demonstrates the presence of antibody globulin on the surface of the cells. Uncoated cells have always given a negative result. The positive direct Coombs reaction found in cases of acute acquired haemolytic anaemia is therefore taken to indicate the presence of antibody globulins on the surface of the cells. The presence of antibodies in the serum of these patients lends further support to this assumption.

The antibodies present in the serum and in the eluates in acquired haemolytic anaemia have previously been regarded as unspecific, or pan-agglutinins. The diagnosis of the specificity of an antibody depends naturally to a great extent on the size of the available panel of test cells. In our case the diagnosis could only be made through the availability of a cDE/cDE donor. As this subtype is comparatively rare among the British population (1.99%) (Race *et al.*, 1948), it is conceivable that even a large laboratory might not have this particular test cell available. As Kidd (1949) has pointed out, these antibodies were regarded as anti-species-specific, as in his cases no cell could be discovered which would not have been acted upon by the eluates. The test cell which permitted the diagnosis of the specificity of the antibody in our patient is, however, usually available in specialized laboratories. An antibody defining, for instance, the very frequent antigen Tj^a (Levine *et al.*, 1951) would react with all available test cells, as this antigen is so widely distributed and a cell lacking it might not be available at all. A pan-agglutinin would therefore be assumed.

Further difficulties might arise if several antibodies were formed against a variety of antigens. Very carefully performed absorption experiments and choice of test cells would be necessary to differentiate the various antibodies contained in any one serum or eluate. Multiple antibody formation is, of course, well known (Callender, Race, and Paykoc, 1945; Malone and Cowen, 1950).

It has been assumed in the past that no organism will form an antibody against an antigen carried on any of its own circulating cells (horror autotoxicus). This was taken to be a basic "law" of serology (Boyd, 1947). Our case has shown that an organism is capable of forming an antibody active against its own antigens, and the serology of acquired haemolytic anaemia would seem to show that this happens in all these cases. The antibodies attached to the cells and/or present in the serum of these patients must have combined with one or more antigens on the cell surface. Our case is exceptional in one way only—namely, that we were able to define the antigen precisely. Dameshek and Schwartz (1940) have shown that injection of antibodies directed against the cells of their experimental animals produce haemolytic anaemias typical in all respects. It is conceivable that the presence of antibodies in patients with acquired haemolytic anaemia may be the cause of these anaemias, though cases have been known in which, in spite of the persistent direct Coombs reactions, patients have had complete remissions. Further work would seem to be necessary to elucidate fully the mechanism of these types of haemolytic anaemias.

The presence of these antibodies in the serum and on the cells of the patients suffering from acquired haemolytic anaemia is difficult to explain. A tentative explanation might perhaps be offered as follows. It is a common experience that many haemolytic anaemias of the acute and acquired type are initiated by an acute febrile episode sometimes accompanied by the signs and symptoms of an upper respiratory tract infection. One might postulate that these initial febrile episodes have been caused by an agent, possibly viral, which carries an antigen similar in its structure to that present on the cells of the patient. An antibody formed in response to the stimulus of this infecting agent might cross-react with an antigen chemically related to the antigen which stimulated its production. There need not be a biological relationship between the two antigens. Many viral and rickettsial infections are known to produce antibodies which cross-react with unrelated organisms—Weil-Felix reaction and biological false Wassermann re-

actions after vaccinia and other viral infections (Moore and Mohr, 1952). (In this connexion one might point out that unspecific positive Wassermann reactions are not uncommon in cases of acquired haemolytic anaemia.) This applies particularly if the infection is severe and if the immunizing stimulus has been applied repeatedly. It is common serological experience that repetitive stimulation with one agent produces antibodies which are less specific than antibodies produced with fewer immunizing stimuli. It could also be possible that this antibody production may be initiated by a different pathological process, and the case of Levine *et al.* which produced the anti-Tj^a was shown to be suffering from a neoplasm which specifically absorbed the antibody, the patient's cells lacking the antigen.

