems evident that for advancement to be made in the understanding of this phenomenon, the beliefs about acute confusion must be examined critically.

Lexicon of Acute Confusion

Nomenclature. The study of acute confusional states is confounded by its lexicon (Foreman, 1986; Francis & Kapoor, 1990; Lipowski, 1983a). A long-standing debate has existed as to which term—*acute confusion, de-lirium,* or *transient cognitive impairment*—is the diagnostic label that would lead to appropriate diagnosis and treatment of this condition. As noted earlier, numerous overlapping, and inconsistently used and defined terms are used interchangeably as synonyms. Whether the differences among these terms are ones of semantics or subtle variations in phenomena awaits data and interpretation (Foreman, 1991).

Throughout this chapter *acute confusion* will be used. This term requires no translation for bedside practitioners, and does not connote etiology. Additionally, the term acute confusion represents more closely what is observed clinically by nurses—a syndrome versus a single disease state (Vermeersch, 1991).

Definition of Terms. In this review the following terms are defined:

- Acute confusion is a transient state of cognitive impairment: it is a syndrome manifested by simultaneous disturbances of consciousness, attention, perception, memory, thinking, orientation, and psy-chomotor behavior that develop abruptly and fluctuate diurnally (Foreman, 1991). The primary deficit is one of attention.
- Cognition is comprised of three components: perception, memory, and thinking (Lipowski, 1983a).
- *Cognitive impairment* is a class of states of dysfunctional cognition of which there are two main types: (a) global cognitive impairment in which all three components of cognition are simultaneously impaired

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(e.g., delirium); and (b) specific global impairment in which only one or predominately one component of cognition is impaired (e.g., amnestic disorder or organic hallucinosis) (Lipowski, 1990).

- *Delirium* is a transient state of global cognitive impairment. The diagnostic criteria for delirium are (a) reduced ability to maintain attention to external stimuli and to shift appropriately attention to new external stimuli; (b) disorganized thinking; and (c) at least two of the following: (a) reduced level of consciousness; (b) perceptual disturbances; (c) disturbance of the sleep-wake cycle; (d) increased or decreased psychomotor behavior; (e) disorientation to person, place, or time; or (f) memory impairment. These clinical features of delirium develop over a short period (usually hours to days) and tend to fluctuate diurnally (American Psychiatric Association, 1987).
- *Mental status* is the thinking sphere of behavior including sensorium and cognitive functions. Components of mental status include state of consciousness, attention, orientation, memory, calculation, abstraction, judgment, insight, use of language, general knowledge, personal appearance, and thought form, content, and process (Foreman, 1987; Fraser, 1988; Strub & Black, 1985).

The Nature of Acute Confusion

The major feature of acute confusion appears to be its transiency. Because this is a post hoc determination, differentiating acute confusion from other more chronic forms is problematic. Although consensus regarding nomenclature is lacking, there is agreement about the fundamental nature of acute confusion (American Psychiatric Association, 1987; Foreman, 1986, 1989, 1991; Nagley & Dever, 1988; Williams et al., 1979, 1985a, 1985b). The onset is acute or subacute depending on cause, whereas the course is short with diurnal fluctuations in symptoms. It gets worse at night and on awakening, and lasts from several hours to less than 1 month. Patients have reduced awareness. impaired attention span, and fluctuating levels of alertness. They exhibit impaired orientation and memory; thinking is disorganized, and perceptions are distorted. They are distracted easily, have disturbed or reversed sleepwake cycles, and may be hyperkinetic or hypokinetic. (Foreman, 1986). The features of the condition are useful for differentiating acute confusion from chronic forms of cognitive impairment, such as the dementias (Foreman, 1986; Foreman & Grabowski, 1991). However, validation of these clinical features has received limited scientific study (Foreman, 1991).

Foreman (1991) identified five dimensions of acute confusion that approximated those identified in the literature. The five factors (cognition, orientation, motoric behavior, memory, and higher integrative functions) accounted for only one-third of the variance in confused behavior, indicating that there is much more to acute confusion than is represented by these five factors. However, these five factors were extremely sensitive and specific in identifying a state of acute confusion (Foreman, 1991), whereas those aspects most often used by practicing nurses—alertness and orientation (Brady, 1987; Rasmussen & Creason, 1991)—were not diagnostic of acute confusion (Foreman, 1991).

The variable manifestations of acute confusion across individuals and the fluctuating nature of the condition within one person across time increases the complexity of the phenomenon (Foreman, 1986). There is some speculation that this variability is a function of the underlying etiologic agent(s), or that there are subtypes or different patterns of acute confusion (Lipowski, 1983a; Neelon et al., 1989). Lipowski (1983a) described three variants of acute confusion based on the behavior, verbal and nonverbal, exhibited by the individual. The three variants are hyperkinetic, hypokinetic, and mixed. The hyperkinetic variant is the "classic" representation of the acutely confused patient and is characterized by hyperarousal of the autonomic system, psychomotor hyperactivity, marked excitability, and a tendency toward hallucinations. Conversely, the hypokinetic variant is often misdiagnosed as it conflicts with the stereotypic notion of acute confusion. The hypokinetic variant is characterized by reduced psychomotor activity, lethargy, somnolence, apathy, and reduced arousal and excitability. The mixed variant involved a fluctuating state between these two variants.

