Operations for the Relief of Visceral Pain

I cannot deal with all the operations that have been devised for the relief of purely visceral pain. I shall deal with the pain of angina pectoris, and with that which arises from disease of the pelvic viscera, because these two types illustrate the anatomical and the physiological principles involved in surgical treatment.

ANGINA PECTORIS

Let us leave on one side the mechanism of production of anginal pain as well as the desirability or not of its suppression, and try to work out a satisfactory procedure for its relief. I have already stated that the greatest concentration of pain fibres from a viscus is to be found in the sympathetic strands in its vicinity. In the case of the heart these are in the cardiac plexus, but a direct attack on this is obviously out of the question. After the first concentration we must look for a "spread" over sympathetic roots; this is over a vertical extent, the upper boundary of which is the junction of the superior cardiac nerve with the superior cervical ganglion, the lower boundary the ramus communicans of the fifth thoracic nerve. To extirpate all the nerves in this spread is obviously undesirable. If it be remembered, however, that the highest available sympathetic connexion to the spinal cord is by way of the ramus communicans of the first thoracic nerve, the vertical extent of attack can be greatly reduced.

Attention may be focused on the posterior roots of the upper five thoracic nerves, but rhizotomy of these would be a formidable operation for so poor a surgical risk. It has been found possible, however, to block afferent cardiac fibres outside the spinal canal by injecting alcohol into and around the upper five thoracic ganglia of the sympathetic chain on the left side. This is not a serious procedure; many good results have followed its application, and I have myself used it with satisfaction. The plan is a compromise between the ideal one of attacking at the most concentrated zone, with high risk, and attacking over a wider front, at a low risk.

PELVIC VISCERAL PAIN

The first "zone of concentration" of sympathetic fibres from pelvic viscera is conveniently—perhaps too conveniently—open to surgical attack as the presacral nerve in front of the fifth lumbar vertebra. In it pass afferent fibres connected with the bladder, the rectum, and certain of the internal genital organs; and it is usually easy enough to resect, with a minimum operative risk. Before employing presacral neurectomy for pain of pelvic origin, it is necessary to be satisfied that the strand does not contain extrinsic nerves which transmit impulses indispensable to the proper functioning of any viscus. Such fibres appear to be absent, for presacral neurectomy is followed by singularly little disturbance of function; indeed, the only really undesirable sequel is sterility, though not impotence, in the male.

The operation has been applied with success in certain cases of intractable cystitis, and in the palliative treatment of inoperable malignant disease of the bladder. It has also been utilized for the relief of intractable dysmenorrhoea, when the pain is of a colicky type; these patients must be very carefully selected. It is, however, by no means certain that the beneficial effects of the operation are directly due to the division of pain fibres alone, although these are undoubtedly present in the nerve. Probably the division of vaso-constrictor fibres that traverse the nerve diminishes the intensity of symptoms due to inflammatory disease by improving the blood supply of the diseased part; and in "obstructive" dysmenorrhoea the efforts of the uterus to get rid of its

contents may be assisted by a reduction in the tone of the cervix as a result of division of some of its extrinsic nerve supply.

Summary

1. There are available a number of surgical procedures which have for their sole object the abolition of pain.
2. Certain of these constitute the treatment of choice for the appropriate condition.
3. None of these operations is to be employed without an assessment of its possible remote effects.
4. Certain of these operations might well be employed more frequently in malignant disease, and at an earlier stage.

FAMILIAL ACHOLURIC JAUNDICE SIMULATING LEDERER'S ANAEMIA

BY

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The differentiation between acholuric jaundice and the acute haemolytic anaemia associated with the name of Lederer may present considerable difficulty, a fact which is well exemplified by the following case records.

Case I

A boy, aged 10, complained on April 19th, 1934, of pain in the left side of the upper abdomen; he felt sick, and vomited repeatedly throughout the day, the vomitus being green and watery. During the following day he was slightly better, but had similar vomiting at night. He did not sleep well, complaining of headache, and became delirious. On April 21st his condition was worse, and he was admitted to hospital. He had been noted to be feverish and to have dark-coloured urine. His bowels had been regular, but were loose on admission, following Gregory's powder.

