THE EFFECT OF ACUTE ALCOHOLISM ON THE SERUM AMYLASE OF NORMAL PERSONS

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Pancreatitis is common in the south-western Cape, where it is claimed[1] that 60% of cases are related to alcohol, mainly in the form of cheap locally-produced wine and brandy. Although an association between pancreatitis and long-standing alcoholic over-indulgence is well recognized,[2-5] the effect of acute alcoholic intoxication on the normal pancreas has received little attention. In this study the effect of acute alcoholic intoxication, using cheap local wine and brandy, on the serum amylase of normal individuals has been examined.

Material and Methods

Subjects. 20 healthy male volunteers aged 21 - 27 years.

Quantity and type of alcohol. 10 subjects consumed a cheap, natural semi-sweet white wine, in 180 ml. aliquots (6 oz.), and the remaining 10 consumed a very cheap brandy, in 35 ml. aliquots (1⅛ oz.) with water if desired. Quantity and rate of alcohol consumption were recorded for each individual and regulated to a steady intake over a 2½-hour period. Degree of intoxication and occurrence of vomiting were also recorded.

Serum amylase. Each sample of 10 ml. of venous blood was withdrawn into a heparinized syringe, centrifuged and the serum refrigerated immediately. Serum amylase was measured by the method of Pimstone.[6] Estimations were performed in duplicate and recorded as the average of these 2 results in Somogyi units (normal 20 - 140 units). Where duplicate estimations differed by 30 or more units, they were repeated.

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PROCEDURE

A control blood sample was taken 2 hours before drinking commenced (4 subjects were not available for this) and all subjects had a meal, following which a further control blood sample was taken. Drinking then proceeded for 2½ hours, when the first post-alcohol sample was taken. The subjects thereafter resumed their normal routine but presented themselves 12, 18 and 36 hours later for the second, third and fourth post-alcohol samples.

RESULTS

All preprandial and postprandial control values of serum amylase were within normal limits and the meal had no effect on serum amylase values (Fig. 1).

The quantities of alcohol consumed ranged from 1,080 ml. to 2,160 ml. of wine and from 245 ml. to 490 ml. of brandy (Table I). Mean consumption of wine was 1,440 ml. per head, and of brandy 340 ml. per head. These quantities resulted in gross alcoholic intoxication in each individual. Fourteen subjects vomited, 6 before withdrawal of the first post-alcohol sample and the remainder within 1 hour afterwards (Table I).

Post-alcohol amylase levels both during acute intoxication and at 12, 18 and 36 hours afterwards fell within the normal range (Table II) and no over-all trend was evident within this range (Fig. 1).

The serum amylase levels of the 6 subjects vomiting before the first post-alcohol blood sample show no immediate effect

of vomiting on serum amylase (Fig. 2), nor does vomiting appear to have had any effect on serum amylase at the 12, 18 and 36-hour intervals (Fig. 3). Since serum amylase might only be affected by larger quantities of alcohol, the results of the 3 heaviest drinkers in each group have been presented separately (Fig. 4). Their alcohol intake was 1,620 ml. - 2,160 ml. of wine, and 395 ml. - 490 ml. of brandy, yet no effect on serum amylase level, either immediately or later, is evident.

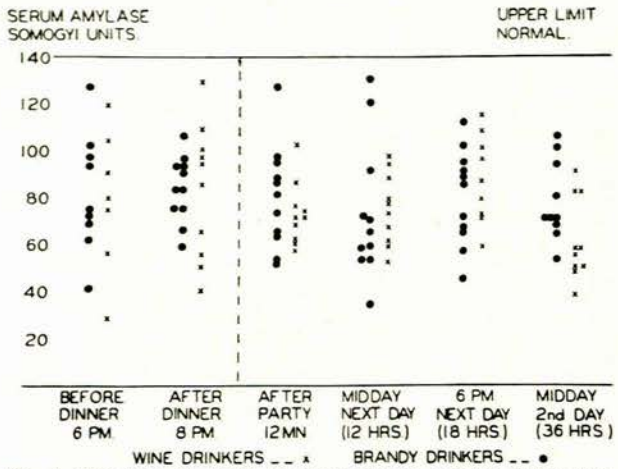

Fig. 1. The range of serum amylase values at intervals before and after acute alcoholic intoxication.

