RECOVERY OF VISION AFTER TWENTY-SEVEN YEARS

BY

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It has recently been my good fortune to be able to operate successfully on a woman, aged 35, who had been suffering from double lamellar cataract (as illustrated in Fig. 3) of an opacity such that she had only been able to distinguish between light and dark since she was 8 years old. Such an opportunity must be of very rare occurrence, and, consequently, there is no routine procedure for such cases ; in the circumstances I think that a few notes on my experience may be of some interest.

The patient was employed as a telephone operator, and

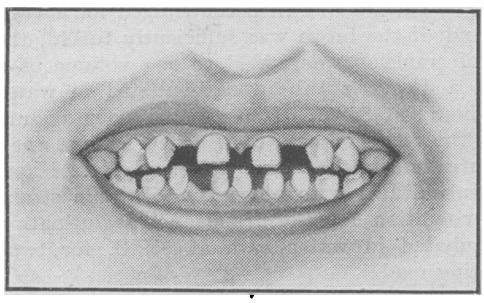

FIG. 1.

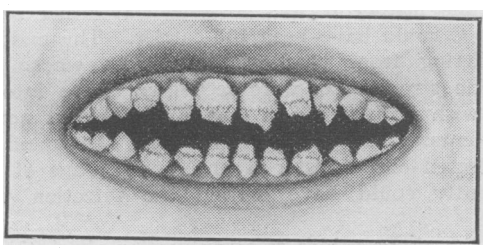

FIG. 2.

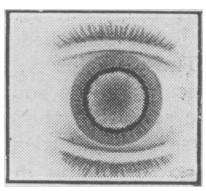

FIG. 3.

performed her duties quite satisfactorily, but her general health was not good ; her complexion was bad, and she had little interest in life or in her personal appearance. When a very small child she had been able to see sufficiently well to learn her alphabet and elementary reading and writing, always holding her book very close to her eyes, as she was, in addition, highly myopic.

With regard to her present condition, her teeth illustrate well the defective enamel so frequently found in association with lamellar cataract ; they are shown in the accompanying drawing (Fig. 1). Fig. 2 was drawn from a case of a boy, aged 14, on whom I operated for lamellar cataract some time ago; it clearly demonstrates the exposed dentine, affecting, as it almost invariably does, the central and lateral incisors, the canines, and first molars of the permanent dentition in upper and lower jaws ; this is found to be worn away in the adult.

The presence of about 10 dioptres of myopia, as measured around the limit of the cataract with a dilated pupil, made me fear the possibility of a detached retina if any operation was performed involving a large opening in the eyeball. I decided to operate by needling only, to proceed by small stages, and to open the eye only in case of urgent need. On each occasion I used a Ziegler knife—one only—and wore a binocular loupe to observe the detail of the operation. On the first occasion I made one long cut only ; on the second, two crescentic cuts, joining at each end ; this was done to ensure the escape of the contained portion of lens matter and, at the same time, to limit the total amount of lens matter which could escape at one time and so obviate glaucoma. The technique proved to be most satisfactory.

On the day following the second operation I applied two leeches to control the small rise in tension which was inevitable ; in this way all trouble from increased tension was avoided. Following the second operation three further needlings were performed on the right eye and four on the left ; on each occasion the loupe was worn and the procedure adapted to the condition found.

The result was as follows:

$$\text{Right with } \frac{+\ 4.00\ \text{DS}}{+\ 1.00\ \text{DC Axis } 85°} = \frac{6}{6} \text{ partly.}$$

$$\text{Left with } \frac{+\ 4.00\ \text{DS}}{+\ 1.50\ \text{DC Axis } 90°} = \frac{6}{6} \text{ partly.}$$

With + 3.50 DS added each eye = J 1.

I consider that the failure to read 6/6 fully is due to imperfect development of macular function, as there is no medial obstruction or fundus disease.

Since the patient has been able to see, my efforts to improve her general health have met with remarkable success ; she now has a good complexion, has energy and interest in life, and the natural pleasure of being able to choose her own clothes and attend to her own personal appearance has transformed her whole mental outlook. She has learned to write, has become a shorthand-typist, and hopes before long to leave her switchboard for another post, to which she looks forward with much interest.

The illustrations are from drawings prepared by Theodore Hamblin, Ltd., opticians, Wigmore Street.