Summary

A case of acquired haemolytic anaemia presenting a variety of interesting clinical details is described.

The development of an anti-e in the presence of the homologous red-cell antigen without stimulation by transfusion or blood injection was shown.

The serological findings were discussed and a tentative explanation was offered.

We wish to thank Dr. R. R. Race and Miss Ruth Sanger for the great help and encouragement they have given and for checking the results. Our thanks are also due to Dr. A. E. Mourant and his staff for their help and readily offered co-operation. We would also like to thank Professor A. P. Thomson, under whose care this patient was admitted to the General Hospital, Birmingham, for permission to publish.

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DIAGNOSIS OF "SEDORMID" PURPURA

BY

D. KERR GRANT, M.B., M.R.A.C.P.

(From the Clinical Research Unit, Royal Prince Alfred Hospital, Sydney)

Thrombocytopenic purpura following ingestion of the hypnotic "sedormid" (allylisopropylacetylurea) by susceptible individuals is a well-known clinical entity. The pathogenesis of this syndrome was made clearer by the investigations of Ackroyd (1949b, 1949c, 1949d) on three patients who had recovered from such attacks. As a result of his researches, he put forward the hypothesis that thrombocytopenia in these patients was associated with a circulating substance in the blood which, in the presence of sedormid, caused lysis of platelets (Ackroyd, 1949d). This lysis he observed to take place *in vitro*, using suitable preparations of the patients' blood. An alternative method of demonstrating platelet lysis was by using the indirect approach of estimating clot retraction either of whole blood or of plasma. In his patients clot retraction was very much, and consistently, reduced in the presence of sedormid. The value of such tests as these obviously lies in enabling confirmation of the provisional diagnosis to be made without the necessity of

giving a test dose of the drug by mouth. This last method, which in the absence of satisfactory *in vitro* tests is the only way of arriving at a definite diagnosis, is not without danger. Use of this test is also limited, as in one of our cases, by the patient's refusal to submit to a test dose.

It is important from the practical point of view, as well as academically, to make the correct diagnosis in suspected cases of sedormid purpura. In the absence of special measures, as outlined above, the historical sequence of events is all-important in diagnosis. The history of sedormid administration may, however, be merely coincidental. It is therefore of great advantage to be able to reassure the patient that no recurrence will take place provided no more sedormid is taken. Inability to give this reassurance demands a further search for an aetiological basis for the purpura.

In two years six cases with the provisional diagnosis of purpura due to sedormid were referred to this unit. I attempted to confirm the diagnosis in all, both by *in vitro* tests using, in the main, clot-retraction estimations, following the method of Ackroyd (1949a), and by skin patch tests. Test doses were used only in some of the cases. In two cases alone was unequivocal confirmation obtained from *in vitro* tests. The possible reasons for this failure are discussed, and a modified approach to the *in vitro* investigation of patients suspected of having sedormid purpura is suggested.

Case 1

A married woman, separated from her husband, was first seen in October, 1949, when she was aged 29. She had taken 1½-2 tablets of sedormid every night without fail for the previous seven months, the last dose of two tablets two nights before admission. After having taken the tablets for about two months, she noticed that she bruised very readily with slight trauma and sometimes even without apparent trauma. She had never before bruised easily. In addition, she developed a reddish speckled rash, particularly on the legs. The bruising and rash persisted with little change, except that in the ten days before admission both became more pronounced. A menstrual period one week before admission was more profuse than any she had experienced before. A blood film examined a few days before admission showed platelets so scanty as to be almost absent.

On admission several bruises were found; there was a petechial rash over the body, mainly on the legs, and slight oozing occurred from the gums. The positive-pressure capillary fragility test (Bell, Lazarus, and Munro, 1940) was moderately positive. A patch test of sedormid in propylene glycol, after the method of Ackroyd (1949b), was applied for three and a half days. There was no petechial reaction. Three days after admission her platelet count was 60,000, and after a further week it was 300,000. One week later, when the platelet count was 290,000, the clot retraction of whole blood by the method of Macfarlane (1939) was carried out, both in the presence of sedormid and with a normal-saline control. In the presence of sedormid, clot retraction was reduced by 16%. She did not develop any further bruises or petechiae after admission, and her capillary fragility returned to normal. The patient was discharged home.