When serum amylase of each individual is plotted against quantity of alcohol consumed no relationship between the two is found, while intoxicated or at 12, 18 and 36 hours later (Fig. 5). This applies even to those who did not vomit and therefore undoubtedly retained the whole amount consumed.

DISCUSSION

The events occurring in the 5 - 15 years' heavy drinking which usually precede alcoholic pancreatitis are unknown.

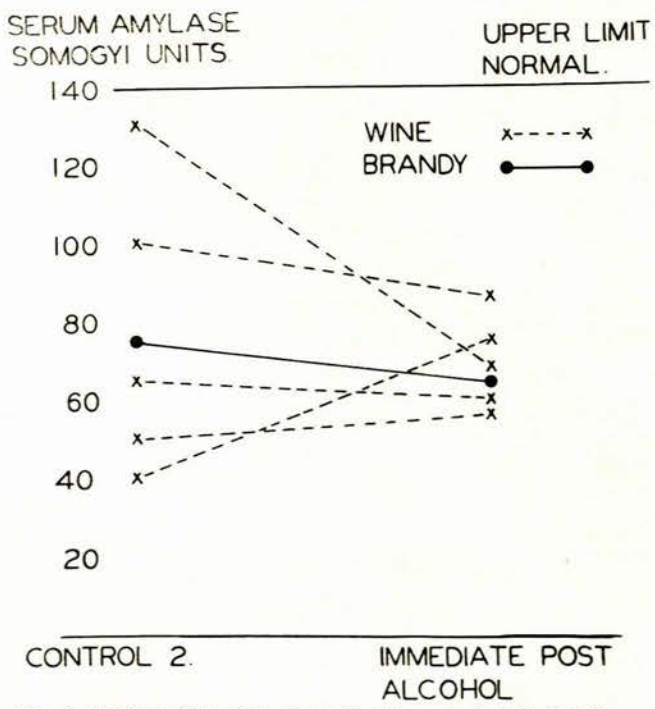

Fig. 2. The immediate effect of vomiting in acute alcoholic intoxication on serum amylase.

Structural changes of uncertain significance have been described in the pancreas of chronic alcoholics, and have included inspissated secretion in the ducts,[5] localized areas of cyst formation with patchy fibrosis,[5] chronic sclerosing pancreatitis,[6] inter- and intra-lobular fibrosis,[8] localized fat

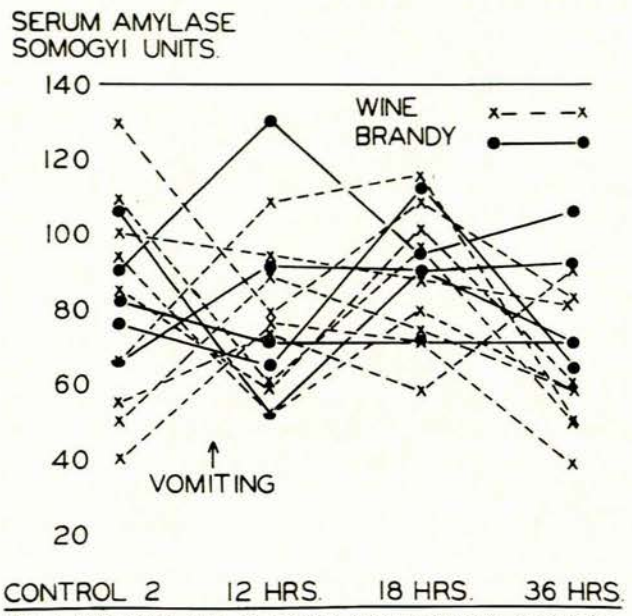

Fig. 3. The remote effect of vomiting in acute alcoholic intoxication on serum amylase.