Neelon et al. (1989) identified three patterns of acute confusion, with manifestations reflecting underlying causal agents. The first pattern, "cognitive restricted," results from environmental challenges such as sensory deprivation or overload typical of acute care hospitals. The second pattern, "physiologic instability," has fluctuating symptomatology and arises from pathophysiologic states such as hypoxemia. The third pattern, "metabolic instability," is manifested by motor symptoms typically observed in encephalopathies and results from the toxic challenges of impaired hepatic or renal function, or from the adverse effects of multiple pharmacologic agents (prescribed and over the counter).

Obtaining knowledge about the nature of this condition is obstructed by designs that fail to reflect its central aspects. For example, many study designs were cross-sectional and rarely included multiple daily observations of patients, yet diurnal fluctuations of behavior are a hallmark of acute confusion.

The literature on acute confusion is largely atheoretic; one exception is the work of Neelon et al. (1985, 1989, 1991, 1992) who applied a human information-processing approach to the study of acute confusion.

Cumulatively, the beliefs, lexicon, and nature of acute confusion pose serious obstacles to the development of knowledge about this problem (Foreman, 1991; Lipowski, 1983a, 1990; Vermeersch, 1991). The professional focus of the beliefs, the semantic muddle, and the largely atheoretic study of acute confusion have led to what has been referred to as "conceptual chaos" (Foreman, 1991). This state of conceptual chaos undermines the search for knowledge about acute confusion. For knowledge development and innovation in patient care issues to progress, conceptual clarity must occur.

CLINICAL ISSUES IN THE STUDY OF ACUTE CONFUSION

The clinical issues in the study of acute confusion pose serious obstacles to the application to practice and further generation of knowledge of the condition. The clinical issues discussed in this section include (a) methods for detecting acute confusional states, (b) the etiologic basis of acute confusion, (c) the epidemiology of acute confusion, (d) outcomes of acute confusion, and (e) the care of patients who are acutely confused or have the potential to be.

Methods for Detecting Acute Confusion

Detection of acute confusion is often the only indication of physical illness in the elderly (Foreman, 1984, 1986; Francis & Kapoor, 1990; Lipowski, 1990). Therefore, it is important to diagnose. However, most cases of acute confusion escape detection (Cameron, Thomas, Mulvihill, & Bronheim, 1987; Gehi, Weltz, Strain, & Jacobs, 1980).

The haphazard and incomplete assessment of cognitive function by physicians and nurses leads to underdetection of acute confusion. In studying nurses, 43% (Lucas & Folstein, 1980) and 72% (Palmateer & McCartney, 1985) of the cases of acute confusion were not identified in hospitalized elders. Similar findings exist with samples of physicians. Thirty percent (DePaulo & Folstein, 1978) to 79% (Garcia, Tweedy, & Blass, 1984; McCartney & Palmateer, 1985) of cases were not recognized. These investigators concluded that the underdetection occurred because clinicians did not use standardized methods of cognitive assessment. No study was located that tested the hypothesis that routine use of standardized and systematic methods of evaluation improved the accuracy and timeliness of detecting acute confusion.

Methods have been developed to detect accurately and promptly patients

who are acutely confused. However, there are competing requirements of maintaining psychometric rigor while maintaining clinical feasibility and patient acceptability. Some maintain that an acceptable instrument must be a screen and also usable to monitor cognitive function over time (Foreman, 1991; Vermeersch, 1991). However, it might be better to have two valid instruments rather than one.

Instruments currently used for detecting acute confusion fall into the following categories: (a) mental status questionnaires, (b) symptom checklists, (c) clinical interviews, and (d) psychomotor tests (Fraser, 1988; Levkoff, Liptzin, Cleary, & Evans, 1991; Nelson, Fogel, & Faust, 1986).

Mental status questionnaires, also known as bedside cognitive screening instruments are preferred by physicians. Several variants of the mental status questionnaire (Kahn, Goldfarb, Pollack, & Peck, 1960) were designed to have these qualities: sufficiently sensitive to detect a minor problem in cognitive function, sufficiently specific to exclude the fringes of normal functioning, able to characterize the specific nature of the impairment (e.g., acute confusion vs. dementia vs. depression), while remaining clinically feasible (i.e., acceptable to the patient, and quick and easy to administer and interpret by clinicians). The most frequently used mental status questionnaires are Pfeiffer's (1975) Short Portable Mental Status Questionnaire (SPMSQ), Folstein's (Folstein, Folstein, & McHugh, (1975) Mini-Mental State Examination (MMSE), and Jacobs' (Jacobs, Bernhard, Delgado, & Strain, 1977) Cognitive Capacity Screening Examination (CCSE). Although these questionnaires are considered the best available, only a dichotomous discrimination of patients is possible (i.e., discrimination between impaired and not impaired). Finer discriminations are not reliable (Foreman, 1987; Smyer, Hofland, & Jonas, 1979).

