Although there is no evidence at Cambridge that undergraduate suicides have increased in number in recent years, certain post-war developments may have had an adverse effect. One of these has been the increase in the number of students, which adds to the difficulties of supervision even though the tutorial body may have been strengthened to some extent; in the period 1948 to 1957 the numbers at Cambridge have increased by over 1,200. In a big university the detection of the earliest stages of mental ill-health must almost inevitably fall on those with whom undergraduates are in daily contact. When it is suspected that trouble is brewing the patient can be guided to where specialized help can be obtained. The increase in the number of undergraduates must mean that tutors find it more difficult to get to know each man personally so that they are in a position to detect small, but possibly significant, changes in personality or performance. Nor do present economic trends make this part of a tutor's work any easier. That cases of mental ill-health are sometimes not detected in the earliest stages is unfortunate, but under present conditions is not surprising.

The crux of the problem has recently been admirably summed up by one of Her Majesty's coroners: "The truth is that suicide is a most complex phenomenon and highly unpredictable; our knowledge of its roots is scanty indeed" (Thurston, 1958). Student suicides are an end stage of the problem of mental health in universities about which undoubtedly far too little is known even by those most directly concerned. Figures of incidence of mental ill-health have been given for certain universities (Still, 1954; Malleson, 1954), but none are available for those universities with the highest incidence of suicide. Information and statistics are the bricks and mortar of prevention, and until mental disease in university students is regarded in the same way as any other illness, as a misfortune and not something of a stigma, it is unlikely that much progress will be made in preventing its occurrence.

Summary

The incidence of undergraduate suicides at the older English universities during a 10-year post-war period is higher than it is in other comparable groups.

The factors which may have had an influence on the rate are discussed.

The need is stressed for more information about the incidence and the various ecological factors which affect the mental health of undergraduates.

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SECRETION OF BLOOD GROUP ANTIGENS AND PEPTIC ULCER

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We have shown that in the Liverpool area there is a higher frequency of blood group O in patients with duodenal ulcer than in a control group taken from the general population (Clarke *et al.*, 1955). In gastric ulcer, on the other hand, we found no evidence for an association with group O, despite the fact that Aird *et al.* (1954) and several subsequent writers, discovered it in other populations. Later we showed that there was a higher frequency of non-secretors of ABH substances among duodenal ulcer patients in Liverpool than in a control series from the general population (Clarke *et al.*, 1956). In a family study of duodenal ulcer sibships, however, where the unaffected sibs acted as controls, we found no evidence that group O individuals were significantly more likely to have an ulcer than their A, B, or AB sibs. The evidence regarding the association between non-secretion and duodenal ulcer within families was equivocal, but with the collection of further sibships the association has now almost disappeared (Clarke, 1959).

The present paper is concerned with the following aspects of the subject: (1) the ABH secretor frequencies in gastric and duodenal ulcer in Liverpool; (2) an examination of the apparent liability to duodenal ulcer of individuals with different ABO blood groups and secretor status; and (3) a review of some of the hypotheses on the nature of the association between ABH non-secretion and duodenal ulcer and the presentation of fresh evidence on the subject.

Secretor Character in Duodenal and Gastric Ulcer

Table I gives the results of our investigations, started in 1954, and Table II the statistical analysis. It will be seen that whilst there is a striking association between duodenal ulcer and ABH non-secretion, there is no evidence that in Liverpool there is any association between gastric ulcer and ABH non-secretion; moreover, there is a significant difference between duodenal and gastric ulceration with respect to the frequencies of ABH non-secretion (36.6% in duodenal and 27.5% in gastric ulcer; controls 24.3%).