CONDITION ON ADMISSION

On admission he was seen to be a well-developed but rather thin boy, showing an extreme degree of pallor of the lips and mucous membranes. The skin was of a slightly yellowish colour, and there was a definite icteric tinge in the conjunctivae. He was very restless, with sighing respirations. The temperature was 102°, pulse 140, and respirations 25. The spleen was enlarged, extending beyond the costal margin medially to within an inch of the mid-line and downwards to about the level of the umbilicus. It was firm in consistence and slightly tender, but no friction could be distinguished. The liver was not enlarged, and there were no signs of enlarged lymph glands. The heart appeared slightly dilated, and loud blowing systolic murmurs could be heard all over the praecordia. No abnormalities were found in the other systems. The urine contained urates, but chemical and microscopical examination revealed no evidences of blood, albumin, sugar, or acetone.

The faeces contained no occult blood. The blood findings were: red blood corpuscles 1,500,000 per c.mm.; haemoglobin 18 per cent.; colour index 0.6; and white blood cells 6,000. The blood film showed small, well-filled cells. There was slight variation in size; the majority appeared somewhat smaller than normal, but regular in shape. No primitive cells of either the red or the white series were present. Reticulocytes numbered less than 1 per cent. The fragility of the cells did not appear to be increased, the range being 0.45 to 0.35. Van den Bergh test, direct reaction delayed positive; icteric index 10.

BLOOD TRANSFUSION AND SPLENECTOMY

On April 22nd the patient was transfused with ten ounces of citrated blood. Following the transfusion the boy was less dyspnoeic, but there was no improvement in the blood count and no reticulocyte response. Accordingly he was

given a second similar transfusion four days later, after which he made a rapid improvement, with a reticulocyte crisis, a drop in temperature to normal, and a slowing of the pulse rate. With the improvement there was a rise in the colour index and a slight diminution in the size of the spleen. Ten days after the second transfusion the fragility range was found to be 0.5 to 0.4. Three weeks after the second transfusion the patient's blood level became stationary, and a permanent reticulocytosis of 15 to 20 per cent. was found to be present along with a more definite jaundice.

The patient was seen from time to time until October, 1934, when at last the parents consented to splenectomy, as the general condition was obviously deteriorating with a progressive anaemia. Splenectomy was performed by Professor Wilkie, and at the same time a thickened gall-bladder containing several small dark faceted stones was removed. Following the operation the patient made rapid improvement. The progress of the case is shown in Table I.

TABLE I

Date	Day of Disease	R.B.C. in millions	Haemo-globin %	Colour Index	W.B.C. in Thousands	Reticulo-cytes %	Fragility Range	Icterus Index	
April 22	4	1.50	18	0.6	6.0	<1	0.45-0.35	10	Transfusion, 10 oz.
" 23	5	1.25	18	0.7		<1			
" 24	6	1.22	16	0.6		<1			
" 26	8								Transfusion, 10 oz.
" 27	9				7.5	4			
" 28	10		21			2			
" 29	11		23			15			
" 30	12	1.80	32	0.9		18			
May 2	14		37			15			
" 3	15					11			
" 5	17					7			
" 6	18	2.18	45	1.0			0.5-0.4		
" 10	22	2.60	54	1.0	5.2	3			
" 15	27	3.40	63	0.9	6.0				
" 24	36	3.32	63	0.9					
" 27	39					15			
" 28	40					21			
June 1	44	3.10	58	0.9		16	0.6-0.45	22	Left hospital
July 11	81	2.70	50	0.9		16			
Aug. 22	126	2.60	50	0.9		15	0.6-0.4	14	
Oct. 9	174	2.40	45	0.9		16			
" 23	188								Splenectomy
Nov. 8	204	4.53	80	0.9	11.0	<1	0.6-0.4		

Case II

A boy, aged 3, a brother of the patient in Case I, was admitted to hospital on May 8th, 1934. On May 1st the child, who had been in perfect health, started vomiting. During the week before admission he steadily became more and more listless, without showing any definite signs or making any complaints. He was swinging an irregular temperature between 99° and 103°, and was noted to be becoming progressively paler.

