present only in such small amounts as to be removed non-specifically by absorption.

The combination of the three antibodies found does not appear to have been described before. It could arise by ordinary immunization only in a person of the rare genotype *cdE/cdE*. It is, moreover, interesting to note that only donors of this genotype would have been compatible with this patient, who belonged to the commonest of all Rh phenotypes. He had antibodies against three of his own four main Rh antigens.

Summary

The serum of an untransfused patient of Rh phenotype *CcDee* (probable genotype *CDe/cde*) suffering from chronic leukaemia and secondary haemolytic anaemia was found to contain the Rh antibodies anti-C, anti-D, and anti-e.

REFERENCES

- Dockeray, G. C., and Sachs, H. (1941). *J. Path. Bact.*, 52, 203.
Race, R. R., and Sanger, Ruth (1958). *Blood Groups in Man*, 3rd ed., p. 327. Blackwell Scientific Publications, Oxford.
Weiner, W. (1957). *Brit. J. Haemat.*, 3, 2-6.
Bartley, D. A., Clekhorn, T. E., Marson, F. G. W., and Meynell, M. J. (1953). *Brit. med. J.*, 2, 125.

Medical Memoranda

A Psychological Analysis of the Munchausen Syndrome: Request for Information

Following the research initiated in 1954 into the Munchausen syndrome by Collie and Grygier,¹⁰ we are interested in ascertaining the present whereabouts of the undermentioned patients whom we have investigated and who are well-known examples of the condition.

Using the principles adopted by Asher,³ we have deliberately altered the patients' names, but those doctors who encounter them will, we hope, have no difficulty in recognizing them from the descriptions and pseudonyms.

Case 1.—A man, now aged 23-25, generally uses the name Austin Capp, but occasionally Chambers, Stephenson, Prince, or Rolls. He is asthenic, fair, about 5 ft. 8 in. (173 cm.) in height and of average intelligence, and generally gives an address in High Wycombe. He is admitted following abdominal pain or symptoms of meningism and has had numerous laparotomies and lumbar punctures, usually discharging himself from hospitals. His abdomen is a mass of scars. He exhibits a mixed abdominal and neurological picture of Munchausen's syndrome.³ He has mainly been admitted to hospitals in the London area, but his circuit includes those in the rest of England and Scotland. We last encountered him in Aylesbury in June, 1957. His case has been briefly reported by Sanderson²² and referred to by Chapman.³

Case 2.—A woman, now aged 39, generally uses the name Winifred Keats and Winifred or Joyce Blokeman, but more recently Joyce Allcrook, and occasionally Joyce Morris. She is auburn-haired, with a freckled face, 4 ft. 10 in. (147 cm.) in height and extremely obese—17 stone (108 kg.)—and on admission usually gives an address in the neighbourhood of the hospital. She has bilateral opaque nerve fibres and congenital colobomata, and bifrontal burr-holes due to a prefrontal leucotomy in 1952. Her abdomen is a mass of scarring. She is a mixed example of the abdominal and neurological varieties of the syndrome, and is generally admitted complaining of severe abdominal pain or with symptoms suggesting pituitary or hypothalamic disturbance. More recently she has occasionally presented herself as a cardiological problem. She usually discharges herself, and confines her activities to London and the suburbs. We last heard of her in North London in December, 1957. She has previously been reported on four occasions.^{6, 7, 13, 20}

Case 3.—An Irishman, now aged 31, generally uses the name Ernest John Mortlake, but occasionally Chars, McCreary, Nylon, Kellogg, and Finch. He has been described as short, beetle-browed, with thick, dark hair, and

the appearance of a mental defective, and is often known as "The Baron." He is frequently found having collapsed in a pool of blood near the hospital of his choosing, and may tell onlookers that he is on his way to a monastery. He is an example of the pure haemorrhagic variety of the syndrome and generally presents himself with bleedings from the ears, nose, mouth, and urinary tract, together with renal colic. He is generally thought initially to be suffering from a severe haemorrhagic disorder, and exhibits a remarkable affinity for pethidine. He has two abdominal scars—one in the R.I.F. and the other in the left hypochondrium due to an alleged splenectomy. There are scars of numerous "cut-down" incisions. He has been admitted to hospitals all over the British Isles and perhaps in France. We last heard of him in Hertfordshire in January, 1958. There are many references to him in the literature, including a detailed account by Grant.^{1, 2, 4, 5, 8, 9, 11, 12, 14-19, 21, 22-25, 27}