The degree of association between both duodenal and gastric ulcer and group O varies from place to place (Roberts, 1957). In Liverpool, where an association of group O with duodenal ulcer but not with gastric ulcer was found, non-secretion is similarly associated with duodenal ulcer and not with gastric ulcer. It is therefore important to establish whether in other localities the degree of association between the two diseases and group O is correlated with that between the diseases and non-secretion. If a correlation be found it would suggest that both group O and non-secretion were working in the same way and that their mode of action is possibly immunological. If no

TABLE I.—*Secretor Status of Series of Controls* (851), *Duodenal Ulcers* (1,014), *and Gastric Ulcers* (138), *Not Random for Blood Group*

Phenotype	Control		Duodenal Ulcer		Gastric Ulcer	
	Males	Females	Males	Females	Males	Females
O secretor ..	131	135	284	60	28	18
O non-secretor ..	35	43	168	40	9	9
A secretor ..	165	117	194	35	28	15
A non-secretor ..	58	44	94	23	8	7
B secretor ..	47	29	37	13	4	6
B non-secretor ..	16	7	30	5	2	3
AB secretor ..	11	9	17	3	1	—
AB non-secretor	—	4	10	1	—	—

TABLE II.—*Statistical Analysis of Data in Table* I

	d.f.	χ^2	Probability
Comparison of frequency of non-secretors between:			
1. The various ABO blood group classes in:			
Control males	2	1·43	>0·3
,, females	2	0·68	>0·7
Duodenal ulcer males ..	2	3·41	>0·1
,, ,, females ..	2	1·30	>0·5
Gastric ulcer males ..	1	0·01	>0·9
,, ,, females ..	1	0·01	>0·9
(In the first four of the above comparisons, because of the small numbers, AB has been combined with B; in the fifth and sixth comparisons AB and B have been combined with A.)			
2. Males and females:			
Control	1	0·34	>0·5
Duodenal ulcer	1	0·29	>0·5
Gastric ,, ...	1	1·37	>0·2
3. Control and duodenal ulcer	1	32·54	<0·001
4. ,, ,, gastric ,,	1	0·66	>0·3
5. Duodenal ,, ,, ,,	1	3·96	<0·05

correlation be found it would suggest either that the two factors work in different ways (probably neither immunological) or that stratification of the population is responsible for at least one of the associations. Stratification seems very unlikely (see Roberts, 1957), but it has not formally been disproved.

Liability to Duodenal Ulcer of Different Blood Group and Secretor Phenotypes

Previous assessments of the risk to individuals of the different blood group and secretor phenotypes of developing duodenal ulcer (*Brit. med. J.*, 1958) have been arrived at by calculations based on series giving blood group frequencies and a separate series, such as that in Table I, giving secretor status, but not random for blood group (Clarke *et al.*, 1956; Wallace *et al.*, 1958). No one, so far as we know, has published a random series of ulcer patients and controls. The data in Table III are from 521 consecutive duodenal ulcer patients collected between January 1, 1956, and March 31, 1958, and are independent of the series of duodenal ulcer patients reported by Clarke *et al.* (1955), but not independent of those in Table I.

TABLE III.—*Phenotypes in Series of Duodenal Ulcer Patients and Controls, Random for Blood Group and Secretor Status*

Phenotype	Control Series		Duodenal Ulcers		Per Cent Incr. or Decr. on Control
	No.	%	No.	%	
O secretor	245	36·03	186	35·70	− 0·9
A ,, ...	204	30·00	124	23·80	−20·7
O non-secretor ..	70	10·30	98	18·81	+82·6
A ,, ..	75	11·03	62	11·91	+ 7·9
B secretor	54	7·94	19	3·63	−54·3
B non-secretor ..	15	2·21	19	3·63	+64·25
AB secretor.. ..	14	2·06	10	1·94	− 5·88
AB non-secretor ..	3	0·44	3	0·58	+31·82
Total ..	680	100·01	521	100·00	

TABLE IV.—*Apparent Liability to Duodenal Ulcer of Different Phenotypes*

Phenotype	Expected Duodenal Ulcer	Controls	"Risk"
O non-secretor	174	70	2·49: 1
A, B, and AB non-secretor ..	149	93	1·60: 1
O secretor..	331	245	1·35: 1
A, B, and AB secretor	272	272	1·00: 1

The 680 controls are a random series of doctors, technicians, students, nurses, and Liverpool Territorial Army soldiers collected between September 1, 1955, and March 31, 1958. The same techniques were employed in scoring both patients and controls, an extract of the seeds of *Ulex europaeus* being used throughout as an anti-H. It can be seen that the four categories of ABH non-secretor all show an increased incidence in the disease group, whilst the four ABH secretor phenotypes would each appear to have some degree of protection, thus indicating that the secretor locus is having a bigger effect on duodenal ulcer than the ABO locus.