ACUTE HAEMORRHAGIC PANCREATITIS

REPORT OF FOUR CASES

BY

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Although acute pancreatitis is perhaps not to be considered as one of the very rare causes of the acute abdomen, in general hospital statistics it appears to be infrequent. The occurrence of four cases in one year in the practice of a single surgeon, and the fact that they were all extremely typical of this condition, may justify recording them. The first two of these cases were verified at operation ; the second two, unfortunately, were fatal, without operation, and in neither was a necropsy permitted. The symptoms in these latter cases were, however, so characteristic as hardly to admit of doubt. The first patient was a girl, aged 17 years, who was operated on and died, chiefly, I believe, because the tube draining her gall-bladder came out and allowed the escape of stones and bile into the abdomen, in this manner adding the factor of infection to what was, in my opinion, an aseptic inflammation of the pancreas. The second patient recovered. She was a woman, aged 50, who was also operated on in the same manner, and presented at operation much the same appearances. In both these patients the gall-bladder, while showing no definite signs of inflammation, contained numbers of very small cholesterol calculi, the type which, according to the theory of Opie and Archibald, might lodge at the bile papilla, leaving the opening of the duct of Wirsung patent behind it, and so permitting the regurgitation of bile into the pancreas.

CASE I

Miss Charlotte P., aged 17 years, was a shop assistant. Four days prior to admission she had a sudden attack of severe epigastric pain, accompanied by persistent vomiting and constipation. The patient was sent in as a case of acute intestinal obstruction. On admission, she was obviously extremely ill, complaining of agonizing pain in the epigastrium. There was tenderness below the right costal margin and in the epigastrium, and rigidity over the upper abdomen. Tenderness was also present in the left costo-vertebral angle. Her face was flushed. Temperature 100°, pulse 126, respiration 24.

On November 13th, 1930, the abdomen was opened by a right upper paramedian incision, and a quantity of blood-stained fluid escaped. Extensive fat necrosis was scattered all over the omenta. The pancreas, which was exposed by tearing through the gastro-colic omentum, was found to be swollen and to resemble a piece of raw beef-steak. The gall-bladder was moderately distended, and contained a quantity of small round yellow cholesterol stones about the size of a hemp seed. The operation was rapidly concluded by fixing a tube in the gall-bladder with a purse-string suture. No attempt was made to remove all the stones, and a tube was passed down to the pancreas after scratching through the peritoneum over it. The abdomen was closed with through-and-through silkworm-gut sutures.

Blood sugar at this time was 0.2, the diastatic index of urine being 40. For a week the patient made great improvement and appeared to be on the road to recovery. On November 21st the tube draining the gall-bladder either came out of itself or was pulled out, and from that time she went downhill. On November 29th she was seized with agonizing abdominal pain. The abdomen was then reopened, and it was found that the bile and small gall-stones had leaked freely into the abdomen. The fat necrosis present at the original operation had disappeared. The pancreas was exposed with some difficulty, and was found to be surrounded by a quantity of blood-stained fluid. The diastatic index of this fluid was 500 ; ten days later the patient died, four weeks after the first operation.

Commentary

In my opinion the sequence of events was as follows. A small stone lodged at the papilla and bile was injected up the pancreatic duct, setting up an aseptic inflammation. With drainage of the pancreas and relief of pressure in the common duct by draining the gall-bladder no further bile reached the pancreas. All her symptoms were relieved, and recovery would probably have ensued, but when the tube came out of the gall-bladder, with the resultant escape of bile into the peritoneum and abdominal wound, an aseptic process in the pancreas became converted into a septic one, with a fatal result.

CASE II

Mrs. Mary J., aged 50 years, a housewife. For years the patient had suffered from attacks of epigastric pain radiating to the back, accompanied by nausea and flatulence. There was no jaundice at any time. Two days before admission to the Cardiff Royal Infirmary she had a severe attack of epigastric pain. The day after admission (August 19th, 1930), I was asked to see her by the house-surgeon, Mr. Morgan, as he thought she was a case of acute pancreatitis. Her face was cyanosed, especially round the lips. She was very dyspnoeic, speaking in gasps and with acute pain. Temperature 98°, pulse 100, respiration 40. The abdomen was distended and rigid. There was acute tenderness in the epigastric and umbilical regions, and at the left costo-vertebral angle.

The abdomen was opened by a right upper paramedian incision on August 20th, when a quantity of blood-stained fluid escaped. Numerous patches of fat necrosis were scattered over the omenta. The gall-bladder, containing a quantity of very small yellow stones, was opened and drained. The pancreas, exposed through the gastro-colic omentum, was swollen and red, and a tube was placed in contact with it. The patient steadily improved from the time of the operation, and was discharged on October 13th free from symptoms.

