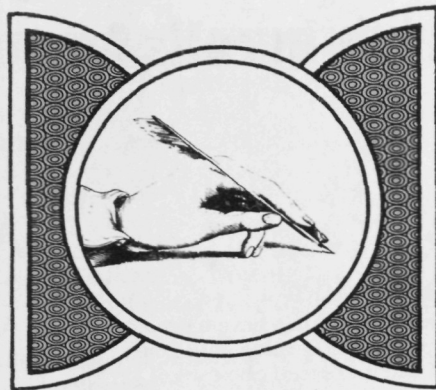


Letters to the Editor



The Journal welcomes Letters to the Editor; if found suitable, they will be published as space allows. Letters should be typed double-spaced, should not exceed 400 words, and are subject to abridgment and other editorial changes in accordance with journal style.

Correction

To the Editor:

An initial typing error resulted in the misspelling of Dr. George Engel's name throughout my paper "The Biopsychosocial Model: Whose Legacy?" in the October 1982 issue of this Journal (*J Fam Pract* 15:811, 1982). My apologies to Dr. Engel and to the Journal readers.

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Etiology of Sudden Infant Death Syndrome

To the Editor:

Has the ghost of the long-discredited "auto-intoxication" come back to haunt us? The Japanese have reported auto-intoxication in adults by the production of ethanol in the gut due to fermentation by yeast-like organisms.¹ With some degree of humor, this condition has been termed the "autobrewery syndrome." A total of 32 cases were reported between 1952 and 1972.

Since it is known that ethanol is a central nervous system depressant that can cause death from respiratory arrest, and since infants and children appear to be much more sensitive than adults to ethanol,² it has occurred to me that the autobrewery syndrome may be a cause of sudden infant death syndrome (SIDS).

Infants are usually supplied ample amounts of carbohydrates, especially lactose. *Candida* species may "colonize" the infant as early as delivery. Consequently, it is not unreasonable to speculate that an infant may be exposed to low levels of ethanol either from its own intestinal production or a nursing mother or from chronic alcohol ingestion by a nursing mother. Acetaldehyde, an ethanol metabolite, is much more effective than ethanol in inhibiting potassium-stimulated brain respiration in vitro.³ Interestingly, acetaldehyde is also found in high concentrations in cigarette smoke. These levels are maintained for several minutes after smoking.⁴

The fetal alcohol syndrome has been publicized extensively in recent years.^{5,6} Neuropathological

studies have identified structural alterations in the brains of infants exposed to alcohol in utero. It seems reasonable to search for morphological alterations in infant brains exposed to alcohol in early postnatal life. Most recently, reticular dendritic spines have been described in autopsy material from the apex of the pons to the cauda of the medulla in 17 of 19 infants who died from SIDS.⁷

Why the dendritic spines were present or what their relationship to SIDS might be are open for speculation and animal experimentation. Perhaps the autobrewery syndrome or other means of chronic exposure to low concentrations of ethanol and acetaldehyde may be a cause of SIDS. Ethanol has been found in the blood and spinal fluid of 5.9 percent of SIDS infants.⁸

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Evaluation of Depression

To the Editor:

A study was undertaken in this program to enhance the understanding of the use of a psychiatric consultant in the education of family practice residents concerning the evaluation and treatment of depressed patients. Thirty consecutive patients diagnosed with depression by second- and third-year residents in family practice were evaluated. The initial chart note (index visit) was audited by two consulting psychiatrists. Prospective patient contacts were undertaken to both verify and elaborate upon the family physicians' data base.

Among the 15 different chief

complaints recorded, only 9 were in reference to a mood complaint. Past medical histories were recorded in 16 cases (53 percent). Nine (23 percent) included physical disorders that may present with behavioral symptoms (somatopsychic illness),¹ albeit in only one case was the relevance charted. Sixteen patients had a history of psychiatric or chemical dependency in a first-degree relative at follow-up; only seven (44 percent) were detected by the residents. A medication history revealed that 15 of the 24 drugs have been associated in the literature with iatrogenic behavioral disturbance.² In 43 percent of cases the resident had recorded a past psychiatric history.

A physical examination (usually limited to one to three organ systems relevant to the system review) was recorded in 21 of 30 patients.

Three individuals (14 percent) had new findings, ie, a supraventricular tachyarrhythmia, hepatomegaly, and pharyngitis.

