1957; Armstrong, Gray, Race, and Thompson, 1957; Levine, Celano, and Griset, 1958; Boorman and Zeillich, 1958). In four of the six cases the antigen was proved to be inherited. Formaggio (1953) mentions in passing an AB with β in the serum, but no details are given. The two remaining cases look very like our seven. The following is taken from Stratton and Renton (1958):

"Case 28. Mrs. B. L. appeared to be AB β . Her A antigen was normal and her serum contained an apparently normal anti-B but no anti-A. Her cells were agglutinated at 16° C. by 13 out of 52 group A sera, and this agglutination was inhibited by the saliva of group B individuals only. Her cells were not agglutinated by 36 group AB sera.

"Case 29. Mr. H. R. also appeared to be AB β with a normal A antigen and apparently normal anti-B in the serum. His cells were agglutinated by 34 out of 36 group A sera at 16° C. The reactions varied from + to +++ in strength and were inhibited by group B saliva only. His cells were not agglutinated by 25 group AB sera. The reactions in this case were stronger than in the preceding one."

Dr. Stratton and Dr. Renton have kindly allowed us to quote further details about their patients. Mrs. B. L., aged 76 (in 1956), was suffering from a strangulated femoral hernia; one son was A β , the other appeared to be O, but his sample was infected and the results were not considered finally conclusive. Mr. H. R. died in 1956, aged 59; in 1946 he had carcinoma of the colon, and in 1956 a leg was amputated for gangrene; two brothers and a sister were group O and his wife and child were group B.

The A antigen is known to be affected by blood disease; this was demonstrated by van Loghem, Dorfmeier, and van der Hart (1957) and proved by Stratton, Renton, and Hancock (1958). The two patients so far reported were group A, but their red cells lost the faculty of being agglutinated by α . Such damage to an existing antigen seems easier to understand than does the acquisition, which we are describing, of a new one. It is striking that the acquired antigen should mimic that produced by the allele B, though had it not done so it would probably have escaped notice.

The acquisition of a B-like antigen by people of group A may be associated with age or disease or both. If the change could be brought about by disease alone it should have been recognized before in younger patients; the change certainly cannot happen to young or middle-aged healthy people, or it would surely have been recognized long ago in blood donors. Five of the seven patients had cancer; this could mean that the acquisition is associated with the disease or some condition secondary to the disease, such as anaemia or abnormalities of the plasma, or it could simply mean that cancer is a very common cause of the blood grouping of old people.

The observations here reported raise a number of questions that only more work may answer. We ought to know whether the change can happen to healthy old people. In any case, many more patients with the B-like antigen will surely be found, and biochemical investigation of the plasma as well as further serological studies may give some clue to the cause of this change.

Summary

Seven examples of blood group A β (β) are described which were peculiar because the red cells were agglutinated by a proportion of β sera. This reaction was inhibited by B and AB secretor saliva but not by

A or O secretor saliva. The β in the serum of the seven patients appeared to be normal. The saliva of three of the patients was tested: two of them were secretors and their saliva contained A and H but not B.

The absence of group O persons amongst the seven patients is highly significant.

Evidence is produced that the B-like antigen is not inherited but is an acquired character. The acquisition may be connected with old age or illness or both.

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A WEAK B ANTIGEN, PROBABLY ACQUIRED

BY

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Variants of the B blood-group antigen are very much rarer than variants of A, and there are no clear-cut subgroups of B corresponding to A β and A γ . Race and Sanger (1958) reviewed the few cases mentioned in the literature, and Cameron *et al.* (1959) have, since the present investigation was completed, given an account of several cases which appear similar to the one here described; we are indebted to them for allowing us to read their paper before publication.

The patient, a married woman aged 57, was admitted to the Royal Sussex County Hospital in April, 1958, suffering from carcinoma of the colon, with liver metastases. She was initially grouped as A and transfused with group A blood without ill effects. When further transfusion was needed a new test appeared to show that the patient's group was AB. It

was then found that her cells were agglutinated by some only of the (pooled) anti-B sera available for routine use.

It was first thought that these results might be due to the presence of an additional antibody in some of the anti-B sera, reacting with a rare, possibly familial, type of antigen on the red cells.

Serological Tests on Patient's Cells

At this stage the case was referred to the Blood Group Reference Laboratory, where the grouping serum had been prepared. Here it was confirmed that the patient's cells were agglutinated by the anti-B sera that had originally been found to agglutinate them. It was shown that agglutination was not due to an antibody against any of the common antigens of the main blood-group systems other than ABO, or to anti-C^x, E^w, V, Mg, Be^a, Wr^a, Vel, Mi^a, or V^w.