When seen four months later there had been no further purpuric episodes. Menstrual periods had again become scanty. Her platelet count was 310,000. After a further three months she was readmitted, having for the past two weeks had a recurrence of the rash on her arms and bruises on her legs and arms. A recent menstrual period had again been associated with excessive loss. She denied, however, taking any more sedormid. Her platelet count on

admission was 220,000, and after a few days her petechiae and bruises had subsided. She then had a menstrual period, normal for her, lasting only a few hours. A patch test was again negative. Clot-retraction studies by the method of Macfarlane showed very slight reduction in the presence of sedormid. When these tests were repeated about a week later, using the method of Ackroyd (1949a), no reduction could be demonstrated. During the following five months she developed occasional crops of petechiae, mainly on the wrists and hands, and occasionally suffered apparently spontaneous bruising. However, her platelet count was always of the order of 200,000, and, apart from "spots" on the skin which might or might not have been fading petechiae, no purpuric manifestations were ever observed by us.

In June, 1951, she was admitted for trial of sedormid by mouth. Patch test and intradermal tests with the drug were negative, and no *in vitro* sensitivity could be demonstrated. She was first given a test dose of half a tablet (0.125 g.). No fall in platelet count was observed. The next morning she was given two tablets. No fall in platelet count or reduction in plasma clot retraction was observed, and there were no purpuric symptoms or signs. After a further interval of five months she was again admitted for a trial of sedormid in bigger dosage. A total of 67 tablets was given over a period of 14 days. Her platelet count and clot retraction remained unchanged. Aspiration of sternal marrow revealed no abnormality.

Case 2

A man, aged 33 when first seen in June, 1950, began to take sedormid in March, 1949, consuming 20 tablets during that and the succeeding month. About that time he noticed on both legs below the knees a dark "spotty" rash, which passed away quite rapidly. He did not take any more tablets, apparently, until the middle of May, 1950, when in four weeks he took 20 tablets. There was then an interval of a few days and he again took sedormid. After a further eight tablets he noticed a recurrence of the rash on the legs. There was no history of ready bruising. He was not sure when he had last taken sedormid, but it was probably two days before admission. Apart from a florid petechial rash on the lower legs, no abnormality was found on physical examination. Capillary fragility by the positive-pressure test was normal, and the platelet count was 190,000. Two weeks after admission clot retraction by the method of Macfarlane was significantly reduced (greater than 10%) in the presence of sedormid. Repetition of these studies four days later showed no such reduction. Clot retraction by the method of Ackroyd after a further two days again showed no reduction with sedormid. The petechiae subsided without any further lesions appearing. The patient reported on one occasion, two months after discharge from hospital, and apparently had had no recurrence of the "rash."

Case 3

A married woman, aged 48 when first seen in March, 1951, had been taking sedormid tablets on rare occasions over the past ten months, and seven days before admission she again took one tablet at night-time. On the following day she developed petechiae on legs and arms, the shoulders, and the interscapular region. There was gross spontaneous bruising in both thighs, the right upper arm, and the left forearm. She was found to have thrombocytopenia. On examination the bruises and petechiae were seen to be subsiding, and no fresh lesions were appearing. Plasma clot retraction by the method of Ackroyd revealed no *in vitro* sensitivity to sedormid. A patch test was negative. She was not seen again for eight months. In the interim, left nephrectomy had been performed for hydro-nephrosis without untoward incident. She had had no further petechiae. Repetition of plasma-clot-retraction tests again gave negative results. Platelet counts were of the order of 160,000 to 180,000.