In order to calculate the liability to ulcer of the different phenotypes, Dr. Richard Doll has suggested that one should take as a baseline the least susceptible group. He has compiled for us Table IV, in which the numbers of groups B and AB, being too small for any great reliance to be placed on them, have been combined with group A. The figures for "expected duodenal ulcer" in each phenotype in this table were obtained by multiplying the number of duodenal ulcers in Table III by 272/153, so as to make equal the numbers of duodenal ulcers and controls in the A, B, and AB secretor phenotype. It can be seen that group O non-secretors would appear to be 2.49 times more liable, and the A, B, and AB non-secretors 1.60 times more liable to develop duodenal ulcer than are A, B, and AB secretors. Taken at their face value, the data suggest that the liability to ulceration due to being simultaneously O and non-secretor is more than would be expected from the additive effects of the two phenotypes, and indicate that they interact with one another. However, the analysis reveals that the excess liability is not statistically significant.

It must be emphasized, however, that these assessments have been made using the general population as a control. When this type of analysis is applied within families the results are very different. Table V shows

TABLE V.—*Numbers of Duodenal Ulcer Propositi and Oldest Unaffected Sibs Examined, Subdivided into Phenotypes*

Phenotypes	Either Sex		Same Sex	
	Ulcer	Sib	Ulcer	Sib
O non-secretor	42	37	20	20
B ,, ...	5	7	4	4
AB ,, ...	4	3	2	1
A ,, ...	28	28	13	13
O secretor	90	91	47	46
AB ,, ...	6	5	5	3
A ,, ...	59	61	26	31
B ,, ...	15	17	10	9
Total ..	249	249	127	127

the numbers of the eight phenotypes in a series of duodenal ulcer patients, random for blood group and secretor status, who also have an unaffected sib whom we have studied. The phenotypes of the sibs are also shown—one for each of the patients, and where more than one unaffected sib has been tested the oldest has been used as the control. The phenotypes are in the order of susceptibility deduced from Table III, and one would expect to have relatively more ulcers in the top half of the table and more controls in the bottom half. Even remembering the expected correlation between the phenotypes of sibs, the almost exact equality of the numbers of the different phenotypes is remarkable. Both the patients and their unaffected sibs have much higher

group O and non-secretor frequencies than the general population, and this must mean that their mothers and/or their fathers had high O and high non-secretor frequencies. Since non-secretor frequencies are probably very uniform throughout Europe this is very unlikely to be due to their being from a particular racial strain, and therefore it is unlikely to be coincidental that one or more of their offspring have developed duodenal ulcer.

While we think that the sibship results are remarkable, yet the data are not extensive enough to prove a discrepancy between them and the results using the general population as a control. Because of this, and in view of the almost universal finding of the association with group O and the finding with non-secretor in Scotland (Wallace *et al.*, 1958), we propose to consider possible biochemical or serological explanations for the associations.

Nature of the Association Between ABH Non-secretion and Duodenal Ulcer

Several hypotheses have been put forward which might explain the relationship between duodenal ulceration, group O, and ABH non-secretion. Thus Aird (1955) thought that the various blood group substances might confer different degrees of protection against ulcerogenic agents, while Clarke *et al.* (1956) suggested that there might be a differential protection depending on the interrelationship between ABH and Lewis blood group secretion. The hypothesis that the excess of group O in duodenal ulcers is due entirely to H substance protecting less than A or B substances is refuted by the present data (Table IV), since, in the absence of H secretion (that is, in non-secretors), there should be no difference in the liability of O and non-O people. In fact, in our data there is a significant excess of group O amongst the duodenal ulcer non-secretors ($\chi^2=4.1$; $P<0.05$) compared with the control non-secretors. In neither hypothesis was it indicated whether the basis of the protection was immunological or mechanical—that is, coating of the duodenal mucosa by the mucoid blood group substances. Cain (1957) put forward a suggestion which would depend on an immunological mode of action.

We can now present the results of two investigations which throw some light on the question of a purely mechanical explanation of the susceptibility of ABH non-secretors.