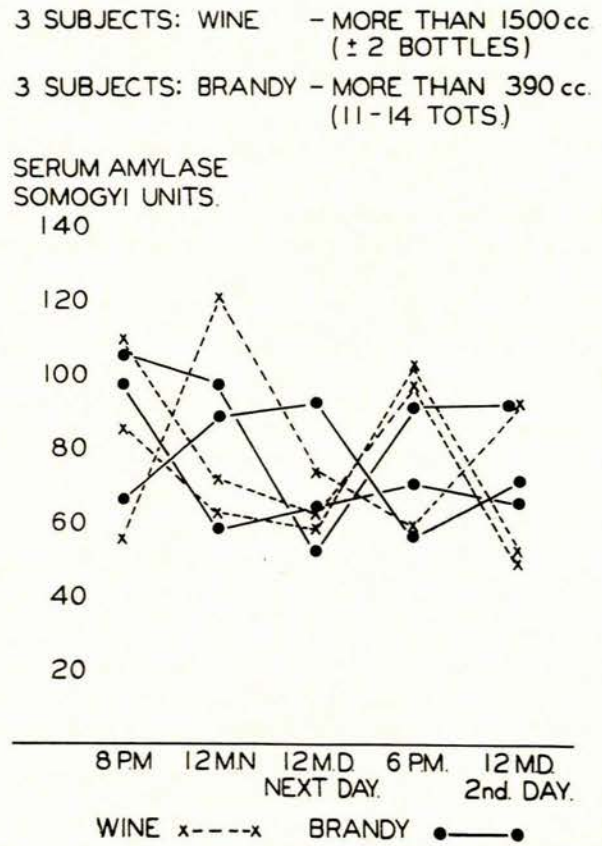

Fig. 4. Effect of very heavy alcohol consumption on serum amylase.

necrosis,[9] duct metaplasia[3,5] and unspecified chronic changes.[10]

The fibrotic changes might arise in alcoholics on a nutritional basis analogous to ethionine pancreatitis, or pancreatic fibrosis of kwashiorkor, but no evidence exists that this is so.[2] In kwashiorkor this fibrosis does not predispose to acute pancreatitis, and a clinical impression exists that increasing pancreatic fibrosis in alcoholics lessens the severity of subsequent attacks of acute pancreatitis.

TABLE I. TYPE AND QUANTITY OF LIQUOR CONSUMED AND OCCURRENCE OF VOMITING

Wine drinkers				*Brandy drinkers*			
		Vomiting				*Vomiting*	
Subject	*Quantity consumed (ml.)*	*Before spec. I*	*After spec. I*	*Subject*	*Quantity consumed (ml.)*	*Before spec. I*	*After spec. I*
1	1,080	yes		11	245		
2	1,080	yes		12	245		
3	1,080	yes		13	280		
4	1,440		yes	14	280		yes
5	1,440			15	280		
6	1,440	yes		16	350	yes	
7	1,440	yes		17	350		yes
8	1,620		yes	18	395		yes
9	1,620		yes	19	490		yes
10	2,160		yes	20	490		

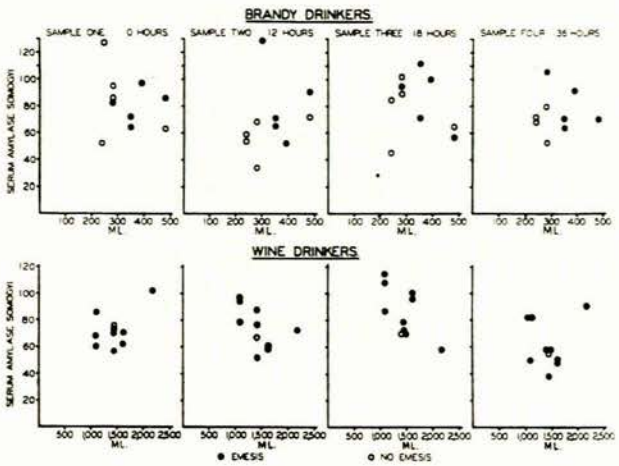

Fig. 5. Relationship between quantity of liquor consumed and serum amylase at various intervals after acute alcoholic intoxication.

Alternatively, the fibrotic changes might follow repeated subclinical episodes of pancreatitis[9,11] localized to 1 or 2 lobules. This might be due to duct obstruction or spasm combined with hypersecretion,[9,12,13] or to a direct toxic action of alcohol or contaminants[14] on the pancreas. Usually asymptomatic or resembling alcoholic gastritis, a particularly severe episode would present as acute pancreatitis. This concept is supported by Domzalski and Wedge[11] who took random blood samples from 50 chronic alcoholics within 48 hours of their last drink, and found raised serum amylase levels in 12, whereas only 1 of 50 non-alcoholic controls had a raised level. Moreover, Weiner and Tennant[10] in a series of 52 autopsies on subjects acutely alcoholic at the time of death, have reported acute pancreatic changes in 25 and chronic changes in 2, but unfortunately the changes were not described.