Numerous validation studies (see Table 1.1) of these instruments have been conducted with samples of individuals of varying racial (white, black, Hispanic); residential (community residing, hospitalized and institutionalized); health status (healthy, or with medical, neurologic, or psychiatric illnesses); and socioeconomic backgrounds. Findings of these studies made clear that the diagnostic precision of mental status questionnaires is limited (Anthony, LeResche, Niaz, Von Korff, & Folstein, 1982; Levkoff et al., 1991; Smyer et al., 1979; Wolber, Romaniuk, Eastman, & Robinson, 1984). It is difficult to establish whether this lack of diagnostic precision was a function of the properties of the instruments (i.e., of invalidity and unreliability) or of the acute confusion (i.e., of instability).

Also of concern with mental status questionnaires is the consolidation of findings into a composite or total score. This procedure reduces information and implies that attentional, memory, language, and other deficits are all equivalent. To surmount this concern, Kiernan, Mueller, Langston, and Van Dyke (1987) developed the Neurobehavioral Cognitive Status Examination

 Table 1.1
 Validation Studies of the Various Instruments for Detecting

 Acute Confusion
 Figure 1

Short Portable Mental Status Questionnaire (Pfeiffer, 1975) Anthony, LeResche, Niaz, Von Korff, & Folstein, 1982 Dalton, Pederson, Blom, & Holmes, 1987 Erkinjuntti, Sulkava, Wikstrom, & Autio, 1987 Fillenbaum & Smyer, 1981 Foreman, 1987 Haglund & Schukit, 1976 Pfeiffer, 1975 Smyer, Hofland, & Jonas, 1979 Wolber, Romaniuk, Eastman, & Robinson, 1984 Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975) Bird, Canino, Stipec, & Shrout, 1987 Bleecker, Bolla-Wilson, Kawas, & Agnew, 1988 Dick, Guiloff, Stewart, Blackstock, Bielawska, & Marsden, 1984 Escobar, Burnam, Karno, Forsythe, Landsverk, & Golding, 1986 Fillenbaum, Hughes, Heyman, George, & Blazer, 1988 Folstein, Folstein, & McHugh, 1975 Foreman, 1987 Jorm, Scott, Henderson, & Kay, 1988 Magaziner, Bassett, & Hebel, 1987 O'Connor, Pollitt, Hyde, Miller, Brook, & Reiss, 1989 O'Connor, Pollitt, Treasure, Brook, & Reiss, 1989 Paveza, Cohen, Blaser, & Hagopian, 1990 Teng & Chui, 1987 Teng, Chui, Schneider, & Metzger, 1987 Cognitive Capacity Screening Examination (Jacobs, Bernard, Delgado, & Strain, 1977) Foreman, 1987 Gehi, Weltz, Strain, & Jacobs, 1980 Jacobs, Bernard, Delgado, & Strain, 1977 Kaufman, Weinberger, Strain, & Jacobs, 1979 Omer, Foldes, Toby, & Menczel, 1983 Strain, Fulop, Lebovits, Ginsberg, Robinson, Stern, Charap, & Gany, 1988 Webster, Scott, Nunn, McNeer, & Varnell, 1984 Neurobehavioral Cognitive Status Examination (Kiernan, Mueller, Langston, & van Dvck, 1987) Kiernan, Mueller, Langston, & van Dyck, 1987 Schwamm, van Dyck, Kiernan, Merrin, & Mueller, 1987 Confusion Assessment Method (Inouye, van Dyck, Alessi, Balkin, Siegal, & Horwitz, 1990) Foreman, Pompei, Lee, Ross, & Rudberg, 1991 Inouye, van Dyck, Alessi, Balkin, Siegal, & Horwitz, 1990

Table 1.1(continued)

Clinical Assessment of Confusion-A (Vermeersch 1990) Foreman, 1989, 1991 Foreman, Pompei, Lee, Ross, & Rudberg, 1991 Kautz, Cheung, & Walker, 1991 Vermeersch, 1990 Neecham Confusion Scale (Neelon, Champagne, & McConnell, 1985) Neelon, Champagne, & McConnell, 1985 Neelon, Champagne, McConnell, 1985 Neelon, Champagne, McConnell, Carlson, & Funk, 1991, 1992 Miller, 1991 Siemsen, Miller, Newman, & Lucas, 1992

(NCSE) in which the multiple domains of cognitive function are evaluated independently, and a profile of function is generated. The NCSE provides greater and more specific information to the clinician; a cognitive profile is generated of the individual's cognitive abilities and disabilities that is said to be specific to the neuropsychiatric condition (e.g., senile dementia of the Alzheimer's type or hepatic encephalopathy) (Kiernan et al., 1987). Psychometric studies are acceptable (Kiernan et al., 1987; Schwamm et al., 1987), and results are promising. Additional limitations of the NCSE are that testing requires greater time to complete than other mental status questionnaires and poses considerable burden for the more impaired.