Amounts of Blood Group Substances in the Saliva of Secretors and Non-secretors of ABH

Professor W. T. J. Morgan has pointed out to us that since non-secretors of ABH usually secrete much more Le^a than do ABH secretors, there may not be a great difference in the amount of blood group substance in the gastro-intestinal tract of secretors and non-secretors. In an attempt to test whether or not this is so, one of us (D. A. P. E.) has investigated the fucose content of saliva from 213 ABH secretors and 119 non-secretors of different ABH blood groups (fucose is a rare sugar found in large amounts in blood group substances; only very small amounts have been found elsewhere in the human body). A modification of Gibbons's (1955) method for methyl pentose was used. All subjects were free of duodenal and gastric ulcer symptoms.

It will be seen from Tables VI and VII that the ABH non-secretors produce a lower mean salivary concentration of fucose than the ABH secretors, and that the B secretors have a higher mean concentration than the O secretors. There are significant correlations between fucose content and serological activity, with the exception of A activity in A secretors (Table VIII).

TABLE VI.—*Mean Log_{10} Salivary Fucose Concentrations for Non-ulcer Salivas*

Phenotype	No.	Mean Log_{10} Salivary Fucose	Standard Error of Mean	Antilog of Mean (μg./ml. Fucose)
O secretor	94	1·9009	0·01812	79·60
A ,, ...	62	1·9257	0·021137	84·28
B ,, ...	57	1·9758	0·026756	94·58
O non-secretor ..	57	1·8187	0·026800	65·88
A ,, ..	53	1·8091	0·02522	64·43
B ,, ..	9	1·7561	0·03806	57·03

TABLE VII.—*Comparison of Means of Log_{10} Salivary Fucose Values by Means of "t" Test*

Phenotypes Compared	Value of "t"	Degrees of Freedom	Significance of "t"
O secretor –B secretor	2·399	149	$p<0.02$
A ,, –B ,, ...	1·481	117	$p>0.10$
O ,, –O non-secretor ..	2·631	149	$p<0.01$
O ,, –A non-secretor ..	2·995	145	$p<0.01$
O ,, –B ,, ..	2·418	101	$p<0.02$
A non-secretor–B ,, ..	0·651	60	$p>0.10$
O ,, –B ,, ..	0·902	64	$p>0.10$

TABLE VIII.—*Correlation Between Log_{10} Salivary Fucose Content and Haemagglutination-Inhibition Titre*

Phenotype	Serological Activity Examined	Type of Antibody	No. of Salivas Examined	Correlation Coefficient r	Significance of r
Controls:					
O secretor ..	H	*Ulex*	94	0·5479	$p<0.001$
A ,, ..	A	Human	62	0·1482	$p>0.10$
A ,, ..	H	*Ulex*	62	0·5202	$p<0.001$
B ,, ..	B	Human	57	0·2785	$p<0.05$
B ,, ..	H	*Ulex*	57	0·3315	$p<0.02$
O non-secretor	Le^a	Human	52	0·3275	$p<0.02$
Duodenal ulcer:					
O secretor ..	H	*Ulex*	82	0·5374	$p<0.001$
O ,, ..	H	Eel	88	0·4957	$p<0.001$

TABLE IX.—*Calculation of Total Salivary Blood Group Content*

Phenotype	Mean Salivary Fucose μg./ml. (Note A)	Subdivision of Mean Salivary Fucose Content. μg./ml. (Note B)	Fucose Content of Pure Blood Group Substances (Note C)	Blood Group Substance Content. μg./ml. (Note D)	Total Salivary Blood Group Substance Content. μg./ml.
O, A, and B non-secretor ..	64·5	All Le^a	12%	537·5	537·5
O secretor ..	79·6	Lewis: 37·1	12%	309·2	521·7
		H : 42·5	20%	212·5	
A ,, ..	84·3	Lewis: 37·1	12%	309·2	562·6
		H : 15·9	20%	79·5	
		A : 31·3	18%	173·9	
B ,, ..	94·6	Lewis: 12·4	12%	103·3	557·5
		H : 4·5	20%	22·5	
		B : 77·7	18%	431·7	