The present study has, however, not demonstrated either immediate or delayed change in serum amylase following acute alcoholic debauch in 20 normal young subjects, even after vomiting. This confirms the findings of a similar study by J. G. Myhre.[15] Pancreatitis is so rare and alcoholic excess so common that it would have been surprising if each alcoholic spree resulted in pancreatic damage in normal persons. Even among heavy drinkers pancreatitis occurs at random, which suggests that a permissive event induces their susceptibility to pancreatitis.

TABLE II. SERUM AMYLASE LEVELS AT INTERVALS BEFORE AND AFTER ACUTE ALCOHOLIC INTOXICATION

Subjects	*Control 1*	*Control 2*	*Immediate post-alcohol*	*12-hour post-alcohol*	*18-hour post-alcohol*	*36-hour post-alcohol*
			Wine drinkers			
1	104	129	68	79	108	82
2	119	100	86	94	87	82
3	×	65	60	97	115	50
4	56	94	71	52	79	58
5	28	96	76	67	71	55
6	×	40	73	77	71	38
7	×	50	57	88	73	58
8	79	109	71	61	96	48
9	90	85	62	59	101	50
10	74	55	102	73	58	91
			Brandy drinkers			
11	93	83	52	59	85	68
12	69	94	126	53	45	72
13	71	93	95	34	89	80
14	127	90	82	130	95	106
15	74	75	86	68	102	53
16	62	75	64	65	112	64
17	×	82	72	71	71	71
18	102	106	97	52	90	92
19	40	66	88	91	57	71
20	97	59	63	70	65	×

× No blood sample taken.

After the first attack, susceptibility to repeated attacks of alcoholic pancreatitis is established.[2] The nature of the change which establishes susceptibility is unknown. It is possible that alcoholic excess alone can cause recurrent attacks in a pancreas damaged by the first episode of pancreatitis. However, repeat attacks do not follow particularly great alcohol intake for that person, suggesting rather that susceptibility depend on recurrence of the permissive factor.

The curious relationship between alcohol and pancreatitis is well explained by McCutcheon's interesting theory[16] that chronic alcoholic excess causes atrophy and incompetence of valvules in the proximal end of the pancreatic duct, permitting reflux of duodenal content and a retrojection pancreatitis. However, atrophy of valvules in alcoholics has yet to be demonstrated and only circumstantial evidence in support of this theory exists.

SUMMARY

No effect, immediate or late, on the serum amylase levels of 20 normal persons was found following acute intoxication with cheap wine or brandy. The implication of this in relation to the aetiology of pancreatitis is discussed.

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REFERENCES

1. Marks, I. N. and Bank, S. (1963): S. Afr. Med. J., **37**, 1039.
2. Richman, A. (1956): Amer. J. Med., **21**, 246.
3. Howard, J. M. and Ehrlich, E. W. (1960): Ann. Surg., **152**, 135.

4. *Idem* (1961): Surg. Gynec. Obstet., **113**, 167.
5. Clark, E. (1942): Amer. J. Dig. Dis., **9**, 428.
6. Pimstone, N. R. (1965): Clin. Chem. (in the press).
7. Meyers, W. K. and Keefer, C. S. (1934): New Engl. J. Med., **210**, 1376.
8. Cross, O. and Guleke, N. (1924): *Op cit.*[7]
9. Rich, A. R. and Duff, G. L. (1936): Bull. Johns Hopk. Hosp., **58**, 212.
10. Weiner, H. A. and Tennant, R. (1938): Amer. J. Med. Sci., **196**, 167.
11. Domzalski, C. A. and Wedge, B. M. (1948): Amer. J. Clin. Path., **18**, 43.
12. McCowan, J. J., Butsch, W. and Walters, W. (1936): J. Amer. Med. Assoc., **106**, 2227.
13. Egdahl, A. (1907): Bull. Johns Hopk. Hosp., **18**, 130.
14. Bennett, I. L., Cary, F. H., Mitchell, G. L. and Cooper, M. N. (1953): Medicine (Baltimore), **52**, 431.
15. Myhre, J. G. (1949): Thesis, University of Minnesota.
16. McCutcheon, A. D. (1964): Gut, **5**, 260.