Apart from the elimination of these possibilities, the details of the patient's blood-group antigens outside the ABO system are of no obvious relevance. It ought nevertheless to be mentioned that she had a weak E (Rh) antigen; however, one child has a normal E apparently inherited from her, her husband being E-negative. The apparent weak E may possibly have been a result of transfusion.

The patient's cells gave normal A₁ reactions with numerous samples of anti-A and anti-A₁ sera. They were agglutinated by 7 out of 20 unselected pooled high-titre anti-B sera as issued for grouping purposes, and by 7 out of 20 unselected anti-B sera from individual donors, the reactions (in both cases) varying from ++ to w. There were usually very large numbers of unagglutinated cells, and so the possibility of the patient being a blood-group chimera was considered, though the appearance was not in fact one of tight clumps in a sea of unagglutinated cells, such as is shown by mixed blood samples in chimeras and post-transfusion specimens, but weak patchy agglutination of the type sometimes given by anti-Lewis sera. The cells were agglutinated in a similar manner by eight out of nine sera from group O donors from which the anti-A had been removed by absorption with A₁ cells. They gave a weak positive indirect antiglobulin test with a human immune anti-B serum in which the saline anti-B had been neutralized with B secretor saliva. They were not agglutinated by an immune anti-B of rabbit origin, nor by "anti-B₁" serum obtained from Dr. R. E. Rosenfield and produced by partial absorption of anti-B serum with AB cells.

Samples were tested on two occasions; at an interval of two weeks; neither gave any reaction with 10 sera from AB blood, and it was most unlikely, therefore, that this was a case of polyagglutinability.

Absorption Experiments with Patient's Cells

An anti-B serum (No. 252) which had reacted strongly with the patient's cells was absorbed with them (one volume packed cells to one volume of serum). Further portions of the same serum were absorbed with normal A₁, B, and A₁B cells as controls. The absorbed sera thus obtained were titrated in parallel with an unabsorbed sample against the patient's cells and against normal A₁, B, and A₁B cells, samples being matched for age (from the time of bleeding) and strength of antigens. Similar experiments were carried

out with an anti-A serum, and with an anti-A+anti-B serum (from a group O donor). The titration results were scored in the manner recommended by Callender and Race (1946). The titres and scores are shown in the Table. It will be seen that the patient's cells

Comparative Titrations of Unabsorbed and Absorbed Sera Against Patient's Cells and Controls. Each Titre is Followed by the Score, in Parentheses, Calculated as Described by Callender and Race (1945)

Cells Used in Titration	Titre Before Absorp- tion	Titre After Absorption with				
		A ₁ Cells	B Cells	A ₁ B Cells	Patient's Cells	O Cells
Anti-A Serum (No. 277)						
A ₁ ..	128 (82)	0 (0)	128 (78)	0 (0)	0 (0)	
B ..	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
A ₁ B ..	32 (64)	0 (0)	32 (58)	0 (0)	0 (0)	
Patient ..	128 (82)	0 (0)	64 (70)	0 (0)	0 (0)	
Anti-B Serum (No. 252)						
A ₁ ..	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
B ..	128 (80)	64 (68)	0 (0)	0 (2)	32 (58)	
A ₁ B ..	32 (52)	16 (52)	0 (0)	0 (0)	16 (46)	
Patient ..	2 (16)	2 (14)	0 (0)	0 (0)	0 (0)	
Anti-A + Anti-B Serum (No. 48)						
A ₁ ..	128 (80)	0 (0)	64 (72)	0 (0)	0 (0)	0 (0)
B ..	128 (78)	64 (76)	0 (0)	0 (0)	32 (56)	0 (0)
A ₁ B ..	128 (78)	16 (44)	32 (62)	0 (0)	4 (26)	0 (0)
Patient ..	128 (82)	2 (12)	64 (68)	0 (0)	0 (0)	0 (0)

reduced the titration score of anti-B sera No. 252 with normal B cells 10 units more than did A₁ cells. After absorption with the patient's cells this serum no longer agglutinated a sample of her cells, whereas that absorbed with A₁ cells still did so, the score initially 16 being lowered only two units by the absorption.

Samples of this anti-B serum, which agglutinated the patient's cells strongly, and of another anti-B, which failed to agglutinate them, were absorbed by her own cells, comparative absorption being performed with normal B cells. The cells were then subjected to elution by heating at 56° C. In all cases some anti-B was eluted. With the serum which agglutinated the patient's cells in saline these cells gave up more anti-B than did normal B cells. With the serum which failed to agglutinate them little anti-B was given up by either kind of cells, and there was little difference between the two eluates.

Tests on Patient's Serum

The patient's serum contained anti-B. It was tested against a number of samples of normal A and AB cells and found to agglutinate most B and AB samples in saline at 37° C., to give weak agglutination of all samples in the presence of cysteine-activated papain, and strong reactions with all samples at 20° C. and 4° C., both in saline and in albumin. Under none of these conditions did it agglutinate the patient's own cells, nor weak B cells of a type apparently similar to that described by Moullec *et al.* (1955).