A. Mean salivary fucose contents are from Table VI.

B. The mean salivary fucose concentrations have been ascribed to the various blood group substances as follows: (1) Non-secretors of ABH are presumed to secrete Lewis substance only. This is assumed to be Le^a. (2) H activity and log_{10} fucose concentration are correlated in O secretors. The intercept of the regression line is taken to represent fucose due to the Lewis substance present. This (when antilogged) is 37·1 μg./ml. The fucose content of this Lewis substance is taken to be that of Le^a—that is, 12%. (3) Since fucose correlates with H activity in all ABH secretors the ratios of fucose due to H substance in the various ABH secretors are taken to be the same as the ratios of the mean anti-H (*Ulex*) inhibiting power—namely, B sec.: A sec.: O sec.:: 1·68: 5·97: 16·00 μg./ml. Therefore the fucose due to H in A secretors is $42·5 \times 5·97 \div 16·00 = 15·9$ μg./ml., and the fucose due to H in B secretors is $42·5 \times 1·68 \div 16·00 = 4·5$ μg./ml. (4) Grubb (1951) showed that the approximate ratio of Le^a activity in ABH secretors was: groups A and O: groups B and AB:: 3: 1. It is assumed that A secretors and O secretors have equal amounts of Lewis substance in their salivas. Since 37·1 μg. of fucose per ml. was held to be due to Lewis in O secretors, the same amount is held to be present in A secretors. In B secretors the Lewis substance is held to account for $37·1 \div 3 = 12·4$ μg. of fucose per ml. This Lewis substance is assumed to have the same fucose content as Le^a. (5) The questions of (*a*) Lewis non-secretor persons and (*b*) Le^b secretion have been ignored in compiling this table.

C. The percentages of fucose are those in pure blood group substances obtained from ovarian cyst fluids (W. T. J. Morgan, 1958, personal communication).

D. Figures of blood-group-substance content are calculated by multiplying the figure of the mean salivary fucose content by the reciprocal of the fraction of fucose in the pure substance—for example, for non-secretors $64·5 \times 100 \div 12 = 537·5$ μg./ml.

The total salivary concentration of blood group substance has been computed for the different phenotypes, and the results are given in Table IX. The reasons for ascribing proportions of the mean salivary fucose concentration to the various blood group substances are given as footnotes to Table IX.

It will be seen that, as judged by the mean $\log_{10}$ salivary fucose content, the total amount of blood group substance is of the same order in each of the blood group and secretor subdivisions. It is true that the estimated proportions of different blood group substances present in each type of individual used in our calculation are only an approximation, but a radical change in the proportions ascribed is required materially to influence the figures computed for total blood group substance. So great a change would seem to be inconsistent with independent serological findings (see Race and Sanger, 1958).

The results of the biochemical investigations (see Tables VIII and IX) show that the fucose content is a good index of the total amount of blood group substances if the ABO blood group and secretor status is known. Fucose estimations indicate that there is little difference in the amount of blood group substances in the saliva of secretors and non-secretors. A full account of this work will be published elsewhere.

Comparison of Duodenal Ulcer Patients and Controls for Titre of Blood Group Substances and for Fucose Content of Saliva

If the ABH secretion itself protects mechanically against ulcer, secretors who develop an ulcer may tend to produce less blood group substances in their watery secretions than secretors who do not develop one. To test this hypothesis we investigated a secretor propositus and one of his unaffected secretor sibs who had the same blood group, to see if the titre of antigen was different in the two individuals. In 98 sib pairs studied the propositus secreted less antigen in 27 pairs, more in 29 pairs, and the same amount in the remaining 42 pairs. Thus these data again do not support the hypothesis of mechanical protection by the mucoid ABH substances, but are consistent with an all-or-nothing effect.

It will be seen from Table X that we have also compared the titres of the group-specific substances in the patients' saliva with the titres in the sibs and in some population controls. There is no evidence of a difference between the patients and their unaffected sibs. There is, however, a highly significant difference between the patients and their sibs on the one hand and the general population on the other, the mean titre of the general population sample being less with each of the antigens. Although the same standard salivas were used as technique controls throughout, it may be that this difference is not real but due to technical factors, as the controls were titred at a different time from the patients and their sibs. Therefore little reliance need be attached to this finding until the matter has been reinvestigated, but if later work shows that the secretor ulcer patients indeed secrete more than the general population secretors, this also would militate against the hypothesis of mechanical protection. There is apparently less variability in the controls than in the duodenal ulcer patients and their sibs (see also the findings with fucose below).

