We (Coppen *et al*, 1978) and others (e.g. Tuomisto and Tukiainen, 1976) have shown that the transport of 5-HT into the platelets of patients with a depressive illness is impaired. This abnormality is reveresed upon successful treatment with lithium (Coppen *et al*, 1980). We have also shown that this 5-HT transport system is impaired in the platelets of patients who had suffered from a migraine attack within 5 days of the estimation of the kinetics of 5-HT accumulation (Coppen *et al*, 1979).

The results from these platelet experiments do indeed suggest that there are abnormalities of 5-HT transport in depressive illness and in migraine and reinforce the association proposed by Garvey *et al* between depression, headaches and 5-HT.

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References

ABOU-SALEH, M. T. & COPPEN, A. (1983) Subjective side-effects of amitriptyline and lithium in affcetive disorders. *British Journal of Psychiatry,* **142,** 391–7.

COPPEN, A., SWADE, C. & WOOD, K. (1978) Platelet 5-hydroxytryptamine accumulation in depressive illness. *Clinical Chimeca Acta,* **87,** 165–8.

—— —— —— (1980) Lithium restores abnormal platelet 5-HT transport in patients with affective disorders. *British Journal of Psychiatry,* **136,** 235–8.

—— —— —— & CARROLL, J. D. (1979) Platelet 5-hyroxytryptamine accumulation and migraine. *Lancet, ii,* 914.

KUDROW, L. (1977) Lithium prophylaxis for chronic cluster headache. *Headache,* **17,** 15–18.

MATHEW, N. T. (1977) Lithium therapy in cluster headache. *Headache,* **17,** 92–3.

TUOMISTO, J. & TUKIAINEN, E. (1976) Decreased uptake of 5-hyroxytryptamine in blood platelets from depressed patients. *Nature,* **262,** 596–8.

VITAMIN SUPPLEMENT TO ALCOHOLIC BEVERAGES

DEAR SIR,

Some of the more serious side effects of alcohol abuse can be reversed or ameliorated by the prompt use of thiamine, preventing the progression of neurological lesions and reversing those lesions in which permanent structural changes have not yet occurred (Victor, 1976). The thiamine deficiency arises because of its excessive utilisations as an essential co-enzyme in intermediary metabolism of carbohydrates (in the decarboxylation of pyruvic acid and α-ketoglutarate to acetyl-CoA and succinyl-CoA respectively, and for the transketolase reactions of the hexose monophosphate shunt (Robinson, 1966). This would be consistent with the precipitation of Wernicke's encephalopathy following glucose infusion and upon refeeding prisoners of war or patients following a starvation diet (Drenick, Joven and Swenseid, 1966). Prophylactic vitamins are protective (Strauss, 1935). Changes in fermentation techniques may contribute to the neurological problems because of the virtual elimination of yeast in most beers. A dose-related prophylaxis could be achieved by compulsory thiamine addition to alcoholic beverages, in the same manner as vitamin A and D supplements to margarine.

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References

DRENICK, E. J., JOVEN, C. B. & SWENSEID, M. E. (1966) Occurrence of acute Wernicke's encephalopathy during prolonged starvation for the treatment of obesity. *New England Journal of Medicine,* **274,** 937–9.

ROBINSON, F. A. (1966) *The Vitamin Co-factors of Enzyme System.* New York: Pergamon.

STRAUSS, M. B. (1935) Etiology of "alcholic" polyneuritis. *Journal of Medical Science,* **189,** 378–82.

VICTOR, M. (1976) The Wernicke-Korsakoff Syndrome. In *Handbook of Clinical Neurology* (eds. P. J. Vinken and G. W. Bruyn). Amstersdam, New York and Oxford: North Holland Publishing.

CAVERNOSAL ALPHA-BLOCKADE: A WARNING

DEAR SIR,

I was very interested to read Professor G. S. Brindley's article (*Journal,* September, **143,** 332–7), as my company manufactures the preparation of phenoxybenzamine used in the study. Some general interest (*Medical News,* November 10, 1983 and *The Times,* November 18, 1983) has, not surprisingly, been generated by this new technique of injecting small doses of phenoxybenzamine into the corpus cavernosum, to treat erectile impotence. Professor Brindley and I have therefore agreed that attention should be drawn to the possibilities for toxicity of the drug when used in this way.