On admission, he was seen to be a plump, well-developed boy. He appeared desperately ill, the skin being of a greyish-yellow colour, and unusually transparent. The lips and mucous membranes were very pale. Temperature was 100°, pulse 140, and respirations 20. He lay quite still in bed, apparently too ill to resent examination. The spleen was enlarged, and extended two fingerbreadths below the costal margin. It was smooth, rounded, and freely movable. There was no obvious enlargement of lymph glands, nor was the liver increased in size. The heart was slightly dilated, and soft blowing systolic murmurs were heard all over the praecordia. No abnormalities were found in the other systems. The urine contained no albumin, blood, sugar, etc.,

and no abnormalities were found on microscopical examination.

The blood findings were: red blood cells 1,700,000 per c.mm.; haemoglobin 27 per cent.; colour index 0.8; white blood cells 50,000. The film showed gross anisocytosis; a great proportion of the cells were microcytes, but there were also large numbers of megalocytes, the majority of which were polychromatic. Many megaloblasts and normoblasts were present. The white cells were mostly young polymorphs, but there were considerable numbers of myelocytes present in addition. Reticulocytes numbered 5 per cent.

On the day following admission a blood transfusion of twelve ounces was given, and this was followed by a dramatic improvement in the patient's general condition. The temperature and pulse rate at once began to settle, and there was a rapid rise in the red blood count and the haemoglobin, associated with a reticulocyte crisis. The spleen quickly reduced in size until it was just palpable at the costal margin. A week after transfusion all abnormal cells had disappeared from the peripheral blood; the cells were more uniform in size, and appeared larger. Fragility tested three weeks after the transfusion showed the range 0.5 to 0.35, and two weeks later 0.55 to 0.4, while a permanent reticulocytosis of about 8 per cent. was maintained. A month after discharge the child's condition had deteriorated, and 41 per cent. reticulocytes was found. The parents, however, would not consent to splenectomy. Table II shows the progress of the case.

TABLE II

Date	Day of Disease	R.B.C. in Millions	Haemo-globin %	Colour Index	W.B.C. in Thousands	Reticulo-cytes %	Fragility Range	Icterus Index	
May 8	8		27		50.0	5			Transfusion, 12 oz.
" 9	9	1.7	27	0.8	44.0	8			
" 10	10	2.35	44	0.9	12.0	12			
" 11	11					19			
" 12	12					40			
" 13	13					33			
" 14	14	3.20	62	0.97	8.0	8			
" 21	21	3.63	68	0.94	6.0				
" 29	29	3.80	72	0.95	6.2	7	0.5-0.35	15	
June 2	33					5			
" 11	42	3.86	72	0.92	7.4	8	0.55-0.4		Left hospital
July 11	72	3.10	50	0.8		41			

PREVIOUS AND FAMILY HISTORY

In the case of the elder boy it had been noted that he was always a little pale, and less active than the rest of the family. He had often had bilious attacks with vomiting, which kept him from school. The younger child, on the other hand, had always been perfectly healthy, and had a rosy complexion. There was an indefinite history of jaundice in the paternal grandmother, who had died many years previously. The father suffered from jaundice on several occasions until 1917, when an enlarged spleen was removed in a military hospital; since that time he has had no symptoms. His blood, examined on October 9th, 1934, showed, however, a slight increase in fragility, the range being 0.5 to 0.4. No other abnormality was found in the blood examination, and there was no increase of reticulocytes. Two other children in the family were apparently healthy, but permission to examine them was refused.

Discussion

When admitted the condition of the elder boy was diagnosed as acholuric jaundice on account of the splenomegaly, the blood film appearances of small well-filled regular cells, and the history of the father's jaundice treated by splenectomy. On the finding of a normal fragility and no reticulocytosis the diagnosis was revised

to that of acute haemolytic anaemia. Later, when the blood level became stationary at just over three million red cells and the jaundice became more marked in conjunction with a permanent reticulocytosis with marked increase of fragility, a reversion to the original diagnosis was made. The history of bilious attacks probably indicates the occurrence of previous less severe haemolytic crises.

The younger boy presented the features usually said to occur in acute haemolytic anaemia, as first described by Lederer in 1925.¹ The typical characteristics were pyrexia, leucocytosis, and reticulocytosis with severe anaemia of sudden origin without any obvious cause. The majority of the published records of acute haemolytic anaemia describe the anaemia as being hyperchromic in type,^{2,3,4} although low colour index cases have been described.⁵ In the present cases the colour index rose following transfusion. In previous reports regarding Lederer's anaemia the fragility has been found to be increased at the time of admission, and has then diminished until normal when cured. The reverse was found in these cases, the cells showing an increasing fragility during the period of observation.