The salivary fucose contents of O secretors and O non-secretors with duodenal ulcer have been determined (Table XI); they have log normal distributions. Comparison of means (Table XII) shows that: (*a*) the O secretors, with and without duodenal ulcer, have means which are not significantly different; (*b*) similarly, the O non-secretors with and without ulcer have means which are not significantly different; and (*c*) both series of O non-secretors are significantly lower than both series of O secretors.

TABLE XI.—*Mean $\log_{10}$ Salivary Fucose Contents of Duodenal Ulcer Patients*

Phenotype	No. Examined	Mean $\log_{10}$ Fucose Value	Standard Error of Mean	Antilog of Mean (μg. ml. Fucose)
O secretor	89	1·9339	0·022643	85·88
O non-secretor ..	50	1·7545	0 036055	56·82

TABLE XII.—*Comparisons of Mean $\log_{10}$ Salivary Fucose of Duodenal Ulcer and Non-ulcer Subjects by Means of "t" Test*

Phenotypes Compared	Value of "t"	Degrees of Freedom	Significance of "t"
D.U. O sec.: D.U. O non-sec.	4·427	137	$p<0·0001$
,, : non-D.U. O sec.	1·144	181	$p>0·10$
,, : ,, non-sec. ..	3·245	144	$p<0·01$
D.U. O non-sec.: non-D.U. O sec. ..	4·051	142	$p<0·001$
,, ,, : non-D.U.O. non-sec. ..	1·451	105	$p>0·10$

The variances of the $\log_{10}$ salivary fucose concentrations are significantly higher in the duodenal ulcer subjects than in the non-ulcer subjects, both in secretors ($p<0.01$) and in non-secretors ($p<0.01$), and these results indicate that there is a real difference in variance of blood group substances in saliva between duodenal ulcer subjects and controls. This difference may be due simply to a greater variability of saliva flow induced by the disease, or by the circumstances at the time of collection of the samples.

The $\log_{10}$ salivary fucose content in the duodenal ulcer O secretors correlates with the titre of H substance using two different antibodies, eel serum and *Ulex* extract (Table VIII). The points where the two regression lines intercept the ordinate are not significantly different from each other nor from that of the non-duodenal ulcer O secretors.

Thus the serological data suggest that within any one blood group there is no difference between the amount of A, B, or H substances secreted by secretors with duodenal ulcer and their sibs. The

TABLE X.—*Results of Agglutination-inhibition Tests of Saliva of Duodenal Ulcer, Sib, and General Population Secretors*

Blood Group	Antisera or Lectin		Total No.	Titre of Saliva, 1 in										
				2	4	8	16	32	64	128	256	512	1,024	2,048
O	*Ulex*	D.U.	54	—	—	—	—	6	10	15	9	11	2	1
		Sibs	54	—	—	—	—	5	11	10	15	9	3	1
		Controls	92	—	—	2	3	17	27	36	6	1	—	—
A	Anti-A	D.U.	35	—	—	—	1	4	12	9	5	3	1	—
		Sibs	35	—	—	—	—	3	10	7	8	5	2	—
		Controls	58	—	—	1	1	16	20	15	4	1	—	—
A	*Ulex*	D.U.	35	—	—	1	8	13	9	—	4	—	—	—
		Sibs	35	—	—	1	6	13	8	3	3	1	—	—
		Controls	58	—	—	5	28	19	6	—	—	—	—	—
B	Anti-B	D.U.	10	—	—	—	—	—	2	—	1	3	2	2
		Sibs	10	—	—	—	—	—	1	—	2	3	3	1
		Controls	57	—	—	—	—	1	6	12	16	19	3	—
B	*Ulex*	D.U.	10	—	1	3	2	1	3	—	—	—	—	—
		Sibs	10	—	2	2	2	2	1	—	1	—	—	—
		Controls	57	1	18	36	2	—	—	—	—	—	—	—

biochemical evidence goes even further in suggesting that there is no difference in the total amounts of blood group substance between all duodenal ulcer patients and controls. It would seem, therefore, that if the association between group O, non-secretion, and duodenal ulcer is causal (but not due to a pleotropic effect of the genes) the most probable explanation can be found in the differences between the antigenic properties of the A, B, H, and Lewis group-specific substances, and this would imply that there is an immunological factor in the pathogenesis of duodenal ulcer (see Clarke *et al.*, 1957).