Case 4

A widow, aged 55 when first seen in April, 1951, had been taking sedormid off and on for about four and a half years, and ten months before being seen began to take 1-1½ tablets regularly every night. After about one month she began to develop large bruises on her arms and legs, apparently unrelated to trauma. Six months later she was told that sedormid might be responsible for her symptoms, and she ceased to take the drug. After this the tendency to easy bruising went away and did not return.

Repeated tests on plasma for *in vitro* sensitivity were all negative, as were patch tests. She was therefore admitted to hospital for test dosage. After an initial dose of half a tablet (0.125 g.) had been taken without effect, the dosage was continued. In 14 days she had had 21½ tablets. There was no subjective or objective evidence of thrombocytopenic purpura.

Case 5

A married woman aged 30 was first seen on August 17, 1951. She had started to take sedormid some six to nine months previously, and thinks that she consumed, in all, the contents of three to four boxes containing 20 tablets each. The tablets were taken at quite irregular intervals. On August 9, after an interval of some weeks, she recommenced taking sedormid more regularly than she had ever done before—namely, one tablet every night. On the evening of August 13 she took two tablets. Early next morning, at about 4 o'clock, she awoke to find blood clot inside her lower lip. A few hours later she noticed spots like "blood blisters" on her fingers and hands. By the afternoon bruises had begun to appear on her arms, legs, and body, quite unrelated to trauma, and she suffered a spontaneous nose-bleed. Normally she does not bruise easily, nor is she subject to nose-bleeding. "Blood spots" and bruises continued to appear throughout that day and into the following day. She had taken no more tablets. On her admission to hospital she was seen to have haematuria, in addition to the above findings, and a little bright blood was noticed in her stools. She had felt nauseated but had not vomited. A blood film was examined and platelets were almost absent. Some 24 hours later blood was taken, using 3.2% sodium citrate as anticoagulant (Ackroyd, 1949c). Plasma-clot-retraction tests were carried out in triplicate, using a saturated sedormid solution in normal saline against normal saline controls (Ackroyd, 1949c). The results were as follows: saline control, 90% retraction; sedormid preparation, 40% retraction.

There was no further bleeding, bruising, or spots, and on August 20 plasma clot retraction gave the following results: saline control, 89% retraction; sedormid preparation, 76% retraction. The next day the platelets numbered 300,000 per c.mm. A patch test was applied to the inner surface of the right arm, consisting of a saturated solution of sedormid in propylene glycol (Ackroyd, 1949b), and a similar control patch of propylene glycol was applied to the left arm. The patch test was examined (after 48 hours) on August 22. There was no reaction at all in either patch or control. Plasma clot retraction was repeated on this day with the result: saline control, 93% retraction; sedormid preparation, 92% retraction. In all these preparations the plasma was allowed to stand in contact with the test solution for 15 minutes before clotting with thrombin.

She was next seen as an out-patient on October 23. She had been in perfect health, with no recurrence of purpura. She had had no more sedormid. Her platelet count was 170,000 per c.mm. and her blood count was normal in all other respects. Platelet counts on October 30 and November 5 were 380,000 and 160,000 per c.mm. respectively. On the latter day plasma clot retraction was repeated: saline control, 90% retraction; sedormid preparation, 93% retraction. On November 13 the platelet count was 150,000 per c.mm. and plasma clot retraction was saline control, 89%; sedormid preparation, 88%. It had been

proposed to administer a small test dose of pure allyl-isopropylacetylurea corresponding to about one-eighth of a tablet (Moeschlin, 1942). The patient declined this and further tests, and she has not been seen since.

Case 6

A widow aged 69 was first seen on January 16, 1952. Some one and a half to two years previously she had taken an unknown number of sedormid tablets over a period of a few weeks. She then changed her sedative to phenobarbitone, which she took regularly every night. Of the old stock of sedormid there remained two or three tablets in a bottle, which was then filled with phenobarbitone tablets. In the course of consumption of the contents of this bottle she thinks that two, or possibly three, tablets of sedormid were taken, one on each evening of January 11 and 12, and possibly again on the 13th. About mid-day on the 13th small black lumps like "blood blisters" appeared on her tongue. Red spots and bruises then began to develop spontaneously all over her body. Her tongue bled and purpuric spots appeared on her palate and pharynx, making swallowing painful. She had never before been subject to any abnormal bleeding tendency.