Case 4.—A man, now aged 40, uses the name Arthur Flitworth. He is good-looking, carefully dressed, a public-school man with a smooth manner. He is superficially plausible and exhibits flagrant pathological lying, usually asserting that he is engaged on important business, and he has enough background knowledge to keep up the pretence. He has facial scarring together with scars of bilateral craniotomy and frontal sinusotomy, the former operation carried out in Canada. He gives a history of a head injury which followed an air crash, gunshot wound, or bomb explosion. His story varies according to the hospital, and he sometimes complains of C.S.F. rhinorrhoea which resulted in his craniotomy. He is an example of the neurological variety of the syndrome and is generally admitted following epistaxes and a succession of epileptic seizures which do not appear to yield to massive sedation. More recently he has been admitted to a few hospitals complaining of pain in the chest and haemoptysis which is thought to be self-induced, and he generally discharges himself after having caused considerable disturbance in the wards. He has been admitted to hospitals and neurosurgical units throughout the country and in Canada, and was last heard of at the Brook Hospital in December, 1957.

Case 5.—A woman, present age 49, generally uses the name Elsie Shackleton and Elsie de Coverley, but also a great many other names, including Joan Summer, Jean Hops, Joan Lark, Joan de Coverley, Joan Malkin, Joan Morris, Elsie Layton, Elsie Jeans, Elsie Jackman, etc. She is Case 3 of Asher's original article³ and has also been referred to by Wilks.²⁶ She is 5 ft. 1 in. (155 cm.) tall, weighs 7½ stone (47.6 kg.), has a sallow complexion, and frequently gives a previous history of a number of fictitious operations for stomach trouble performed at the Royal Devon and Exeter Hospital. She is an example of the abdominal variety of the syndrome, and is usually admitted following severe abdominal pain and a history of recent haematemeses. Her abdomen is a mass of scars, but in addition she has mitral stenosis, and more recently has gained admission to hospitals with anginal pain and breathlessness, and is sometimes thought to have had a myocardial infarct. She now has a left Horner's syndrome resulting from a left cervical ganglionectomy that was carried out for cardiac pain in 1955. Most of her admissions have been to hospitals in London and the Home Counties, and her last known appearance was at Lambeth Hospital in September, 1957.

Case 6.—A woman, now aged about 37, and Case 2 of Asher's original article.³ She uses three names—Elsie Packoma, Margaret Coke, and Elsie Silverborough. Her abdomen is a mass of scars and she is an example of the abdominal type of the syndrome. Her itinerary includes hospitals in the London and Manchester areas, and she would sometimes maintain that her previous operations had been done by a "house-doctor" in Houston, Texas. She was last heard of by us in March, 1951, at St. James's Hospital, Portsmouth.

We have traced over 120 admissions on one of these patients, and the others do not lag far behind. To our knowledge they still continue their peregrinations, and we should like to follow up their histories.

If any of these individuals are at present hospital in-patients or arrive in the surgeries or casualty departments, it would be much appreciated if the hospital concerned could notify us as soon as possible (write, or phone VIGILANT 6611). We would then arrange either to visit the patients directly, or, alternatively, they could be admitted to Banstead Hospital.