Other concerns about mental status questionnaires are that (a) performance is strongly influenced by age, educational level, ethnicity, and language (Bird, Canino, Stipec, & Shrout, 1987; Bleecker, Bolla-Wilson, Kawas, & Agnew, 1988; Escobar, Burnam, Karno, Forsythe, Landsverk, & Golding, 1986; Fillenbaum, Hughes, Heyman, George, & Blazer, 1988; Folstein et al., 1975; Jorm, Scott, Henderson, & Kay, 1988; Magaziner, Bassett, & Hebel, 1987). Older, more poorly educated minority persons whose primary language is not English perform less well; (b) distinction cannot be made between acute and chronic impairment; (c) all aspects of acute confusion are not measured by mental status questionnaires (e.g., psychomotor behavior, perceptual disturbances); and (d) responses are heavily verbal, and, thus cannot be used with nonverbal persons (e.g., those intubated and aphasic) (Foreman, 1987; Levkoff et al., 1991; Smyer et al., 1979; Wolber et al., 1984). More recently, Levkoff and colleagues (1991) and Vermeersch (1991) have questioned the validity of mental status questionnaires because they are based on antiquated (e.g., organic brain syndromes) rather than on contemporary (e.g., transient cognitive impairment) conceptualizations.

Behavioral, psychomotor, and symptom rating scales were developed to

surmount many of the preceding identified limitations of mental status questionnaires. Instruments in these categories depend on the observations rather than the testing of acutely confused patients. Several instruments in this category exist: (a) the Confusion Rating Scale (Williams, Ward, & Campbell, 1988); (b) the Clinical Assessment of Confusion—A (Vermeersch, 1990); (c) the Delirium Rating Scale (Trzepacz, Baker, & Greenhouse, 1988); (d) the Confusion Assessment Method (Inouye, van Dyke, Alessi, Balkin, Siegal, & Horwitz, 1990); (e) the Neecham Confusion Scale (Neelon, Champagne, & McConnell, 1985); (f) the Delirium Symptom Interview (Albert et al., 1992); (g) the hand-held tachistoscope (Pauker, Folstein, & Moran, 1978); and (h) the Trailmaking Tests A and B (Reitan, 1958). These instruments were designed to detect acute confusion rather than some other phenomenon such as "mental status"; reflect objectively and consistently the essential aspects of acute confusion; accurately and promptly identify confusional behavior in all patients, especialy those not able to be evaluated using a questionnaire or interview format (e.g., individuals who are noncommunicative, persons who have sensory or physical impairments, and those in whom behavior changes rapidly or in whom manifestations of cognitive problems might be subtle); minimize the response burden on the patient; and facilitate use by nonpsychiatrically trained clinicians.

Despite good intentions and fairly rigorous validation testing, limitations of measurement persist. Many of the scales rely on clinical judgments for assessment, scoring, and interpretation, thus introducing subjectivity and unreliability (Kautz, Cheung, & Walker, 1991; Levkoff et al., 1991). Many of the behaviors to be observed are not specific to acute confusion (e.g., slurred speech, demanding behavior, and restlessness); therefore specificity is low (Foreman, Pompei, Lee, Ross, & Rudberg, 1991). Some procedures in these scales cannot be performed by acutely confused patients because such individuals cannot attend to directions (Anthony et al., 1985; Levkoff et al., 1991; Trzepacz et al., 1988). Educational and age biases persist (Pauker et al., 1978). Not all aspects of acute confusion are measured by the instruments, and the distinction cannot be made between acute and chronic conditions (Levkoff et al., 1991). Hence the need for additional valid and reliable instruments persists. Future instrument development should be deductive rather than empirical to ensure inclusion of all essential features of the phenomenon of acute confusion (Cameron et al., 1987; Inouye et al., 1990; Johnson et al., 1990; Trzepacz et al., 1988). Intensive and rigorous clinical testing is needed to demonstrate minimization of respondent burden, utility with noncommunicative patients, clinical feasibility for monitoring of changes in patient status, and ease of administration and interpetation for clinicians. Vermeersch (1991) suggested that two types of instruments may be needed: one to detect the onset of acute confusion, and another to monitor and determine the severity of the acute confusion. To date, no such instrument(s) has been developed.

Etiologic Basis of Acute Confusion

Multiple and disparate methods have been used to study the etiologic basis of acute confusion. Yet, no study fully met the criteria necessary for reaching definitive conclusions regarding the causal mechanisms behind acute confusion. Correlational designs have been used almost exclusively in the study of this condition. Thus, much is known about the various conditions surrounding the genesis of acute confusion, but little definitive and incontrovertible knowledge exists about causal relationships. As it seems unlikely that all plausible rival hypotheses could be eliminated or controlled without trivialization, it is improbable that definitive, incontrovertible evidence of the causal relationships of acute confusion will be found.