The next day the blood count was normal, apart from a platelet count of 48,000 per c.mm. Purpuric spots and bruises continued to appear, and a further platelet count on January 16 gave 15,000 per c.mm. Occult blood was present in the faeces on this day, but there was no haematuria.

On the 17th her platelet count was 67,000 per c.mm. and plasma-clot-retraction tests were saline control, 83%; sedormid preparation, 18%. Next day her platelets numbered 126,000 per c.mm. and clot retraction was saline control, 84%; sedormid preparation, 33%. A patch test, applied on January 17, was negative after 96 hours. She had now ceased to develop fresh purpuric lesions, and platelet counts continued to rise, being 200,000 per c.mm. on January 21 and 371,000 and 360,000 on succeeding days. Clot-retraction tests still showed sensitivity on January 19: saline control, 83%; sedormid preparation, 54%; but by January 23 had become normal: saline control, 87%; sedormid preparation, 86%. After one month, during which time she had been free of all symptoms, clot-retraction tests were repeated and, to our surprise, gave the following result: saline control, 86%; sedormid preparation, 56%. The patient stated that she had not knowingly taken sedormid in the interim. Unfortunately, it has not been possible to perform further tests, and this apparent return of *in vitro* sensitivity to sedormid remains unconfirmed and unexplained.

Discussion

There are certain obvious criticisms which arise at once in discussion of these cases. The most obvious and most important one is that they may not in fact represent cases of purpura due to idiosyncrasy to sedormid. This diagnosis is, in the main, a presumptive clinical one, with the exception of those cases in which, if we accept the validity of Ackroyd's work, positive *in vitro* tests have been obtained. At first I was reluctant to administer a test dose to these cases in view of the possibility of arousing a dangerous, even fatal, reaction. For some time I persisted in this attitude, even in the face of negative *in vitro* and patch tests, for I felt that failure to produce positive findings here might have been due to technical imperfections. This aspect still remains, perforce, a matter for possible criticism.

With regard to the disappointingly negative results when test-dosing was at length employed, I was encouraged to some extent by reading of the experience of Falconer and Schumacher (1940). These authors gave test doses of half a tablet and two tablets on successive days to a patient with sedormid purpura who had been free of purpuric manifestations for scarcely a week. A strong positive response was obtained after the second test dose. A little more than a

year later the same patient was again given sedormid. On this occasion, however, it was not until a total of $11\frac{1}{2}$ tablets had been taken over a period of nine days that haemorrhagic phenomena associated with marked thrombopenia appeared. In most reported cases it seems that the test dose has been administered shortly after recovery from an attack of purpura, and this may account for a correspondingly high proportion of positive results.

In Case 1 the first test doses were not given until some 20 months had elapsed after sedormid had apparently last been taken. Case 4, which was a very mild case, showing bruising as the only symptom, was not given a test dose until eight months after the last dose of sedormid. It seems not beyond possibility that such intervals of time might have allowed an acquired sensitivity to sedormid to disappear completely.

If, then, we assume that sensitivity to sedormid by mouth can diminish considerably, or even disappear altogether with the passage of time, this reasoning may surely apply equally well to *in vitro* tests. Ackroyd (1949b) says, "Hypersensitivity, once it has been established, persists for an unknown, but often considerable, period," and refers to one of his cases in which hypersensitivity was found nine years after the original attack of purpura. It appears (Ackroyd, 1949d) that he was able to demonstrate *in vitro* sensitivity in his patients, at periods ranging from 28 months in his Case 3 and 50 months in his Case 1 to some 112 months in his Case 2. The interval in his Case 2 was probably less than this, for he mentions inducing an experimental attack of purpura after December, 1940, the last recorded time of an attack due to sedormid.