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REFERENCES

- ¹ Alderson, D. A., and Henriques, C. Q., *Brit. med. J.*, 1956, 1, 462.
- ² Armitage, G. H., *Lancet*, 1951, 1, 743.
- ³ Asher, R., *ibid.*, 1951, 1, 339.
- ⁴ ———, *Brit. med. J.*, 1955, 2, 1271.
- ⁵ Balmforth, G. V., and Pollock, J. A., *ibid.*, 1955, 2, 1330.
- ⁶ Barker, J. C., and Grygier, T. G., *Lancet*, 1957, 2, 1069.
- ⁷ Bass, B. H., and Selson, M. G., *ibid.*, 1957, 2, 1006.
- ⁸ Bolam, R. J., *ibid.*, 1951, 1, 743.
- ⁹ Chapman, J. S., *J. Amer. med. Ass.*, 1957, 165, 927.
- ¹⁰ Collie, I. F., and Grygier, T. G., *Lancet*, 1954, 2, 1284.
- ¹¹ Cook, P. J., *ibid.*, 1951, 1, 743.
- ¹² Drury, M. I., *J. Irish med. Ass.*, 1952, 30, 141.
- ¹³ Essenhigh, D. M., and Peachey, R. D. G., *Lancet*, 1957, 1, 638.
- ¹⁴ Gatenby, P. B. B., *J. Irish med. Ass.*, 1952, 30, 102.
- ¹⁵ ———, *Brit. med. J.*, 1955, 2, 1207.
- ¹⁶ Gawn, R. A., and Kauffmann, E. A., *ibid.*, 1955, 2, 1068.
- ¹⁷ ———, *ibid.*, 1955, 2, 1329.
- ¹⁸ Grant, A. P., *J. Irish med. Ass.*, 1952, 31, 299.
- ¹⁹ Young, A. G., *Lancet*, 1951, 1, 744.
- ²⁰ Heathfield, K. W. G., *ibid.*, 1957, 1, 533.
- ²¹ Irvine, R. E., *Brit. med. J.*, 1955, 2, 1207.
- ²² Sanderson, P. H., *Lancet*, 1957, 1, 483.
- ²³ Short, I. A., *ibid.*, 1951, 1, 639.
- ²⁴ ———, *Brit. med. J.*, 1955, 2, 1206.
- ²⁵ Staffurth, J. S., *ibid.*, 1955, 2, 1207.
- ²⁶ Wilks, J., *Lancet*, 1951, 1, 475.
- ²⁷ Jones, E. W., *Brit. med. J.*, 1955, 2, 1271.

Acute Barbitone and Bromide Poisoning with Unusual Features

Severe poisoning with barbiturate usually causes some or all of the following: coma, absent reflexes, depressed respiration (usually with a slow rate of breathing, deep and stertorous or shallow, often with cyanosis and sometimes pulmonary oedema), variable pulse rate, frequently low blood pressure, lowering of body temperature, and dehydration if coma has persisted for some time. The clinical findings are very variable, depending on the type of barbiturate swallowed, the dose in relation to size, duration of poisoning in relation to a previous meal, and the age and previous state of health of the patient. The findings detailed in the following case history were unusual enough to cause difficulty.

CASE REPORT

A male schizophrenic aged 44 had complained of cough for many years. Pulmonary tuberculosis and dust disease had been excluded, but he had a mild chronic bronchitis. During February and again in April, 1956, while in a mental hospital, he was found to have a slight tachycardia (70–86 a minute), labile hypertension 120/80 to 160/100 mm. Hg, respiratory rate 24–28 a minute, and a normal x-ray appearance of the chest.

On July 6, 1957, he was put to bed at 11 p.m. after an episode of mental disturbance; at 7 o'clock the next morning he was found unconscious; at 11.30 a.m. his doctor was called and visited him promptly. The patient was then in coma, all reflexes except the conjunctival were absent, the left leg was more flaccid than the right, he was incontinent of urine, the pupils were dilated, the pulse was slow and of fair volume, and he was frothing copiously at the mouth. The doctor wrote: "The whole condition is suggestive of barbiturate poisoning, but I can find no source for this. He has been on a bromide-barbitone sodium mixture, but has not taken excess of this." He was admitted to hospital at 1 p.m., when his condition was found to be essentially unchanged, and in addition it was noted that all reflexes were absent, anaesthesia was complete, the eyes and mouth were dry and foul, the abdomen was silent, the bladder was not palpable, and the skin was dry; axillary temperature was 103° F. (39.4° C.), pulse 86, B.P. 170/110, and respiratory rate 80. Cardiac sounds were remote, and harsh sounds con-

ducted throughout the chest, but there was no pulmonary oedema. The facies was blue-grey in colour.