Nonetheless, what has been discovered about the etiologic basis of acute confusion is relatively consistent. First, it is clear that patients who are older (Blachy & Starr, 1964; Fields, MacKinzie, Charlson, & Perry, 1986; Golinger, Peet, & Tune, 1987; Jordan, Wilkinson, & Giuffre, 1991; Raway, 1991; Rockwood, 1989; Williams et al., 1979, 1985b); sicker (Blachy & Starr, 1964; Evans, 1987; Fields et al., 1986; Foreman, 1989; Francis, Martin, & Kapoor, 1990; Pompei, Foreman, Ross, Lee, & Rudberg, 1991; Rockwood, 1989); and cognitively impaired (Evans, 1987; Gustafson et al., 1988; Koponen, Hurri, Stenback, & Reikkinen, 1987; Koponen, Hurri, Stenback, Matilla, Soininen, & Riekkinen, 1989; Pompei et al., 1991; Rockwood, 1989) are more vulnerable to acute confusion during hospitalization.

Second, acute confusion generally has multiple rather than single causes (Foreman, 1989; Francis et al., 1990; Jolley, 1981) that span the spectrum of human illnesses. Hence, shifts in physiologic parameters are not perceived as clinically significant. This lack of perceived clinical significance emanates from lack of knowledge about precise limits of normal physiologic functioning, pharmacotherapeutics specific to the elderly, and interactive effects of medications. Thus, what is perceived as a situation within normal limits may in fact be an abnormal clinical state with accompanying acute confusion.

Third, there is general agreement about the most prevalent conditions associated with the genesis and presence of acute confusion. Although the designs of the studies failed to meet criteria for making definitive causal inferences, authors generally interpreted the relationships between these conditions and acute confusion as causal. Various pharmacologic agents were the most frequently identified cause of acute confusion (Foreman, 1989; Francis et al., 1990), especially agents with anticholinergic properties (Berrgren et al., 1987; Blazer, Federspiel, Raya, & Schaffner, 1983; Brannstrom, Gustafson, Norberg, & Winblad, 1989; Dellasega, 1990; Dickson, 1991; Golinger et al., 1987; Miller, Richardson, Jyu, Lemay, Hiscock, & Keegan, 1988; Mondimore, Damlouji, Folstein, & Tune, 1983; Purdie, Honigman, & Rosen, 1981; Summers, 1978; Tune, Holland, Folstein, Damlouji, Gardner, & Coyle, 1981), or those that have central nervous system effects (Berrgren et al., 1987; Brannstrom et al., 1989; Dellasega, 1990; Dickson, 1991; Purdie et al., 1981; Savageau et al., 1982; Sirois, 1988). The second most prevalent identified etiology was infection (Blank & Perry, 1984; Dickson, 1991; Francis et al., 1990; Levkoff et al., 1988; Morse & Litin, 1971; Purdie et al., 1981; Rabins & Folstein, 1982; Rockwood, 1989; Sadler, 1981), especially urinary tract and respiratory infections. However, it is unknown if the causal mechanism is a function of the infective agent or the hyperthermic response to the infective process. Fluid (Egerton & Kay, 1964; Francis et al., 1990; Gardner, 1984; Seymour, Henschke, Cape, & Campbell, 1980) and electrolyte imbalances, especially sodium (Dickson, 1991; Foreman, 1989; Francis et al., 1990; Morse & Litin, 1969; Purdie et al., 1981; Rockwood, 1989), and potassium (Foreman, 1989; Morse & Litin, 1969; Sirios, 1988), were identified as the third most common cause of acute confusion. Metabolic disturbances such as azotemia, alterations in pH, and nutritional deficiencies are the fourth most common cause (Dickson, 1991; Foreman, 1989; Francis et al., 1990; Gardner, 1984; Levkoff et al., 1988; Morse & Litin, 1969; Neelon et al., 1991; Rockwood, 1989).

It also has been shown that the etiologic basis of acute confusion varies across the trajectory of illnesses (Quinless, Cassese, & Atherton, 1985; Williams et al., 1985b); the nature of the health problem (i.e., medical vs. surgical); and the setting (Dellasega, 1990; Roberts & Lincoln, 1988). However, it is unclear whether this variability was entirely a function of the phenomenon itself or an artifact of the assessment techniques used to describe the phenomenon. Variables that were significant in studies with univariate designs dropped out when examined with multivariate designs (e.g., PaO₂). Also, some variables were significant for some patient populations and not for others (e.g., hypothermia and hemorrhage for surgical patients). Hence, there is disagreement as to whether the study of acute confusion should occur within narrowly defined patient populations (e.g., elderly patients undergoing surgery for traumatic hip fracture) or within more general populations (e.g., general medical patients). Those favoring narrow populations wish to control competing hypotheses, whereas the others asserted that acute confusion has universal characteristics; studies of narrow medical diagnostic categories may generate artifacts specific to that medical diagnostic category.

Debate continues about the relationships between sensory impairment, the environment, and acute confusion. Some argued that the relationship between sensory impairment and acute confusion is causal, whereas others insisted it is coincidental. Extremes in environmental characteristics are common with acute confusion, but it is not known if environmental factors are causative.