CASE III

S. E., aged 46 years, was a police constable. On June 20th, 1930, he was seen by me in consultation with Dr. Anderson of Penygraig, who diagnosed an attack of gall-stone colic, the symptoms being severe pain in the epigastrium passing round to the right scapular region, with vomiting. The attack had come on the previous night. When I saw him it appeared to be passing off, and the patient, who was afraid of operation, was endeavouring to make light of it. He was extremely fat, and it was very difficult to make anything out by examining the abdomen, except that there was tenderness in the gall-bladder region ; but the history appeared pretty definitely to indicate biliary colic, and I had him admitted to my ward at the Royal Infirmary the following day. He had had a similar attack a week previously. The abdominal symptoms were now less in evidence, and as he was found to have some consolidation at the base of the right lung and was a very unfavourable subject for operation, he was transferred to Professor Kennedy's ward in the Medical Unit. The note states that there was then "tenderness in the right hypochondrium, and the right upper rectus was slightly rigid," and that there was "dullness, fine rales, and pleuritic rub in right axillary region and behind." I did not see the patient again, but was told by Professor Kennedy that the following day he was slightly cyanosed, and this condition became more marked the day after. The pulse varied from 112 to 136. Blood sugar, 400 mg. per cent. On June 25th, 1930, he was seized with severe abdominal pain, and was breathless and drowsy. He then became extremely cyanosed, and died the same day. A post-mortem examination was refused.

Commentary

Although not verified by operation or post-mortem there seems little doubt that this was a case of acute pancreatitis ; with this opinion Professor Kennedy is in agreement. Two attacks of biliary colic, a remission followed by sudden agonizing abdominal pain and rigidity, rapid pulse, marked cyanosis, and the presence of a very high blood sugar, would seem conclusive evidence. The patient was extremely fat and an alcoholic, and operation would probably have been unsuccessful, especially in view of commencing pneumonia.

CASE IV

With the view I hold of etiology of acute pancreatitis, this case is of particular interest to me. Mrs. P., a housewife, aged 34, was a patient of Dr. Nelson of Treherbert, who was called in by a midwife to a case of prolonged labour. Dr. Nelson found no evidence of pregnancy, and diagnosed intestinal obstruction. The patient was suffering from pains resembling those of parturition ; the lower abdomen was distended ; and there was obstinate constipation and occasional vomiting. This condition had continued for a fortnight. The patient was then sent into the Treherbert Hospital, where, on July 2nd, 1930, I opened the abdomen under general anaesthesia by a lower median incision. I found that she had had a subtotal hysterectomy, and that a loop of small intestine had become fixed to the cervical stump and was acutely kinked. It was separated, and the raw areas on the intestine and in the pelvis were peritonized. I happened to pass my hand to the upper abdomen and felt the gall-bladder, which was of normal size and consistency, but contained a quantity of small gall-stones, and I remarked then that these were just the sort of stones which might cause acute pancreatitis. As it was impossible to deal with the gall-bladder through a lower abdominal incision, and, since her condition did not warrant doing anything further at the time, the abdomen was closed.

Three days later, July 5th, the patient was seized with acute abdominal pain ; there was some distension, and she vomited a dark brown fluid. On July 7th she had severe pain in the back, and diarrhoea. On the following day she became cyanosed. Pulse 120, temperature subnormal. Vomiting of brown fluid and diarrhoea continued. On July 9th I was asked to see her, although I was told her condition was desperate. What struck one at once was her appearance. With all the symptoms of collapse she had a bright pink flush on her cheeks. Her pulse was 130, temperature 97°. There was acute tenderness at the pancreatic point above the umbilicus and in the left costo-vertebral angle. The diagnosis of acute pancreatitis appeared evident, but no persuasion would induce

her to let me operate again. The next day she became wildly delirious and was in agonizing pain, still passing dark brown fluid per rectum. She died that night. I feel convinced that after the gall-bladder had been handled a small stone worked its way down the cystic duct and lodged at the ampulla, causing regurgitation up the duct of Wirsung.

Commentary.

Although the diagnosis, unfortunately, was confirmed neither by operation nor by post-mortem, the symptoms were so typical as not to admit of doubt. As to the cause the theory of infection seems untenable. No focus of infection was present in the abdomen, the gall-bladder showed no signs of active inflammation, and, at the operation for the relief of obstruction, the bowel was not opened, the operation resolving itself into the separation of an adhesion.

REMARKS

In three of these cases gall-stones were present, all of very small size, such as might block the opening of the common bile duct without at the same time blocking the opening of the duct of Wirsung.