The family practice residents recommended a psychotropic drug in 17 cases. Fourteen recommendations were acted upon. Tricyclic antidepressant doses were initiated at a mean of 57.7 mg; with one exception, the drug of choice was from the tertiary amine group. Duration of treatment ranged from three days to four months with a mean of 2.7 follow-up visits. Ten of the 13 patients subjectively reported a positive response to their tricyclic antidepressant. These were cases where mean dosage and length of treatment was 112 mg and 5.3 weeks, respectively. This compares to 75 mg and 1.1 weeks

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References: 1. Craig JW: Clinical implications of the new diabetes classification. *Postgrad Med* 68 (No. 4):122-133, October 1980. 2. Yalow RS, Berson SA: Immunoassay of endogenous plasma insulin in man. *J Clin Invest* 39:1157-1175, July 1960.

BRIEF SUMMARY DIABINESE® (chlorpropamide) Tablets

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HYPOGLYCEMIA, IF IT OCCURS, MAY BE PROLONGED.

Adverse Reactions: Usually dose-related and generally respond to reduction or withdrawal of therapy. Generally transient and not of a serious nature and include anorexia, nausea, vomiting and gastrointestinal intolerance; weakness and paresthesias.

Certain untoward reactions associated with idiosyncrasy or hypersensitivity have occasionally occurred, including jaundice (rarely associated with severe diarrhea and bleeding), skin eruptions rarely progressing to erythema multiforme and exfoliative dermatitis, and probably depression of formed elements of the blood. With a few exceptions, these manifestations have been mild and readily reversible on the withdrawal of the drug. Diabinese should be discontinued promptly when the development of sensitivity is suspected.

Jaundice has been reported, and is usually promptly reversible on discontinuance of therapy. THE OCCURRENCE OF PROGRESSIVE ALKALINE PHOSPHATASE ELEVATION SHOULD SUGGEST THE POSSIBILITY OF INCIPENT JAUNDICE AND CONSTITUTES AN INDICATION FOR WITHDRAWAL OF THE DRUG.

Leukopenia, thrombocytopenia and mild anemia, which occur occasionally, are generally benign and revert to normal, following cessation of the drug.

Cases of aplastic anemia and agranulocytosis, generally similar to blood dyscrasias associated with other sulfonylureas, have been reported.

BECAUSE OF THE PROLONGED HYPOGLYCEMIC ACTION OF DIABINESE, PATIENTS WHO BECOME HYPOGLYCEMIC DURING THERAPY WITH THIS DRUG REQUIRE CLOSE SUPERVISION FOR A MINIMUM PERIOD OF 3 TO 5 DAYS, during which time frequent feedings or glucose administration are essential. The anorectic patient or the profoundly hypoglycemic patient should be hospitalized.


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among nonresponders, suggesting that duration and dose of the drugs were critical outcome variables ($P < .05$).

Of note was the brief follow-up of newly prescribed patients (2.7 visits) in this study. These appeared attributable to patient attrition where residents scheduled as-needed follow-ups. Ongoing psychiatric disturbance (68 percent) often exists at a six-month follow-up,³ underscoring the importance of such a practice.

The underutilization by family practice residents of past personal and family histories and physical and mental status examination was evident. Training of primary care physicians in psychiatry should include comprehensive interview techniques, awareness of self and peer attitudes toward the mental health field, recognition of impact of illness on the patient, the interrelation of psychic and somatic factors, principles of controlled psychotropic management, the role of supportive therapies, and of what constitutes a successful intervention and follow-up in behavioral medicine.⁴ Experience in such case management principles during training under supervision of a liaison psychiatrist is currently being employed in this training program. Inclusion of the liaison psychiatrist at a training clinic adds relevance for the family practice resident that may not be seen with traditional psychiatric inpatient-based rotations.

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Treatment of Alcoholism and Depression

To the Editor:

The paper on alcoholism and depression is a valuable addition to the Family Practice literature (*Sedlacek DA, Miller SI: A framework for relating alcoholism and depression. J Fam Pract* 14:41, 1982). The phenomenon the author describes in the recovering alcoholic is one that I frequently observe in my clinical practice with alcoholic patients. The urge to treat the patients with some sort of "magic drug" to relieve the symptoms, as the authors discuss in the postacute withdrawal syndrome, should be resisted; instead the patient should be referred to an AA sponsor or other recovering person who can provide the reassurance that the symptoms will abate, usually in 24 to 72 hours. In my experience these symptoms are intensified by the recovering person's excessive ingestion of coffee or other stimulant-containing substances.

An additional comment: the authors¹ cautions against medicating the alcoholic in the early withdrawal stage is particularly important in tricyclic antidepressant use, for it can depress seizure threshold.

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