Epidemiology of Acute Confusion

The reported incidence and prevalence rates of acute confusion varied widely from a low of 8% in postoperative patients just before discharge from the hospital (Williams et al., 1979) to a high of 85% in terminally ill cancer patients (Massie, Holland, & Glass, 1983). This disparity in incidence and prevalence rates resulted from variability in the conceptual definition, measurement, precision of diagnostic criteria, diagnostic aids, and diagnostician. Generally, the more conservative the conception and operation, and the more sensitive and specific the measures, the lower the estimates of prevalence and incidence (e.g., studies of delirium diagnosed by a psychiatrist using the Diagnostic and Statistic Manual, 3rd ed., rev., criteria for delirium (Cameron et al., 1987; Inouye et al., 1990; Johnson et al., 1989; Trzepacz et al., 1988) are low). More liberal methods were associated with higher estimates of prevalence and incidence. Additionally, the presentation and manifestation of acute confusion among individuals and within an individual across time, methods of case finding, setting, and patient population influenced the findings about the incidence and prevalence of acute confusion.

Prevalence on Admission to the Hospital. The prevalence of acute confusion on admission to acute care hospitals has not been studied widely, but the findings of the few studies are consistent. Williams et al. (1979) reported a prevalence of 16% at admission for surgical repair of traumatic hip fracture. More recently, a prevalence of 16% also was reported in a sample of general medical patients (Francis et al., 1990) and surgical patients (Wanich, Sullivan, Gottleib, & Johnson, 1991). There has been speculation that acute confusion at admission results from the underlying health condition for which the individual was hospitalized (Foreman, 1989, 1991).

Incidence During Hospitalization. During hospitalization, the incidence of acute confusion ranged from 6% (Francis et al., 1990) to 55% (Chisholm, Deniston, Igrisan, & Barbus, 1982). Typically, the incidence ranges between 20% and 40% (e.g., Cavanaugh, 1983; Foreman, 1989, 1991; Neelon et al., 1991; Roberts & Lincoln, 1988; Schor et al., 1990; Williams et al., 1979, 1985a, 1985b). As mentioned previously, some of the variability in the incidence may be explained by methodological inconsistencies. However, assuming that the true incidence of acute confusion during hospitalization is much greater than that upon admission, it would be logical to conclude that many cases of acute confusion are iatrogenic or nosocomial in nature, and, therefore, preventable (Foreman, 1989, 1991). Nevertheless, Foreman, Thies, and Anderson (1991) found no differences in various physiologic parameters between patients who were admitted acutely confused and those who became acutely confused later in the course of hospitalization.

Postoperative Incidence. Estimates of the postoperative incidence of acute confusion ranged from 15% in the first 24 hours after surgery (Jordan et al., 1991; Summers, 1978) to a high of 72% in cardiac surgery patients (Sadler, 1981). The cause of the acute confusion has been attributed to intraoperative hypothermia (Gardner, 1984; Sadler, 1981; Wragg, Dimsdale, Moser, Daily, Dembitsky, & Archbold, 1988), hemorrhage (Morse & Litin, 1969, 1971; Savageau, Stantor, Jenkins, & Klein, 1982), and the anticholinergic effect of various medications (Berrgren et al., 1987; Blazer et al., 1983; Brannstrom et al., 1989; Dellasega, 1990; Dickson, 1991; Golinger et al., 1987; Mondimore et al., 1983; Purdie et al., 1981; Summers, 1978; Tune et al., 1981).

Historically, the incidence of acute confusion was reported as higher in surgical than medical patients (Levkoff, Besdine, & Wetle, 1986), which may reflect a more conservative surgical attitude. However, recent data (Pompei et al., 1991) indicated that the incidence of acute confusion is the lowest in patients undergoing elective general surgical procedures (approximately 10% to 20% incidence), moderate in patients with general medical conditions (approximately 22% to 50%), and highest in patients with critical (requiring immediate medical or surgical intervention) or terminal illness (approximately 58% to 85%).

Incidence at Discharge From the Hospital. It seems reasonable that discharge from the hospital would occur only after the resolution of the acute confusion. However, such is not the case. Furstenberg and Mezey (1987) reported 29% of elderly patients admitted for the surgical repair of a fractured hip were discharged while acutely confused. Although the exact figure is unknown, Rogers et al. (1989) reported many of the subjects they studied remained acutely confused at the time of discharge. Dellasega (1990) reported that 46% of elderly patients admitted to a visiting nurse service had some degree of acute confusion.

Onset and Duration of Acute Confusion. The onset of acute confusion during hospitalization occurs shortly after admission (Berrgren et al., 1987; Chisholm et al., 1982; Egerton & Kay, 1964; Foreman, 1989, 1991; Johns, Large, Masterton, & Dudley, 1974; Morse & Litin, 1971; Pompei et al., 1991; Raway, 1991; Wanich et al., 1991; Williams et al., 1979; Wragg et al., 1988). Onset has been reported to range from the first 24 hr to the 6th day of hospitalization. Modal day of onset across studies is day 2, or between 24 and 48 hr of hospitalization. Few instances of the onset of acute confusion have been reported beyond the 6th day of hospitalization.