Conclusions

Gastric and duodenal ulcer in Liverpool behave differently with respect to the frequency of secretion and non-secretion of ABH blood group substances, there being no apparent association between non-secretion and gastric ulcer.

The excess of group O individuals in a duodenal ulcer series compared with a general population control is as great in the secretor as in the non-secretor patients. Group O non-secretors are more than twice as liable to develop duodenal ulcer as secretors of groups A, B, and AB.

Evidence is presented that the fucose content of a saliva specimen is a good index of its content of total blood group substances, provided the ABO blood group and ABH secretor status is known. Fucose estimations in saliva specimens indicate that the various types of ABH secretors and non-secretors all secrete about the same total amount of blood group substances.

There is little difference between the mean salivary titre of A, B, and H substances of secretors with duodenal ulcer and their unaffected sibs. The mean salivary fucose concentration, and hence the total blood group substance concentration, are the same in duodenal ulcer patients as in controls. These results suggest that the protection enjoyed by ABH secretors is not due to the blood group substances protecting quantitatively. The most probable explanation of the associations found between group O, non-secretion, and duodenal ulcer is that there is an immunological factor in the pathogenesis of duodenal ulcer.

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EXCESS BLOOD-GROUP SUBSTANCE A IN SERUM OF PATIENT DYING WITH CARCINOMA OF STOMACH

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The following observations are reported because they may throw some light on the association between blood group A and carcinoma of the stomach (Aird, Bentall, and Roberts, 1953).

Case Report

Mrs. J. was admitted to the Sheffield Royal Infirmary after a severe haematemesis, and it was necessary to select blood because of her anaemic and shocked condition. The duty pathologist grouped Mrs. J., by both tile and tube techniques (Dunsford and Bowley, 1955), and obtained the following results: anti-A, − ; anti-B, − ; O serum, (+) ; A cells, − ; B cells, V ; O cells, − ; auto-control, −. These results suggested that Mrs. J. was a rare subgroup of A—possibly A_1 (Dunsford and Aspinall, 1952)—and the sample was immediately referred to one of us (I. D.) for opinion. On regrouping the same sample of blood, the following results were obtained: anti-A, V ; anti-B, − ; O serum, V ; A cells, − ; B cells, V ; O cells, − ; auto-control, −. It will be observed that Mrs. J.'s blood now gave the reactions expected of a group A blood. Following a telephone discussion with the pathologist carrying out the original grouping, the only difference in method and reagents used lay in the fact that the cell grouping done at the Sheffield Royal Infirmary was on unwashed red cells, whilst the grouping at the Blood Transfusion Service Laboratories was on washed cells—that is, cells freed from the patient's serum by centrifuging through three aliquots of isotonic saline. This suggested that Mrs. J.'s serum contained enough A substances to neutralize the anti-A antibodies before they could agglutinate her red cells.

This possibility was investigated, and to one volume of Mrs. J.'s washed red cells (4% in saline) was added one volume of her serum, and this mixture was then tested with standard anti-A, anti-B, and group O sera. As a control Mrs. J.'s 4% washed red cells were mixed with an equal volume of saline, and this mixture was tested with the same antisera. The results obtained are shown in Table I. It will be seen that the addition of Mrs. J.'s serum to her red cells inhibits the action of anti-A grouping serum and greatly reduces the action of group O sera.

TABLE I

	Anti-A (Batch No. 973)	Group O (Batch No. 92)
Mrs. J.'s cells in saline plus Mrs. J.'s serum	—	w
,, ,, ,, ,, ,, saline	V	V
Controls	V (A_2)	V
	— (O)	—