The first case, that of the elder boy, may be looked upon as one of acholuric jaundice showing several unusual features. Cases have previously been described with a normal fragility, and Gänsslen⁶ states that 10 per cent. of acholuric jaundice subjects show a normal fragility. Thus the initial normal fragility in the present case is not unique. The absence of any reticulocytosis until after the second transfusion must, however, be rare, as the reticulocyte count is stated always to be raised in the disease.^{4,7} Barber⁸ has, however, reported a case with a reticulocyte count of only 0.5 per cent. The low colour index on admission is also uncommon, and suggests some error in technique, since the film showed the cells to be well filled though small in diameter. The rapid improvement following splenectomy is a usual finding, as is the presence of gall-stones at operation.

The younger boy would have been regarded as a typical case of Lederer's anaemia if his brother had not been found to have acholuric jaundice. The very mild degree of icterus present in association with such a profound degree of haemolysis is surprising, a fact which has been previously discussed.⁹ The leucocytosis with primitive cells in the blood film is more suggestive of a Lederer's anaemia, but sometimes occurs in acholuric jaundice.¹ The very acute onset of anaemia with pyrexia in two brothers at about the same time suggests an infective origin, but as in previous cases careful bacteriological examination of the stools and urine failed to reveal any haemolytic organisms. No septic foci were discovered, and in neither case was there evidence of external or internal bleeding. Splenectomy is obviously indicated for the younger child, who has shown a mild relapse since discharge, and is liable to another acute crisis with a possibly fatal outcome.

Summary

1. The cases are reported of two brothers showing simultaneous acute haemolytic crises in unsuspected acholuric jaundice.
2. The elder showed the unusual features of an initial normal fragility, an increased fragility developing later, and an absence of reticulocytosis.
3. The younger showed the typical features of an acute Lederer's anaemia.
4. Both responded well to blood transfusion, with a subsequent return to continued haemolysis of mild degree.
5. The elder was successfully treated by splenectomy.

I wish to record my thanks to Professor D. M. Lyon for permission to report these cases, which were treated in his wards.

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A NOTE ON PRE-OPERATIVE PREPARATION OF CASES WITH GRAVE INTRA- ABDOMINAL HAEMORRHAGES

BY

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INFIRMARY

The outlook on acute abdominal surgery has undergone considerable change within recent years. Formerly, attention centred largely upon the lesion which had initiated the crisis, and the patient was hurried from the ambulance to the operating room for what was indeed an emergency operation.

An increasing realization that the general condition of the patient is one of the most important factors which determine the ultimate success of any form of treatment has brought with it a much wider outlook in emergency surgery. Measures directed towards combating the shock which is such a prominent feature of many acute abdominal conditions are now established as a routine, and it is appreciated that two or three hours devoted to supplying warmth and fluids (frequently by the intravenous route) will so improve the condition of the patient that operation can usually be undertaken without anxiety and with a much increased chance of a smooth convalescence. An operation undertaken in a patient whose general condition is so precarious that there is serious anxiety that it may not be successfully completed presupposes ultimate failure, even if the patient does actually survive the operation itself.

The value of pre-operative measures in the treatment of grave intra-abdominal haemorrhages has apparently not received such wide recognition. Frequently, indeed, the greater the exsanguination and circulatory collapse the more urgently is the operation for control of the haemorrhage undertaken. It is certainly remarkable that this procedure is so frequently successful, assisted perhaps by the post-operative infusion of saline or by reintroduction of the lost blood.

The extreme circulatory collapse seen in the subjects of massive intra-abdominal haemorrhage is clearly due to the rapid depletion of the blood volume, associated with a failure of the compensating function of the tissue fluids. There is, in addition, a not inconsiderable element of shock consequent upon the irritation of the peritoneum by the blood within it. The only reasonable method of improving the patient's condition is to increase the blood volume by the administration of considerable quantities of fluid.

Rationale of Fluid Administration

From animal experiment it is known that the intravenous administration of saline equal in quantity to the blood which has been lost will bring about a considerable, though temporary, recovery of the blood pressure.