Controversy continues relative to the time of onset of acute confusion. Lipowski (1989) maintains that acute confusion often begins at night. Engel (1989), however, contends that it is not so, but that acutely confused patients, "whose level of awareness is already impaired, are likely to become further disoriented, frightened, and disturbed in the dark and quiet of the night, and hence behave in ways more likely to attract the attention of the staff" (p. 264).

Scant information was available about the duration of a state of acute confusion. Gustafson et al. (1991) recently reported the duration of acute confusion in patients postsurgery for the repair of hip fracture as less than 7 days. Pompei et al. (1991) reported the modal duration of an acute confusional state as approximately 3 to 4 days; cases of acute confusion lasting 7 days were rare.

Although desirable, more precise conclusions about the incidence, prevalence, onset, and duration of acute confusion are not possible. This imprecision is a result of methods that vary among studies and that are incongruent with the characteristics of the phenomenon. First, because the methods and criteria for case finding vary, what has been identified as acute confusion in one study may not be in another. Also, varying methods of detection were associated with varying degrees of sensitivity and specificity; more sensitive and specific methods were able to detect acute confusion sooner than those that are less sensitive and specific. As a result, different methods generated different findings about the incidence and prevalence of acute confusion.

Because it is agreed that acute confusion is dynamic (i.e., the clinical features fluctuate over the course of a day), multiple measurements over a 24-hr period would be expected. However, most investigators assessed the patients once per day (e.g., Foreman, 1989, 1991) and others measured acute confusion every other day (e.g., Williams et al., 1985). With widely spaced data points it is difficult to draw valid and reliable conclusions about the dynamic nature of acute confusion. Although more frequent measurements would provide more complete information about acute confusion, more frequent measurements are intrusive, add to respondent burden, lead to higher rates of subject attrition and misclassification (false positives owing to fatigue and false negatives owing to learning effects). Unobtrusive techniques might be helpful to improve sensitivity and specificity of measurement.

Outcomes of Acute Confusion

Findings about outcomes of acute confusion consistently showed that acute confusion is a marker of poor prognosis (Dickson, 1991; Eagles, Beattie, Restall, Rawlinson, Hagen, & Ashcroft, 1990; Fields, MacKenzie, Charlson, & Sax, 1986; Flint & Richards, 1956; Furstenberg & Mezey, 1987; Levkoff

et al., 1986, 1988; Pompei et al., 1991; Rabins & Folstein, 1982; Rogers et al., 1989; Schor et al., 1990; Thomas, Cameron, & Fahs, 1988; Trzepacz, Teague, & Lipowski, 1985; Weddington, 1982). Outcomes included length of hospital stay, morbidity, mortality (inhospital and posthospitalization), and discharge disposition.

The length of hospitalization is protracted for patients experiencing some degree of acute confusion while hospitalized. Overall, patients with acute confusion were hospitalized about twice as long as nonconfused patients. This is true in recent as well as older studies, and in studies of both surgical and medical patients. Pompei et al. (1991) found that nonconfused elderly patients were hospitalized the same number of days as the diagnostic-related groups (DRG) reimbursement coverage. Conversely, patients who met specific criteria for acute confusion were hospitalized 4 days more than allowable by DRG. Schor et al (1991) reported similar findings.

Mortality rates for acutely confused patients were consistent across studies. Acutely confusioned elders were 3 to 5 times more likely to die than those who were not. Pompei et al. (1991) reported an inhospital mortality rate of 14% for acutely confused patients and less than 5% for comparable patients; in the 3 months following discharge from hospital the rates were 24% versus 10%, respectively. Schor et al. (1991) reported similar mortality rates: 26% versus 9%, and Rogers et al. (1989) reported 54% versus 15%.

Morbidity was higher in patients who were acutely confused. Acutely confused patients were more likely to experience adverse reactions to treatment, complications from the original illness(es), have a more protracted recuperative phase, and were less likely to return to preillness level of function (both cognitive and physical). Thus, acute confusion was a major reason for the nursing home placement of these patients (Zarit & Zarit, 1983).

Some argue that it was not the acute confusional state per se that resulted in poorer outcomes, but that acutely confused patients were more severely ill. However, in the study of Pompei et al. (1991), severity of illness failed to explain the variance in outcomes experienced by acutely confused patients. Hence, it may be impossible to conclude how morbidity and acute confusion are related. Does the acute confusion precipitate greater comorbidity; is greater morbidity reflective of greater physiologic instability that in turn is the cause of the acute confusion; or is there some interactive relationship between these two events? Further research is warranted.