Much mucus was sucked from the throat and pharynx and oxygen was given by mask. Samples of blood, urine (2 oz. —57 ml.—by catheter), and C.S.F. (clear, under increased pressure) were withdrawn and sent for analysis. As oxygen therapy did little to relieve the cyanosis and the tachypnoea defeated attempts at manual assistance to respiration, it was decided to inject the contents of 1 ampoule (125 mg.) of the soluble sodium salt of bemegride ("megimide sodium") in 5 ml. of saline intravenously. This compound has a dramatic effect in dogs anaesthetized with pentobarbitone sodium and may increase respiratory minute-volume by eight times within one minute of injection, but its effects in man have not so far been reported.

There was an immediate improvement. Coma lessened; a sluggish light reflex appeared in smaller pupils; reaction to firm supraorbital or plantar stimulation returned; he groaned, grimaced, and babbled words after a few minutes; muscle tone improved greatly; and cyanosis lessened. The B.P. was 160/90, pulse 120, and respirations 60. Within 45 minutes these signs of improvement had gone.

Analytical results were as follows. Urine: sp. gr., 1030, acid; albumin-positive; a trace of sugar; no barbiturate, salicylate, or bromide. C.S.F.: clear, no cells, protein 56 mg./100 ml.

In view of the failure to find barbiturate and because of the marked tachypnoea, fever, and hypertension, 1 million units of benzylpenicillin was given, suction repeated, and the patient transferred to the ward for observation. Thereafter his condition deteriorated, and by 8 p.m. he was in deep coma, flaccid, anaesthetic; reflexes were absent. The pulse was 110–65 and respirations 60/90; the chest was full of rales; B.P. was 110/70; he was sweating heavily and had a temperature of 100° F. (37.8° C.). A glucose-saline drip was instituted and bemegride, 0.4 g., and amiphenazole ("daptazole"), 50 mg., were injected in the course of an hour. Reflexes returned within 30 minutes; pulse slowed to 100, but rose after two hours to 120 and fell below 100 only after 48 hours. Blood pressure declined to 90/60, then rose steadily to 120/60 in three hours and 170/110 in two days. The respiratory rate fell to 50 and in three hours to 30; next day it rose to 35, then declined slowly as fever resolved over three days.

After two hours he could sit up with assistance and take fluids by mouth. On July 8 blood examination showed: normal electrolytes and alkali reserve; Hb, 16.6 g./100 ml.; W.B.C., 23,300 (polymorphs 79%); glucose 147 mg./100 ml.; urea, 80 mg./100 ml.; Wassermann and Kahn reactions negative. The first urine specimen was loaded with albumin, had a trace of sugar, and was like pomegranate juice in colour. Unfortunately the pigment was not determined. X-ray films of the chest and E.C.G. were normal. The patient improved steadily on penicillin-streptomycin treatment and was transferred to a mental hospital after five days.

COMMENT

In 354 consecutive cases of barbiturate poisoning observed personally, no patient has had a respiratory rate over 35 a minute, a temperature above 100° F. (37.8° C.), or blood pressure above 140/90 mm. Hg. The abnormal findings and apparent absence of urinary barbiturate or bromide delayed the diagnosis. Bemegride sodium was dramatic in its effect but is hardly diagnostic; bemegride treatment was very effective when used. Blood taken at 2 p.m. on July 7 contained 10 mg. of barbitone per 100 ml. and on July 15 18.75 mEq./l. (150 mg. per 100 ml.) of bromide. He admitted to having taken 5 g. (75 gr.) of barbitone sodium and 10 g. (150 gr.) of sodium bromide at midnight on July 6 in 120 ml. (4 fl. oz.) mixture.

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