First, as a general point, I should make it clear that apart from Professor Brindley's work, I know of no animal or human experience of this use of phenoxybenzamine. As far as I am aware, such use is not officially 'licensed' by any government regulatory agency.

There are also some specific points about the properties of the drug which may be relevant. Phenoxybenzamine is not easily soluble, and the concentrate we produce is an acidified mixture of ethanol and propylene glycol containing 100 mg phenoxybenzamine in 2 ml for intravenous use after dilution. This solution (pH 2.7-2.8) is a local irritant even when diluted according to the directions in the package insert if, for instance, it leaks from a vein. With regard to repeated long term use, it may be relevant that phenoxybenzamine is mutagenic in some *in vitro* tests (Anon, 1983). Phenoxybenzamine by intraperitonal injection in the rat has been reported (like many other substances when given by this unphysiological route) to give rise to sarcoma (National Cancer Institute, 1978), and formal oral carcinogenicity studies in the rat are in progress. Conversely, phenoxybenzamine orally has been in use for 24 years without evidence of human carcinogenicity emerging.

The relevance of these facts to the use Professor Brindley has described is entirely unknown, but they should be borne in mind when considering the risks and benefits of treating individual patients. Smith Kline & French Laboratories do not endorse this use of 'dibenyline injection'.

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References

ANON (1983) *Drug Therapy Bulletin,* **21,** 15–16.

NATIONAL CANCER INSTITUTE (1978) *Carcinogenesis Technical Report.* Series No. 72. U. S. Department of Health, Education and Welfare.

MANIA AS A SEQUEL TO A ROAD TRAFFIC ACCIDENT

DEAR SIR,

A 23 year old, single female was in a car crash in March 1981. She was a front seat passenger, wearing a seat belt, in her fiance's car which was involved in a head-on-collison with a bus. She received multiple lacerations to her face and neck, fractures of the transverse processes of 3rd and 4th lumbar vertebrae and compression of the body of L3. Abdominal pain was followed by shock. A diagnosis of an intra-abdominal bleed, confirmed by peritoneal tap, was treated conservatively.

She was unable to remember details for a period of time preceeding her accident and was described as quite confused and amnesic for a major part of her 12 day stay in the general hospital. When told that her boyfriend was in another hospital, she enquired if he too had been in an accident. On discharge home her family found her to be talking continuously. She was normally a quiet, easy going girl, who had to be encouraged to get out of bed in the morning. Now she started getting up at 6.00 a.m. and told her mother that she wanted to pull her weight in the house and that she was going to do the housework. She was unable to sit down, and continually tried to do all the cleaning in the house. Six weeks following discharge from hospital she returned to work and her employers contacted the family, with concern, as she was described as overactive in her work. Two weeks later her mother noticed her behaviour becoming stranger. She was restless at home and talked more than usual. She became religous and claimed that she was 'Mary' and then later that she was the 'Virgin Mary'. She started spending money in what was described as a 'sillier manner'. She claimed to have special powers.

At this stage she was admitted to a psychiatric hospital on a Temporary Certificate. On admission she maintained that she felt great and wanted to be with her husband, Brendan, who had died 'on The Cross' the previous week, that she had two children and that she had special powers that would cure other patients. She admitted to auditory hallucinations coming from a dustbin, saying the voice was that of her husband, Brendan, telling her that she was terrific and great. On testing, her concentration and memory were poor. She was unable to give the months of year or an accurate history of events. She was distractable and exhibited flight of ideas. She was disinhibited on the ward.

She was treated with a mixture of Lithium, Haloperidol intravenously and Chlorpromazine orally. Psychological testing revealed no brain damage and an IQ on the Borderline Mental Handicap Level. An EEG showed mild abnormalities (a repeat 4 months later was normal).

Family history revealed that both parents were alive, healthy and in their 50's. Father was described as a heavy drinker. Her birth weight had been 8 lb and her milestones were normal. School, where she was described as middle of her class, ended at 13 years. She had a good work record and was in her present job in a factory for 6 years. She made friends easily and had an active social life, described as normal. She had had no previous psychiatric illness or psychiatric contact. Her maternal aunt in Australia was thought to have suffered from a post-natal depression.

Her mania required approximately 4 months inpatient treatment with a return of elation early on when her medication was reduced. Final resolution of the elation was followed by mild depressive mood swings. She has been maintained on a small dose of Perphenazine, usually 4 mg. at night, and in the last year requires sleeping medication for some days prior