Large amounts of A and H substances were present in the patient's saliva, but no B substance could ever be detected although tests were performed on several different occasions and on different saliva samples.

At this point in the investigations, when only samples from the patient herself had been tested, it appeared possible that weak B antigen was due to an abnormal allelic gene at the ABO locus, possibly manifesting itself especially weakly because of the accompanying A₁ gene, the serological expression of which appeared to be normal. The strong anti-B in her serum and the absence of B substance in her saliva were, however, evidence against this hypothesis.

The possibility of her being a chimera appeared unlikely because of the variable reactions with anti-B sera and the apparent absence of signs of heterogeneity for any of the other blood-group systems. Investigations of the patient herself were cut short by her death a few weeks after they began.

Genetical Study

An investigation of the family put a completely different complexion upon the case. Fortunately a number of relatives were available and proved most co-operative. Their blood groups are shown in the pedigree. In the case of the ABO, Rh, and MNS

Pedigree of patients' family, showing blood groups. For the ABO, Rh, and MNS groups the phenotype is given first, followed by the genotype where known. The arrow indicates the proband. She has a weak B-like antigen, apparently acquired. E antigen weak. N.T.=Not tested.

systems the phenotype is shown first, followed by the genotype where this can be deduced. The most unexpected and important finding was that the patient, whose husband was group O, had an A₂ daughter. This result was confirmed on a fresh specimen. The cells were tested with several anti-H as well as anti-A₁ sera on both occasions, and there is no doubt that this daughter is truly A₂. The patient's genotype must therefore be A₁A₂.

The finding that her sister was a normal A₁B revived the possibility that the patient might be a chimera, but the family were all certain that she had not had a twin brother or sister. The cells of her group A₁ daughter and son were used to absorb anti-A and anti-B sera. They behaved as ordinary A₁ cells, and did not remove anti-B, whereas those of the patient's group A₁B sister removed them completely: the patient's own cells removed very little indeed.

Discussion

The genetical investigation leaves virtually no doubt that the patient's genotype was A₁A₂ and hence that her B-like antigen was not the product of a gene at the ABO locus. The seven similar cases, all of group A₁, investigated by Cameron *et al.* (1959) and two, probably similar, which they quote from the literature, leave little doubt that the antigen in such cases is acquired as a result of old age or disease, though its first appearance has not yet been observed in any individual. Though five of the seven patients had cancer the authors do not consider that this association is proved to be specific: as they point out, cancer is a common reason for blood-grouping old people. However, the present case adds a further example of this phenomenon associated with cancer.

The present case, like all the others, is phenotypically of group A₁, but, being of genotype A₁A₂, differs from

the three others whose genotypes could be ascertained, and who were all A₁O.

Summary

The presence is described of a B-like antigen on the red cells of a patient of blood group genotype A₁A₂, suffering from carcinoma of the colon.

We thank the members of the family for their kind co-operation and their willingness to donate blood samples on more than one occasion. We also thank Dr. T. Cleghorn, Dr. J. W. Lacey, Dr. D. Lehan, and Surgeon Lieutenant K. J. McDonald, R.N., for obtaining blood samples from various members of the family.

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INTESTINAL DIVERTICULA

BY

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The majority of intestinal diverticula consist of hernial protrusions of the mucous membrane and submucous layers through the muscular coats of the bowel (primary type). In this inquiry, which is based on a study of patients encountered during a nine-year period, one case of acute diverticulitis of the caecum is reported; otherwise only propulsion diverticula are considered.

Diverticula of Duodenum

The total number of patients encountered with duodenal diverticula is unknown, but 10 in whom such diverticula have been demonstrated during routine abdominal investigations and without other demonstrable abnormality have been studied. Seven were female and three male, and the average age was 52 years. They all complained of flatulent dyspepsia, but the clinical findings did not permit differentiation from cholecystitis or chronic peptic ulcer. The disability was judged sufficient to warrant operation in only one of these patients, and pre-operative radiographs had appeared to show the diverticulum arising from the second part of the duodenum close to its junction with the first part. At operation it was traced behind the duodenum and pancreas to its origin from the postero-superior aspect of the third part close to its commencement. Five years after diverticulectomy this patient was found to have regained weight lost before operation, but still complained of occasional dyspepsia.

Two of these patients presented with haematemesis and melaena. One was a woman aged 60 who recovered without operation. Barium-meal x-ray examination revealed two diverticula arising from the fourth part of the duodenum (Fig. 1), and an associated colonic diverticulosis (Fig. 2). The second was a man aged 73 in whom colonic diverticula had been demonstrated three years previously. This patient died after repeated haematemeses and melaena over a period of 10 days. Post-mortem examination revealed four