In the *British Medical Journal*,[3] I published a series of twelve cases of acute necrosis of the pancreas, six of these being my own. In ten of these, gall-stones were present in the gall-bladder ; in several it is noted that the stones were small. In one case with large stones in the gall-bladder, a small stone was found impacted at the ampulla of Vater. While not for a moment suggesting that all cases of acute pancreatitis are due to the impaction of a stone at the ampulla, I think there is no doubt that in many, if not most, of the acute fulminating cases the cause is the retrojection of bile or duodenal contents into one or other of the pancreatic ducts. In the early stages of the condition the evidence of infection is very slight in many cases. Graham, who at one time was an adherent of the theory of the lymphatic origin of pancreatitis, seems now to have changed his view, and in his recent work on the gall-bladder[1] quotes the investigations of Kodama[2] on the dog. Kodama found that dye injected into the subserosa of the gall-bladder passed in the lymphatic vessels over the pancreas to a gland at the commencement of the portal vein, and did not enter the substance of the pancreas ; nor does it appear probable that infection is likely to pass against the direction of the lymph flow. The argument against the views of Maugeret, Deaver, and others has been very fully and clearly discussed by Fiske Jones,[4] and I need not recapitulate it here. It should, I think, carry conviction to those who are not irretrievably wedded to the elusive haemolytic streptococcus. He also points out, what one notes in Graham's work, that it is very difficult to make out whether the upholders of this theory are referring to the above condition or to the chronic interstitial pancreatitis which may conceivably have a lymphatic origin. I quote his remark on this subject: "It is impossible to read the articles of Deaver and others who accept Maugeret's theory as to the cause of pancreatitis, and those of Archibald and others who believe in Opie's theory, without feeling that they are discussing two different diseases."[1]

The question is of more than academic interest, for correct treatment depends, to a large extent, on the view we take of the causation.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

EFFECT OF INTRAVENOUS MAGNESIUM SULPHATE ON THE BLOOD PRESSURE IN URAEMIA

The treatment of uraemia in acute glomerular nephritis by the intravenous injection of magnesium sulphate was originally described by Blackfan and Hamilton (*Boston Med. Surg. Journ.*, October, 1928). They reported eleven cases of children in whom the treatment was used. Of these, eight recovered, and three, in whom it was not used until the terminal stages, died. Blackfan and Hamilton used the treatment in the acute glomerular type of nephritis, characterized clinically by the acute onset, the slight degree of oedema, and a rise in the blood pressure, together with haematuria and albuminuria. This glomerular type shows a marked tendency to progress to uraemia. They found that the degree of oedema and haematuria, and even of oliguria, was of no great significance, provided that the systolic blood pressure remained below 130 mm. But if the blood pressure commenced to rise above 130, with, as often occurred, vomiting, headaches, and visual disturbances, they regarded magnesium sulphate treatment as indicated. Their technique was the slow intravenous infusion of 1 per cent. magnesium sulphate (2 grams of the crystalline salt, $MgSO_4 + 7H_2O$, in 100 c.cm. of distilled water).

CASE HISTORY

A boy, aged 9, was admitted on February 22nd, 1931, with a history of vomiting and sore throat of three days' duration. On examination he was found to have the rash, throat, tongue, and enlarged glands of a typical third-day case of scarlet fever, with a temperature of 99° F. and a pulse rate of 112. His urine was then free from albumin. The disease followed a normal course until March 16th (twenty-fifth day of disease), when the routine urinary examination showed that albumin was present. The next day red blood corpuscles, and granular and epithelial casts, were seen microscopically, and the patient's face was noticed to be pale and puffy. A diagnosis of acute glomerular nephritis was made. The bowels were freely opened by the regular administration of jalap, pil. colocynth, and sodium sulphate. However, on March 19th (fourth day of the nephritis) he had six convulsions, lasting from three to eight minutes each, spread over an hour and a half, with complete unconsciousness between. He was then treated by venesection, and chloral and potassium bromide per rectum. Two hours later the patient was still semi-conscious, with a blood pressure of 148/132 ; 150 c.cm. of 1 per cent. magnesium sulphate was then sterilized and given intravenously by the gravity method. The following systolic blood-pressure readings from the hourly chart kept show the effect (the diastolic was found to follow the systolic): after one hour, 148 ; three hours, 146 ; six hours, 122 ; eight hours, 128. By the next morning the systolic blood pressure was found to be 150, and intravenous magnesium sulphate was repeated. The effect on the blood pressure was: after one hour, 144 ; three hours, 144 ; six hours, 122 ; eight hours, 128. The following morning (sixth day of nephritis) the reading was 108, and in the evening 126. After that, except for one reading of 134 on the sixth day of the nephritis, it settled at 120, gradually falling to 112. From the onset of the nephritis the urinary output varied from 8 to 19 ounces until March 24th, when 38 ounces were passed, after eight days of partial suppression. The magnesium sulphate did not affect the quantity of urine. Red blood corpuscles disappeared from the urine on the fourteenth day of the nephritis, and albumin on the nineteenth day. The patient appeared to have suffered no permanent damage to the kidneys, having no recurrence of albuminuria on leaving his bed, and being perfectly well on discharge, ten weeks from admission.