Care of Patients Who are Acutely Confused

Intervention studies to prevent or manage acute confusion are few (Bay, Kupferschmidt, Opperwall, & Speer, 1988; Budd & Brown, 1974; Chatham, 1978; Fields et al., 1986; Gustafson et al., 1991; Langland & Panicucci, 1982; Lazarus & Hagens, 1968; Miller, 1991; Moore, 1977; Moore et al.,

1991; Nagley, 1986; Owens & Hutelmyer, 1982; Wanich et al., 1991; Williams et al., 1985b). Most interventions, especially those conducted by nurse investigators, have manipulated psychosocial variables, testing the influence of such strategies as providing orientation, clarification, and meaning to the patient's immediate environment (Bay et al., 1988; Budd & Brown, 1974; Chatham, 1978; Langland & Panicucci, 1982; Wanich et al., 1991; Williams et al., 1985b), continuity between patient and caregiver (Williams et al., 1985b), and anticipatory information about acute confusion (Owens & Hutelmyer, 1982). These strategies alone led to a lower incidence of acute confusion, fewer complications, a shorter hospitalization, and lessened the physiologic response to the experience. However, the incidence and consequences of acute confusion persisted at significant levels. Hence, it seems reasonable that these psychosocially oriented interventions are a necessary but insufficient intervention for acute confusion. Similarly, Gustafson et al. (1991) tested an intervention that was primarily physiologically based (e.g., oxygen therapy to prevent/minimize hypoxia and prevent perioperative hypotension). This intervention also led to a reduction in the incidence and severity of acute confusion, shortened the duration of the event, minimized complications, and shortened the length of hospitalization. Mortality and disposition on discharge were unaffected, and the incidence of acute confusion remained high, although reduced. As with psychosocial strategies, purely physiologically based interventions appear insufficient treatments for acute confusion. Interventions that incorporate both physiologic and psychosocial strategies seem a reasonable approach for future research.

Lived Experience of Acute Confusion

Acute confusion has been studied from the professionals', that is, the outsiders' perspectives. No accounts exist of the lived experience of acute confusion. Although such study is vital, many previously acutely confused elders are unable to recall the event; still others find it too unsettling to discuss it with others (Foreman, 1990). Yet, without insight into the subjective meaning and experience of acute confusion, a comprehensive understanding of this phenomenon may not be attainable. Qualitative or phenomenologic research into acute confusional states could provide valuable insights.

DIRECTIONS FOR FUTURE RESEARCH

Much remains unknown about acute confusion. It is clear that this is a prevalent and life-threatening condition among the elderly ill. Little theoretic

or conceptual development has occurred, yet conceptualization is basic to knowledge generation. Thus, for any appreciable progress to occur in knowledge development relative to acute confusion, theoretic and conceptual efforts must increase and conceptual clarity must be achieved.

Some semblance of consensus must be reached relative to nomenclature to facilitate the communication of ideas. At a minimum, investigators must make explicit the conceptual and operational definitions and assumptions foundational to the study of acute confusion.

Cumulatively, the beliefs, lexicon, and nature of acute confusion pose serious obstacles to the development of knowledge about this important health problem (Foreman, 1991; Lipowski, 1983a, 1990; Vermeersch, 1991).

The nature of acute confusion also remains unclear because study designs fail to incorporate fundamental characteristics of acute confusion. For example, investigators ignore diurnal fluctuations in symptomatology. Studies must be designed to examine all variables causally implicated in the pathogenesis of acute confusion. Hence, designs should be (a) multivariate to include variables that incorporate relevant physiologic, psychologic, sociologic, and environmental factors; (b) continuous to capture the diurnal fluctuation of symptomatology; and (c) time series to follow elderly individuals not only across the trajectory of illness but through recovery to "health," and across all health care delivery settings. Interactive effects must be examined. Clinical, as opposed to statistical, significance also must be considered. Designs should incorporate sensitive and severity of cognitive deficits that comprise the phenomenon called acute confusion.

Additionally, phenomenologic approaches to the study of acute confusion must be undertaken to complement the knowledge gained through more traditional research approaches. Such study is difficult, but without insight into the human experience of acute confusion, it is doubtful that a comprehensive understanding of this phenomenon can be attained, or that effective methods of caring for such individuals can be devised.

Although considerable energies have been spent in developing measures for detecting acute confusion, there is need for improvement. The search continues for instrumentation that is (a) practical—easy to administer and interpret, nonburdensome for respondents, thus permitting frequent administrations, and effective in mute patients; (b) sensitive and specific—producing few false-positive or -negative misclassifications, and resistant to cultural, racial, and educational effects; and (c) discriminating—able to detect minor cognitive deficits, distinguish acute confusion from dementia and depression, and detect acute confusion superimposed on dementia or depression.

The incidence and prevalence of acute confusion is well documented, but little is known about the natural history of acute confusion. Yet information about timing and duration is crucial for developing cost-effective and efficacious interventions. Definitive information about the causes rather than correlates of acute confusion is needed. Accurate and clinically useful models or profiles of patients at risk for developing acute confusion, and profiles or models of patients who are resistant to acute confusion are needed. These predictive models or profiles should be able to be individualized, and should provide direction for preventing and managing patients with acute confusion.

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