The usefulness of both *in vitro* tests and the giving of a test dose in confirming a provisional diagnosis of sedormid purpura depends, therefore, on the length of time that sensitivity persists after subsidence of the purpura. If this interval is relatively long, to be measured in months or years, then there is no need for undue haste in performing the tests. If, on the other hand, the sensitivity passes off in a matter of days, as it certainly appeared to do in Case 5 above, and probably in Case 6, then *in vitro* tests should preferably be carried out as soon as possible and at the height of the purpuric attack. In Case 5 the first estimation of plasma clot retraction was performed four days after the last dose of sedormid. Clot retraction in the control specimens was normal, but in the presence of sedormid it was reduced by 50%. Three days later the reduction had fallen to 13%, and two days after this there was no reduction. All subsequent tests were negative. Case 6 followed an essentially similar pattern, except that there was an apparent return of sensitivity one month after tests had become normal. These results indicate a steadily decreasing sensitivity over a period of a few days. After experience with these cases, I referred again to our Cases 1 and 2. In both of these a reduction of clot retraction in the presence of sedormid (using the method of Macfarlane) had occurred. As this reduction had been found only during the earlier stages of the purpura, and later vanished entirely, I had been inclined to discount the results. In view of the later findings in Cases 5 and 6, I now feel that perhaps the results in Cases 1 and 2 were valid after all.

I may mention here, in passing, that I have never had a positive sedormid response in clot-retraction estimations performed on patients with florid purpura due to other causes—for example, aplastic anaemia, leukaemia.

My findings, therefore, are somewhat at variance with those of Ackroyd. From his work it would appear that there need be no undue haste in the attempt to make a retrospective diagnosis of sedormid purpura, because of the long duration of sensitivity in his patients. From my work it seems that the sensitivity may be of relatively short duration. To avoid, therefore, the possibility of, as it were, "false negative" *in vitro* tests, I venture to suggest that such tests be carried out as soon as the provisional diagnosis of thrombocytopenic purpura due to idiosyncrasy to sedormid has been made.

Summary

The use of *in vitro* tests in the diagnosis of sedormid purpura is discussed.

Evidence is presented that sensitivity to sedormid, presumed to be due to a plasma factor in the blood of patients recovered from sedormid purpura, may be of short duration.

It is therefore suggested that *in vitro* tests be carried out on the patient as soon as the provisional diagnosis of sedormid purpura is made, and preferably at the height of the purpura.

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AN OUTBREAK OF TRICHINIASIS IN BARRY, GLAMORGAN

IMPROVED TECHNIQUE FOR DEMONSTRATION OF TRICHINELLA IN MUSCLE

BY

A. DUNCAN EVANS, M.B., B.Ch., B.Sc., Dip.Bact.

AND

MARY LENNOX, M.B., B.Ch., B.Sc., D.P.H.

(From the Public Health Laboratory, Cardiff, and the
Public Health Department, Barry M.B.)

In May, 1952, an outbreak of trichiniasis occurred in the Borough of Barry, Glamorgan (population 41,000), in which at least 25 people were affected as a result of eating pork sausage-meat. We report this outbreak not only because of its size but also because the infected pigs came from a district which has been incriminated on six previous occasions, resulting in 47 human cases. Hitherto, trichinellae have not been demonstrated in pigs or rats in the district (Howell, 1941, 1942), but, arising out of the inquiries into the present outbreak, it was possible to demonstrate live trichinellae in rats and pigs from the suspected area. We do not propose to review the literature of trichiniasis in Britain, as this has been done recently by Young (1947).

The Outbreak

On May 3, 1952, two persons were seen by their doctors with symptoms suggestive of trichiniasis. Inquiries amongst the other practitioners in the area revealed a further 23 patients with a similar illness. All except one admitted eating sausage, sausage-meat, or Scotch eggs within three weeks of the onset of symptoms. The sausage and sausage-